

TESTING THE ASYMMETRIC INHIBITION MODEL:
FRONTAL EEG ASYMMETRY DOES NOT PREDICT INHIBITORY CONTROL
OF EMOTIONAL DISTRACTORS

BY

ROSANNA ELLEN MOODY

A thesis
submitted to the Victoria University of Wellington
in fulfillment of the requirements for the degree of
Master of Science

Victoria University of Wellington
2016

Abstract

Frontal electroencephalographic (EEG) asymmetry is a reliable marker of psychopathology vulnerability, yet the mechanisms underlying this relationship remain unclear. There is accumulating evidence that frontal asymmetry reflects individual differences in ability to use cognitive control to regulate emotional processing. This thesis provides the first test of the asymmetric inhibition model (Grimshaw & Carmel, 2014), which holds that frontal asymmetry reflects ability to engage valence-specific inhibitory control mechanisms supported by dorsolateral prefrontal cortex (dlPFC): left dlPFC inhibits negative distractors and right dlPFC inhibits positive distractors. Frontal asymmetry was tested as a predictor of ability to inhibit distracting emotional images. Frontal asymmetry was measured at rest and during emotional challenge, which is argued to provide a more powerful measure of individual differences (capability model; Coan, Allen, & McKnight, 2006). Emotional challenge was induced using a stressful serial subtraction task, verified to be effective in Study 1, followed by a silent speech preparation task, during which EEG was recorded. An irrelevant distractor paradigm measured ability to inhibit emotional distraction; participants identified a target letter within a central symbol array while attempting to inhibit positive, negative and neutral peripheral images (Study 2). Overall, positive and negative images were more distracting than neutral images. Critically, neither resting nor emotional challenge frontal asymmetry predicted distraction by positive, negative or neutral images, suggesting that frontal asymmetry does not reflect ability to inhibit irrelevant emotional distractors. Thus, the asymmetric inhibition model was not supported. This thesis provides the first direct test of the relationship between frontal EEG asymmetry and inhibitory control of emotion, paving the way for future explorations into this relationship. These findings add to a growing literature attempting to elucidate the cognitive mechanisms underlying frontal asymmetry in order to better understand the etiology of psychopathology.

Acknowledgements

First and foremost, I'd like to thank my supervisor, Dr. Gina Grimshaw. I am truly grateful for your patience, wisdom and guidance throughout this process and throughout the time I have known you. You have inspired me as a scientist and as a teacher. I have learned so much from you and I feel truly privileged to have had you guiding me through this process. I would also like to thank Dr. David Carmel for his valuable input into this project.

To my family, you have always supported me, bolstered my spirits when things felt impossible and helped me believe I would get through this. To Mum, thank you for helping me keep perspective, for your compassionate, patient listening and for your voice of reason - you know me better than I care to admit sometimes. To Dad, thank you for showing me the value of both hard work and creativity and for gently nurturing and challenging my inquisitive mind. To George and Kate, you two are an inspiration and have shown me the meaning of true grit. To my grandparents, thank you for making me feel so loved and for all your encouragement and support. Grandpa, your faith in me has helped me believe in myself. You always take an active interest in what I'm doing – thank you for the engaging discussions and for helping me to stay grounded. Gran, thank you for showing me the beauty of an open mind, and the value of a dose of mischief! You are amazing. To Callum, thank you for being my rock. You are always there when I need you and always make me feel better. Thank you for looking after me all those times when I was too tired to do it myself. I truly couldn't have got through this without you. To my friends, Clara, Claire, Zoe, Jake, Sarah, Zénobie, Ellie and Paul, thank you for cheering me up and keeping me sane, and for putting up with an often-absent (physically, mentally, and emotionally) flatmate and friend.

To my friends in the Cognitive and Affective Neuroscience Lab, thank you for making science fun. I have hugely valued having you all to share ideas with. Special thanks to Angus for your stats advice and for opening my eyes to the marvels of excel, to Amy for your support in the writing process, and to Michael for your assistance as the “evaluator” and as my research partner. To Hazel, your friendship has been a constant comfort. Thank you for reminding me to look after myself and for the pipe-cleaner animal distractions. Laura and Michael - you are the best! Thank you so much for all your support throughout my thesis and for your never-ending encouragement and friendship. I could not have done this without you.

This thesis was made possible thanks to financial support from the Royal Society of New Zealand Marsden Fund, a Victoria University of Wellington Master's (by thesis) scholarship and a Tertiary Education Union Crozier scholarship. I am hugely grateful for this financial support.

Table of Contents

Abstract.....	i
Acknowledgements.....	ii
Table of Contents.....	iii
List of Figures.....	vi
List of Tables.....	viii
Introduction.....	1
Frontal Asymmetry and Psychopathology Vulnerability.....	2
Early Theories of Frontal Asymmetry.....	3
The Need to Shift Towards a Cognitive Model of Frontal Asymmetry.....	4
Frontal Cortex and Cognitive Control.....	4
Frontal Cortex and Cognitive Control of Emotion.....	5
Cognitive Control and Psychopathology.....	5
Cognitive Models of Frontal Asymmetry.....	6
The Capability Model.....	6
The Asymmetric Inhibition Model.....	7
Support for the Asymmetric Inhibition Model.....	8
Clinical research.....	8
Neuroimaging research.....	8
Electrophysiological research.....	9
The Present Thesis.....	10
Study 1.....	13
Method.....	14
Design.....	14
Participants.....	15
Materials.....	15
Counting Task Procedure.....	15
Self-Report Ratings.....	15
Procedure.....	16
Physiology Data Recording and Reduction.....	16
Data Analysis.....	17
Results and Discussion.....	18
Physiological Response.....	18
Subjective Response.....	20

Summary.....	23
Study 2.....	23
Method.....	25
Design.....	25
Participants.....	25
Procedure.....	26
Materials.....	27
Irrelevant Distractor Paradigm.....	27
Stress Induction.....	30
Questionnaires.....	30
Physiology Data Recording and Reduction.....	32
Electroencephalography (EEG)	32
Heart Rate.....	33
Results and Discussion.....	34
Irrelevant Distractor Paradigm.....	34
Response Times.....	34
Accuracy.....	37
Summary.....	37
Stress Induction.....	38
Emotional Challenge.....	38
Relative Frontal EEG Asymmetry.....	43
Individual Differences Analyses.....	46
An Individualised Measure of Distraction.....	46
Relationships with Depression and Anxiety Symptoms.....	47
Testing the Asymmetric Inhibition Model.....	49
General Discussion.....	52
A Different Cognitive Control Mechanism.....	53
A Different Aspect of Inhibitory Control.....	54
Statistical Power.....	56
Broad Contributions to the Literature.....	58
The Modified Counting Task: A Validated Emotional Challenge Induction.....	58
Depression and Anxiety Symptoms.....	58
Frontal Asymmetry and Emotional Challenge.....	59
Limitations.....	60

Conclusions.....	61
References.....	62
Appendices.....	85

List of Figures

Figure 1. Electrocardiogram (ECG) electrode placement (white: negative electrode; grey: positive electrode; black: ground electrode) in Study 1.....	17
Figure 2. Heart rate (in beats per minute) in Study 1.....	19
Figure 3. Electrodermal activity (in micro-Siemens, μ S) in Study 1.....	20
Figure 4. Change in subjective mood (mean difference between post- and pre-counting ratings) in Study 1. Mood was rated from 0 to 100. Positive mood change values indicate greater reported mood experience after the counting task than at the beginning of the session.....	22
Figure 5. An example of a trial procedure in the irrelevant distractor paradigm in Study 2. Participants identified the target (X or N) in the letter display, whilst attempting to ignore distractor images. Distractors were presented on 50% of trials, either above or below the display. An error tone was presented for an incorrect response or no response. The image depicted above is for display purposes only; image stimuli were selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008). Display is not to scale.....	29
Figure 6. Electrocardiogram (ECG) electrode placement (white: negative electrode; grey: positive electrode; black: ground electrode) in Study 2.....	33
Figure 7. Response times (in milliseconds) for distractor-absent and distractor-present trials in the irrelevant distractor paradigm in Study 2.....	36
Figure 8. Distraction indices (distractor-present RT – distractor-absent RT, in milliseconds) for the irrelevant distractor paradigm in Study 2.....	36
Figure 9. Accuracy rates (%) for distractor-absent and distractor-present trials in the irrelevant distraction paradigm in Study 2.....	38
Figure 10. Heart rate (in beats per minute) in Study 2. Emotional challenge heart rate was measured during silent speech preparation, following a stressful serial subtraction task.....	40
Figure 11. Subjective mood in Study 2, rated before and after stress induction (a stressful serial subtraction task, followed by silent speech preparation). Mood was rated on a scale from 0 to 100, with higher ratings indicating greater mood experience. Ratings made at the beginning of the session are not presented here for ease of presentation.....	42

- Figure 12. Mean frontal asymmetry scores in Study 2, calculated using the formula: $\ln(F4) - \ln(F3)$. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task..... 45
- Figure 13. Individual frontal asymmetry data, depicting the relationship between baseline resting and emotional challenge frontal asymmetry, calculated using the formula: $\ln(F4) - \ln(F3)$. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task..... 46
- Figure 14. The relationship between distraction (negative, neutral, positive) and frontal asymmetry in study 2, calculated using the formula: $\ln(F4) - \ln(F3)$. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task. Distraction is measured in residuals (calculated by regressing distractor-present RTs on distractor-absent RTs for each valence), which indicate distraction relative to the sample average. Residual distributions have a mean of zero, so more positive values indicate greater distraction than the sample average and more negative values indicate less distraction than the sample average..... 51
- Figure 15. Example of the display screen for the central distractor paradigm (see Gupta et al., 2015). The image depicted is for display purposes only; positive, negative and neutral image stimuli would be selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008). Display is not to scale..... 55

List of Tables

Table 1. Mean Physiology Measurements (with Standard Deviations) in Study 1.....	18
Table 2. Mean Self-Reported Mood Ratings (with Standard Deviations) in Study 1.....	21
Table 3. Mean Task Experience Ratings (with Standard Deviations) in Study 1.....	22
Table 4. Study 2 Procedure Timeline.....	27
Table 5. Mean Ratings (with Standard Deviations) for the Image Stimuli Used in the Irrelevant Distractor Paradigm.....	28
Table 6. Study 2 Questionnaire Scores.....	31
Table 7. Study 2 Response Time Means (in Milliseconds) for the Irrelevant Distractor Paradigm.....	35
Table 8. Mean Accuracy (%) for the Irrelevant Distractor Paradigm in Study 2.....	37
Table 9. Mean Heart Rate and Mood Ratings (with Standard Deviations) in Study 2.....	39
Table 10. Self-report Ratings of the Stress Induction in Study 2.....	43
Table 11. Frontal EEG Asymmetry Scores in Study 2.....	44
Table 12. Zero-order Correlations Between Frontal Asymmetry (FA), Questionnaire Scores, and Residual Distraction Indices (RDIs) in Study 2.....	48

Testing the Asymmetric Inhibition Model:

Frontal EEG Asymmetry Does Not Predict Inhibitory Control of Emotional Distractors

Psychological disorders are among the leading causes of disease and disability worldwide (World Health Organization [WHO], 2013). With over 600 million people suffering from depression and anxiety, the WHO (2016) has declared mental health to be a global priority. The development of successful prevention strategies is critical and is dependent on our ability to understand psychological processing in vulnerable individuals. One established marker of vulnerability to psychopathology is hemispheric asymmetry in frontal brain activity. Frontal asymmetry is most commonly associated with vulnerability to emotional disorders: greater rightward frontal asymmetry (greater relative activity over right than left frontal cortices; RFA) has been linked to mood disorders like depression and anxiety (Coan & Allen, 2004; Thibodeau, Jorgensen, & Kim, 2006), while greater leftward frontal asymmetry (greater relative activity over left than right frontal cortices; LFA) has been linked with disorders characterised by impulsivity and sensitivity to reward, like addiction (Balconi, Finocchiaro, & Canavesio, 2014) and attention-deficit hyperactivity disorder (ADHD; Keune, Wiedemann, Schneidt, & Schönenberg, 2015).

Despite decades of research, it is still unclear why these patterns of frontal asymmetry mark vulnerability to psychopathology. Traditional theories hold that frontal asymmetry reflects individual differences in affective functioning, that is, how we process, experience and respond to emotion (Davidson, 1998; Harmon-Jones, 2003; Harmon-Jones & Allen, 1997; Heller, 1993; Heller & Nitschke, 1998; Heller, Nitschke, Etienne, & Miller, 1997). However, there is accumulating evidence that the mechanisms underlying the relationship between frontal asymmetry and psychopathology vulnerability are cognitive; that is, frontal asymmetry may reflect individual differences in cognitive processes that modulate affective functioning (Grimshaw, Foster, & Corballis, 2014; Miskovic & Schmidt, 2010; Pérez-Edgar, Kujawa, Nelson, Cole, & Zapp, 2013).

One recent theory argues that frontal asymmetry reflects individual differences in ability to use cognitive control to inhibit emotion. The asymmetric inhibition model (Grimshaw & Carmel, 2014) holds that the frontal cortex is anatomically specialised for inhibiting emotional stimuli: the left side inhibits negative stimuli and the right side inhibits positive stimuli. Frontal asymmetry therefore reflects disproportionate activity in these systems and thus disproportionate ability to inhibit different valences of emotional stimuli. In this thesis, I tested the asymmetric inhibition model to elucidate the cognitive mechanisms that link frontal asymmetry to psychopathology vulnerability. Using an

individual differences approach, I explored the relationship between an individual's pattern of frontal asymmetry and their ability to exert inhibitory control over emotional distractors.

Frontal Asymmetry and Psychopathology Vulnerability

Frontal asymmetry, most commonly measured in relative frontal alpha power, has long been associated with vulnerability to psychopathology. Alpha power is measured using EEG, which records oscillatory neuronal activity across different frequency bands. Alpha is commonly used as an inverse index of relative frontal cortical activity (see Allen, Coan, & Nazarian, 2004) because alpha power (8-13 Hz) decreases as cortical activity increases (Klimesch, 1999; Pfurtscheller, Stancak, & Neuper, 1996). For example, greater alpha power over the left than right frontal regions infers that there is less left, relative to right, frontal activity. Relative EEG activity is measured as an asymmetry score by subtracting left alpha power from right alpha power (usually after a log transformation); thus, positive asymmetry scores indicate LFA and negative scores indicate RFA. Typically measured while the participant rests, frontal asymmetry scores show high internal reliability and stability over time (Mathewson et al., 2015; Salinsky, Oken, & Morehead, 1991) and are thus considered to reflect dispositional processing tendencies that may indicate psychopathology vulnerability (Davidson, 1998; Tomarken, Davidson, Wheeler, & Doss, 1992).

The relationship between frontal asymmetry and psychopathology vulnerability is well established. The majority of research has investigated emotional vulnerabilities associated with a greater rightward pattern of asymmetry. Greater RFA (i.e., lower LFA) has been linked to higher levels of trait negative affect (Tomarken & Davidson, 1994), increased trait rumination (Nusslock et al., 2011), increased shyness, less sociability (Schmidt, 1999), greater social withdrawal (Cole, Zapp, Nelson, & Pérez-Edgar, 2012), greater neuroticism (Uusberg, Allik, & Hietanen, 2015), and poor regulation of negative emotions (Tooley, 2015; Wheeler, Davidson, & Tomarken, 1993). Greater RFA is also considered a marker of vulnerability to mood disorder, found not only in individuals currently experiencing depression and anxiety (Henriques & Davidson, 1991; Mathersul, Williams, Hopkinson, & Kemp, 2008; Thibodeau et al., 2006), but also in those with a history of depression (Henriques & Davidson, 1998; Gotlib, Ranganathand, & Rosenfeld, 1998), in the infants of depressed mothers (Field & Diego, 2008), and in those with a genetic risk (Bismark et al., 2010) or a family history of mood disorder (Feng et al., 2012; Goldstein et al., 2016). Critically, greater RFA has been linked to future depression and anxiety in individuals with no history of mood disorder (Blackhart, Minnix, & Kline, 2006; Nusslock et al., 2011),

suggesting that this pattern of asymmetry reflects pre-existing vulnerability to developing an emotional disorder, rather than being a symptom of current disorder or a ‘scar’ from previous disorder.

A smaller body of research has investigated emotional vulnerabilities associated with a greater leftward pattern of asymmetry. Greater LFA (i.e., lower RFA) has been linked to higher levels of trait positive affect, anger and aggression (Niv et al., 2015), increased risk-taking (Black et al., 2014; Telpaz & Yechiam, 2014), increased sensation seeking (Santesso et al., 2008), greater reward sensitivity (Balconi et al., 2014), and greater impulsivity (Gable, Mechin, Hicks, & Adams, 2015). Greater LFA has also been associated with psychopathology, particularly with disorders characterised by impulsivity and reward sensitivity, including ADHD (Keune et al., 2015) and addiction (Balconi et al., 2014; Gapin, Etnier, & Tucker, 2009). Taken together, these findings provide evidence for a critical link between frontal asymmetry and psychopathology. If we can understand the mechanisms underlying this link, we may gain valuable insight into the development of psychological disorders.

Early Theories of Frontal Asymmetry

Two theories have dominated the literature in their attempts to explain the relationship between frontal asymmetry and psychopathology: the valence hypothesis and the motivational direction hypothesis. These theories are based on the view that frontal asymmetry represents a dispositional tendency to respond to situations with a certain affective or motivational style (Davidson, 1998).

According to the valence hypothesis (Berntson, Norman, & Cacioppo, 2011; Heller, 1993; Heller & Nitschke, 1998; Tomarken et al., 1992), the frontal cortex is responsible for affective experience: the left side grounded in positive emotion and the right side grounded in negative emotion. Frontal asymmetry is argued to reflect an imbalance or bias in emotional experience. The valence hypothesis was proposed to explain the link between frontal asymmetry and vulnerability to mood disorders; individuals with greater RFA are posited to experience more negative than positive emotion, making them vulnerable to disorders like depression and anxiety. However, the model does not clearly explain how a bias to experience more positive over negative emotion (i.e., greater LFA) would make an individual vulnerable to disorders characterised by impulsivity and reward sensitivity (e.g., addiction).

According to the motivational direction hypothesis (Harmon-Jones, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), the frontal cortex directs action in response

to emotional stimuli: the left side guiding approach behaviours (e.g., embracing a loved one, or shouting at a belligerent teenager) and the right side guiding withdrawal behaviours (e.g., fleeing from an aggressive animal, or seeking solitude after bad news). This model holds that frontal asymmetry reflects propensity to engage in such behaviours; that is, individuals with greater RFA would be more prone to withdraw from an emotional situation, making them vulnerable to disorders like depression and anxiety, while those with greater LFA would be more prone to approach the situation, making them vulnerable to disorders characterised by impulsivity and reward sensitivity.

The Need to Shift Towards a Cognitive Model of Frontal Asymmetry

The valence and motivational direction hypotheses have dominated frontal asymmetry theory for decades and been important catalysts in generating frontal asymmetry research. However, they have been criticised for being overly simplistic and descriptive, failing to account for cognitive functioning associated with frontal brain areas (see Grimshaw & Carmel, 2014; Miller, Crocker, Spielberg, Infantolino, & Heller, 2013). In light of growing evidence that links frontal asymmetry with cognitive processing, there is an increasing focus on more cognitive accounts of frontal asymmetry.

Frontal cortex and cognitive control. There is accumulating evidence that frontal EEG activity reflects engagement of cognitive mechanisms in lateral prefrontal cortex (lPFC), an area primarily associated with cognitive control (Braver, Paxton, Locke, & Barch, 2009; Miller & Cohen, 2001), or “the ability to regulate, coordinate, and sequence thoughts and actions in accordance with internally maintained behavioural goals” (Braver, 2012, p. 106). Neuroimaging and EEG source localisation studies have connected emotional biases, vulnerability, and disorder to asymmetries in lPFC, particularly dorsolateral PFC (dlPFC; Auerbach, Stewart, Stanton, Mueller, & Pizzagalli, 2015; Engels et al., 2010; Grimm et al., 2008; Herrington et al., 2005; Herrington et al., 2010; Koslov, Mendes, Pajtas, & Pizzagalli, 2011; Pizzagalli, Sherwood, Henriques, & Davidson, 2005; Shackman, McMenamin, Maxwell, Greischar, & Davidson, 2009). These findings suggest that frontal EEG asymmetry reflects activity from dlPFC, an area responsible for guiding attention according to current goals, updating and shifting attention towards potentially relevant information and employing inhibitory control to ignore or suppress irrelevant information from further processing (Hasegawa, Peterson, & Goldberg, 2004; Kane & Engle, 2002; MacDonald, Cohen, Stenger, & Carter, 2000; Ochsner, Silvers, & Buhle, 2012; Shimamura, 2000; Suzuki & Gottlieb, 2012; Weissman, Roberts, Visscher, & Woldorff, 2006). Frontal asymmetry may therefore reflect individual differences in activation of cognitive control

mechanisms that regulate emotional processing, rather than trait-like tendencies toward emotional experience or motivational response.

Frontal cortex and cognitive control of emotion. Cognitive control is a vital component of emotional processing. We need cognitive control to manage distraction from salient stimuli (e.g., Forster & Lavie, 2008a) and emotional stimuli are highly salient (Yiend, 2010; Pourtois, Schettino, & Vuilleumier, 2013). Within an attentional system designed to prioritise relevant information for further processing, emotional stimuli have the advantage of biological relevance; it is adaptive for emotional information to jump to the front of the attention queue in survival situations, demanding focus when an aggressive animal is approaching or a potential mate is flaunting their wares in your direction. However, often emotional information is not critical for survival; cognitive control then becomes vital to regulate processing of these potent stimuli to avoid distraction and enable effective emotion regulation.

Through its fundamental involvement in cognitive control, IPFC plays a critical role in regulating emotional processing (Davidson, 2000; Dolcos, Iordan, & Dolcos, 2011; Gray, Braver, & Raichle, 2002; Ochsner & Gross, 2005; Ochsner et al., 2012; Pessoa, 2008, 2009). Though IPFC has not been directly implicated in the generation of emotional experience or preparation to approach or withdraw (Craig, 2009; Gu, Hof, Friston, & Fan, 2013; Lang & Bradley, 2010; Ochsner et al., 2012; Pessoa, 2013; see Steele & Lawrie, 2004, for a meta-analysis), activity here critically influences structures that are involved in these functions, including the amygdala and insula (Ochsner, Bunge, Gross, & Gabrieli, 2002; Pessoa, 2015). dlPFC works in concert with ventrolateral PFC to modulate perception and interpretation of emotional stimuli, helping us to ignore distracting emotional stimuli in our environment (e.g., Bishop, Duncan, Brett, & Lawrence, 2004) and regulate our emotions via strategies like cognitive reappraisal (Drabant, McRae, Manuck, Hariri, & Gross, 2009; Ochsner & Gross, 2005; Ochsner et al., 2012; but see Kompus, Hugdahl, Öhman, Marklund, & Nyberg, 2009). Considering that dlPFC plays an important role in regulating emotional processing and that frontal EEG asymmetry has been localised to dlPFC, it is plausible that frontal asymmetry marks emotional vulnerability because it reflects individual differences in cognitive regulation, or control, of emotion.

Cognitive control and psychopathology. Individual differences in cognitive control have been reliably linked with differences in emotional experience. Cognitive control deficits are evident in many emotional disorders. Impaired cognitive control is one of the most promising endophenotypes of depression (Goldstein & Klein, 2014; Snyder, 2013;

Webb et al., 2016), with individuals showing multifaceted deficits in inhibition, shifting, updating, visual and verbal working memory and verbal fluency (Snyder, 2013), while anxiety is associated with impairments in attentional shifting and inhibitory control (for reviews, see Eysenck & Derakshan, 2011; Eysenck, Derakshan, Santos, & Calvo, 2007). Both emotional dysfunction and poor cognitive control are considered core components of drug addiction (Groman & Jentsch, 2012; Kalivas & Volkow, 2005; Verdejo-García, Pérez-García, & Bechara, 2006) and ADHD (Barkley, 1997; Brown, 2006; Seymour et al., 2015; Walcott & Landau, 2004). Both disorders are associated with impairments in inhibitory control (Forster, Robertson, Jennings, Asherson, & Lavie, 2014; Moeller et al., 2014), set shifting, cognitive flexibility (Goldstein et al., 2004), verbal fluency and working memory (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Kübler, Murphy, & Garavan, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

Ineffective use of cognitive control mechanisms manifest as emotion processing biases in these disorders, such as poor attentional disengagement from negative stimuli in depression (Armstrong & Olatunji, 2012; Beevers, Clasen, Enock, & Schnyer, 2015; Everaert, Grahek, & Koster, 2016; Koster, De Lissnyder, Derakshan, & De Raedt, 2011), increased attentional capture by threatening stimuli in anxiety (see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007), heightened reward sensitivity in addiction (Balconi et al., 2014; Groman & Jentsch, 2012) and increased attentional capture by positive stimuli in ADHD (Seymour et al., 2015), which in turn maintain symptoms of emotional disorder (Beevers et al., 2015). Individual differences in cognitive control in healthy individuals have also been linked to vulnerability to developing emotional psychopathologies and predict future increases in symptoms (Everaert et al., 2016; Pe, Brose, Gotlib, & Kuppens, 2016; Rudaizsky, Basanovic, & MacLeod, 2014; Zetsche & Joormann, 2011; for reviews, see De Raedt & Koster, 2010; Groman & Jentsch, 2012; Joormann & D'Avanzato, 2010; Mathews & MacLeod, 2005). Thus, there is compelling support for the idea that frontal asymmetry is linked to psychopathology because it represents individual differences in propensity or ability to exert cognitive control over emotion.

Cognitive Models of Frontal Asymmetry

The capability model. The first model to interpret frontal asymmetry in the context of cognitive processes was Coan, Allen, and McKnight's (2006) capability model. This model holds that patterns of frontal activity are driven by the current situation, and reflect an individual's capability to respond adaptively to the situation, activating emotion regulation

mechanisms when needed and inhibiting emotional responses when appropriate. In line with this idea, frontal asymmetry seems to be a more powerful and reliable measure of individual differences when measured during situations of emotional challenge (i.e., during a task that elicits an emotional reaction, such as a stress response) than at rest (i.e., the traditional method; Coan et al., 2006; Stewart, Coan, Towers, & Allen, 2011, 2014). Thus, rather than reflecting a certain affective style or tendency to engage in certain motivational behaviours, frontal asymmetry may mark psychopathology vulnerability through differential capability to *manage* (or control) one's response to emotional stimulation. Although the capability model does not describe the specific mechanisms through which such emotional "management" might occur, it provides an important shift of focus toward cognitive explanations for correlates of frontal asymmetry.

The asymmetric inhibition model. Grimshaw and Carmel (2014) recently put forward a more specified model of frontal asymmetry that proposes that inhibitory cognitive control mechanisms underlie the relationship between frontal asymmetry and psychopathology vulnerability. The asymmetric inhibition model holds that frontal asymmetry reflects relative activity in a bilateral cognitive control system based in dlPFC. Within this system, each hemisphere specialises in the control of different types of emotional information, the left side controlling negative (or withdrawal-related) information and the right side controlling positive (or approach-related) information. The authors operationalise their model in terms of distractor inhibition. Inhibitory control mechanisms enable us to effectively suppress irrelevant stimuli from further processing; thus, when inhibitory mechanisms are less effective, distraction becomes more likely. The asymmetric inhibition model holds that left dlPFC is responsible for inhibiting negative distractors and right dlPFC is responsible for inhibiting positive distractors. Frontal asymmetry is thus proposed to reflect ability to engage different types of emotional control. Less activity in left frontal areas (i.e., RFA) is argued to reflect less effective inhibition of negative than positive information, leading to increased vulnerability to disorders like depression and anxiety, while less activity in right frontal areas (i.e., LFA) is argued to reflect less effective inhibition of positive than negative information, leading to increased vulnerability to disorders like addiction and ADHD. The asymmetric inhibition model is based on clinical (e.g., Cisler & Koster, 2010; De Raedt & Koster, 2010; Field & Cox, 2008), neurological (e.g., Bishop et al., 2004; Engels et al., 2010; Herrington et al., 2010), and electrophysiological (e.g., Grimshaw et al., 2014; Pérez-Edgar et al., 2013) research that

links frontal asymmetry, cognitive control, and psychopathology, though researchers are yet to directly test it.

Support for the Asymmetric Inhibition Model

Clinical research. The asymmetric inhibition model (Grimshaw & Carmel) holds that left and right frontal areas inhibit negative and positive information, respectively. In support of a valence-specific inhibitory control system, some inhibitory control deficits in psychological disorders linked to frontal asymmetry appear to be valence-specific. Depression and anxiety (linked to greater RFA) are characterised by poor inhibition of negative (but not positive) distractors (for reviews, see Cisler & Koster, 2010; Derakshan & Eysenck, 2009; Gotlib & Joormann, 2010; Snyder, 2013). For example, Joormann and Gotlib (2008) showed that depressed individuals have greater difficulty inhibiting irrelevant negative words than healthy controls, but have a similar ability to inhibit irrelevant positive words. Conversely, addiction and ADHD (linked to greater LFA) are characterised by poor control over positive information and associated with poor inhibition of positive distractors (Franken, Kroon, Wiers, & Jansen, 2000; Hester, Dixon, & Garavan, 2006; for a review, see Garavan & Hester, 2007), compared to negative distractors (Seymour et al., 2015). Valence-specific biases in cognitive control also predict risk of future disorder in healthy individuals (see De Raedt & Koster, 2010; Field & Cox, 2008; Joormann, Talbot, & Gotlib, 2007). These studies suggest that, like frontal asymmetry, valence-specific control deficits indicate a trait-like cognitive vulnerability to psychopathology. Therefore, frontal asymmetry may be associated with psychopathology vulnerability because it reflects individual differences in valence-specific control.

Neuroimaging research. Although the relationship between frontal asymmetry and inhibition of emotional distractors has not yet been directly examined, lateralised activity in dlPFC has been linked to inhibition of positive and negative distractors. Neuroimaging research shows increased activation in left (but not right) dlPFC during inhibition of irrelevant negative (compared to neutral) distractors (Compton et al., 2003; Herrington et al., 2010) and links activity in right dlPFC with control of positive distractors (Beauregard et al., 2001; Herrington et al., 2010). Altered asymmetries while attempting to inhibit emotional stimuli have been associated with depression (Herrington et al., 2010) and anxiety (Engels et al., 2007), with difficulties inhibiting negative stimuli linked to insufficient activation of left dlPFC (Bishop et al., 2004; Engels et al., 2010). For example, Bishop et al. (2004) found decreased left dlPFC activation in high trait-anxious individuals preparing to inhibit

irrelevant threat-related images. Together, these neuroimaging studies provide indirect support for the asymmetric inhibition model.

Electrophysiological research. There is some EEG evidence that frontal asymmetry is involved in cognitive control. A few studies have found that frontal asymmetry predicts attentional biases to emotion, which may arise from failure to inhibit emotional stimuli. Miskovic and Schmidt (2010) found greater resting RFA to predict attentional biases to angry (but not happy) face cues, using a modified Posner spatial cueing task (Posner & Cohen, 1984). Similarly, Grimshaw et al. (2014) found that individuals with strong RFA showed an attentional bias for angry (but not happy) faces in a dot-probe task, in which bilateral face cues precede a target probe. Individuals with strong LFA did not show attentional biases. Using a similar dot-probe task, Pérez-Edgar et al. (2013) found attentional biases to angry faces were predicted by frontal asymmetry shift. Participants who increased RFA showed a bias towards angry faces and away from happy faces, whereas those who increased LFA showed no attentional biases. Notably, both Pérez-Edgar et al. and Grimshaw et al. (2014) found frontal asymmetry to predict biases only when faces were presented for a longer duration (i.e., when participants had more time to process them), supporting the idea that frontal asymmetry is linked to controlled, top down aspects of attentional processing. Taken together, these three studies show that frontal asymmetry is negatively related to attentional bias for angry (but not happy) faces (i.e., greater RFA, greater attentional bias). Although none of these studies found a link between frontal asymmetry and attentional biases for positive emotional stimuli, happy faces are inherently less potent than angry faces (Grimshaw & Carmel, 2014), and thus may not be effective “positive” stimuli to test attentional biases to positive emotion.

These studies (Grimshaw et al., 2014; Miskovic & Schmidt, 2010; Pérez-Edgar et al., 2013) support the idea that frontal asymmetry reflects individual differences in valence-specific control processes. However, they were not designed to specifically test inhibitory control. Participants were not asked to ignore the emotional stimuli and it was not necessarily beneficial for them to do so. Indeed, in all three studies emotional stimuli appeared in target locations (i.e., an attended location), and in one study the emotional faces predicted cue locations (Miskovic & Schmidt, 2010). To specifically test inhibitory control of emotion, the emotional stimuli must be task-irrelevant (i.e., distractors) so that it is clearly beneficial to ignore them.

The Present Thesis

An understanding of the mechanisms underlying frontal asymmetry could provide valuable information about the etiology of psychological disorder and the processes contributing to vulnerability, yet our understanding of these mechanisms is still limited. Though frontal asymmetry is traditionally thought to mark vulnerability because it reflects biases in emotional processing, contemporary accounts suggest that these biases may be an indirect effect of ability to engage cognitive control. In this thesis, I tested the asymmetric inhibition model (Grimshaw & Carmel, 2014), which holds that frontal asymmetry reflects an asymmetric ability to use lateralised valence-specific inhibitory control mechanisms; left frontal areas are proposed to inhibit negative distraction and right frontal areas are proposed to inhibit positive distraction. Previous research has linked frontal asymmetry to attentional biases for emotion, but no one has yet tested whether frontal asymmetry predicts inhibitory control over emotion. To address this question, I used an individual differences study to test whether frontal asymmetry predicts ability to inhibit positive and negative distractors.

Ability to inhibit emotional distraction was assessed using an irrelevant distractor paradigm previously used in our lab and originally adapted from Forster and Lavie (2008a). Participants viewed a central array of letters and identified a target amongst them. On some trials, a distracting image (positive: erotic scenes of couples; negative: scenes of bodily mutilations; neutral: scenes involving people) was presented, either above or below the array; participants were told to ignore the images and focus on the letter task. Slower response times on trials when an image is present indicate distraction. This paradigm elicits robust emotional distraction effects (Grimshaw, Kranz, Carmel, Moody, & Devue, 2016; Kranz, 2015; Maddock, Harper, Carmel, & Grimshaw, 2016; Murphy, 2016); that is, participants show greater distraction from both positive and negative images than neutral images.

There are several advantages of using this irrelevant distractor paradigm to study inhibitory control. First, it tests inhibitory control processes more directly than tasks used previously to test the relationship between frontal asymmetry and cognitive biases (e.g., dot-probe and spatial cueing). Unlike other tasks, this paradigm presents distracting images that are completely unrelated to the central task in visual appearance and location and require no response (i.e., they are entirely irrelevant); it is therefore beneficial to inhibit the images and participants are encouraged to do so in order to enhance performance. Further, experimental studies show that emotional distraction in this task is subject to factors that are known to influence cognitive control. For example, distraction drops dramatically when distractors are

expected frequently (Grimshaw et al., 2016; Kranz, 2015; Murphy, 2016), or when motivation to perform well on the letter task is high (Maddock et al., 2016). Both expectancy and motivation are argued to affect cognitive control (Botvinick & Braver, 2015; Bugg & Crump, 2012; Chiew & Braver, 2011), suggesting that this task elicits use of control to inhibit distractors.

Second, this paradigm uses arousal-matched positive and negative stimuli and elicits similar distraction effects for both valences (Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2015; Murphy, 2016), in line with literature showing that positive and negative stimuli capture attention to a similar extent when equated for arousal (Most, Smith, Cooter, Levy, & Zald, 2007; Vogt, De Houwer, Koster, Van Damme, & Crombez, 2008). Therefore, this task should be a better test of cognitive biases to positive emotion than those using emotional faces (see Grimshaw & Carmel, 2014) or low arousal positive images (e.g., Augst, Kleinsorger, & Kunde, 2014).

Third, this task elicits larger emotion effects than spatial cueing and dot-probe tasks (often used to detect attentional biases). Behavioural biases are very small in these tasks and are found inconsistently in healthy individuals. Indeed, a meta-analysis revealed that, on average, healthy controls do not show a threat-related bias for dot-probe or spatial cueing paradigms (Bar-Haim et al., 2007). Individual differences in ability to use valence-specific control mechanisms may be more evident in a task that elicits larger, more robust emotion effects in healthy individuals (i.e., placing greater demands on control processes), for both positive and negative emotional stimuli.

Lastly, a “non-emotional” version of the irrelevant distractor paradigm has been shown to be sensitive to individual differences in distractibility and is proposed to measure an “attention-distractibility trait” (Forster & Lavie, 2016; Forster, Robertson, Jennings, Asherson, & Lavie, 2014). Performance on the task has been linked with vulnerability to ADHD (Forster & Lavie, 2016), a disorder characterised by poor inhibitory control (Boonstra et al., 2005; Willcutt et al., 2005) and greater LFA (Keune et al., 2015). Thus, this task has already been shown to be sensitive to individual differences in cognitive vulnerability. In sum, the irrelevant distractor paradigm provides a sensitive measure of ability to inhibit emotional distractors.

Frontal asymmetry was measured at rest and during emotional challenge. Though the majority of frontal asymmetry research measures asymmetry while participants rest (the traditional framework), Coan and colleagues (2006; Stewart et al., 2014) have shown that frontal asymmetry is a more powerful indicator of individual differences when measured

during emotional challenge (the capability framework). Challenge asymmetries account for substantially greater individual variance than resting asymmetries, and are less affected by different recording parameters (e.g., reference mode), a leading cause of inconsistent findings in the resting frontal asymmetry literature (e.g., Blackhart, Kline, Donohue, LaRowe, & Joiner, 2002; Kline, Blackhart, & Joiner, 2002, Thibodeau et al., 2006). Coan et al. (2006) suggest that emotional challenge asymmetries are more powerful and reliable than resting asymmetries due to better control over mental behaviour during recording, as participants are given a specific task to occupy their mental processing. Conversely, in resting procedures, participants are simply asked to rest, leading to variable levels and types of mental activity (Binder et al., 1999; McKiernan, Kaufman, Kucera-Thompson, & Binder, 2003). Emotional challenge frontal asymmetry has been shown to be a stronger and more robust marker of emotional disorder (Stewart et al., 2014; see Meyer et al., 2015, for a review) and personality traits indicative of psychological vulnerability (Cole et al., 2012). To enable hypothesis testing within both a traditional and a capability framework, I measured frontal asymmetry at rest and during emotional challenge.

Study 1 piloted a method of inducing emotional challenge. In a stress induction paradigm, participants performed a difficult mental arithmetic task while being socially evaluated (Kirschbaum, Pirke, & Hellhammer, 1993). This paradigm was implemented in Study 2, which tested the asymmetric inhibition model: frontal asymmetry was measured at rest and during emotional challenge and tested as a predictor of ability to inhibit emotional distraction in the irrelevant distractor paradigm.

Participants were young women with no current diagnosis of depression or anxiety, as the goal of this study was to identify cognitive vulnerability associated with frontal asymmetry, independent of symptoms of current disorder. The sample was limited to young women, because this group is at particularly high risk for emotional disorder (Kessler et al., 2005; Rohde, Lewinsohn, & Klein, 2013), so should show variation in cognitive vulnerability. Further, the relationship between frontal asymmetry and psychopathology vulnerability is robust in women (Thibodeau et al., 2006), but less so in men (Miller et al., 2002; Smit, Posthuma, Boomsma, & De Geus, 2007; Stewart, Bismark, Towers, Coan, & Allen, 2010).

The asymmetric inhibition model (Grimshaw & Carmel, 2014) proposes that right IPFC is responsible for inhibiting positive stimuli and left IPFC is responsible for inhibiting negative stimuli. If so, frontal asymmetry should predict ability to inhibit distractors on the irrelevant distractor paradigm accordingly. As more positive values of frontal asymmetry

indicate greater LFA and more negative scores indicate greater RFA, frontal asymmetry should be positively correlated with distraction by positive images and negatively correlated with distraction by negative images. Greater LFA (lower RFA) should predict more positive distraction and greater RFA (lower LFA) should predict more negative distraction.

According to the capability model (Coan et al., 2006), frontal asymmetry is a stronger, more reliable predictor of emotional vulnerability during emotional challenge than at rest. Any relationships between frontal asymmetry and distraction levels were expected to be stronger, if not solely evident, when asymmetry was measured during emotional challenge than when measured at rest.

Study 1

The purpose of Study 1 was to develop and validate a paradigm to induce emotional challenge, to enable testing of the asymmetric inhibition model (Grimshaw & Carmel, 2014) within a capability framework in Study 2. The capability model (Coan et al., 2006) argues that frontal asymmetry is a more powerful and reliable measure of individual differences when measured during emotional challenge, than when measured at rest. Previous capability research has induced emotional challenge through various methods, including directed emotional facial expressions (e.g., Coan et al., 2006; Stewart et al., 2011, 2014), passive viewing of distressing videos (e.g., Dennis & Solomon, 2010; Papousek et al., 2014), auditory presentation of human emotional expressions (e.g., Papousek, Freudenthaler, & Schulter, 2011; Papousek, Reiser, Weber, Freudenthaler, & Schulter, 2012), working memory tasks with threat of shock (e.g., Goodman, Rietschel, Lo, Costanzo, & Hatfield, 2013), and speech preparation (e.g., Cole et al., 2012; Pérez-Edgar et al., 2013). To ensure that the emotional challenge paradigm was as effective as possible, I used an adapted version of the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), widely used for over 20 years to induce stress (for a review, see Kudielka, Hellhammer, & Kirschbaum, 2007). The TSST has been shown to elicit a strong stress response (for a meta-analysis, see Dickerson & Kemeny, 2004) and provides a more intense, and naturalistic, experience of emotional challenge than many of the tasks previously used in capability research.

In the classic TSST paradigm, participants prepare for and give a speech in front of a panel of judges, then perform a serial subtraction task, during which they count backwards in large increments (e.g., serially subtract 17 from 2023) for five minutes. Participants show a clear stress response to the classic application of these tasks, revealed by greater cortisol levels, accelerated heart rate and increased electrodermal activity (e.g., Hofmann et al., 2005; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004; Lin, Lin, Lin, & Huang,

2011), as well as deteriorated mood (e.g., Ellenbogen, Schwartzman, Stewart, & Walker, 2002; Wahlström, Hagberg, Johnson, Svensson, & Rempel, 2002). The strongest responses result when the tasks are both uncontrollable (perceived failure at the task despite best efforts) and socially evaluated (real or potential negative judgement of performance by others; Dickerson & Kemeny, 2004).

Study 1 piloted a modified version of the TSST's serial subtraction ("counting") task for use in Study 2, to fine-tune implementation and evaluate efficacy. This task was chosen because it is quick and easy to implement alongside the irrelevant distractor paradigm and comprises elements of both uncontrollability and social evaluation (via monitoring and scripted feedback from an "evaluator") to maximise emotional challenge. Emotional response to the task was inferred from physiological responses (heart rate and electrodermal activity) and subjective mood reports (stress, anger, worry, sadness, happiness), measured to ensure that participants actually *felt* emotionally challenged, over and above any effects of cognitive load. Emotional response was measured as the change in physiological activity and subjective mood from an initial rest period to the counting task. A control group was included to ensure that any change in physiology and mood between the resting and counting tasks was due to the stressful nature of the task, and not simply cognitive engagement or motor movements associated with speech production. Controls completed an easy counting task without monitoring. Heart rate and electrodermal activity were expected to increase from rest to counting in both groups due to task engagement, but if the modified TSST counting task is successful at inducing stress, then these increases should be greater for the stress group. Subjective stress ratings were expected to increase significantly after the counting task for the stress group only. The other mood ratings were included to explore other potential mood effects; mood was generally expected to deteriorate, but specific predictions were not made.

Method

Design

A mixed experimental design was employed to test the efficacy of the counting task. Response to the counting task was compared between the stress group and the control group. Heart rate and electrodermal activity were compared between the resting period and counting task. Subjective response was measured in self-reported mood ratings (stress, anger, worry, sadness, happiness), compared before and after the counting task.

Participants

Twenty-five undergraduate women were randomly assigned to either the stress group ($n = 13$) or control group ($n = 12$). Participants were 18-24 years old ($M_{stress} = 19.30$ years, $SD_{stress} = 1.83$; $M_{control} = 18.56$ years, $SD_{control} = 1.13^1$), and were right-handed, fluent English speakers with no known hearing impairments, normal or corrected-to-normal vision and no current diagnosis of depression or anxiety. These criteria were chosen so that participant characteristics would be similar to Study 2. All participants gave written informed consent and received course credit in exchange for their time. This study was approved by the Human Ethics Committee of Victoria University of Wellington, New Zealand.

Materials

Counting task procedure. The stress group completed a counting task adapted from the classic TSST procedure (Kirschbaum et al., 1993). Participants were told that they would complete a mental arithmetic task assessing working memory ability and verbal intelligence. Their behaviour during the task would be recorded via video-camera for future assessment and they would be monitored by an ‘evaluator’, who is a psychologist trained to assess verbal and non-verbal behaviour (to increase the social evaluation aspect of the task). After setting up the camera, the experimenter exited the room and the evaluator (a mature male research assistant) entered. The evaluator maintained a neutral, professional demeanor throughout the session and spoke from scripted lines (see Appendix A). He told the participant to count backwards from 2023 in steps of 17 aloud, as quickly and accurately as possible. As they counted, he timed them using a stopwatch, made notes on a clipboard, and enforced a restart after every error. He also pressed them to count faster, particularly for those who were skilled at the task, to maximise feelings of uncontrollability. The evaluator ended the task after five minutes.

The control group performed a similar, but easier task. Participants were left alone (no evaluator or camera) to count forwards from zero in steps of five, aloud. The experimenter re-entered after five minutes to end the task. This task was designed to match the stress group’s task in movement and speech, but to be low in stress (i.e., performance is controllable and not socially evaluated).

Self-report ratings. Changes in subjective mood over the session were assessed using visual analog scales, administered via an online questionnaire forum (Qualtrics) on a Dell Precision T1600 computer with an Alienware AW2310 24” digital monitor. Five aspects of

¹ Specific age data were unsuccessfully recorded for 6 participants (3 controls). Recruitment screening procedures ensured all participants were aged 18-24.

current mood were assessed (e.g., *Indicate how stressed you feel on the ruler below*) using a 100-point slider, from 0 (e.g., *No stress at all*) to 100 (e.g., *As stressed as I could be*), with the slider initially centered between the two points. Each of the five moods (stress, anger, worry, sadness, happiness) were rated in this way, in randomised order. This simple scale discriminates stress levels to a similar level as a questionnaire, has good construct validity (Lesage, Berjot, & Deschamps, 2012) and has been used to assess mood change in capability research (e.g., Meyer et al., 2014).

Two ratings of task experience were also collected (*The experiment task was stressful for me; I found the experiment task to be a challenge*) on 100-point sliders from 0 (*Not at all*) to 100 (*Extremely So*)², always presented in the same order. These rating statements have been used previously to assess efficacy of TSST procedures (e.g., Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999).

Procedure

Participants were run individually. On arrival, they were told that we would be studying their physiological responses to cognitive tasks. Participants first rated their current mood. The ECG and electrodermal electrodes were then attached and the importance of minimising movement artifacts explained. Participants rested for four minutes with their eyes closed while baseline physiology was recorded. Procedure then diverged for each group; the stress group completed the TSST-adapted counting task and the control group completed the low stress counting task. Participants then completed the final set of mood and task ratings and were debriefed. The session lasted around 45 minutes.

Physiology Data Recording and Reduction

To measure heart rate, electrocardiogram (ECG) was recorded using three disposable adhesive Ag-AgCl foam ECG electrodes (Kendall Meditrac, Tyco Healthcare), placed according to a Lead II system (see Figure 1). ECG was amplified using an ML408 Dual Bio Amp/Stimulator (ADInstruments, Australia). Electrodermal activity, a measure of sympathetic nervous system activation, was recorded using MLT116F GSR electrodes attached to the medial phalanges of the index and ring fingers of the right hand. Electrodermal activity was amplified using an ML116 GSR Amp (ADInstruments, Australia).

The amplified analogue signals were converted to digital using a PowerLab 16/30 amplifier, sampled at a rate of 1 kHz. Samples were recorded and processed by LabChart

² Participants also made two ratings of task controllability. These results were not presented in this thesis due to participant confusion over question wording.

Pro 8.0 software (ADInstruments, Australia) on a Dell Optiplex 9020 computer, running Windows 7 Enterprise operating system. ECG data was filtered offline (band-pass filter: 8-40 Hz). Artifacts were identified by visual inspection of the heart rate data; data points within a time window spanning two R-wave spikes either side of the artifact were excluded from both heart rate and electrodermal activity data. Heart rate was then calculated using the inter-beat interval (time between R-wave spikes), converted to beats per minute, and averaged across the four-minute rest period and across the five-minute counting period for each participant. Electrodermal activity (recorded in micro-Siemens) was averaged across the rest period and counting period for each participant and compared, a procedure used commonly in electrodermal research (e.g., Wagner & Abaied, 2016). As electrodermal data showed no skew or kurtosis, raw averages were used in analyses.

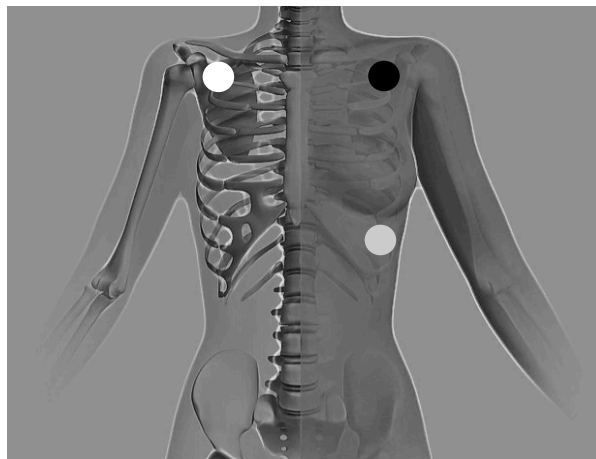


Figure 1. Electrocardiogram (ECG) electrode placement (white: negative electrode; grey: positive electrode; black: ground electrode) in Study 1.

Data Analysis

Statistical analyses were conducted using SPSS (Statistical Package for Social Sciences) version 22 (SPSS Inc, Chicago). Statistical significance was set at $p < .05$, with $p < .1$ considered marginally significant for predicted effects only. Statistical corrections were applied for heterogeneity of variance and sphericity violations (Greenhouse Geisser). Effect sizes for independent comparisons were calculated using Cohen's d (mean difference divided by the pooled standard deviation). Paired comparisons can produce large effect sizes due to strong correlations between measurements, resulting in a power advantage for repeated measures designs over independent designs when using Cohen's d . Thus, for paired comparisons, the correlation between measurements was included in the effect size

calculation to correct for dependence between means and thereby produce Cohen's d comparable to that of independent comparisons (Dunlap, Cortina, Vaslow, & Burke, 1996). All figures include 95% confidence intervals, adjusted for within-subjects comparisons where appropriate (Morey, 2008).

Results and Discussion

To assess the efficacy of the counting task at inducing emotional challenge, mean heart rate, electrodermal activity and subjective mood ratings were compared between the two tasks (resting, counting) and between the two groups (stress, control). Group means and standard deviations are presented in Tables 1 and 2.

Table 1

Mean Physiology Measurements (with Standard Deviations) in Study 1

Measure	Control group		Stress group	
	Resting	Counting	Resting	Counting
Heart rate ^a	85.84 (13.42)	95.03 (10.63)	81.85 (11.05)	102.34 (19.15)
Electrodermal activity ^b	-1.65 (3.13)	5.14 (4.06)	-0.74 (1.58)	8.73 (4.05)

^aIn beats per minute. ^bIn micro-Siemens (μ S).

Physiological Response

Heart rate increased from resting to counting, with the stress group showing a substantially greater increase than controls (see Figure 2). A two-way mixed model analysis of variance (ANOVA), with task (resting, counting) as the within-subjects independent variable, group (stress, control) as the between subjects independent variable and heart rate as the dependent variable, showed heart rate to be significantly faster during the counting task than the resting period, $F(1, 23) = 38.24, p < .001, \eta_p^2 = 0.62$, but no main effect of group, $F < 1$. These findings were qualified by a significant task x group interaction, $F(1, 23) = 5.54, p = .028, \eta_p^2 = 0.19$. Heart rate increased from resting to counting for both groups, but to a greater extent for the stress group, $t(12) = 5.09, p < .001, d = 1.17$, than for controls, $t(11) = 3.78, p = .003, d = 0.73$.

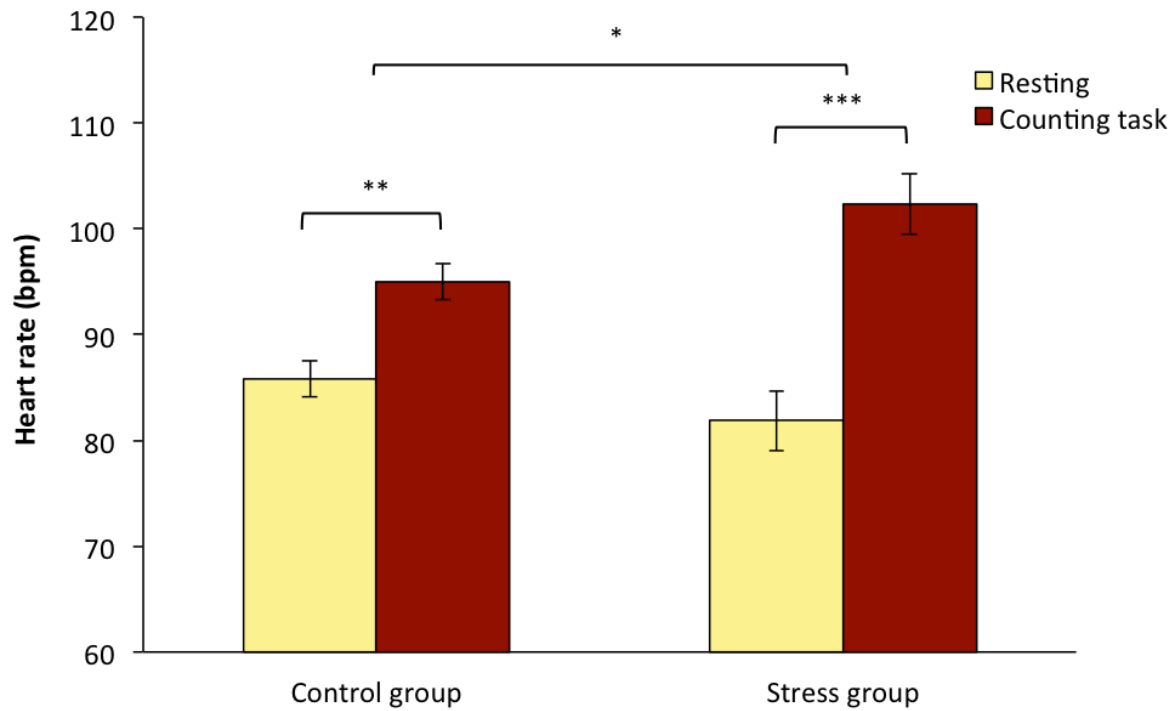


Figure 2. Heart rate (in beats per minute) in Study 1. Error bars represent 95% confidence intervals for within-subject comparisons. * $p < .05$. ** $p < .01$. *** $p < .001$.

Electrodermal activity also increased from resting to counting, with the stress group showing a slightly greater increase than controls (see Figure 3). Electrodermal activity levels were significantly greater during the counting task than the resting period, $F(1, 23) = 109.24, p < .001, \eta_p^2 = 0.83$, and marginally greater for the stress group than controls, $F(1, 23) = 4.26, p = .051, \eta_p^2 = 0.16$. Both main effects were qualified by a predicted marginal task x group interaction, $F(1, 23) = 2.95, p = .099, \eta_p^2 = 0.11$. Electrodermal activity increased from rest to counting for both groups, but more so for the stress group, $t(12) = 8.64, p < .001, d = 2.93$, than for controls, $t(11) = 6.17, p < .001, d = 1.85$. This TSST-adapted counting task therefore showed the predicted increases in heart rate and electrodermal activity, relative to the control group, suggesting that the task successfully induced a physiological stress response.

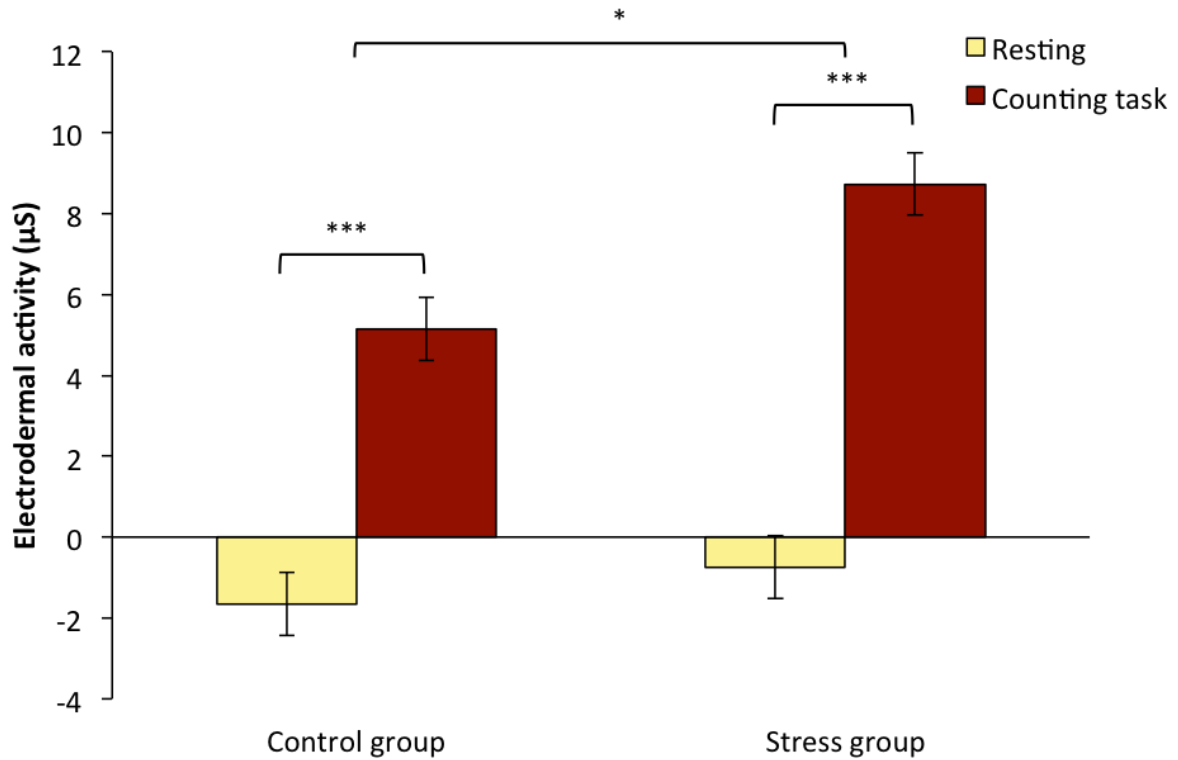


Figure 3. Electrodermal activity (in micro-Siemens, μS) in Study 1. Error bars represent 95% confidence intervals for within-subject comparisons. $*p < .05$. $***p < .001$.

Subjective Response

While controls showed little change in mood across the session, the stress group reported greater levels of subjective stress, anger, worry and sadness, and lower levels of happiness after the counting task than at the beginning of the session (see Figure 4). Mood changes were analysed in a two-way mixed model multivariate analysis of variance (MANOVA), with task (resting vs counting) as the within-subjects independent variable, group (stress vs control) as the between-subjects independent variable, and the five mood ratings (stress, worry, anger, sadness, happiness) as the dependent variables. Neither of the main effects approached significance, F 's < 1.3 . Group significantly interacted with task, using Pillai's trace, $V = 0.57$, $F(5, 19) = 4.93$, $p = .005$. The task x group interaction was significant for ratings of stress, $F(1, 23) = 6.64$, $p = .017$, $\eta_p^2 = 0.22$, anger, $F(1, 23) = 6.73$, $p = .016$, $\eta_p^2 = 0.23$, and happiness, $F(1, 23) = 9.65$, $p = .005$, $\eta_p^2 = 0.30$, but did not reach significance for sadness, $p = .095$, or worry, $p = .178$. Paired t-tests revealed that while the stress group showed significant increases in stress, $t(12) = 2.23$, $p = .046$, $d = 0.37$, and anger, $t(12) = 2.93$, $p = .013$, $d = 0.10$, and decreases in happiness, $t(12) = 3.00$, $p = .011$, d

= 0.26, the control group showed no change in stress, $p = .180$, anger, $p = .704$, or happiness, $p = .192$. In sum, mood deteriorated during the counting task (stress and anger increased, happiness decreased) for the stress group compared to controls, indicating that the TSST-adapted counting task successfully induced both physiological and subjective emotional challenge.

Table 2

Mean Self-Reported Mood Ratings (with Standard Deviations) in Study 1

Mood rating	Control group	Stress group
Stress		
Start of session	58.17 (25.79)	52.92 (26.95)
After counting task	48.42 (26.96)	67.46 (22.68)
Anger		
Start of session	14.92 (20.33)	11.38 (19.19)
After counting task	13.08 (15.54)	31.46 (26.53)
Worry		
Start of session	45.08 (28.34)	38.15 (30.77)
After counting task	44.58 (25.05)	50.54 (27.31)
Sadness		
Start of session	38.67 (33.18)	20.15 (27.49)
After counting task	34.33 (28.14)	32.00 (27.33)
Happiness		
Start of session	52.58 (25.52)	64.38 (21.28)
After counting task	58.75 (20.77)	50.92 (18.09)

Note. Mood was rated on a scale from 0 to 100, with higher scores indicative of greater current mood experience.

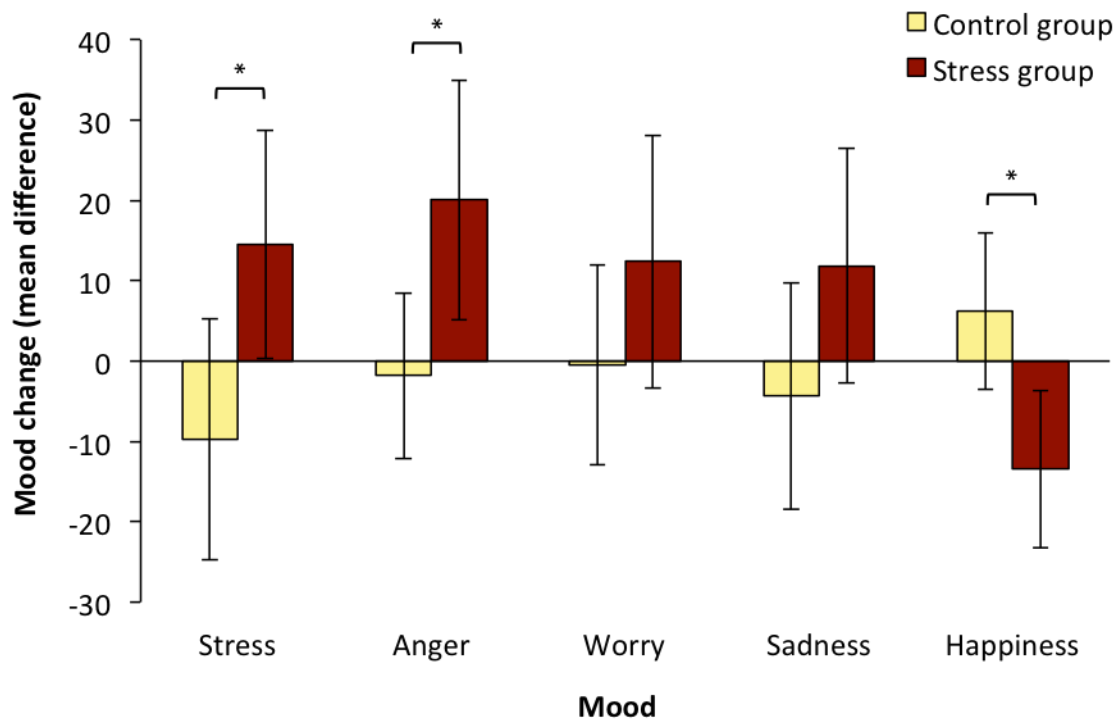


Figure 4. Change in subjective mood (mean difference between post- and pre-counting ratings) in Study 1. Mood was rated from 0 to 100. Positive mood change values indicate greater reported mood experience after the counting task than at the beginning of the session. Error bars represent 95% confidence intervals. $*p < .05$.

Furthermore, the stress group rated the counting task as significantly more stressful, $t(15.090) = 5.97, p < .001, d = 2.46$, and challenging, $t(14.356) = 10.18, p < .001, d = 4.20$, than controls (see Table 3), providing additional evidence that the counting task was successful at inducing emotional challenge.

Table 3

Mean Task Experience Ratings (with Standard Deviations) in Study 1

Statement	Control group	Stress group
The experiment task was stressful for me	24.33 (28.92)	78.69 (13.14)
I found the experiment task to be a challenge	15.50 (23.26)	88.92 (9.53)

Note. Statements were rated on a scale from 0 to 100, with higher ratings indicative of greater endorsement of the statement.

Summary

Study 1 showed that the TSST-adapted counting task was effective at inducing emotional challenge. The stress group reported finding the task highly stressful and difficult and showed larger heart rate and electrodermal activity responses relative to controls, replicating other studies' findings (e.g., Goodman et al., 2013; Kudielka et al., 2004; Lin et al., 2011). The combination of increases in self-reported stress and in objective physiological indices of stress provides good evidence that the adapted TSST counting task was in fact inducing emotional challenge. Subjective anger also increased, while happiness decreased, suggesting that the counting task effected several facets of emotional challenge.

It is possible that changes in physiology during the counting task may occur in response to an increase in cognitive load rather than stress. Both heart rate and skin conductance increase during cognitively demanding tasks (e.g., Engström, Johanssen, & Östlund, 2005; Kennedy & Scholer, 2000; Turner & Carroll, 1985; Wilson & Eggemeier, 1991). While it can be difficult to distinguish effects of stress and cognitive load on physiological responses (i.e., they are not mutually exclusive; stress is cognitively demanding and cognitively demanding tasks can be stressful), subjective mood ratings in the present study indicated that the task is indeed stressful. For the purposes of this thesis, separation of cognitive load and stress was deemed unnecessary. Capability effects have been elicited using cognitively demanding tasks (e.g., Cole et al., 2012; Goodman et al., 2013; Pérez-Edgar et al., 2013) and, indeed, some level of cognitive demand may be inherent and even essential to successful capability induction. As subjective mood ratings indicated that participants subjectively experienced emotional challenge, the counting task was deemed effective for use in Study 2.

Study 2

To elucidate the cognitive mechanisms underlying frontal asymmetry, Study 2 tested whether frontal asymmetry predicts ability to inhibit emotional distractors, as outlined by the asymmetric inhibition model (Grimshaw & Carmel, 2014). This model unites two sets of literature: one that associates frontal asymmetry with cognitive control mechanisms in IPFC and the other that associates valence-specific processing biases with emotional disorders linked to distinct patterns of frontal asymmetry. Frontal asymmetry is proposed to reflect individual differences in the ability to use valence-specific inhibitory control mechanisms: left frontal areas are posited to inhibit negative information and right frontal areas are posited to inhibit positive information. Study 2 tested this hypothesis; frontal EEG asymmetry was tested as a predictor of ability to differentially inhibit positive and negative

distractors. A non-clinical sample was recruited to enable study of individual differences in control that may explain the relationship between frontal asymmetry and vulnerability to psychopathology.

Ability to inhibit emotional distractors was assessed using an irrelevant distractor paradigm, in which participants identified a target letter (X or N) within a symbol array (five o's) while attempting to ignore peripheral images (erotic scenes of couples, scenes of bodily mutilations, and scenes involving people). This task reliably elicits emotional distraction effects; participants are slower to respond when an image is present than when there is no image (Forster & Lavie, 2008a, 2008b), especially when the images are emotional in nature (Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016). Distractors were presented on 50% of trials, blocked by distractor valence (positive, negative, neutral). Previous studies have shown that emotional distraction effects are larger when distractors are infrequent (presented on 25% of trials; Grimshaw et al., 2016; Kranz, 2015; Murphy, 2016) and smaller when distractors are frequent (presented on 75% of trials), suggesting that increasing distractor expectancy elicits more effective use of control. As the present study used an individual differences approach, a distractor frequency of 50% was chosen to maximise individual variability in responding. Previous work in our lab has shown that 50% distractor frequency produces greater variability in response time than 25% distractor frequency, while still inducing substantial emotional distraction (Grimshaw et al., 2016; see Appendix B).

Frontal asymmetry was measured under two conditions: at rest and during emotional challenge. These measures enabled hypothesis testing within a traditional resting asymmetry framework (Davidson, 1998) and a capability framework (Coan et al., 2006), respectively. Emotional challenge was induced after the irrelevant distractor paradigm (negative emotion can influence ability to use cognitive control; e.g., Curci, Lanciano, Soleti, Rimé, 2013), using the counting task piloted in Study 1. Because it was not possible to collect clean EEG recordings while participants were counting, a silent speech preparation task was added after the counting task to maintain the elicited stress response for EEG recording. Speech preparation elicits a reliable stress response (for a review, see Kudielka et al., 2007) and has been used to induce emotional challenge in prior capability research that has linked frontal asymmetry with attention biases to emotion (Pérez-Edgar et al., 2013).

As depression and anxiety symptoms have been associated with both frontal asymmetry (e.g., Thibodeau et al., 2006) and inhibitory control (e.g., Eysenck & Derakshan,

2011; Snyder, 2013), current symptom levels were assessed via self-report questionnaires to enable symptom variance to be controlled for in analyses.

In line with previous findings (Grimshaw et al., 2016), a main effect of emotional distraction (i.e., greater distraction from both positive and negative images than neutral images) was expected. Importantly, if the asymmetric inhibition model is correct and frontal EEG activity reflects valence-specific inhibitory control mechanisms, then frontal EEG asymmetry should predict individual differences in positive and negative distraction. Frontal asymmetry was expected to positively correlate with positive distraction (i.e., greater LFA/lower RFA predicts more positive distraction) and negatively correlate with negative distraction (i.e., greater RFA/lower LFA predicts more negative distraction). As frontal asymmetry represents relative activity between the hemispheres, predictions can also be framed in relative ability to control positive and negative distractors. Greater RFA should be associated with better inhibitory control of positive over negative information, resulting in greater negative (than positive) distraction, while greater LFA should be associated with better inhibitory control of positive than negative information, resulting in greater positive (than negative) distraction. In line with the capability model (Coan et al., 2006), any relationships between frontal asymmetry and distraction were expected to be stronger for frontal asymmetry measured during emotional challenge, than for frontal asymmetry measured at rest.

Method

Design

Study 2 used an individual differences design to test whether frontal asymmetry moderates emotional distraction. Frontal EEG asymmetry was measured at rest and during emotional challenge (during silent speech preparation, following a stressful counting task). Distraction was measured using an irrelevant distractor paradigm, which assessed ability to inhibit positive and negative images. Neutral distractors were also included as a control condition, to enable testing of inhibitory control of emotion, over and above control of images per se.

Participants

Participants were 61 women who met the same criteria as for Study 1, as well as reporting no neurological issues and either heterosexual or bisexual orientation³. Participants provided informed written consent and received course credit or vouchers in compensation.

³ Participants were screened for sexuality because the positive images showed erotic scenes of heterosexual couples, which may be less positive for homosexual and asexual individuals.

This study was approved by the Human Ethics Committee of Victoria University of Wellington, New Zealand. Accuracy on this task was high ($M = 96.26\%$, $SD = 2.86$). Three participants were excluded for low accuracy ($< 80\%$) in one or more of the three valence blocks of the irrelevant distractor paradigm and one participant was excluded due to excessive EEG artifacts during the speech preparation task ($< 75\%$ of epochs providing usable data; Luck, 2005), leaving 57 participants ($M_{age} = 19.09$ years, $SD = 1.31$) for inclusion in analyses.

Procedure

Participants were run individually in an electrically shielded, dimly lit and sound attenuated Faraday chamber. They were seated in an ergonomic chair, which they reclined for the resting phases. After giving consent, participants completed questionnaires assessing current depression and anxiety (Mini Mood & Anxiety Symptom Questionnaire; Depression, Anxiety & Stress Symptom questionnaire; State-Trait Anxiety Inventory - Trait form⁴) and rated their current mood. ECG electrodes were then attached and the EEG electrode cap fitted. The rest of the session comprised four phases: baseline resting, irrelevant distractor paradigm, pre-stress resting, and stress induction. For the resting phases, participants rested while EEG was recorded for four one-minute blocks with eyes open (O) or closed (C); block order was counterbalanced across participants and the same for both resting phases (O-C-C-O or C-O-O-C). The stress induction phase comprised the counting task piloted in Study 1, followed by a silent speech preparation task. Participants rated current mood before and after the stress induction phase and finished the session by rating their experience of the stress induction tasks. ECG and EEG were recorded throughout these phases (see Table 4 for a procedure timeline). The session took approximately two hours.

⁴ The Emotion Regulation Questionnaire (Gross & John, 2003) and Kinsey Scale (Kinsey, Pomeroy & Martin, 1948) were completed after the STAI-T, but were not analysed in this thesis.

Table 4

Study 2 Procedure Timeline

Approximate timeline (hour:minutes)	Task	
0:00	Consent	
0:03	Questionnaires	
0:12	Mood rating	
0:13	Physiology set-up	
0:43	Baseline resting	
0:48	Irrelevant distractor paradigm	
1:39	Mood rating	
1:40	Pre-stress resting	
1:45	Counting task	Stress induction
1:52	Speech preparation	
1:55	Mood rating	
1:56	Task ratings	
1:57	Debriefing	

Note. EEG and ECG were analysed during tasks in boldface.

Materials

The resting procedure, irrelevant distractor paradigm and questionnaires were administered through E-Prime 2.0 (Psychology Software Tools, Pittsburgh PA) on a Dell Precision T3610 computer with a 23" Alienware 2310 LCD digital monitor (1920 x 1080 pixels, 120 Hz vertical refresh rate) and Dell A215 Multimedia speakers.

Irrelevant distractor paradigm. Participants identified a target letter (X or N, size 24, white Arial font, subtending 0.67° of visual angle) amongst five identical non-targets (o, size 8, white Arial font, subtending 0.22° of visual angle), presented within a circular array (radius of 1.75° eccentricity) around fixation. On 50% of trials, a distracting image (subtending 6.68° x 6.68° of visual angle) was presented simultaneously with the array, with

the nearest edge of the image 3.34° above or below fixation. Target identity, target location, distractor presence and distractor location were counterbalanced across trials⁵.

Distractors were thirty-six colour images selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008): 12 positive (erotic scenes of heterosexual couples), 12 negative (human bodily mutilations) and 12 neutral (people doing everyday activities). Within each block of trials, images were randomly presented from the valence set. The set was repeated eight times throughout the block; images were repeated only when all 12 had been presented. Image sets differed in valence rating, $F(2, 33) = 475.62, p < .001, \eta_p^2 = 0.97$, with positive images more pleasant than neutral images, $t(33) = 7.91, p < .001, d = 2.78$, and neutral images more pleasant than negative images, $t(33) = 21.87, p < .001, d = 11.95$. Image sets also differed in arousal rating, $F(2, 33) = 372.64, p < .001, \eta_p^2 = 0.96$. Positive and negative images were matched on arousal, $t(33) = 1.59, p = .122$, and neutral images were lower in arousal than both positive images, $t(33) = 22.81, p < .001, d = 10.58$, and negative images, $t(33) = 24.40, p < .001, d = 9.27$. See Table 5 for valence and arousal ratings. Images were matched for luminance and contrast using Matlab's SHINE Toolbox (Willenbocket et al., 2010). Twelve pixel-scrambled images (1296 pixels), four from each valence set, were created for the practice trials.

Table 5

Mean Ratings (with Standard Deviations) for the Image Stimuli Used in the Irrelevant Distractor Paradigm

Image set	Valence	Arousal
Negative	1.64 (0.21)	6.53 (0.42)
Neutral	5.01 (0.34)	3.07 (0.32)
Positive	6.23 (0.52)	6.31 (0.29)

Note. Image sets were each comprised of 12 images from the International Affective Picture Stimuli (IAPS) database (Lang et al., 2008). Negative images: 3015, 3030, 3059, 3103, 3131, 3140, 3150, 3195, 3550.1, 9253, 9405, 9420. Neutral images: 2026, 2102, 2221, 2305, 2393, 2397, 2411, 2512, 2593, 2595, 2745.1, 2840. Positive images: 4658, 4659, 4660, 4668, 4680, 4690, 4693, 4694, 4695, 4697, 4698, 4800. Ratings can range from 1 to 9, with lower ratings indicative of low arousal and a more “unpleasant” valence and higher ratings indicative of high arousal and a more “pleasant” valence. Ratings for each image, averaged across female raters, were taken from Lang et al. (2008), and averaged.

⁵ Every combination of these parameters was presented randomly and only repeated after all combinations of the set had been presented.

A schematic of trial procedure is presented in Figure 5. Each trial began with a fixation cross for a random duration of 416 to 834 ms, replaced by the array for 100 ms. A fixed response window of 2000 ms began at onset of the letter display, during which participants identified the target letter using the 1 and 2 numberpad keys (key mappings counterbalanced across participants) with right index and middle fingers. A blank screen followed for 310 ms, during which auditory feedback (an error tone) was presented for incorrect responses or no response. A jittered inter-trial interval (206-624 ms) separated trials.

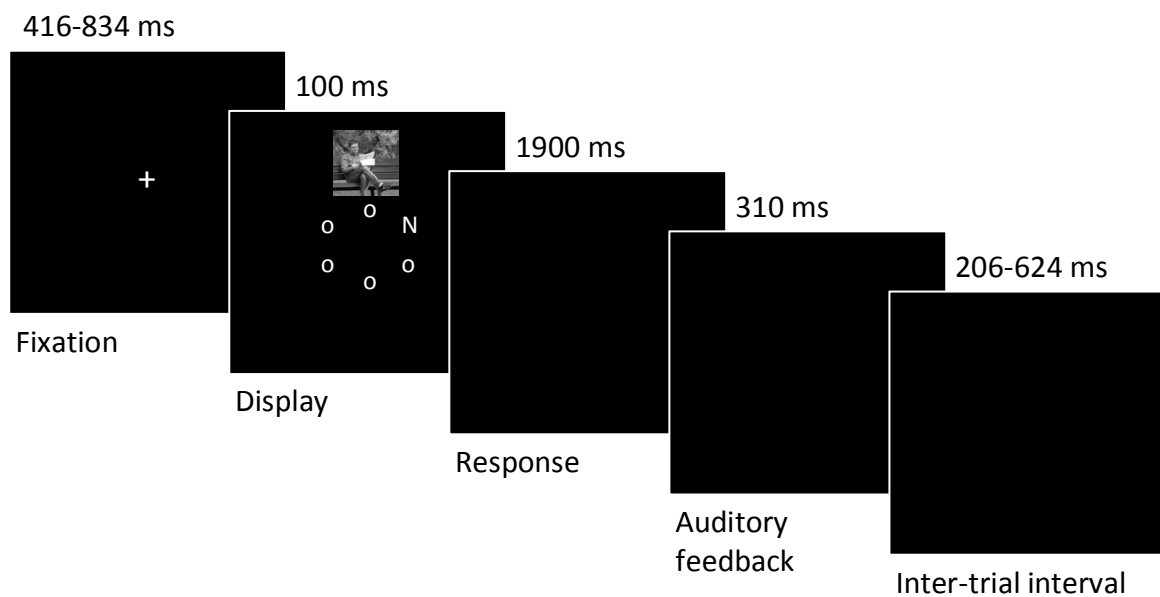


Figure 5. An example of a trial procedure in the irrelevant distractor paradigm in Study 2. Participants identified the target (X or N) in the letter display, whilst attempting to ignore distractor images. Distractors were presented on 50% of trials, either above or below the display. An error tone was presented for an incorrect response or no response. The image depicted is for display purposes only; image stimuli were selected from the International Affective Picture System (IAPS; Lang et al., 2008). Display is not to scale.

Before beginning the task, participants were asked to respond as quickly and accurately as possible. To encourage use of control, participants were explicitly told to ignore the distracting images that would appear on 50% of trials. Participants completed 48 practice trials before starting the main task; distractors were presented at the same frequency, but comprised scrambled images so participants could practice controlling the

images without exposure to valence⁶. The main task comprised 576 trials, divided into three valence blocks of 192 trials, with valence order counterbalanced across participants. To separate the valences, participants were given a one-minute rest period after each valence block, during which they relaxed with their eyes closed. Participants were also given a self-timed break every 96 trials to reduce fatigue effects.

Stress induction. The counting task was run as in Study 1, with some adaptations: (1) to encourage feelings of uncontrollability (to increase stress), participants who were performing well were told they were counting too slowly and restarted; (2) counting time was reduced to 3 minutes to keep within time restraints; (3) a silent speech preparation task was added after the counting task to maintain stress while allowing for EEG recording with minimal movement artifacts. Speech preparation is commonly used as part of the classic TSST (Kirschbaum et al., 1993; Kudielka et al., 2007) and has been validated to effectively induce stress. The task used in the present study was simplified from the original TSST version and is similar to speech preparation tasks used in previous capability research (e.g., Cole et al., 2012; Pérez-Edgar et al., 2013).

After ending the counting task, the evaluator told participants that they should now silently prepare for a three-minute speech about how their performance in the task relates to aspects of their life and personality. After giving the speech, they would rate the previous participant's speech from the video recording and the next participant would do the same for their recording. They were left alone to prepare silently for two minutes, while EEG and ECG were recorded to measure physiological stress response. After rating their current mood, participants were told that the study was running behind schedule so they would not give the speech or rate the previous participant's speech. To check that the stress induction was effective at inducing subjective emotional challenge, participants completed the same self-report mood rating scales and similar task rating scales as in Study 1 (see Table 10).

Questionnaires. Average scores for depression, anxious arousal and trait anxiety were low (see Table 6)⁷.

Depression, Anxiety and Stress Symptom Questionnaire (DASS). Depression was measured using the Depression subscale of the DASS (Lovibond & Lovibond, 1995), which comprises 14 items (see Appendix C). Participants rate the extent to which each item (e.g., *I*

⁶ Distraction does not differ between valences when using pixel-scrambled images (Grimshaw et al., 2016).

⁷ Though only one subscale was analysed for the Mini-MASQ and DASS, participants completed the full version of each questionnaire.

felt sad and depressed) applied to them over the past week on a 4-point scale, from 0 (*Did not apply to me at all*) to 3 (*Applied to me very much, or most of the time*). Past research has shown good reliability, construct validity and convergent validity for the DASS (Lovibond & Lovibond, 1995). The present study showed excellent internal consistency for the Depression ($\alpha = .91$) subscale.

Mini Mood and Anxiety Symptom Questionnaire (Mini-MASQ). Anxious arousal was measured using the Anxious Arousal subscale of the Mini-MASQ (Casillas & Clark, 2000; Clark & Watson, 1995), which comprises 10 items (see Appendix D). Participants rate the extent to which each item (e.g., *Felt like I was choking*) applied to them over the past week on a 5-point scale, from 1 (*Not at all*) to 5 (*Extremely*). Past research has shown good internal consistency ($\alpha = .88-.92$) and convergent reliability for the Mini-MASQ (Lin et al., 2014). The present study showed good internal consistency for the Anxious Arousal subscale ($\alpha = .78$).

State-Trait Anxiety Inventory – Trait Form Y-2 (STAI-T). Trait anxiety was measured using the STAI-T (Spielberger & Gorsuch, Lushene, Vagg, & Jacobs, 1983), which comprises 20 items (e.g., *I feel nervous and restless*; see Appendix E). For each item, participants rate the extent to which they generally feel that way on a 4-point scale, from 1 (*Almost never*) to 4 (*Almost always*). Past research has shown the STAI to have good internal consistency ($\alpha = .90$), test-retest reliability ($r = .70-.76$) and concurrent validity with other anxiety measures (Spielberger et al., 1983). The present study showed excellent internal consistency for the STAI-T ($\alpha = .92$).

Table 6

Study 2 Questionnaire Scores

Questionnaire	Possible score range	<i>M (SD)</i>
DASS - Depression	0-42	5.16 (5.52)
Mini-MASQ - Anxious Arousal	10-50	15.51 (4.53)
STAI-T - Trait Anxiety	20-80	42.96 (10.34)

Note. Mini-MASQ = Mini Mood & Anxiety Symptom Questionnaire; DASS = Depression, Anxiety, & Stress Symptom Questionnaire; STAI-T = State-Trait Anxiety Inventory - Trait Form.

Physiology Data Recording and Reduction

Physiological activity was amplified by Professional BrainAmps, digitized at a sample rate of 1000 Hz and recorded through BrainVision Recorder software (BrainProducts GmbH, Gilching, Germany) via a Dell Precision T1600 computer.

Electroencephalography (EEG). EEG activity was recorded from lycra Quick-Caps (Compumedics NeuroMedical Supplies), embedded with Ag/AgCl electrodes over 28 scalp sites, arranged according to the modified 10-20 system. Signal from each site was referenced online to the left mastoid electrode, then re-referenced offline to the algebraic average of the left and right mastoid electrodes. Eye movements were measured using electrooculography electrodes (horizontal: outer canthi of each eye; vertical: superior and inferior orbit of the left eye). All impedances were kept below 10 k Ω . Electrode impedances were checked between tasks and improved if necessary. Impedances of electrodes of interest (F3, F4) were usually 0-3 k Ω and kept within 1 k Ω of each other.

EEG data was processed offline using BrainVision Analyzer 2.0 software (BrainProducts GmbH, Gilching, Germany). A zero phase-shift Butterworth filter (12 dB/oct) was applied, with a low cut-off of 1 Hz, a high cut-off of 20 Hz⁸ and a notch filter of 50 Hz. Data was segmented into four 1-minute blocks for each of the two resting periods and into one 2-minute block for the emotional challenge (speech preparation) period. Each block was then segmented into 1.024 second epochs, with a 50% overlap to compensate for later application of a Hamming window, giving 464 segments for each resting phase and 233 segments for the emotional challenge period. After baseline correction, automatic artifact rejection was applied for key electrodes (F3, F4) and visually inspected for efficacy. Artifacts were detected 200 ms either side of signal deviations of greater than 100 μ V and affected segments were removed from all channels, leading to an average removal (per participant) of 1 segment ($SD = 2$) for baseline resting, 1 segment ($SD = 1$) for pre-stress resting, and 10 segments ($SD = 13$) for emotional challenge. As blinks have negligible effects on alpha asymmetries (Hagemann, 2004; Hagemann & Naumann, 2001), they were not removed from the data. Alpha power (8-13 Hz) was extracted using a Fast Fourier Transform (10% Hamming window) on the raw power of each of the key electrodes and averaged across all remaining segments.

⁸ These parameters differ from other EEG/ERP literature, which usually apply filters of 0.01-30 Hz. The use of conventional parameters resulted in a large loss of data ($n = 9$) for the speech preparation period due to movement artifacts. As only alpha power (8-13 Hz) was examined in this thesis, slightly less conservative parameters were chosen to reduce initial loss of data ($n = 1$), without compromising data quality.

Frontal asymmetry scores were calculated for alpha power using the formula: $\ln(F4) - \ln(F3)$. These electrode sites were chosen as they are the most commonly used in both resting and capability-driven frontal EEG asymmetry research (e.g., Allen et al., 2004; Coan et al., 2006; Cole et al., 2012; Dennis & Solomon, 2010; Papousek et al., 2014; Pérez-Edgar et al., 2013; for a review, see Coan & Allen, 2004), show stronger effects than lateral or composite sites in relation to current mood disorder (Thibodeau et al., 2006), and have been linked to asymmetries in dlPFC via EEG source localisation (Shackman et al., 2009). Positive asymmetry scores reflect greater alpha power over right than left frontal areas, and negative scores reflect greater alpha power over left than right frontal areas. As alpha is commonly interpreted as the inverse of cortical activity, positive scores reflect LFA, while negative scores reflect RFA (Allen et al., 2004).

Heart rate. ECG was recorded using a Lead II system, with a slightly different configuration than in Study 1 (to reduce movement artifacts; see Figure 6). Data was referenced online to the left mastoid electrode, then submitted to a linear derivation (positive electrode – negative electrode) and re-referenced to the ECG ground electrode offline, using BrainVision Analyzer 2.0 software (BrainProducts GmbH, Gilching, Germany). Further processing was conducted via the ECG module of LabChart 8.0 (ADInstruments, New South Wales, Australia), using the same procedure as Study 1. Heart rate data was averaged across each resting period and the emotional challenge period.

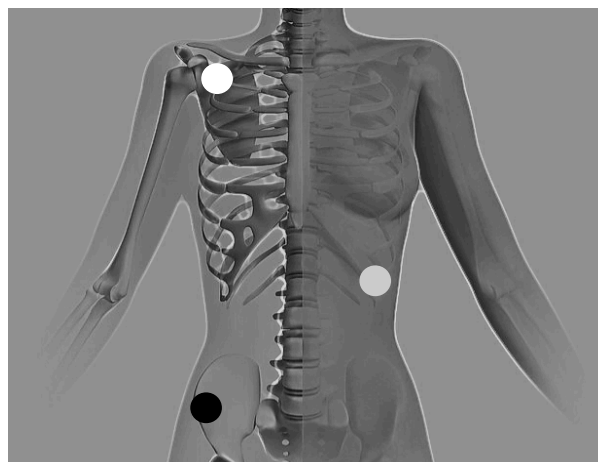


Figure 6. Electrocardiogram (ECG) electrode placement (white: negative electrode; grey: positive electrode; black: ground electrode) in Study 2.

Results and Discussion

Analyses are presented in three sections. First, behavioural data from the irrelevant distractor paradigm was tested for evidence of emotional distraction. Second, the efficacy of the stress induction was tested and its effects on frontal asymmetry explored. Third, individual difference analyses were conducted, including testing the primary research question by assessing whether frontal asymmetry (at rest and during emotional challenge) predicted emotional distraction. Statistical analysis software, significance thresholds, heterogeneity and sphericity corrections, and effect size calculations were the same as for Study 1. All figures include 95% confidence intervals, adjusted for within-subjects comparisons (Morey, 2008).

Irrelevant Distractor Paradigm

Behavioural data was examined to test whether the irrelevant distractor paradigm produced the expected emotional distraction effects. Trials with incorrect responses or RTs faster than 200 ms (indicative of anticipatory responding) were excluded from RT analyses, leading to an average removal of 22 trials ($SD = 17$) per participant. The trial response window limited maximum RT to 2000 ms, so no upper cut-off on outliers was imposed.

Mean RTs and accuracy rates were calculated for each valence block. As distraction is typically greater for both positive and negative blocks compared to neutral blocks (Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016), quadratic effects were examined by entering the neutral block as the middle variable in analyses. Quadratic relationships test directly for an emotional distraction effect (greater distraction from emotional than neutral images, regardless of valence) and were only examined for predicted effects. To control for potential order effects, valence block order was included as a between-subjects independent variable.

Response times. Distraction was evident across all valence blocks, particularly for emotional blocks, with slower responses on distractor-present trials than distractor-absent trials (see Table 7 and Figure 7). A 3 (block valence: negative, neutral, positive) x 2 (distractor presence: present, absent) x 6 (block order) mixed measures ANOVA revealed that RTs were significantly slower on distractor-present trials than distractor-absent trials, $F(1, 51) = 68.51, p < .001, \eta_p^2 = 0.57$. There were no main effects or interactions with block order, F 's < 1 , and the valence x distractor presence interaction was not significant, $F(2, 102) = 2.23, p = .113$. However, there was a significant quadratic effect for the valence x distractor presence interaction, $F(1, 51) = 4.22, p = .045, \eta_p^2 = 0.08$, indicating that emotional

distraction was greater than neutral distraction (see Figure 8). Follow-up tests showed significant distraction effects for each valence: RTs were slower for distractor-present trials (than distractor-absent trials) for negative blocks, $t(56) = 5.64, p < .001, d = 0.22$, positive blocks, $t(56) = 5.82, p < .001, d = 0.20$, and neutral blocks, $t(56) = 3.53, p = .001, d = 0.14$. Paired t-tests comparing RT difference scores (distractor-present RT – distractor-absent RT) revealed marginally greater distraction during negative blocks, $t(56) = 1.75, p = .086, d = 0.35$, and positive blocks, $t(56) = 1.82, p = .074, d = 0.33$, compared to neutral blocks. Distraction in positive and negative blocks did not significantly differ, $t(56) = 0.18, p = .861$.

Table 7

Study 2 Response Time Means (in Milliseconds) for the Irrelevant Distractor Paradigm

	Valence block		
	Negative	Neutral	Positive
Distractor-absent trials	568 (69)	570 (70)	571 (77)
Distractor-present trials	588 (83)	581 (76)	590 (87)
Distraction ^a	19 (26)	11 (23)	19 (24)

Note. Standard deviations are presented in parentheses.

^adistractor-present RT - distractor-absent RT.

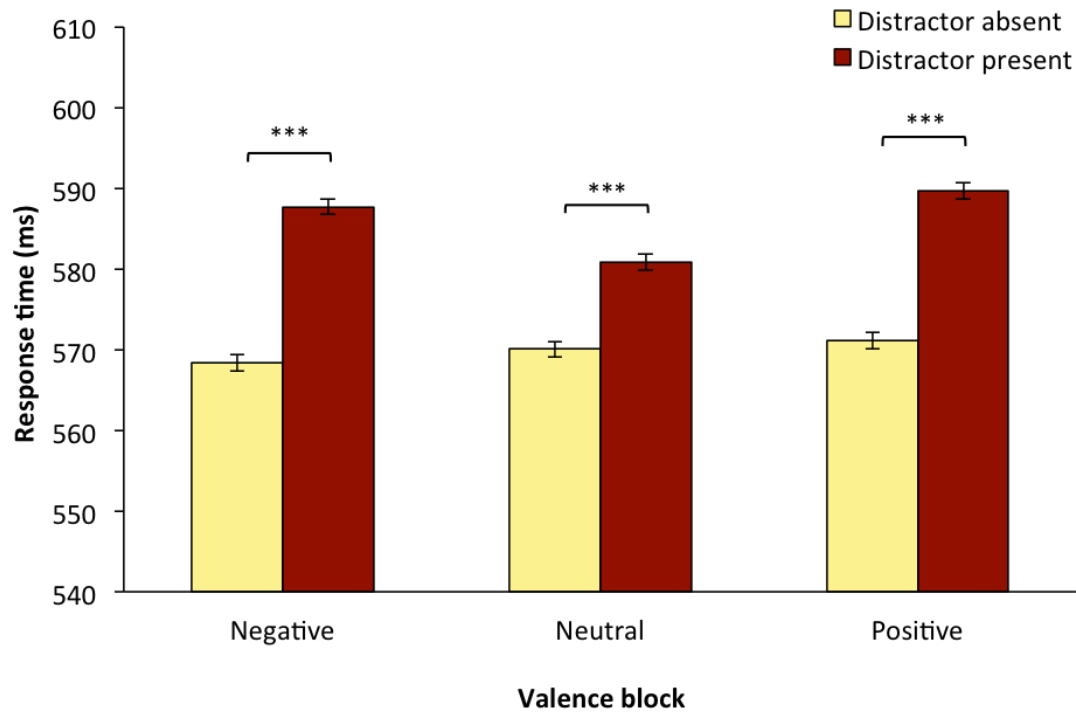


Figure 7. Response times (in milliseconds) for distractor-absent and distractor-present trials in the irrelevant distractor paradigm in Study 2. Error bars represent 95% confidence intervals for within-subject comparisons. *** $p < .001$.

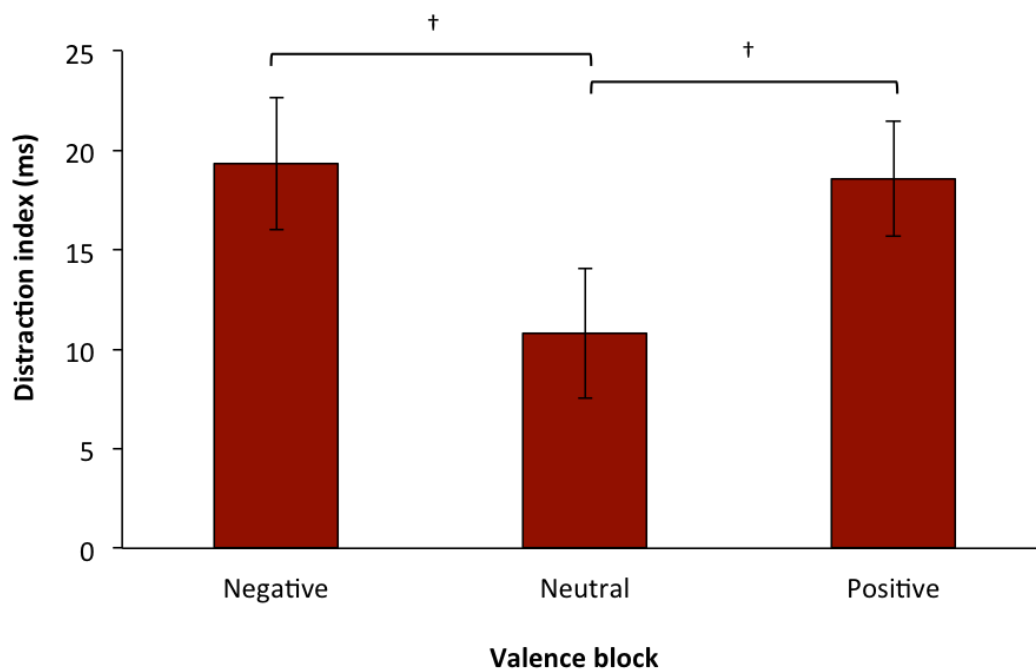


Figure 8. Distraction indices (distractor-present RT – distractor-absent RT, in milliseconds) for the irrelevant distractor paradigm in Study 2. Error bars represent 95% confidence intervals for within-subject comparisons. † $p < .1$.

Accuracy. Accuracy is typically high in the irrelevant distractor paradigm (90-99%; Forster & Lavie, 2008a, 2008b; Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016) and specific accuracy effects were not predicted. However, accuracy was examined to test for a speed/accuracy trade-off. Overall accuracy was high (see Table 8). Accuracy rates (%) were entered into a valence x distractor presence x block order ANOVA. There was a marginal valence x distractor presence interaction, $F(2, 102) = 2.54, p = .084, \eta_p^2 = 0.05$, explained by poorer accuracy for distractor-present trials than distractor-absent trials in the negative condition (see Figure 9). There were no effects of block order, F 's < 2. This pattern is inconsistent with a speed/accuracy trade-off, and consistent with the RT data showing that negative images are distracting and disrupt performance.

Table 8

Mean Accuracy (%) for the Irrelevant Distractor Paradigm in Study 2

	Valence block		
	Negative	Neutral	Positive
Distractor-absent trials	96.69 (3.28)	95.96 (3.47)	96.55 (3.06)
Distractor-present trials	95.82 (4.32)	95.87 (3.46)	96.68 (2.95)

Note. Standard deviations are presented in parentheses.

Summary. The irrelevant distractor paradigm showed clear evidence of distraction for all image types. Distraction was greater for emotional than neutral images, indicating that the task provides a good measure of susceptibility to distraction and is sensitive to emotion, supporting previous findings (Fox, Yates, & Ashwin, 2012; Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016; Yates, Ashwin, & Fox, 2010). Positive and negative images were similarly distracting, supporting the idea that positive and negative stimuli elicit similar distraction when matched for arousal (Most et al., 2007; Vogt et al., 2008). Accuracy data revealed that these effects could not be accounted for by a speed/accuracy trade-off.

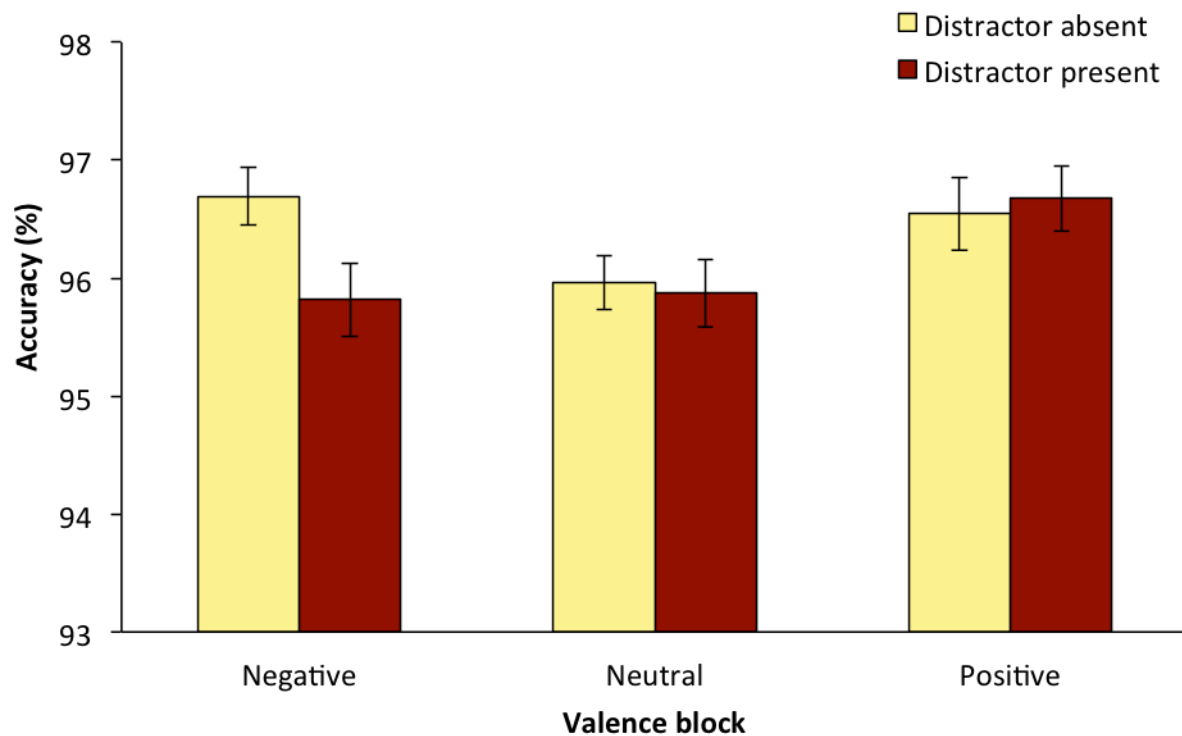


Figure 9. Accuracy rates (%) for distractor-absent and distractor-present trials in the irrelevant distraction paradigm in Study 2. Error bars represent 95% confidence intervals for within-subject comparisons. $*p < .05$.

Stress Induction

Emotional challenge. To test the efficacy of the stress induction at inducing emotional challenge, changes in heart rate and subjective mood were assessed across the study session. Means and standard deviations are presented in Tables 9 and 10.

Table 9

Mean Heart Rate and Mood Ratings (with Standard Deviations) in Study 2

Recording period	Heart rate	Stress	Anger	Worry	Sadness	Happiness
Baseline ^a	73.57 (9.21)	38.81 (24.86)	10.00 (17.86)	31.63 (27.29)	15.70 (21.26)	59.88 (22.51)
Pre-stress ^b	69.23 (9.47)	26.28 (24.35)	15.05 (20.55)	19.49 (20.53)	16.00 (19.21)	50.88 (20.05)
Emotional challenge ^c	80.18 (12.93)	50.72 (25.20)	22.70 (26.04)	45.47 (25.09)	17.30 (18.82)	39.81 (19.96)

Note. Heart rate was measured in beats per minute. Mood was rated on a scale from 0 to 100, with higher ratings indicating greater mood experience.
^aHeart rate was averaged across a rest period, before the irrelevant distractor paradigm; mood ratings were collected prior to this period. ^bHeart rate was averaged across a rest period, before the stress induction (but after the irrelevant distraction task); mood ratings were collected prior to this period. ^cHeart rate was averaged across a silent speech preparation period (following a stressful counting task); mood ratings were collected after this period.

Heart rate. Heart rate was measured during three periods of interest: baseline resting (before the irrelevant distractor paradigm), pre-stress resting (after the irrelevant distractor paradigm and before the stress induction), and emotional challenge (following the counting task, during speech preparation). Across the two rest periods, heart rate showed excellent test-retest reliability, $r(55) = .84, p < .001$. Heart rate significantly differed across recording periods, $F(2, 112) = 48.14, p < .001, \eta_p^2 = 0.46$, decreasing from baseline to pre-stress resting, $t(56) = 6.27, p < .001, d = 0.46$, and elevating during emotional challenge compared to baseline resting, $t(56) = 4.87, p < .001, d = 0.57$, and pre-stress resting, $t(56) = 9.04, p < .001, d = 0.92$ (see Figure 10). This finding suggests that the stress induction successfully induced physiological stress, replicating the findings of Study 1 and consistent with previous stress literature (e.g., Kudielka et al., 2004). The initial decrease in heart rate likely reflects participants becoming more comfortable in the study setting over time, as well as remaining stationary for a long period, and suggests that the increase in heart rate during emotional challenge cannot be accounted for by time passing.

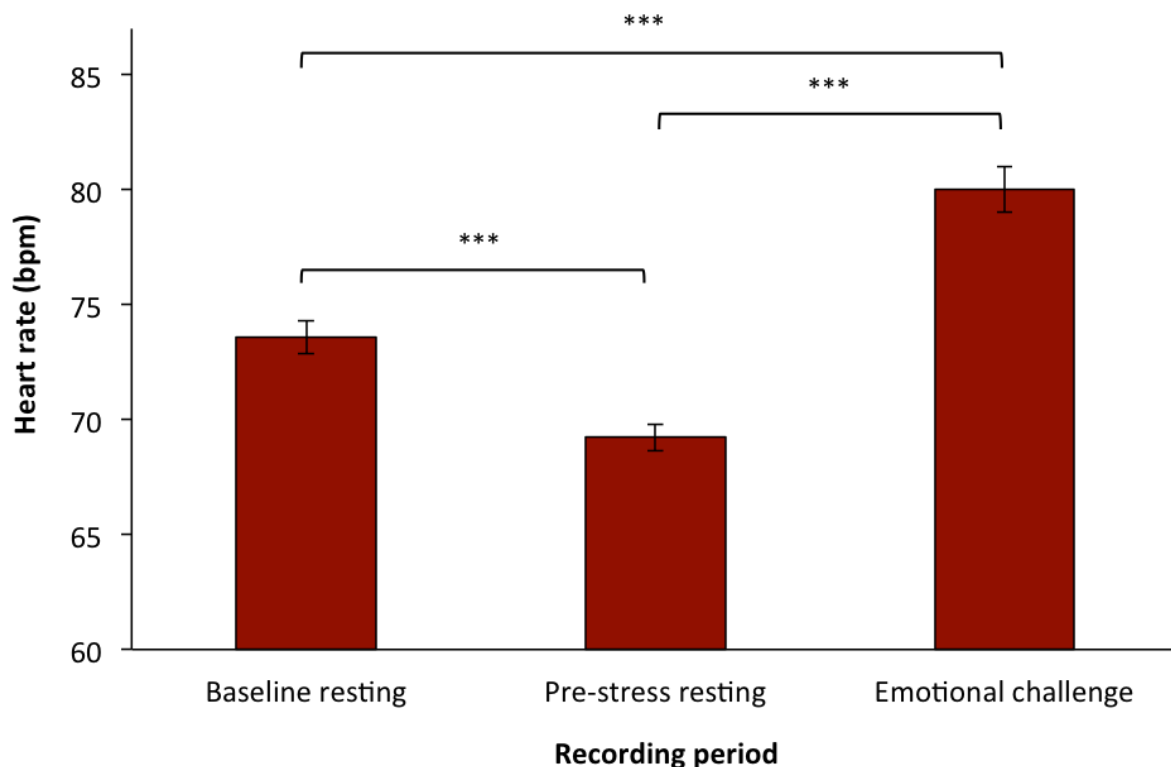


Figure 10. Heart rate (in beats per minute) in Study 2. Emotional challenge heart rate was measured during silent speech preparation, following a stressful serial subtraction task. Error bars represent 95% confidence intervals for within-subject comparisons. *** $p < .001$.

Subjective mood. Mood was rated before the irrelevant distractor paradigm, after the irrelevant distractor paradigm (but before the stress induction), and after the stress induction. Ratings were moderate to low throughout the session (see Figure 11). A repeated-measures MANOVA revealed that mood ratings significantly changed across the session, using Pillai's trace, $V = 0.75$, $F(10, 47) = 13.80$, $p < .001$. Separate univariate tests revealed significant changes for stress, $F(2, 112) = 35.89$, $p < .001$, $\eta_p^2 = 0.39$, anger, $F(1.661, 3.006) = 10.16$, $p < .001$, $\eta_p^2 = 0.15$, worry, $F(1.780, 99.707) = 29.42$, $p < .001$, $\eta_p^2 = 0.34$, and happiness, $F(1.808, 101.259) = 34.48$, $p < .001$, $\eta_p^2 = 0.38$, but not for sadness, $F < 1$.

Prior to the stress induction, there were significant decreases over time in both stress, $t(56) = 4.08$, $p < .001$, $d = 0.51$, and worry, $t(56) = 3.56$, $p = .001$, $d = 0.50$. Post-stress induction, ratings significantly increased again for both stress, $t(56) = 10.05$, $p < .001$, $d = 0.99$, and worry, $t(56) = 9.23$, $p < .001$, $d = 1.12$. Anger was initially stable, $p = .065$, but increased post-stress induction, $t(56) = 3.28$, $p = .002$, $d = 0.31$. Happiness initially decreased, $t(56) = 3.52$, $p = .001$, $d = 0.42$, and decreased further post-stress induction, $t(56) = 5.55$, $p < .001$, $d = 0.55$.

The stress induction was successful at creating subjective emotional challenge, particularly affecting stress and worry and, to a lesser extent, anger and happiness. The decrease in stress and worry ratings pre-stress induction (likely due to participants feeling more comfortable in the study context over time) indicate that the effects of the stress induction on these emotions cannot be accounted for by gradual changes over time. As initial anger ratings were stable over time, post-stress induction increases in anger also cannot be accounted for by time passing. As happiness ratings decreased gradually over the session, the decreased ratings after the stress induction could be due to time passing, rather than the stress induction. However, as Study 1 found significant decreases in happiness after the stress induction task compared to the control task (which were matched for duration), the decrease in happiness in Study 2 is likely at least partially caused by the stress of the counting task. The stress induction did not seem to effect emotional challenge through sadness, as levels remained stable throughout the session. Changes in mood ratings replicate those found in Study 1, with an additional finding of increased worry, potentially due to the addition of the speech preparation task.

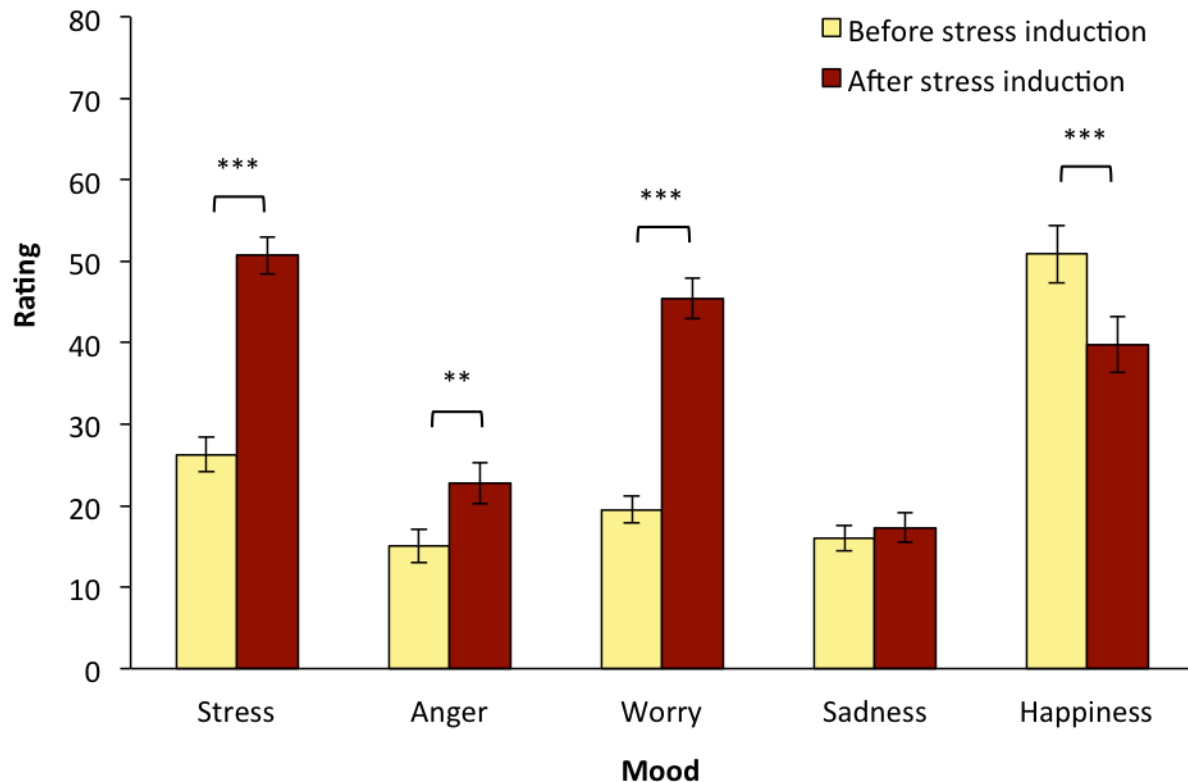


Figure 11. Subjective mood in Study 2, rated before and after stress induction (a stressful serial subtraction task, followed by silent speech preparation). Mood was rated on a scale from 0 to 100, with higher ratings indicating greater mood experience. Ratings made at the beginning of the session are not presented here for ease of presentation. Error bars represent 95% confidence intervals for within-subject comparisons. ** $p < .01$. *** $p < .001$.

Participants reported feeling highly embarrassed and judged during the counting task, and found it to be a highly challenging and stressful task, replicating findings for the experimental group in Study 1. Although the speech preparation task was rated only moderately stressful and challenging, physiological and subjective stress responses were still evident during and after speech preparation, suggesting that this task effectively maintained stress. The difference in ratings between the two tasks (see Table 10) suggests that the current study may have induced greater emotional challenge by including both tasks, than previous capability studies that have used only speech preparation (e.g., Cole et al., 2012; Pérez-Edgar et al., 2013).

Table 10

Self-report Ratings of the Stress Induction in Study 2

Statement of task experience	<i>M (SD)</i>
Counting task	
The counting task was stressful for me	78.72 (16.48)
I found the counting task to be a challenge	84.00 (12.37)
I felt embarrassed during the counting task	75.18 (20.64)
I felt like I was being judged in the counting task	73.12 (21.93)
Speech preparation task	
The speech task was stressful for me	56.63 (26.81)
I found the speech task to be a challenge	55.84 (26.14)

Note. Statements were rated from 0 to 100, with higher ratings indicative of greater affirmation.

Summary. The stress induction effectively induced emotional challenge, with participants showing significant increases in heart rate, stress and anger, and a significant decrease in happiness. Participants reported finding the tasks stressful and challenging, and emotional responses were shown to be over and above the effects of time passing. Taken together, these findings replicate the effects of the stress induction in Study 1 by experimental (but not control) participants. Worry also significantly increased in Study 2, potentially attributable to the speech preparation task. Therefore, frontal EEG activity recorded during this stress induction should provide a valid indicator of prefrontal activity during emotional challenge.

Relative frontal EEG asymmetry. Asymmetry scores were examined across the session to explore any effects of the distraction task and emotional challenge (see Table 11). Relative frontal EEG asymmetry remained stable across the two rest periods, showing good test-retest reliability, $r(55) = .79, p < .001$, consistent with previous research (Mathewson et al., 2015; Salinsky et al., 1991). Emotional challenge frontal asymmetry was weakly correlated with both baseline resting asymmetry, $r(55) = .29, p = .027$, and pre-stress resting asymmetry, $r(55) = .28, p = .038$. A one-way repeated measures ANOVA revealed a marginal difference in asymmetry across the three recording periods, $F(1.245, 69.704) = 2.94, p = .082, \eta_p^2 = 0.05$, likely driven by a leftward shift in asymmetry during emotional challenge (see Figure 12). Notably, individual variability was greater during emotional

challenge than during the rest periods (see Figure 13), indicated by a significant violation of the assumption of sphericity (Mauchly's Test), $\chi^2(2) = .39, p < .001$. This finding is consistent with the idea that emotional challenge asymmetries are more powerful at detecting individual differences than resting asymmetries (Coan et al., 2006; Stewart et al., 2014). Implications of these findings are explored in the general discussion.

Table 11

Frontal EEG Asymmetry Scores in Study 2

Recording period	<i>M (SD)</i>
Baseline resting	-0.012 (0.066)
Pre-stress resting	-0.007 (0.076)
Emotional challenge	0.017 (0.114)

Note. Frontal asymmetry scores were calculated from relative frontal alpha power, using the formula: $\ln(F4) - \ln(F3)$. More positive scores indicate greater leftward frontal asymmetry; more negative scores indicate greater rightward frontal asymmetry. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task.

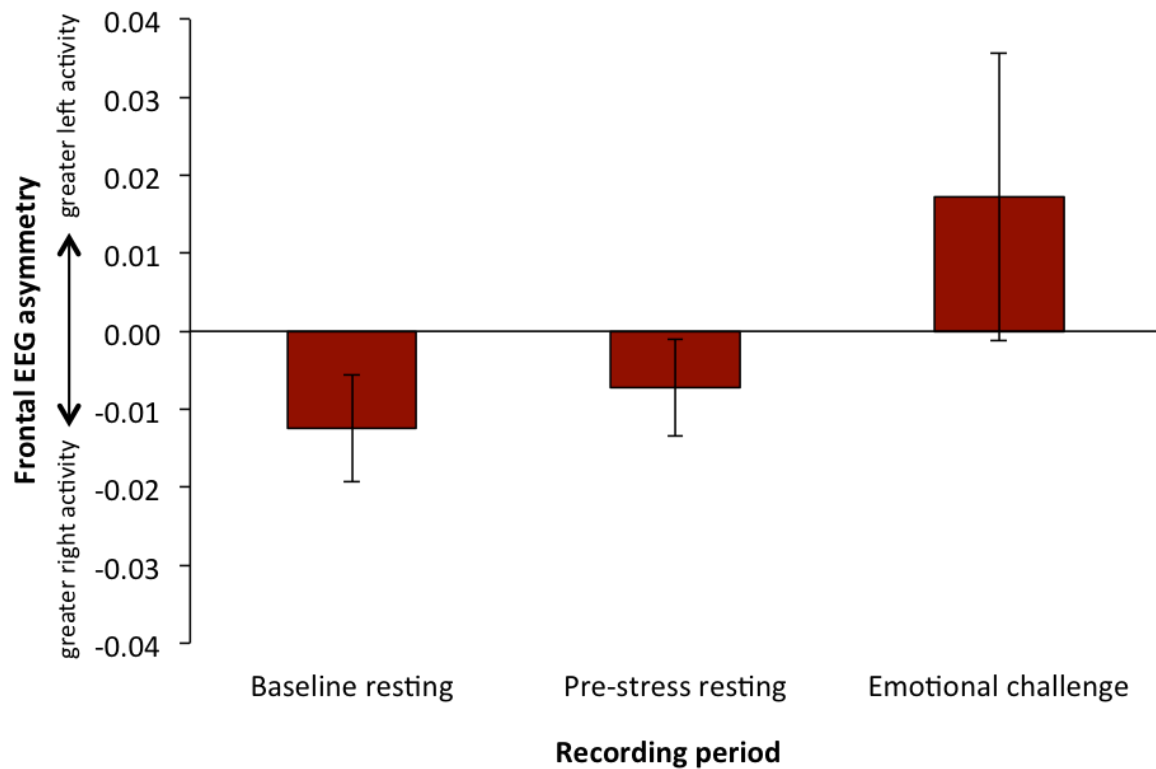


Figure 12. Mean frontal asymmetry scores in Study 2, calculated using the formula: $\ln(F4) - \ln(F3)$. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task. Error bars represent 95% confidence intervals for within-subject comparisons.

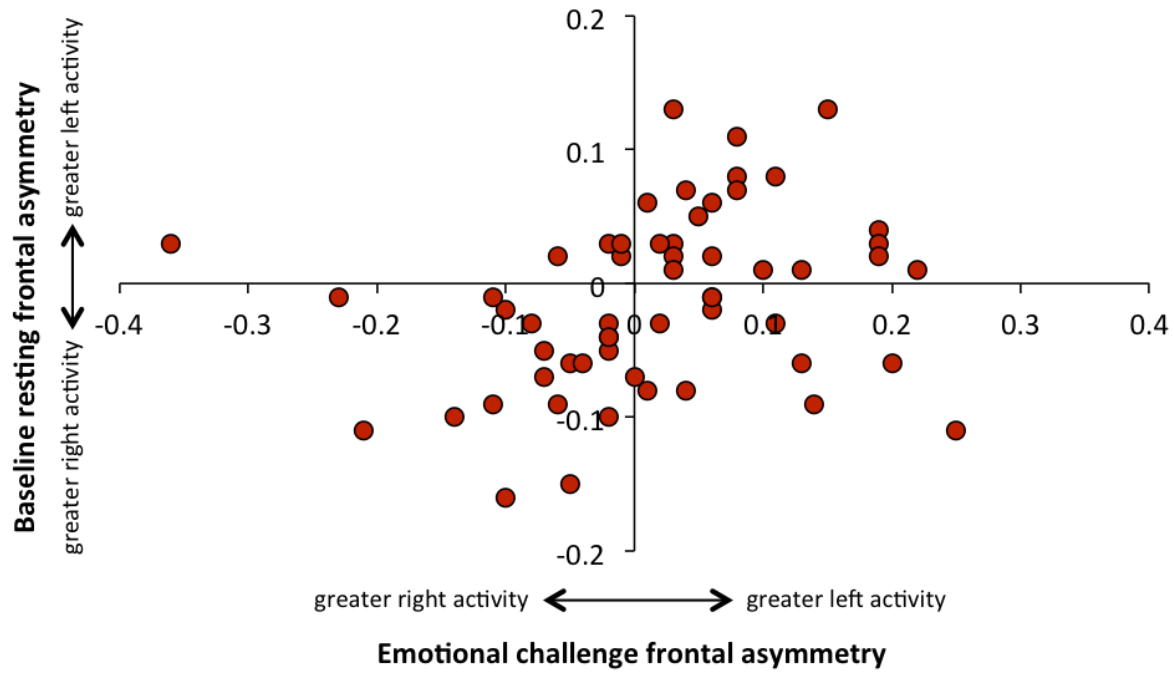


Figure 13. Individual frontal asymmetry data, depicting the relationship between baseline resting and emotional challenge frontal asymmetry, calculated using the formula: $\ln(F4) - \ln(F3)$. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task.

Individual Differences Analyses

Individual differences analyses explored the relationships between frontal asymmetry (baseline resting⁹, emotional challenge), questionnaire scores (depression, anxious arousal, trait anxiety), and distraction (positive, negative, neutral). Because analyses focussed on relationships that have been previously established, corrections for multiple comparisons were not applied.

An individualised measure of distraction. As I employed an individual differences approach to test the asymmetric inhibition model, it was necessary to create a summary variable to capture distraction effects for each participant. Simple difference scores (distractor-present RT – distractor-absent RT) could be calculated to index positive, negative and neutral distraction, but these scores reflect individual differences in distractor-absent trials as well as distractor-present trials. An alternative, more sensitive method is to calculate residual distraction indices (RDIs) by regressing distractor-present RTs on distractor-absent

⁹ The baseline resting measure of asymmetry was used for the comparison resting measure because resting asymmetry is typically measured at the beginning of the study session.

RTs, calculated separately for each valence. Residuals are argued to provide a more reliable and more accurate measurement of an effect than a difference score by statistically removing the variance from the control variable (i.e., distractor-absent trials; see Degutis, Wilmer, Mercado, & Cohan, 2013), which is particularly important in research on individual differences¹⁰. Indeed, distractor-absent and distractor-present RTs were highly correlated in the present study, $r(55) = .99, p < .001$.

This method creates a distribution of distraction (from the sample's distractor-absent and distractor-present RTs) with a mean of zero. Residual scores indicate the extent to which an individual's level of distraction differs from the sample mean; that is, the extent to which their distractor-present RT is greater or lower than would be expected, based on their distractor-absent RT and the average amount of distraction shown by the sample. Positive residual scores indicate greater distraction than the sample average, and negative residual scores indicate lower distraction than the sample average. In this way, RDIs indicate an individual's level of distraction, relative to their own performance on distractor-absent trials, while accounting for individual variance in RT within the sample. Using linear regression, positive ($SD = 23.76$), negative ($SD = 23.00$) and neutral ($SD = 22.93$) RDIs were calculated for each participant for use in further analyses.

Relationships with depression and anxiety symptoms. Relationships between frontal asymmetry, symptoms of mood disorder and inhibitory control have been widely reported in previous literature. To add to this body of research, relationships between these variables were explored before testing the main hypotheses. Zero-order correlations were conducted between frontal asymmetry, questionnaire scores and distraction (see Table 12).

¹⁰ Use of difference scores did not change analysis outcomes.

Table 12

Zero-order Correlations Between Frontal Asymmetry (FA), Questionnaire Scores, and Residual Distraction Indices (RDIs) in Study 2

Measure	1	2	3	4	5	6	7
1. Baseline resting FA							
2. Emotional challenge FA	0.29*						
3. Depression	0.17	0.09					
4. Anxious arousal	0.28*	-0.08	0.34**				
5. Trait anxiety	0.27*	-0.12	0.79***	0.44**			
6. Negative distraction	-0.18	0.18	0.01	0.07	-0.06		
7. Neutral distraction	0.14	-0.19	0.06	0.03	-0.03	-0.25†	
8. Positive distraction	0.16	-0.14	0.01	-0.09	-0.01	-0.04	0.01

Note. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task. Depression was measured using the Depression, Anxiety & Stress Symptom questionnaire. Anxious arousal was measured using the Mini Mood & Anxiety Symptom Questionnaire. Trait anxiety was measured using the trait form of the State-Trait Anxiety Inventory. Distraction was measured in residuals, calculated for each valence by regressing distractor-present RTs on distractor-absent RTs, and indicate distraction, relative to the sample average. Residual distributions have a mean of zero, so more positive values indicate greater distraction than the sample average and more negative values indicate less distraction than the sample average.

† $p < .1$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Baseline resting frontal asymmetry was positively correlated with trait anxiety, $r(55) = .27$, $p = .041$, and anxious arousal, $r(55) = .28$, $p = .034$, but not depression, $p = .208$. These findings indicate that greater resting LFA is associated with greater trait anxiety supporting previous research (e.g., Heller et al., 1997; Mathersul et al., 2008; Nitschke et al., 1999), but also greater somatic anxiety, inconsistent with previous findings that show the opposite pattern (e.g., Nitschke et al., 1999; Wiedemann et al., 1999). Resting frontal asymmetry was not related to depressive symptoms, inconsistent with some research (Pössel, Lo, Fritz, & Seemann, 2008; Wiedemann et al., 1999; see also Nusslock et al., 2015), but consistent with others (Blackhart et al., 2006; Metzger et al., 2004; Papousek et al., 2014; Tooley, 2015). Indeed, the relationship between resting frontal asymmetry and sub-clinical levels of depression and anxiety is generally inconsistent, potentially due to methodological differences (see Thibodeau et al., 2006).

Emotional challenge frontal asymmetry did not significantly correlate with depression or anxiety symptoms, p 's > .3. These findings are inconsistent with research showing greater RFA during emotional challenge to predict greater symptoms of mood disorder (e.g., Harmon-Jones et al., 2002), as well as research showing greater RFA during challenge for clinically depressed and anxious individuals (Davidson, Marshall, Tomarken, & Henriques, 2000; Stewart et al., 2011, 2014; Wiedemann et al., 1999) and depression-vulnerable individuals (Jones, Field, Fox, Davalos, & Gomez, 2001), compared to controls. However, relationships between challenge frontal asymmetry and symptoms of mood disorder are not widely reported.

Depression and anxiety scores did not correlate with distraction, p 's > .5. While many studies report a link between inhibitory control of emotion and depression and anxiety symptoms in non-clinical samples (e.g., Bar-Haim et al., 2007; Goeleven, De Raedt, Baert, & Koster, 2006; Joormann, 2004), others have found no relationship (e.g., Engels et al., 2007; Everaert et al., 2016). Further implications of these findings are covered in the general discussion.

Testing the Asymmetric Inhibition Model. To test the primary research question that frontal asymmetry reflects biases in a specialised inhibitory control system, frontal asymmetry (at rest and during emotional challenge) was assessed as a predictor of emotional distraction. Analyses were performed twice: once using baseline resting frontal asymmetry to test hypotheses within a traditional dispositional framework (Davidson, 1998), and once using emotional challenge frontal asymmetry to test hypotheses within a capability framework (Coan et al., 2006). First, correlations between RDIs and frontal asymmetry were examined for hypothesised relationships between distraction and relative frontal brain activity. Second, an analysis of covariance (ANCOVA) was performed to test whether individual differences in frontal asymmetry predicted emotional distraction. This method tests whether a covariate (frontal asymmetry) moderates the relationship between the independent variable (distractor valence) and dependent variable (distraction). A significant covariate in this case would indicate that frontal asymmetry moderates distraction regardless of valence, while a significant interaction between the independent variable and the covariate would indicate that asymmetry moderates distraction according to valence (the predicted effect). This method enables testing of the relationship between frontal asymmetry and distraction for all valences within one analysis and so is preferable to running separate regression analyses for each valence because it minimises number of comparisons and tests for valence-specific relationships between frontal asymmetry and distraction. Therefore,

ANCOVA provides a sensitive test of whether frontal asymmetry is related to positive and negative distraction, relative to neutral *and* relative to each other. Because depression and anxiety symptoms did not correlate with distraction levels, questionnaire scores were not included as covariates in analyses.

The relationships between baseline resting frontal asymmetry and distraction showed the predicted patterns, with a negative relationship between negative RDIs and asymmetry and a positive relationship between positive RDIs and asymmetry. However, correlational analyses revealed that these relationships were not significant, p 's > .17 (see Table 12). The valence x asymmetry ANCOVA revealed no main effect of frontal asymmetry, $F(1, 55) = 0.31$, $p = .581$, and no asymmetry x valence interaction, $F(2, 110) = 1.97$, $p = .144$, or quadratic effect, $F(1, 55) = 0.76$, $p = .388$ (see Figure 14).

Relationships between emotional challenge frontal asymmetry and distraction were in the opposite pattern to that predicted (and to resting asymmetry), with a positive relationship between negative RDIs and asymmetry and a negative relationship between positive RDIs and asymmetry. Again, correlational analyses revealed that these relationships were not significant, p 's > .16 (see Table 12). The valence x asymmetry ANCOVA showed no main effect of frontal asymmetry, $F(1, 55) = 0.46$, $p = .503$, and no asymmetry x valence interaction, $F(2, 110) = 2.03$, $p = .136$, or quadratic effect, $F(1, 55) = 1.39$, $p = .244$ (see Figure 14).

These findings indicate that neither resting nor emotional challenge frontal asymmetries modulated overall distraction or valence-specific distraction, suggesting frontal asymmetry was not related to ability to inhibit emotional distractors. Though the pattern was in the predicted direction for resting frontal asymmetry, correlations with positive and negative distraction were not significant and the ANCOVA revealed that they did not significantly differ from one another. Therefore, if this effect exists, it is not evident in the present study. Further, the relationship between challenge asymmetry and distraction was in the opposite direction to that of the resting asymmetry, and to what would be predicted by the asymmetric inhibition model. In sum, findings did not support hypotheses and did not support the asymmetric inhibition model (Grimshaw & Carmel, 2014). The hypothesis that emotional challenge frontal asymmetry would be more strongly related to emotional distraction than resting frontal asymmetry was rendered irrelevant because neither measure was related to frontal asymmetry.

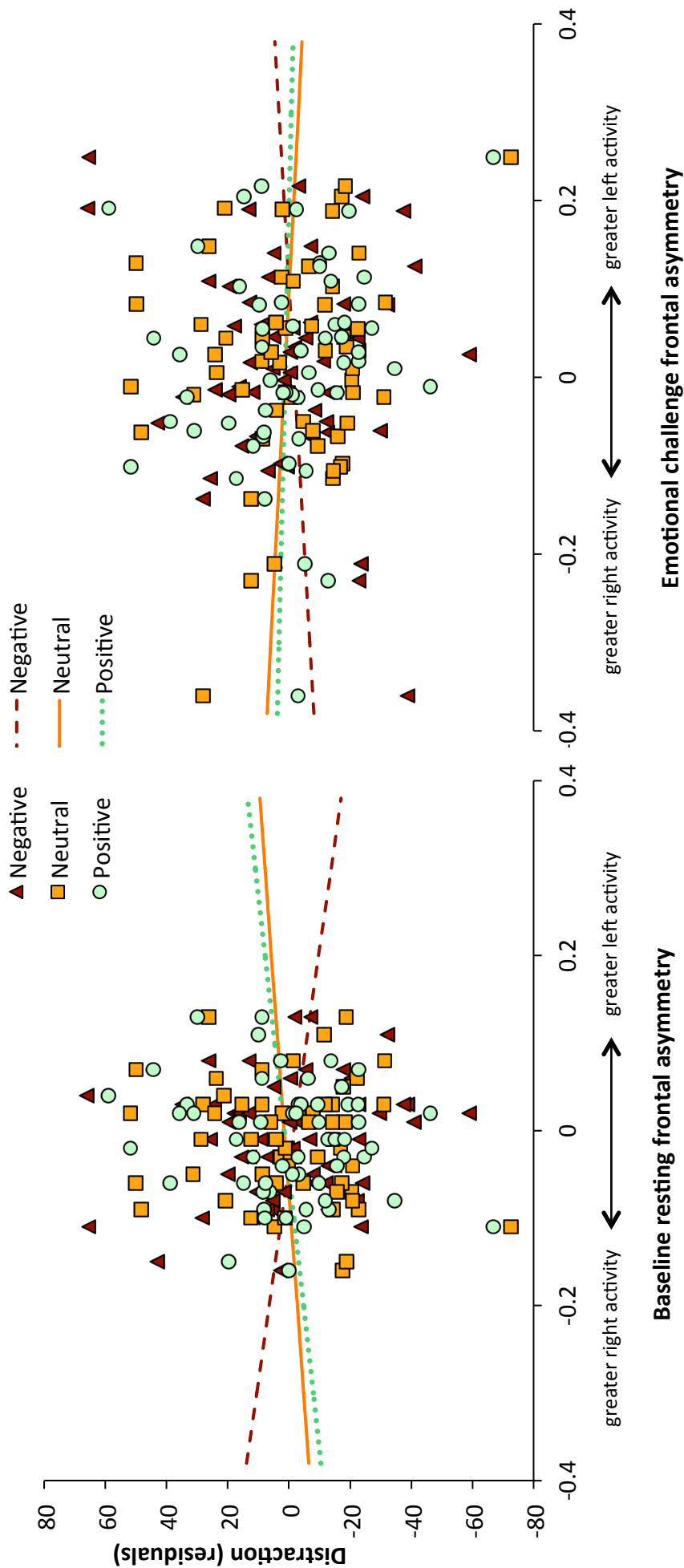


Figure 14. The relationship between distraction (negative, neutral, positive) and frontal asymmetry in Study 2, calculated using the formula: $\ln(F4) - \ln(F3)$. Emotional challenge frontal asymmetry was measured during speech preparation, following a stressful serial subtraction task. Distraction is measured in residuals (calculated by regressing distractor-present RTs on distractor-absent RTs for each valence), which indicate distraction relative to the sample average. Residual distributions have a mean of zero, so more positive values indicate greater distraction than the sample average and more negative values indicate less distraction than the sample average.

General Discussion

Frontal EEG asymmetry is a reliable marker of psychopathology vulnerability (Coan & Allen, 2004; Thibodeau et al., 2006), yet the mechanisms underlying this relationship remain unclear. The asymmetric inhibition model (Grimshaw & Carmel, 2014) holds that frontal asymmetry reflects valence-specialised inhibitory control mechanisms based in dlPFC; left dlPFC is posited to inhibit negative information and right dlPFC is posited to inhibit positive information. Frontal asymmetry therefore represents individual differences in ability to inhibit positive and negative information. The present thesis tested this model, investigating whether ability to inhibit emotional distractors is predicted by frontal asymmetry. Participants performed a simple visual search task at fixation, while attempting to inhibit peripheral distracting images (positive, negative, or neutral). Frontal asymmetry was measured at rest (the traditional approach) and during emotional challenge, purported to produce a stronger and more reliable measure of individual differences due to capturing emotion regulation strategies “in action” (capability model; Coan et al., 2006; see also Stewart et al., 2014). Emotional challenge was induced using a stressful serial subtraction task, verified to be effective in Study 1, and maintained using a speech preparation task while EEG was recorded. If the asymmetric inhibition model is accurate, then frontal asymmetry should predict individual differences in distraction. Frontal asymmetry was expected to negatively correlate with negative distraction (i.e., lower LFA/greater RFA predicts more negative distraction) and positively correlate with positive distraction (i.e., greater LFA/lower RFA predicts more positive distraction).

Results showed the predicted emotional distraction effect; irrelevant positive and negative images captured attention to a greater extent (i.e., were less effectively inhibited) than neutral images, supporting previous research (Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016). However, frontal asymmetry was not related to emotional (or neutral) distraction, when measured at rest or during emotional challenge. Frontal asymmetry therefore did not reflect individual differences in the ability to engage control mechanisms responsible for inhibiting irrelevant emotional (or even neutral) distractors. That is, individuals with different patterns of frontal asymmetry (i.e., RFA vs LFA) were similarly able to inhibit different types of emotional information. These findings do not support the asymmetric inhibition model (Grimshaw & Carmel, 2014). They suggest that inhibitory control of emotional distractors is not the mechanism linking frontal asymmetry to psychopathology vulnerability, adding to a growing literature attempting to

pinpoint the cognitive mechanisms underlying frontal asymmetry in order to better understand the etiology of psychopathology.

There are a few possible explanations for why the present findings did not support the asymmetric inhibition model: (1) frontal asymmetry reflects a cognitive control mechanism other than inhibition (i.e., the model is wrong); (2) frontal asymmetry reflects an aspect of inhibitory control other than that tested in the current study (i.e., the model is right, but the irrelevant distractor paradigm did not tap the right aspect of inhibitory control); or (3) due to methodological factors, the present study lacked statistical power to adequately test the relationship between frontal asymmetry and inhibitory control. These explanations are addressed in turn below.

A Different Cognitive Control Mechanism

One explanation for why the present study did not find a relationship between frontal asymmetry and inhibitory control is that the asymmetric inhibition model is wrong, and frontal asymmetry reflects a different cognitive control mechanism to inhibitory control. Though it is possible that frontal asymmetry reflects emotional (rather than cognitive) processing, as suggested by the valence hypothesis (Berntson et al., 2011; Heller, 1993; Heller et al., 1998; Tomarken et al., 1992) and the motivational direction hypothesis (Harmon-Jones, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), decades of research linking frontal asymmetry with emotional biases have failed to identify an underlying mechanism. Conversely, there is compelling evidence that frontal asymmetry reflects a cognitive mechanism, specialised in emotional control, from clinical (e.g., De Raedt & Koster, 2010; Field & Cox, 2008; Garavan & Hester, 2007; Joormann et al., 2007), neurological (e.g., Beauregard et al., 2001; Bishop et al., 2004; Engels et al., 2007; Herrington et al., 2010), and electrophysiological (e.g., Ambrosini & Vallesi, 2016; Grimshaw et al., 2014; Pérez-Edgar et al., 2013) research.

Frontal asymmetry likely reflects a cognitive mechanism supported by dlPFC (e.g., Grimshaw & Carmel, 2014; Koslov et al., 2011; Pizzagalli et al., 2005; Shackman et al., 2009; but see Gable et al., 2015; Gorka, Phan, & Shankman, 2015). dlPFC has been linked to a number of cognitive processes requiring cognitive control, including task switching (Ambrosini & Vallesi, 2016), working memory (D'Esposito, Postle, & Rypma, 2000; Petrides, 2000), emotion regulation (Goldin, McRae, Ramel, & Gross, 2008; Lévesque et al., 2003; for a review, see Ochsner et al., 2012), and attentional disengagement (Vanderhasselt et al., 2011), all of which are implicated in vulnerability to psychopathologies associated with frontal asymmetry (e.g., Boonstra et al., 2005; Goldstein et al., 2004; Kübler et al., 2005;

Snyder, 2013). These processes require not only inhibitory control, but also other control mechanisms, such as updating and shifting (see Joormann & Tanovic, 2015; Miyake et al., 2000; Schmeichel & Tang, 2015). Thus, it is possible that a control mechanism other than inhibitory control underlies the relationship between frontal asymmetry and psychopathology vulnerability.

A Different Aspect of Inhibitory Control

Alternatively, the asymmetric inhibition model could be accurate, but frontal asymmetry reflects a different aspect of inhibitory control to that measured by the irrelevant distractor paradigm in the present study. There seem to be multiple aspects of inhibitory control, which have been associated with distinct areas of dlPFC (Warren et al., 2013). The irrelevant distractor paradigm requires inhibition of *attentional capture* by distracting stimuli (Forster & Lavie, 2008a, 2008b; Grimshaw et al., 2016). However, psychological disorders associated with frontal asymmetry also show deficits in response inhibition (e.g., Epstein, Johnson, Varia, & Conners, 2001; Joormann & Tanovic, 2015), behavioural inhibition (e.g., Barkley, 1997; de Wit, 2009), and other cognitive control processes requiring inhibition, such as attentional disengagement (e.g., Koster et al., 2011; Proudfit, Bress, Foti, Kujawa, & Klein, 2015; Rudaizky et al., 2014).

In fact, frontal asymmetry has already been linked with attentional disengagement from emotional stimuli (i.e., withdrawing attentional resources from an emotional stimulus and shifting them to the target). Pérez-Edgar et al. (2013) and Grimshaw et al. (2014) both showed frontal EEG asymmetry to predict attentional biases to negative emotion when image cues were presented for a long duration (500 or 1000 ms), but not when presented for a short duration (17 or 250 ms), suggesting that frontal asymmetry reflects late, effortful control processes that facilitate attentional disengagement from emotion. Further, greater activity in right dlPFC when attempting to disengage from negative stimuli has been associated with vulnerability to depression (Vanderhasselt et al., 2011).

It is possible that inhibitory control underlies the relationship between frontal asymmetry and attentional disengagement from emotion. Of course, disengagement also requires control mechanisms other than inhibition (e.g., updating, shifting; see Miyake et al., 2000) and may not always require inhibition (e.g., attention may be captured by another stimulus, drawing attention away without need for inhibition); however, in some circumstances, inhibitory control may be vital for successful disengagement, such as when the distractor is highly potent (e.g., an emotional image). Thus, frontal asymmetry may reflect inhibitory control processes that facilitate attentional disengagement from emotion, rather

than prevent attentional capture, meaning that the asymmetric inhibition model could be right, but was incorrectly operationalised in the present study. Previous tests of the relationship between frontal asymmetry and attentional disengagement from emotion (Grimshaw et al., 2014; Pérez-Edgar et al., 2013) do not provide an adequate test of the asymmetric inhibition model because they do not use positive stimuli of equivalent arousal and, critically, do not explicitly test inhibitory control; that is, it is not discernably necessary to inhibit the emotional distractors.

Future research should test whether frontal asymmetry reflects valence-specific ability to use inhibitory control mechanisms to disengage from emotional stimuli, as an alternative operationalisation of the asymmetric inhibition model. A central distractor paradigm (see Gupta et al., 2015) could be used to directly test inhibitory control aspects of disengagement. In this paradigm, distractor images are presented at fixation, bordered by a letter display (see Figure 15). The distractor automatically captures attention, producing large emotional distraction effects (Gupta, Hur, & Lavie, 2015; Maddock et al., 2016), and inhibitory control is needed to shift attention away from the potent image to the letter display (see Maddock et al., 2016). If this alternative operationalisation of the asymmetric inhibition model is accurate, then frontal asymmetry should predict ability to disengage from positive and negative distractors, in the same directions as predicted in the current thesis. Because disengagement likely requires several control mechanisms (e.g., inhibiting, updating, shifting), further research would then be needed to corroborate the idea that frontal asymmetry specifically reflects the inhibitory component of disengagement. As it is difficult to disentangle control mechanisms from one another, multiple studies using multiple tasks would be vital (e.g., cueing tasks, the AX-CPT, the attention network task; see Miyake et al., 2000, and Miyake & Friedman, 2012, for discussions on disentangling executive functions).



Figure 15. Example of the display screen for the central distractor paradigm. The image depicted is for display purposes only; positive, negative and neutral image stimuli would be selected from the International Affective Picture System (IAPS; Lang et al., 2008). Display is not to scale.

Statistical Power

A final explanation for the findings of the current thesis is that the asymmetric inhibition model is correct (and the irrelevant distractor paradigm targeted the appropriate inhibitory mechanism), but the present thesis did not find evidence to support it due to methodological factors. That is, there may not have been enough statistical power to test the relationship between frontal asymmetry and inhibitory control.

It is possible that the present study failed to capture control biases indicative of psychopathology vulnerability because inhibitory control was not tested during emotional challenge. Emotional challenge was induced after the irrelevant distractor paradigm in the present study to prevent emotional response to the stress induction from influencing task performance. However, task performance may better reflect cognitive vulnerability when measured during emotional challenge. The capability model (Coan et al., 2006) holds that frontal asymmetry is a more powerful measure of cognitive vulnerability during emotional challenge because ability to engage cognitive regulation strategies is measured “online”. If we accept this model as true, and there is compelling evidence to support it (see Dennis & Solomon, 2010; Goodman et al., 2013; Lopez-Duran, Nusslock, George, & Kovacs, 2012; Meyer et al., 2015; Papousek, Freudenthaler, et al., 2011; Papousek, Reiser, et al., 2013; Papousek, Weiss, Perchtold, et al., 2016; Papousek, Weiss, Schulte, et al., 2014; Pérez-Edgar et al., 2013; Stewart et al., 2011, 2014; Verona, Sadeh, & Curtin, 2009; Wacker, Mueller, Pizzagalli, Hennig, & Stemmler, 2013; for a review see Allen & Reznik, 2015), then it logically follows that individual differences in engaging the mechanisms underlying frontal asymmetry might also be better captured during emotional challenge.

In line with this idea, cognitive theories of vulnerability to mood disorder hold that stress activates cognitive biases (Gotlib & Joormann, 2010; see Grimshaw et al., 2014; Pérez-Edgar et al., 2013). Stress is thought to play a critical role in the development of emotional disorders (Monroe & Simons, 1991; Willner, Scheel-Krüger, & Belzung, 2013) through its detrimental effects on cognitive functioning combined with an individual’s impaired ability (cognitive vulnerability) to manage it (Curci et al., 2013; Sinha, 2008; Tooley, 2015). Thus, cognitive vulnerability may be better evident when measured during emotional challenge. Indeed, Grimshaw et al. (2014) administered a negative mood induction (passive viewing of gory images) prior to testing attentional biases to emotion; they may have found a relationship between frontal asymmetry and control biases because the mood induction enabled a more sensitive test of cognitive vulnerability. Failure to activate cognitive vulnerability may account for difficulties detecting cognitive biases in non-clinical populations.

A simple way to re-test the relationship between frontal asymmetry and inhibitory control of emotional distractors would be to administer the stress induction prior to the irrelevant distractor paradigm. The stress induction would need to be altered slightly to enable stress maintenance throughout the task. For example, after completing the stressful counting task, participants would be told that they will next complete a cognitive task (e.g., the irrelevant distractor paradigm), and then give a speech to other (confederate) participants, who will rate their performance. In this way, participants would complete the irrelevant distractor paradigm while anticipating an impending social evaluative test, that is, while under emotional challenge.

Another possible cause of low statistical power in the present study is low individual variability in frontal asymmetry. However, frontal asymmetry was recorded during emotional challenge specifically to provide a more sensitive measure of individual differences than the traditional resting measure. Study 1 and Study 2 showed the counting task to effectively induce emotional challenge, and the speech preparation task to successfully maintain it. Further, frontal asymmetry showed the predicted increase in individual variability when measured during emotional challenge, in line with the capability model (Coan et al., 2006; see also Stewart et al., 2014). Therefore, it is unlikely that low variability in frontal asymmetry reduced statistical power.

On the other hand, there may have been enough variability in frontal asymmetry, but the irrelevant distractor paradigm may have insufficiently captured individual differences in ability to inhibit emotional distractors. Distraction levels and variability were low when compared to our previous studies (Grimshaw et al., 2016; Kranz, 2015; Murphy, 2016). However, Forster and Lavie (2016) showed that a neutral version of the irrelevant distractor paradigm is sensitive to individual differences in distractibility, and to a greater extent than other tasks assessing inhibitory control. Further, the present study found significant distraction effects, consistent with research showing that the irrelevant distractor paradigm is a sensitive test of distractibility (Forster & Lavie, 2008a, 2008b, 2014, 2016), as well as significant emotional distraction, in line with research showing that this paradigm is sensitive to emotional biases (Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016). Thus, the irrelevant distractor paradigm showed the effects of interest, suggesting that there was sufficient power.

Taken together, it seems unlikely that the present thesis did not support the asymmetric inhibition model due to low variability in frontal asymmetry or distraction. Indeed, in these respects, the present study likely had greater statistical power than studies that have

previously found a relationship between frontal asymmetry and attentional control over emotion (Grimshaw et al., 2014; Miskovic & Schmidt, 2010; Pérez-Edgar et al., 2013), as it used a more sensitive measure of frontal asymmetry, found larger behavioural effects, and had a bigger sample size ($N = 57$, compared to 30-45).

It is possible that the irrelevant distractor paradigm is not a sensitive measure of inhibitory control. Participants may be simply directing attentional resources to the target display without engaging inhibitory processes; distraction then occurs when some resources are left unallocated, resulting in attentional capture. However, there is compelling evidence to suggest that inhibitory control is engaged in this task. The irrelevant distractor paradigm was specifically designed to provide a sensitive measure of ability to ignore distractors (Forster & Lavie, 2008a); it is clearly beneficial to task performance to inhibit entirely irrelevant images and participants are encouraged to do so. Distractors clearly capture attention, as evidenced by robust RT interference effects, but accuracy remains high (90-99%; (Forster & Lavie, 2008a, 2008b; Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016)), suggesting that participants are inhibiting the images to enable accurate responding to the target. Further, performance on the task is sensitive to factors known to influence cognitive control, including distractor expectancy (Grimshaw et al., 2016; Kranz, 2015; Murphy, 2016) and motivation to perform well (Maddock et al., 2016), suggesting that the task elicits use of control to prevent and resolve interference effects (see Braver, 2012). Task performance has also been linked to patterns of electrophysiological activity associated with inhibitory control (Kranz, 2015). Thus, while there is no evidence to definitively confirm that the irrelevant distractor paradigm taps inhibitory control, there is good evidence that it does.

Broad Contributions to the Literature

The modified counting task: A validated emotional challenge induction. The efficacy of the counting task at inducing emotional challenge was verified by significant physiological and subjective emotional responses to the task in both Study 1 and 2. These findings support previous research showing the efficacy of serial subtraction tasks in inducing stress (Kudielka et al., 2007; Lin et al., 2011), and validate this particular counting paradigm, modified from the classic TSST paradigm (Kirschbaum et al., 1993), for future use in capability and stress research. Indeed, the counting task was reported to be substantially more stressful and challenging than the speech preparation task in Study 2, suggesting that the counting task may be more effective at eliciting emotional challenge than speech preparation.

Depression and anxiety symptoms. Although this thesis was not designed to test cognitive vulnerability associated with psychopathology, analyses revealed that ability to

control distraction was unrelated to depression and anxiety symptoms. These findings are inconsistent with research associating cognitive control deficits with vulnerability to depression (Goeleven et al., 2006; Joormann, 2004) and anxiety (Bar-Haim et al., 2007), though some studies also report finding no relationship between inhibitory control and symptoms of mood disorder in non-clinical samples (e.g., Engels et al., 2007; Everaert et al., 2016). Indeed, biases in control can be subtle in non-clinical populations, leading to inconsistent findings (see Booth & Peker, 2016). Only one study has tested this relationship using an irrelevant distractor paradigm; Fox et al. (2012) showed that emotional distraction effects differ according to levels of trait anxiety, but only when negative distractors were fear-conditioned. Thus, the distractors in the present study may not be potent enough to elicit biases in a non-clinical population or, as aforementioned, emotional challenge (i.e., threat of shock) may be needed to elicit cognitive biases indicative of vulnerability.

Frontal asymmetry and emotional challenge. Frontal asymmetry showed greater variability during emotional challenge than rest, in line with capability research (Coan et al., 2006; Stewart et al., 2014). Interestingly, frontal asymmetry shifted marginally leftward during emotional challenge. The capability literature shows inconsistencies in the direction of challenge-induced asymmetry shift, with some studies showing an overall rightward shift (Papousek, Reiser, et al., 2013; Papousek, Weiss, et al., 2014), others showing a leftward shift (Hofmann et al., 2005), and others reporting no overall shift (Meyer et al., 2014; Papousek et al., 2011). However, few studies directly test average changes in asymmetry from rest to challenge. The capability model proposes that emotional challenge elicits a more powerful measure of individual differences because it explicitly requires the individual to demonstrate their ability to manage emotion. Leftward shifts in asymmetry are proposed to indicate adaptive emotion regulation processes in the face of stressful situations (Coan et al., 2006; Goodman et al., 2013; Papousek et al., 2014; Pérez-Edgar et al., 2013), perhaps through engagement of control strategies specialised to regulate negative emotion (asymmetric inhibition model; Grimshaw & Carmel, 2014). The shift in the current study did not reach statistical significance and therefore cannot be confidently interpreted. However, the large variability in emotional challenge asymmetry indicates substantial individual variation in mental processing during challenge. To speculate, this variability could reflect differential success at engaging left-lateralised control mechanisms needed for regulation of negative emotion. Future research is needed to test this idea.

Limitations

One limitation of the present study was that individual variability in emotional challenge frontal asymmetry was confounded by mental rehearsal during the speech preparation task. That is, the increased variability in frontal asymmetry scores from rest to emotional challenge may reflect mental rehearsal for the upcoming speech, as well as response to emotional challenge. Activity in left dlPFC is associated with verbal working memory and verbal fluency (e.g., Friston, Frith, Liddle, & Frackowiak, 1993; Gray et al., 2002; Provost, Petrides, Monchi, 2010), both relevant processes for speech preparation; further, greater LFA predicts better performance on tasks assessing these processes (Papousek, Murhammer, & Schulter, 2011; Papousek & Schulter, 2004). However, mental verbalisation is also thought to be central to worry and ruminative thinking (Barlow, 1991; Heller, 1993; see Smith et al., 2016), both of which are associated with greater LFA (Engels et al., 2007; Heller et al., 1997; Hofmann et al., 2005); indeed, Study 2 showed significant increases in worry during the stress induction. Speech preparation may therefore lead individuals with good stress regulation to prepare for the speech and individuals with poor stress regulation to worry about the speech, both of which would be associated with leftward shifts in asymmetry.

Use of the speech preparation task does not invalidate the emotional challenge asymmetry measure; participants still showed physiological and subjective emotional responses. However, challenge asymmetries would provide a stronger measure of individual variability in emotion regulation when measured during a task that does not explicitly require verbal rehearsal. Verbal rehearsal can be removed from the speech preparation task by simply refraining from telling participants the topic of the speech (see Hofmann et al., 2005).

A second limitation of the present study is that sample size restricted analyses to a limited number of variables. Many studies have highlighted the importance of studying interactions between frontal and parietal activity when studying cognitive control of emotion. Frontal and parietal cortices are both considered vital nodes in a distributed network responsible for cognitive control of emotion (Banich et al., 2009; Pessoa, 2008); together, they modulate emotional processing through top-down attentional control (Corbetta & Shulman, 2002; Pessoa, Kastner, & Ungerleider, 2002). Interactions between frontal and parietal EEG asymmetries have been used to predict vulnerability to different emotional disorders (e.g., Heller et al., 1997; Tooley, 2015) and, critically, to predict the nature of attentional biases to emotion (Grimshaw et al., 2014). Thus, it is likely important to consider both frontal and parietal involvement when studying cognitive control of emotion.

Additionally, in line with idea that frontal asymmetry reflects capability to engage emotion regulation mechanisms (capability model; Coan et al., 2006), many researchers are now examining the *shift* in frontal asymmetry from rest to challenge in relation to cognitive vulnerability to psychopathology (see Cole et al., 2012; Meyer et al., 2014; Papousek, Freudenthaler, et al., 2011; Papousek, Reiser, Schulter, et al., 2013; Papousek, Reiser, Weber, et al., 2012; Papousek, Weiss, Perchtol, et al., 2016; Papousek, Weiss, Schulter, et al., 2014). Indeed, Pérez-Edgar et al. (2013) showed that attentional control biases were predicted by frontal asymmetry shift (from rest to challenge), but not by either asymmetry measure alone. Because sample size limited number of comparisons in the present study, I focussed on the comparisons most clearly predicted by the model. Larger scale studies exploring the influence of multiple factors on the relationship between frontal asymmetry and cognitive processing would be valuable to this area of research.

Conclusions

This thesis tested whether frontal EEG asymmetry reflects ability to engage valence-specific inhibitory control mechanisms, as predicted by the asymmetric inhibition model (Grimshaw & Carmel, 2014). In an individual differences study, frontal asymmetry, measured at rest and during emotional challenge, was assessed as a predictor of ability to inhibit irrelevant emotional distractors. Overall, irrelevant emotional images were more distracting than neutral images, supporting previous research (Grimshaw et al., 2016; Kranz, 2015; Maddock et al., 2016; Murphy, 2016), and emotional challenge produced greater individual variability in frontal asymmetry, in line with the capability model (Coan et al., 2006; Stewart et al., 2014). However, neither measure of asymmetry predicted distraction, suggesting that frontal asymmetry does not reflect ability to control attentional capture by emotional distractors.

These findings add to a large literature attempting to elucidate the mechanisms underlying frontal asymmetry in order to better understand the processes that contribute to psychopathology vulnerability, as well as to a growing literature that considers these mechanisms to be cognitive. This study was the first to directly test the relationship between frontal asymmetry and inhibitory control of emotion and paves the way for future investigations into the relationship between frontal asymmetry and cognitive control. Future research should explore whether frontal asymmetry reflects control processes that facilitate disengagement rather than those that limit capture, and consider the critical influence of emotion on cognitive processing when investigating cognitive vulnerabilities associated with frontal asymmetry.

References

- Allen, J. J. B., Coan, J. A., & Nazarian, M. (2004). Issues and assumptions on the road from raw signals to metrics of frontal EEG asymmetry in emotion. *Biological Psychology*, 67(1-2), 183–218. doi:10.1016/j.biopsycho.2004.03.007
- Allen, J. J., & Reznik, S. J. (2015). Frontal EEG asymmetry as a promising marker of depression vulnerability: Summary and methodological considerations. *Current Opinion in Psychology*, 4, 93–97. doi:10.1016/j.copsyc.2014.12.017
- Ambrosini, E., & Vallesi, A. (2016). Asymmetry in prefrontal resting-state EEG spectral power underlies individual differences in phasic and sustained cognitive control. *NeuroImage*, 124, 843–857. doi:10.1016/j.neuroimage.2015.09.035
- Armstrong, T., & Olatunji, B. O. (2012). Eye tracking of attention in the affective disorders: A meta-analytic review and synthesis. *Clinical Psychology Review*, 32(8), 704–723. doi:10.1016/j.cpr.2012.09.004
- Auerbach, R. P., Stewart, J. G., Stanton, C. H., Mueller, E. M., & Pizzagalli, D. A. (2015). Emotion-processing biases and resting EEG activity in depressed adolescents. *Depression and Anxiety*, 32(9), 693–701. doi:10.1002/da.22381
- Augst, S., Kleinsorge, T., & Kunde, W. (2014). Can we shield ourselves from task disturbance by emotion-laden stimulation? *Cognitive, Affective & Behavioral Neuroscience*, 14(3), 1009–1025. doi:10.3758/s13415-013-0243-x
- Balconi, M., Finocchiaro, R., & Canavesio, Y. (2014). Reward-system effect (BAS rating), left hemispheric “unbalance” (alpha band oscillations) and decisional impairments in drug addiction. *Addictive Behaviors*, 39, 1026–1032. doi:10.1016/j.addbeh.2014.02.007
- Banich, M. T., Mackiewicz, K. L., Depue, B. E., Whitmer, A., Miller, A., & Heller, W. (2009). Cognitive control mechanisms, emotion & memory: A neural perspective with implications for psychopathology. *Neuroscience and Biobehavioral Reviews*, 33(5), 613–630. doi:10.1016/j.neubiorev.2008.09.010
- Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin*, 133(1), 1–24. doi:10.1037/0033-2909.133.1.1
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, 121(1), 65–94. doi:10.1037/0033-2909.121.1.65

- Barlow, D. H. (1991). Disorders of emotion. *Psychological Inquiry*, 2(1), 58–71. doi: 10.1207/s15327965pli0201_15
- Beauregard, M., Lévesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 21(18), RC165. Retrieved from <http://www.jneurosci.org/cgi/content/full/5619>
- Beevers, C. G., Clasen, P. C., Enock, P. M., & Schnyer, D. M. (2015). Attention bias modification for major depressive disorder: Effects on attention bias, resting state connectivity, and symptom change. *Journal of Abnormal Psychology*, 124(2), 1–13. doi:10.1037/abn0000049
- Berntson, G. G., Norman, G. J., & Cacioppo, J. T. (2011). Laterality and evaluative bivalence: A neuroevolutionary perspective. *Emotion Review*, 3(3), 344–346. doi:10.1177/1754073911402401
- Binder, J. R., Frost, J. A., Hammeke, T. A., Bellgowan, P. S. F., Rao, S. M., & Cox, R. W. (1999). Conceptual processing during the conscious resting state: A functional MRI study. *Journal of Cognitive Neuroscience*, 11(1), 80–93. doi:10.1162/089892999563265
- Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience*, 7(2), 184–8. doi:10.1038/nn1173
- Bismark, A. W., Moreno, F. A., Stewart, J. L., Towers, D. N., Coan, J. A., Oas, J., ... Allen, J. J. B. (2010). Polymorphisms of the HTR1a allele are linked to frontal brain electrical asymmetry. *Biological Psychology*, 83(2), 153–158. doi:10.1016/j.biopsycho.2009.12.002
- Black, C. L., Goldstein, K. E., Labelle, D. R., Brown, C. W., Harmon-Jones, E., Abramson, L. Y., & Alloy, L. B. (2014). Behavioral approach system sensitivity and risk taking interact to predict left-frontal EEG asymmetry. *Behavior Therapy*, 45, 640–650. doi:10.1016/j.beth.2014.01.006
- Blackhart, G. C., Kline, J. P., Donohue, K. F., LaRowe, S. D., & Joiner, T. E. (2002). Affective responses to EEG preparation and their link to resting anterior EEG asymmetry. *Personality and Individual Differences*, 32(1), 167–174. doi:10.1016/S0191-8869(01)00015-0
- Blackhart, G. C., Minnix, J. A., & Kline, J. P. (2006). Can EEG asymmetry patterns predict future development of anxiety and depression? A preliminary study. *Biological Psychology*, 72(1), 46–50. doi:10.1016/j.biopsycho.2005.06.010

- Boonstra, A. M., Oosterlaan, J., Sergeant, J. A., & Buitelaar, J. K. (2005). Executive functioning in adult ADHD: A meta-analytic review. *Psychological Medicine*, 35(8), 1097–1108. doi:10.1017/S003329170500499X
- Booth, R. W., & Paker, M. (2016). State anxiety impairs attentional control when other sources of control are minimal. *Cognition and Emotion*, 9931, 1–8. doi:10.1080/02699931.2016.1172474
- Botvinick, M., & Braver, T. (2015). Motivation and cognitive control: From behavior to neural mechanism. *Annual Review of Psychology*, 66, 83–113. doi:10.1146/annurev-psych-010814-015044
- Braver, T. S. (2012). The variable nature of cognitive control: A dual-mechanisms framework. *Trends in Cognitive Sciences*, 16(2), 106–113. doi:10.1016/j.tics.2011.12.010
- Braver, T. S., Paxton, J. L., Locke, H. S., & Barch, D. M. (2009). Flexible neural mechanisms of cognitive control within human prefrontal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, 106(18), 7351–7356. doi:10.1073/pnas.0808187106
- Brown, T. E. (2006). Executive functions and attention deficit hyperactivity disorder: Implications of two conflicting views. *International Journal of Disability, Development and Education*, 53(1), 35–46. doi:10.1080/10349120500510024
- Bugg, J. M., & Crump, M. J. C. (2012). In support of a distinction between voluntary and stimulus-driven control: A review of the literature on proportion congruent effects. *Frontiers in Psychology*, 3, 1–16. doi:10.3389/fpsyg.2012.00367
- Casillas, A. & Clark, L. A. (2000, May). The Mini Mood and Anxiety Symptom Questionnaire (MiniMASQ). Poster presented at the 72nd Annual Meeting of the Midwestern Psychological Association, Chicago, IL.
- Chiew, K. S., & Braver, T. S. (2011). Positive affect versus reward: Emotional and motivational influences on cognitive control. *Frontiers in Psychology*, 2(279). doi:10.3389/fpsyg.2011.00279
- Cisler, Josh, M., & Koster, Ernst, H. W. (2011). Mechanisms of attentional biases towards threat in anxiety disorder: An integrative review. *Clinical Psychology Review*, 30(2), 1–29. doi:10.1016/j.cpr.2009.11.003.Mechanisms
- Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological assessment*, 7(3), 309. doi:10.1037/1040-3590.7.3.309

- Coan, J. A., & Allen, J. J. B. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, 67(1-2), 7–49. doi:10.1016/j.biopsycho.2004.03.002
- Coan, J. A., Allen, J. J. B., & McKnight, P. E. (2006). A capability model of individual differences in frontal EEG asymmetry. *Biological Psychology*, 72(2), 198–207. doi:10.1016/j.biopsycho.2005.10.003.A
- Cole, C., Zapp, D. J., Nelson, S. K., & Pérez-Edgar, K. (2012). Speech presentation cues moderate frontal EEG asymmetry in socially withdrawn young adults. *Brain and Cognition*, 78(2), 156–162. doi:10.1016/j.bandc.2011.10.013
- Compton, R. J., Banich, M. T., Mohanty, A., Milham, M. P., Herrington, J., Miller, G. A., ... Heller, W. (2003). Paying attention to emotion: An fMRI investigation of cognitive and emotional stroop tasks. *Cognitive, Affective & Behavioral Neuroscience*, 3(2), 81–96. doi:10.3758/CABN.3.2.81
- Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience*, 3(3), 201–15. doi:10.1038/nrn755
- Craig, A. D. (2009). Emotional moments across time: A possible neural basis for time perception in the anterior insula. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 364(1525), 1933–42. doi:10.1098/rstb.2009.0008
- Curci, A., Lanciano, T., Soleti, E., & Rimé, B. (2013). Negative emotional experiences arouse rumination and affect working memory capacity. *Emotion*, 13(5), 867–80. doi:10.1037/a0032492
- Davidson, R. J. (1998). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition & Emotion*, 12(3), 307–330. doi:10.1080/026999398379628
- Davidson, R. J. (2000). Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *The American Psychologist*, 55(11), 1196–1214. doi:10.1037/0003-066X.55.11.1196
- Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: Regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry*, 47(2), 85–95. doi:10.1016/S0006-3223(99)00222-X
- De Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: A review of underlying processes. *Addiction Biology*, 14(1), 22–31. doi:10.1111/j.1369-1600.2008.00129.x

- Degutis, J., Wilmer, J., Mercado, R. J., & Cohan, S. (2013). Using regression to measure holistic face processing reveals a strong link with face recognition ability. *Cognition*, 126, 87–100. Retrieved from <http://www.elsevier.com/copyright>
- Dennis, T. A., & Solomon, B. (2010). Frontal EEG and emotion regulation: Electrocortical activity in response to emotional film clips is associated with reduced mood induction and attention interference effects. *Biological Psychology*, 85(3), 456–464. doi:10.1016/j.biopsycho.2010.09.008
- Dolcos, F., Iordan, A. D., & Dolcos, S. (2011). Neural correlates of emotion-cognition interactions: A review of evidence from brain imaging investigations. *Journal of Cognitive Psychology*, 23(6), 669–694. doi:10.1080/20445911.2011.594433
- Drabant, E. M., McRae, K., Manuck, S. B., Hariri, A. R., & Gross, James, J. (2009). Individual differences in typical reappraisal use predict amygdala and prefrontal responses. *Biological Psychiatry*, 65(5), 367–373. doi:10.1016/j.biopsych.2008.09.007
- D’Esposito, M., Postle, B. R., & Rypma, B. (2000). Prefrontal cortical contributions to working memory: Evidence from event-related fMRI studies. *Experimental Brain Research*, 133(1), 3–11. doi:10.1007/s002210000395
- De Raedt, R., & Koster, E. H. W. (2010). Understanding vulnerability for depression from a cognitive neuroscience perspective: A reappraisal of attentional factors and a new conceptual framework. *Cognitive, Affective & Behavioral Neuroscience*, 10(1), 50–70. doi:10.3758/CABN.10.1.50
- Derakshan, N., & Eysenck, M. W. (2009). Anxiety, processing efficiency, and cognitive performance: New developments from attentional control theory. *European Psychologist*, 14(2), 168–176. doi:10.1027/1016-9040.14.2.168
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130(3), 355–391. doi:10.1037/0033-2909.130.3.355
- Dunlap, W. P., Jose, J. M., Vaslow, J. B., & Burke, M. J. (1996). Meta-analysis of experiments with matched groups or repeated measures designs. *Psychological Methods*, 1(2), 170–177. doi:10.1037/1082-989X.1.2.170
- Ellenbogen, M. A., Schwartzman, A. E., Stewart, J., & Walker, C.-D. (2002). Stress and selective attention: The interplay of mood, cortisol levels, and emotional information processing. *Psychophysiology*, 39(6), 723–732. doi:10.1111/1469-8986.3960723
- Engels, A. S., Heller, W., Spielberg, J. M., Warren, S. L., Sutton, B. P., Banich, M. T., & Miller, G. A. (2010). Co-occurring anxiety influences patterns of brain activity in

- depression. *Cognitive Affective Behavioral Neuroscience*, 10(1), 141–156. doi:10.3758/CABN.10.1.141.
- Engels, A. S., Heller, W., Mohanty, A., Herrington, J. D., Banich, M. T., Webb, A. G., & Miller, G. A. (2007). Specificity of regional brain activity in anxiety types during emotion processing. *Psychophysiology*, 44(3), 352–363. doi:10.1111/j.1469-8986.2007.00518.x
- Engström, J., Johansson, E., & Östlund, J. (2005). Effects of visual and cognitive load in real and simulated motorway driving. *Transportation Research Part F: Traffic Psychology and Behaviour*, 8(2), 97–120. doi:10.1016/j.trf.2005/04/012
- Epstein, J. N., Johnson, D. E., Varia, I. M., & Conners, C. K. (2001). Neuropsychological assessment of response inhibition in adults with ADHD. *Journal of Clinical and Experimental Neuropsychology*, 23(3), 362–371. doi:10.1076/jcen.23.3.362.1186
- Everaert, J., Grahek, I., & Koster, E. H. W. (2016). Individual differences in cognitive control modulate cognitive biases linked to depressive symptoms. *Cognition and Emotion*, 12, 1–11. doi:10.1080/02699931.2016.1144562
- Eysenck, M. W., & Derakshan, N. (2011). New perspectives in attentional control theory. *Personality and Individual Differences*, 50(7), 955–960. doi:10.1016/j.paid.2010.08.019
- Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion*, 7(2), 336–53. doi:10.1037/1528-3542.7.2.336
- Feng, X., Forbes, E. E., Kovacs, M., George, C. J., Lopez-Duran, N. L., Fox, N. A., & Cohn, J. F. (2012). Children's depressive symptoms in relation to EEG frontal asymmetry and maternal depression. *Journal of Abnormal Child Psychology*, 40(2), 265–276. doi:10.1007/s10802-011-9564-9
- Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug and Alcohol Dependence*, 97(1-2), 1–20. doi:10.1016/j.drugalcdep.2008.03.030
- Field, T., & Diego, M. (2008). Maternal depression effects on infant frontal EEG asymmetry. *The International Journal of Neuroscience*, 118(8), 1081–108. doi:10.1080/00207450701769067
- Forster, S., & Lavie, N. (2008a). Attentional capture by entirely irrelevant distractors. *Visual Cognition*, 16(2-3), 200–214. doi:10.1080/13506280701465049

- Forster, S., & Lavie, N. (2008b). Failures to ignore entirely irrelevant distractors: The role of load. *Journal of Experimental Psychology: Applied*, *14*(1), 73–83. doi:10.1037/1076-898X.14.1.73
- Forster, S., & Lavie, N. (2016). Establishing the attention-distractibility trait. *Psychological Science*, *27*(2), 203–212. doi:10.1177/0956797615617761
- Forster, S., Robertson, D. J., Jennings, A., Asherson, P., & Lavie, N. (2014). Plugging the attention deficit: Perceptual load counters increased distraction in ADHD. *Neuropsychology*, *28*(1), 91–97. doi:10.1037/neu0000020
- Fox, E., Yates, A., & Ashwin, C. (2012). Trait anxiety and perceptual load as determinants of emotion processing in a fear conditioning paradigm. *Emotion*, *12*(2), 236–249. doi:10.1037/a0025321
- Franken, I. H., Kroon, L. Y., Wiers, R. W., & Jansen, A. (2000). Selective cognitive processing of drug cues in heroin dependence. *Journal of Psychopharmacology*, *14*(4), 395–400. doi:10.1177/026988110001400408
- Friston, K. J., Frith, C. D., Liddle, P. F., & Frackowiak, R. S. J. (1993). Functional connectivity: The principal-component analysis of large (PET) data sets. *Journal of Cerebral Blood Flow and Metabolism*, *13*, 5–14. doi:10.1038/jcbfm.1993.4
- Gable, P., Mechin, N., Hicks, J., & Adams, D. (2015). Supervisory control system and frontal asymmetry: Neurophysiological traits of emotion-based impulsivity. *Social Cognitive and Affective Neuroscience*. Retrieved from <http://scan.oxfordjournals.org/content/early/2015/02/11/scan.nsv017.abstract>
- Gapin, J., Etnier, J. L., & Tucker, D. (2009). The relationship between frontal brain asymmetry and exercise addiction. *Journal of Psychophysiology*, *23*(3), 135–142. doi:10.1027/0269-8803.23.3.135
- Garavan, H., & Hester, R. (2007). The role of cognitive control in cocaine dependence. *Neuropsychology Review*, *17*(3), 337–345. doi:10.1007/s11065-007-9034-x
- Goeleven, E., De Raedt, R., Baert, S., & Koster, E. H. W. (2006). Deficient inhibition of emotional information in depression. *Journal of Affective Disorders*, *93*(1-3), 149–157. doi:10.1016/j.jad.2006.03.007
- 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*(6), 577–86. doi:10.1016/j.biopsych.2007.05.031
- Goldstein, B. L., & Klein, D. N. (2014). A review of selected candidate endophenotypes for depression. *Clinical Psychology Review*, *34*(5), 417–27. doi:10.1016/j.cpr.2014.06.003

- Goldstein, R. Z., Leskovjan, A. C., Hoff, A. L., Hitzemann, R., Bashan, F., Khalsa, S. S., ... Volkow, N. D. (2004). Severity of neuropsychological impairment in cocaine and alcohol addiction: Association with metabolism in the prefrontal cortex. *Neuropsychologia*, 42(11), 1447–58. doi:10.1016/j.neuropsychologia.2004.04.002
- Goldstein, B. L., Shankman, S. A., Kujawa, A., Torpey-Newman, D. C., Olino, T. M., & Klein, D. N. (2016). Developmental changes in electroencephalographic frontal asymmetry in young children at risk for depression. *Journal of Child Psychology and Psychiatry*. Advance online publication. doi:10.1111/jcpp.12567
- Goodman, R. N., Rietschel, J. C., Lo, L.-C., Costanzo, M. E., & Hatfield, B. D. (2013). Stress, emotion regulation and cognitive performance: The predictive contributions of trait and state relative frontal EEG alpha asymmetry. *International Journal of Psychophysiology*, 87(2), 115–23. doi:10.1016/j.ijpsycho.2012.09.008
- Gorka, S. M., Phan, K. L., & Shankman, S. A. (2015). Convergence of EEG and fMRI measures of reward anticipation. *Biological Psychology*, 112, 12–9. doi:10.1016/j.biopsycho.2015.09.007
- Gotlib, I., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Revista de Neurologia*, 6, 285–312. doi:10.1146/annurev.clinpsy.121208.131305
- Gotlib, I. H., Ranganathand, C., & Rosenfeld, J. P. (1998). EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion*, 12(3), 449–478. doi:10.1080/026999398379673
- Gray, J. R., Braver, T. S., & Raichle, M. E. (2002). Integration of emotion and cognition in the lateral prefrontal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, 99(6), 4115–4120. doi:10.1073/pnas.062381899
- 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(4), 369–376. doi:10.1016/j.biopsych.2007.05.033
- Grimshaw, G. M., & Carmel, D. (2014). An asymmetric inhibition model of hemispheric differences in emotional processing. *Frontiers in Psychology*, 5(489). doi:10.3389/fpsyg.2014.00489

- Grimshaw, G. M., Foster, J. J., & Corballis, P. M. (2014). Frontal and parietal EEG asymmetries interact to predict attentional bias to threat. *Brain and Cognition*, 90, 76–86. doi:10.1016/j.bandc.2014.06.008
- Grimshaw, G. G., Kranz, L. K., Carmel, D., Moody, R. E., & Devue, C. (2016). *Contrasting reactive and proactive control of emotional distraction: Sex and violence don't always win*. Manuscript submitted for publication.
- Groman, S. M., & Jentsch, J. D. (2012). Cognitive control and the dopamine D₂-like receptor: A dimensional understanding of addiction. *Depression and Anxiety*, 29(4), 295–306. doi:10.1002/da.20897
- Gross, J.J., & John, O.P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, 85, 348–362. doi:10.1037/0022-3514.85.2.348
- Gu, X., Hof, P. R., Friston, K. J., & Fan, J. (2013). Anterior insular cortex and emotional awareness. *Journal of Comparative Neurology*, 521(15), 3371–3388. doi:10.1002/cne.23368
- Gupta, R., Hur, Y.-J., & Lavie, N. (2015). Distracted by pleasure: Effects of positive versus negative valence on emotional capture under load. *Emotion*. doi:10.1037/emo0000112
- Hagemann, D. (2004). Individual differences in anterior EEG asymmetry: Methodological problems and solutions. *Biological Psychology*, 67(1-2), 157–182. doi:10.1016/j.biopsycho.2004.03.006
- Hagemann, D., & Naumann, E. (2001). The effects of ocular artifacts on (lateralized) broadband power in the EEG. *Clinical Neurophysiology*, 112(2), 215–231. doi:10.1016/S1388-2457(00)00541-1
- Harmon-Jones, E. (2003). Clarifying the emotive functions of asymmetrical frontal cortical activity. *Psychophysiology*, 40(6), 838–848. doi:10.1111/1469-8986.00121
- Harmon-jones, E., & Allen, J. J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology*, 106(1), 159–163. doi:10.1037//0021-843X.106.1.159
- Hasegawa, R. P., Peterson, B. W., & Goldberg, M. E. (2004). Prefrontal neurons coding suppression of specific saccades. *Neuron*, 43(3), 415–425. doi:10.1016/j.neuron.2004.07.013

- Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology*, 7(4), 476–489. doi:10.1037/0894-4105.7.4.476
- Heller, W., & Nitschke, J. B. (1998). The puzzle of regional brain activity in anxiety: The importance of subtypes and comorbidity. *Cognition & Emotion*, 12(3), 421–447. doi:10.1080/026999398379664
- Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology*, 106(3), 376–385. doi:10.1037/0021-843X.106.3.376
- Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, 100(4), 535–45. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/1757667>
- Henriques, J. B., & Davidson, R. J. (1997). Brain electrical asymmetries during cognitive task performance in depressed and nondepressed subjects. *Biological Psychiatry*, 42(11), 1039–1050. doi:10.1016/S0006-3223(97)00156-X
- Herrington, J. D., Heller, W., Aprajita, M., Engels, A. S., Banich, M. T., Webb, A. G., & Miller, G. A. (2010). Localization of asymmetric brain function in emotion and depression. *Psychophysiology*, 47(3), 442–454. doi:10.1111/j.1469-8986.2009.00958.x
- Herrington, J. D., Mohanty, A., Koven, N. S., Fisher, J. E., Stewart, J. L., Banich, M. T., ... Heller, W. (2005). Emotion-modulated performance and activity in left dorsolateral prefrontal cortex. *Emotion*, 5(2), 200–207. doi:10.1037/1528-3542.5.2.200
- Hester, R., Dixon, V., & Garavan, H. (2006). A consistent attentional bias for drug-related material in active cocaine users across word and picture versions of the emotional Stroop task. *Drug and Alcohol Dependence*, 81(3), 251–257. doi:10.1016/j.drugalcdep.2005.07.002
- Hofmann, S. G., Moscovitch, D. A., Litz, B. T., Kim, H.-J., Davis, L. L., & Pizzagalli, D. A. (2005). The worried mind: Autonomic and prefrontal activation during worrying. *Emotion*, 5(4), 464–475. doi:10.1037/1528-3542.5.4.464
- Jones, N. A., Field, T., Fox, N. A., Davalos, M., & Gomez, C. (2001). EEG during different emotions in 10-month-old infants of depressed mothers. *Journal of Reproductive and Infant Psychology*, 19(4), 295–312. doi:10.1080/02646830127204
- Joormann, J. (2004). Attentional bias in dysphoria: The role of inhibitory processes. *Cognition & Emotion*, 18(1), 125–147. doi:10.1080/02699930244000480

- Joormann, J., & D'Avanzato, C. (2010). Emotion regulation in depression: Examining the role of cognitive processes. *Cognitive & Emotion*, 24(6), 913–939.
doi:10.1080/02699931003784939
- Joormann, J., & Gotlib, I. H. (2008). Updating the contents of working memory in depression: Interference from irrelevant negative material. *Journal of Abnormal Psychology*, 117(1), 182–192. doi:10.1037/0021-843X.117.1.182
- Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of Abnormal Psychology*, 116(1), 135–143.
doi:10.1037/0021-843X.116.1.135
- Joormann, J., & Tanovic, E. (2015). Cognitive vulnerability to depression: Examining cognitive control and emotion regulation. *Current Opinion in Psychology*, 4, 86–92.
doi:10.1016/j.copsyc.2014.12.006
- Kalivas, P. W., & Volkow, N. D. (2005). The neural basis of addiction: A pathology of motivation and choice. *The American Journal of Psychiatry*, 162(8), 1403–1413.
doi:10.1176/appi.ajp.162.8.1403
- Kane, M. J., & Engle, R. W. (2002). The role of prefrontal cortex in working-memory capacity, executive attention, and general fluid intelligence: An individual-differences perspective. *Psychonomic Bulletin & Review*, 9(4), 637–671. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/12613671>
- Kennedy, D. O., & Scholey, A. B. (2000). Glucose administration, heart rate and cognitive performance: Effects of increasing mental effort. *Psychopharmacology*, 149, 63–71.
doi:10.1007/s002139900335
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 593–602.
doi:10.1001/archpsyc.62.6.593
- Keune, P. M., Wiedemann, E., Schneidt, A., & Schönenberg, M. (2015). Frontal brain asymmetry in adult attention-deficit/hyperactivity disorder (ADHD): Extending the motivational dysfunction hypothesis. *Clinical Neurophysiology*, 126(4), 711–20.
doi:10.1016/j.clinph.2014.07.008
- Kinsey, A. C., Pomeroy, W. B., & Martin, C. E. (1948). *Sexual behavior in the human male*. Philadelphia: W. B. Saunders.
- Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the

- hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61(2), 154–162.
Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10204967>
- Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The “Trier Social Stress Test”: A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28(1-2), 76–81. doi:10.1159/000119004
- Klimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews*, 29(2-3), 169–195. doi:10.1016/S0165-0173(98)00056-3
- Kline, J. P., Blackhart, G. C., & Joiner, T. E. (2002). Sex, lie scales, and electrode caps: An interpersonal context for defensiveness and anterior electroencephalographic asymmetry. *Personality and Individual Differences*, 33(3), 459–478. doi:10.1016/S0191-8869(01)00167-2
- Kompus, K., Hugdahl, K., Ohman, A., Marklund, P., & Nyberg, L. (2009). Distinct control networks for cognition and emotion in the prefrontal cortex. *Neuroscience Letters*, 467(2), 76–80. doi:10.1016/j.neulet.2009.10.005
- Koslov, K., Mendes, W. B., Pajtas, P. E., & Pizzagalli, D. A. (2011). Asymmetry in resting intracortical activity as a buffer to social threat. *Psychological Science*, 22(5), 641–649. doi:10.1177/0956797611403156
- Koster, E. H. W., De Lissnyder, E., Derakshan, N., & De Raedt, R. (2011). Understanding depressive rumination from a cognitive science perspective: The impaired disengagement hypothesis. *Clinical Psychology Review*, 31(1), 138–45. doi:10.1016/j.cpr.2010.08.005
- Kranz, L. S. (2015). *Proactive control of emotional distraction: An ERP investigation (Masters thesis)*. Victoria University of Wellington, Wellington, New Zealand.
- Kübler, A., Murphy, K., & Garavan, H. (2005). Cocaine dependence and attention switching within and between verbal and visuospatial working memory. *The European Journal of Neuroscience*, 21(7), 1984–1992. doi:10.1111/j.1460-9568.2005.04027.x
- Kudielka, B. M., Hellhammer, D. H., & Kirschbaum, C. (2007). Ten years of research with the Trier Social Stress Test (TSST) - Revisited. In P. Harmon-Jones (Ed.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 56-83). New York: Guilford Press. Retrieved from <https://books.google.com/books?hl=en&lr=&id=mKSqxaHGaicC&pgis=1>
- Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in

- humans at different times of day. *Psychoneuroendocrinology*, 29(8), 983–992.
doi:10.1016/j.psyneuen.2003.08.009
- Lang, P. J., & Bradley, M. M. (2010). Emotion and the motivational brain. *Biological Psychology*, 84(3), 437–450. doi:10.1016/j.biopsycho.2009.10.007
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual* (Technical report A-8). Gainesville, FL: University of Florida.
- Lesage, F.-X., Berjot, S., & Deschamps, F. (2012). Clinical stress assessment using a visual analogue scale. *Occupational Medicine*, 62(8), 600–605. doi:10.1093/occmed/kqs140
- Lévesque, J., Eugène, F., Joanne, Y., Paquette, V., Mensour, B., Beaudoin, G., ... Beauregard, M. (2003). Neural circuitry underlying voluntary suppression of sadness. *Biological Psychiatry*, 53(6), 502–510. doi:10.1016/S0006-3223(02)01817-6
- Lin, H.-P., Lin, H.-Y., Lin, W.-L., & Huang, A. C.-W. (2011). Effects of stress, depression, and their interaction on heart rate, skin conductance, finger temperature, and respiratory rate: Sympathetic-parasympathetic hypothesis of stress and depression. *Journal of Clinical Psychology*, 67(10), 1080–1091. doi:10.1002/jclp.20833
- Lin, A., Yung, A. R., Wigman, J. T. W., Killackey, E., Baksheev, G., & Wardenaar, K. J. (2014). Validation of a short adaptation of the Mood and Anxiety Symptoms Questionnaire (MASQ) in adolescents and young adults. *Psychiatry Research*, 215(3), 778–783. doi:10.1016/j.psychres.2013.12.018
- Lopez-Duran, N. L., Nusslock, R., George, C., & Kovacs, M. (2012). Frontal EEG asymmetry moderates the effects of stressful life events on internalizing symptoms in children at familial risk for depression. *Psychophysiology*, 49(4), 510–521. doi:10.1111/j.1469-8986.2011.01332.x
- Lovibond, S.H. & Lovibond, P.F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd. Ed.). Sydney: Psychology Foundation.
- Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: MIT Press.
- MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science*, 288(5472), 1835–1838. doi:10.1126/science.288.5472.1835
- Maddock, A., Harper, D., Carmel, D., & Grimshaw, G. M. (2016). *The influence of motivation on emotional distraction*. Manuscript in preparation.

- Mathersul, D., Williams, L. M., Hopkinson, P. J., & Kemp, A. H. (2008). Investigating models of affect: Relationships among EEG alpha asymmetry, depression, and anxiety. *Emotion, 8*(4), 560–572. doi:10.1037/a0012811
- Mathewson, K. J., Hashemi, A., Sheng, B., Sekuler, A. B., Bennett, P. J., & Schmidt, L. A. (2015). Regional electroencephalogram (EEG) alpha power and asymmetry in older adults: A study of short-term test-retest reliability. *Frontiers in Aging Neuroscience, 7*, 177. doi:10.3389/fnagi.2015.00177
- Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology, 1*, 167–195. doi:10.1146/annurev.clinpsy.1.102803.143916
- McKiernan, K. A., Kaufman, J. N., Kucera-Thompson, J., & Binder, J. R. (2003). A parametric manipulation of factors affecting task-induced deactivation in functional neuroimaging. *Journal of Cognitive Neuroscience, 15*(3), 394–408. doi:10.1162/089892903321593117
- Metzger, L. J., Paige, S. R., Carson, M. A., Lasko, N. B., Paulus, L. A., Pitman, R. K., & Orr, S. P. (2004). PTSD arousal and depression symptoms associated with increased right-sided parietal EEG asymmetry. *Journal of Abnormal Psychology, 113*(2), 324–329. doi:10.1037/0021-843X.113.2.324
- Meyer, T., Quaedflieg, C. W. E. M., Giesbrecht, T., Meijer, E. H., Abiad, S., & Smeets, T. (2014). Frontal EEG asymmetry as predictor of physiological responses to aversive memories. *Psychophysiology, 51*(9), 853–865. doi:10.1111/psyp.12230
- Meyer, T., Smeets, T., Giesbrecht, T., Quaedflieg, C. W. E. M., Smulders, F. T. Y., Meijer, E. H., & Merckelbach, H. L. G. J. (2015). The role of frontal EEG asymmetry in post-traumatic stress disorder. *Biological Psychology, 108*, 62–77. doi:10.1016/j.biopsycho.2015.03.018
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.
- Miller, G. A., Crocker, L. D., Spielberg, J. M., Infantolino, Z. P., & Heller, W. (2013). Issues in localization of brain function: The case of lateralized frontal cortex in cognition, emotion, and psychopathology. *Frontiers in Integrative Neuroscience, 7*, 2. doi:10.3389/fnint.2013.00002
- Miller, A., Fox, N. A., Cohn, J. F., Forbes, E. E., Sherrill, J. T., & Kovacs, M. (2002). Regional patterns of brain activity in adults with a history of childhood-onset

- depression: Gender differences and clinical variability. *The American Journal of Psychiatry*, 159(6), 934–40. doi:10.1176/appi.ajp.159.6.934
- Miskovic, V., & Schmidt, L. A. (2010). Frontal brain electrical asymmetry and cardiac vagal tone predict biased attention to social threat. *International Journal of Psychophysiology*, 75, 332–338. doi:10.1016/j.ijpsycho.2009.12.015
- Miyake, A., & Friedman, N. P. (2012). The nature and organisation of individual differences in executive functions: Four general conclusions. *Current Directions in Psychological Science*, 21(1), 8–14. doi:10.1177/0963721411429458
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex “Frontal Lobe” tasks: A latent variable analysis. *Cognitive Psychology*, 41(1), 49–100. doi:10.1006/cogp.1999.0734
- Moeller, S. J., Honorio, J., Tomasi, D., Parvaz, M. A., Woicik, P. A., Volkow, N. D., & Goldstein, R. Z. (2014). Methylphenidate enhances executive function and optimizes prefrontal function in both health and cocaine addiction. *Cerebral Cortex*, 24(3), 643–653. doi:10.1093/cercor/bhs345
- Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, 110(3), 406–425. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/1758917>
- Morey, R. D. (2008). Confidence intervals from normalized data: A correction to Cousineau (2005), *Tutorial in Quantitative Methods for Psychology*, 4(2), 61–64. Retrieved from <http://pcl.missouri.edu/sites/default/files/morey.2008.pdf>
- Most, S. B., Smith, S. D., Cooter, A. B., Levy, B. N., & Zald, D. H. (2007). The naked truth: Positive, arousing distractors impair rapid target perception. *Cognition & Emotion*, 21(5), 964–981. doi:10.1080/02699930600959340
- Murphy, J. L. (2016). *EEG evidence for the effective proactive control of emotional distractors* (Master’s thesis). Victoria University of Wellington, Wellington, New Zealand.
- Nitschke, J. B., Heller, W., Palmieri, P. A., & Miller, G. A. (1999). Contrasting patterns of brain activity in anxious apprehension and anxious arousal. *Psychophysiology*, 36(5), 628–637. doi:10.1017/S0048577299972013
- Niv, S., Ashrafulla, S., Tuvblad, C., Joshi, A., Raine, A., Leahy, R., & Baker, L. A. (2015). Childhood EEG frontal alpha power as a predictor of adolescent antisocial behavior: A

- twin heritability study. *Biological Psychology*, 105, 72–76.
doi:10.1016/j.biopsycho.2014.11.010
- Nusslock, R., Shackman, A. J., Harmon-Jones, E., Alloy, L. B., Coan, J. A., & Abramson, L. Y. (2011). Cognitive vulnerability and frontal brain asymmetry: Common predictors of first prospective depressive episode. *Journal of Abnormal Psychology*, 120(2), 497–503. doi:10.1037/a0022940
- 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(8), 1215–1229. doi:10.1162/089892902760807212
- Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences*, 9(5), 242–249. doi:10.1016/j.tics.2005.03.010
- 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, E1–24. doi:10.1111/j.1749-6632.2012.06751.x
- Papousek, I., Freudenthaler, H. H., & Schuler, G. (2011). Typical performance measures of emotion regulation and emotion perception and frontal EEG asymmetry in an emotional contagion paradigm. *Personality and Individual Differences*, 51(8), 1018–1022. doi:10.1016/j.paid.2011.08.013
- Papousek, I., Murhammer, D., & Schuler, G. (2011). Intra- and interindividual differences in lateralized cognitive performance and asymmetrical EEG activity in the frontal cortex. *Brain and Cognition*, 75(3), 225–231. doi:10.1016/j.bandc.2010.11.013
- Papousek, I., Reiser, E. M., Schuler, G., Fink, A., Holmes, E. A., Nagl, S., ... Niederstätter, H. (2013). Serotonin transporter genotype (5-HTTLPR) and electrocortical responses indicating the sensitivity to negative emotional cues. *Emotion*, 13(6), 1173–1181. doi:10.1037/a0033997
- Papousek, I., Reiser, E. M., Weber, B., Freudenthaler, H. H., & Schuler, G. (2012). Frontal brain asymmetry and affective flexibility in an emotional contagion paradigm. *Psychophysiology*, 49(4), 489–498. doi:10.1111/j.1469-8986.2011.01324.x
- Papousek, I., & Schuler, G. (2004). Manipulation of frontal brain asymmetry by cognitive tasks. *Brain and Cognition*, 54(1), 43–51. doi:10.1016/S0278-2626(03)00258-6
- Papousek, I., Weiss, E. M., Perchtold, C. M., Weber, H., de Assunção, V. L., Schuler, G., ... Fink, A. (2016). The capacity for generating cognitive reappraisals is reflected in

- asymmetric activation of frontal brain regions. *Brain Imaging and Behavior*. Advance online publication. doi:10.1007/s11682-016-9537-2
- Papousek, I., Weiss, E. M., Schuster, G., Fink, A., Reiser, E. M., & Lackner, H. K. (2014). Prefrontal EEG alpha asymmetry changes while observing disaster happening to other people: Cardiac correlates and prediction of emotional impact. *Biological Psychology*, *103*, 184–194. doi:10.1016/j.biopsycho.2014.09.001
- Pe, M. L., Brose, A., Gotlib, I. H., & Kuppens, P. (2016). Affective updating ability and stressful events interact to prospectively predict increases in depressive symptoms over time. *Emotion*, *16*(1), 73–81. doi:10.1037/emo0000097
- Pérez-Edgar, K., Kujawa, A., Nelson, S., Cole, C., & Zapp, D. J. (2013). The relation between electroencephalogram asymmetry and attention biases to threat at baseline and under stress. *Brain and Cognition*, *82*(3), 337–343. doi:10.1016/j.bandc.2013.05.009
- Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience*, *9*(2), 148–58. doi:10.1038/nrn2317
- Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences*, *13*(4), 160–166. doi:10.1016/j.tics.2009.01.006
- Pessoa, L. (2013). *The cognitive-emotional brain: From interactions to integration*. Cambridge, MA: MIT press.
- Pessoa, L. (2015). Précis on the cognitive-emotional brain. *Behavioral and Brain Sciences*, *38*, 1–66. doi:10.1017/S0140525X14000120
- Pessoa, L., Kastner, S., & Ungerleider, L. G. (2002). Attentional control of the processing of neutral and emotional stimuli. *Cognitive Brain Research*, *15*(1), 31–45. doi:10.1016/S0926-6410(02)00214-8
- Petrides, M. (2000). Dissociable roles of mid-dorsolateral prefrontal and anterior inferotemporal cortex in visual working memory. *Journal of Neuroscience*, *20*(19), 7496–7503. Retrieved from <http://www.jneurosci.org/content/20/19/7496.full>
- Pizzagalli, D. A., Sherwood, R. J., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness: A source-localization study. *Psychological Science*, *16*(10), 805–813. doi:10.1111/j.1467-9280.2005.01618.x
- Pfurtscheller, G., Stancák, A., & Neuper, C. (1996). Event-related synchronization (ERS) in the alpha band - An electrophysiological correlate of cortical idling: A review. *International Journal of Psychophysiology*, *24*(1-2), 39–46. doi:10.1016/S0167-8760(96)00066-9

- Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. *Attention and Performance X: Control of Language Processes*, 32, 531–556. Retrieved from http://www.psych.utoronto.ca/users/ferber/teaching/visualattention/readings/Sept22/1984_Posner_Cohen_Attention&PerformanceX.pdf
- Pössel, P., Lo, H., Fritz, A., & Seemann, S. (2008). A longitudinal study of cortical EEG activity in adolescents. *Biological Psychology*, 78(2), 173–178. doi:10.1016/j.biopsycho.2008.02.004
- Pourtois, G., Schettino, A., & Vuilleumier, P. (2013). Brain mechanisms for emotional influences on perception and attention: What is magic and what is not. *Biological Psychology*, 92(3), 492–512. doi:10.1016/j.biopsycho.2012.02.007
- Proudfit, G. H., Bress, J. N., Foti, D., Kujawa, A., & Klein, D. N. (2015). Depression and event-related potentials: Emotional disengagement and reward sensitivity. *Current Opinion in Psychology*, 4, 110–113. doi:10.1016/j.copsyc.2014.12.018
- Provost, J.-S., Petrides, M., & Monchi, O. (2009). Basal ganglia and frontal involvement in self-generated and externally-triggered finger movements in the dominant and non-dominant hand. *European Journal of Neuroscience*, 29(6), 1277–1286. doi:10.1111/j.1460-9568.2009.06671.x
- Psychology Software Tools, Inc. [E-Prime 2.0]. (2012). Retrieved from <http://www.pstnet.com>
- Rohde, P., Lewinsohn, P. M., & Klein, D. N. (2014). Key characteristics of major depressive disorder occurring in childhood, adolescence, emerging adulthood, adulthood. *Clinical Psychological Science*, 1(1), 1–21. doi:10.1177/2167702612457599
- Rudaizky, D., Basanovic, J., & MacLeod, C. (2014). Biased attentional engagement with, and disengagement from, negative information: Independent cognitive pathways to anxiety vulnerability? *Cognition & Emotion*, 28(2), 245–259. doi:10.1080/02699931.2013.815154
- Salinsky, M. C., Oken, B. S., & Morehead, L. (1991). Test-retest reliability in EEG frequency analysis. *Electroencephalography and Clinical Neurophysiology*, 79(5), 382–392. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/1718711>
- Santesso, D. L., Segalowitz, S. J., Ashbaugh, A. R., Antony, M. M., McCabe, R. E., & Schmidt, L. A. (2008). Frontal EEG asymmetry and sensation seeking in young adults. *Biological Psychology*, 78(2), 164–172. doi:10.1016/j.biopsycho.2008.02.003

- Schmeichel, B. J., & Tang, D. (2015). Individual differences in executive functioning and their relationship to emotional processes and responses. *Current Directions in Psychological Science*, 24(2), 93–98. doi:10.1177/0963721414555178
- Schmidt, L. A. (1999). Frontal brain electrical activity in shyness and sociability. *Psychological Science*, 10(4), 316–320. doi:10.1111/1467-9280.00161
- Seymour, K. E., Kim, K. L., Cushman, G. K., Puzia, M. E., Weissman, A. B., Galvan, T., & Dickstein, D. P. (2015). Affective processing bias in youth with primary bipolar disorder or primary attention-deficit/hyperactivity disorder. *European Child & Adolescent Psychiatry*, 24(11), 1349–1359. doi:10.1007/s00787-015-0686-4
- Shackman, A. J., McMenamin, B. W., Maxwell, J. S., Greischar, L. L., & Davidson, R. J. (2009). Right dorsolateral prefrontal cortical activity and behavioral inhibition. *Psychological Science*, 20(12), 1500–1506. doi:10.1111/j.1467-9280.2009.02476.x
- Shimamura, A. P. (2000). The role of the prefrontal cortex in dynamic filtering. *Psychobiology*, 28(2), 207–218. doi:10.3758/BF03331979
- Smith, E. E., Zambrano-Vazquez, L., & Allen, J. J. B. (2016). Patterns of alpha asymmetry in those with elevated worry, trait anxiety, and obsessive-compulsive symptoms: A test of the worry and avoidance models of alpha asymmetry. *Neuropsychologia*. doi:10.1016/j.neuropsychologia.2016.03.010
- Sinha, R. (2008). Chronic stress, drug use, and vulnerability to addiction. *Annals of the New York Academy of Sciences*, 1141, 105–130. doi:10.1196/annals.1441.030.Chronic
- Smit, D. J. A., Posthuma, D., Boomsma, D. I., & De Geus, E. J. C. (2007). The relation between frontal EEG asymmetry and the risk for anxiety and depression. *Biological Psychology*, 74(1), 26–33. doi:10.1016/j.biopsycho.2006.06.002
- Snyder, H. R. (2013). Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: A meta-analysis and review. *Psychological Bulletin*, 139(1), 81–132. doi:10.1037/a0028727
- Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Steele, J. D., & Lawrie, S. M. (2004). Segregation of cognitive and emotional function in the prefrontal cortex: A stereotactic meta-analysis. *NeuroImage*, 21(3), 868–875. doi:10.1016/j.neuroimage.2003.09.066
- Stewart, J., Bismark, A., Towers, D. N., Coan, J. A., & Allen, J. J. B. (2010). Resting frontal EEG asymmetry as an endophenotype for depression risk: Sex-specific patterns of

- frontal brain asymmetry. *Journal of Abnormal Psychology*, 119(3), 502–512.
doi:10.1037/a0019196
- Stewart, J. L., Coan, J. A., Towers, D. N., & Allen, J. J. B. (2011). Frontal EEG asymmetry during emotional challenge differentiates individuals with and without lifetime major depressive disorder. *Journal of Affective Disorders*, 129(1-3), 167–174.
doi:10.1016/j.jad.2010.08.029
- Stewart, J. L., Coan, J. A., Towers, D. N., & Allen, J. J. B. (2014). Resting and task-elicited prefrontal EEG alpha asymmetry in depression: Support for the capability model. *Psychophysiology*, 51(5), 446–455. doi:10.1111/psyp.12191
- Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science*, 8(3), 204–210.
doi:10.1111/j.1467-9280.1997.tb00413.x
- Suzuki, M., & Gottlieb, J. (2013). Distinct neural mechanisms of distractor suppression in the frontal and parietal lobe. *Nature Neuroscience*, 16(1), 98–104. doi:10.1038/nn.3282
- Telpaz, A., & Yechiam, E. (2014). Contrasting losses and gains increases the predictability of behavior by frontal EEG asymmetry. *Frontiers in Behavioral Neuroscience*, 8, 149.
doi:10.3389/fnbeh.2014.00149
- Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *Journal of Abnormal Psychology*, 115(4), 715–729. doi:10.1037/0021-843X.115.4.715
- Tomarken, A. J., & Davidson, R. J. (1994). Frontal brain activation in repressors and nonrepressors individual differences in anterior eeg asymmetry: Relations with emotion and psychopathology individual differences in repressive-defensive coping styles, *Journal of Abnormal Psychology*, 103(2), 339–349. doi:10.1037/0021-843X.103.2.339
- Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Doss, R. C. (1992). Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. *Journal of Personality and Social Psychology*, 62(4), 676–87. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/1583591>
- Tooley, M. D. (2015). *Emotion regulation and vulnerability to depression: A longitudinal test of the diathesis-stress model* (Doctoral dissertation). Victoria University of Wellington, Wellington, New Zealand.
- Turner, J. R., & Carroll, D. (1985). Heart rate and oxygen consumption during mental arithmetic, a video game, and graded exercise: further evidence of metabolically-

- exaggerated cardiac adjustments? *Psychophysiology*, 22(3), 261–267.
doi:10.1111/j.1469-8986.1985.tb01597.x
- Uusberg, H., Allik, J., & Hietanen, J. K. (2015). Eye contact reveals a relationship between Neuroticism and anterior EEG asymmetry. *Neuropsychologia*, 73, 161–168.
doi:10.1016/j.neuropsychologia.2015.05.008
- Vanderhasselt, M.-A., Kühn, S., & De Raedt, R. (2011). Healthy brooders employ more attentional resources when disengaging from the negative: An event-related fMRI study. *Cognitive, Affective & Behavioral Neuroscience*, 11(2), 207–216.
doi:10.3758/s13415-011-0022-5
- Verdejo-Garcia, A., Perez-Garcia, M., & Bechara, A. (2006). Emotion, decision-making and substance dependence: A somatic-marker model of addiction. *Current Neuropsychopharmacology*, 4(1), 17–31. doi:10.2174/157015906775203057
- Verona, E., Sadeh, N., & Curtin, J. J. (2009). Stress-induced asymmetric frontal brain activity and aggression risk. *Journal of Abnormal Psychology*, 118(1), 131–145.
doi:10.1037/a0014376
- Vogt, J., De Houwer, J., Koster, E. H. W., Van Damme, S., & Crombez, G. (2008). Allocation of spatial attention to emotional stimuli depends upon arousal and not valence. *Emotion*, 8(6), 880–885. doi:10.1037/a0013981
- Wacker, J., Mueller, E. M., Pizzagalli, D. A., Hennig, J., & Stemmler, G. (2013). Dopamine-D2-receptor blockade reverses the association between trait approach motivation and frontal asymmetry in an approach-motivation context. *Psychological Science*, 24(4), 489–497. doi:10.1177/0956797612458935
- Wagner, C. R., & Abaied, J. L. (2016). Skin conductance level reactivity moderates the association between parental psychological control and relational aggression in emerging adulthood. *Journal of Youth and Adolescence*, 45(4), 687–700.
doi:10.1007/s10964-016-0422-5
- Wahlström, J., Hagberg, M., Johnson, P. W., Svensson, J., & Rempel, D. (2002). Influence of time pressure and verbal provocation on physiological and psychological reactions during work with a computer mouse. *European Journal of Applied Physiology*, 87(3), 257–263. doi:10.1007/s00421-002-0611-7
- Walcott, C. M., & Landau, S. (2004). The relation between disinhibition and emotion regulation in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child and Adolescent Psychology*, 33(4), 772–782. doi:10.1207/s15374424jccp3304_12

- Warren, S. L., Crocker, L. D., Spielberg, J. M., Engels, A. S., Banich, M. T., Sutton, B. P., ... Heller, W. (2013). Cortical organization of inhibition-related functions and modulation by psychopathology. *Frontiers in Human Neuroscience*, 7(271), 1–13.
doi:10.3389/fnhum.2013.00271
- Webb, C. A., Dillon, D. G., Pechtel, P., Goer, F. K., Murray, L., Huys, Q. J., ... Pizzagalli, D. A. (2016). Neural correlates of three promising endophenotypes of depression: Evidence from the EMBARC study. *Neuropsychopharmacology*, 41(2), 454–463.
doi:10.1038/npp.2015.165
- Weissman, D. H., Roberts, K. C., Visscher, K. M., & Woldorff, M. G. (2006). The neural bases of momentary lapses in attention. *Nature Neuroscience*, 9(7), 971–978.
doi:10.1038/nn1727
- Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (1993). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. *Psychophysiology*, 30(1), 82–89. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/8416065>
- Wiedemann, G., Pauli, P., Dengler, W., Lutzenberger, W., Birbaumer, N., & Buchkremer, G. (1999). Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Archives of General Psychiatry*, 56, 78–84. Retrieved from <http://archpsyc.jamanetwork.com/article.aspx?articleid=204589>
- Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry*, 57(11), 1336–1346.
doi:10.1016/j.biopsych.2005.02.006
- Willenbockel, V., Sadr, J., Fiset, D., Horne, G. O., Gosselin, F. & Tanaka, J. W. (2010). Controlling low-level image properties: The SHINE toolbox. *Behavior Research Methods*, 42(3), 671–684. doi:10.3758/BRM.42.3.671
- Willner, P., Scheel-Krüger, J., & Belzung, C. (2013). The neurobiology of depression and antidepressant action. *Neuroscience and Biobehavioral Reviews*, 37(10), 2331–2371.
doi:10.1016/j.neubiorev.2012.12.007
- Wilson, G. F., & Eggemeier, F. T. (1991). Psychophysiological assessment of workload in multi-task environments. In D. L. Damos (Ed.), *Multiple-task performance* (pp. 329–360). London: Taylor & Francis.
- World Health Organisation (2013). Mental Health Action Plan 2013–2020. Retrieved from http://apps.who.int/iris/bitstream/10665/89966/1/9789241506021_eng.pdf

- World Health Organization (2016). Mental Disorders (Fact Sheet). Retrieved from <http://www.who.int/mediacentre/factsheets/fs396/en/>
- Yates, A., Ashwin, C., & Fox, E. (2010). Does emotion processing require attention? The effects of fear conditioning and perceptual load. *Emotion, 10*(6), 822–830. doi:10.1037/a0020325
- Yiend, J. (2010). The effects of emotion on attention: A review of attentional processing of emotional information. *Cognition & Emotion, 24*(1), 3–47. doi:10.1080/02699930903205698
- Zetsche, U., & Joormann, J. (2011). Components of interference control predict depressive symptoms and rumination cross-sectionally and at six months follow-up. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(1), 65–73. doi:10.1016/j.jbtep.2010.06.001

Appendix A

Evaluator Script

Counting Task

Note. Italicised text indicates instructions for the evaluator. Sections in parentheses were added in Study 2.

“Hello. I’m Dr. [surname] and I am a psychologist. I am here to assess your verbal intelligence and non-verbal behaviour. You will now complete a working memory task. You are required to count backwards from 2023 in steps of 17. You must count out loud. Your performance will be timed and if you make a mistake, I will tell you to restart. (Also, if you count too slowly, I will tell you to restart.) Therefore, it is vital that you count as quickly as possible without making any errors. I will be observing your behaviour while you do this task, so it’s important that you are facing me and the camera. Remember to keep as still as possible to keep the recording signal clean. Do you understand?” *Clarify if needed.*

“Start counting now from 2023.” *Start stopwatch and look intently at the participant, maintaining eye contact whenever possible. Cross off each number as they say it. If they make a mistake, say:*

“Stop. That was an error. Start again from 2023.” *Make a point of stopping and restarting the stopwatch, noting the timing and that a restart was made on your clipboard. If they are good at the task, are on-a-roll, or don’t seem to be trying, tell the participant to count faster (or to restart for counting too slowly). Use your judgement.*

“You need to count faster.”

(“Stop. You’re counting too slowly. Start again from 2023.”)

After 5 minutes (3 minutes), tell the participant to “Stop counting now”.

Speech Preparation Task (Study 2 Only)

“Your next task is to give a speech about your performance in the counting task. The speech should be three minutes long and you will have three¹¹ minutes to prepare. We will be recording while you prepare so you’ll need to prepare silently in your head and keep as still as possible. You should reflect on your performance in the task.

“Think about how you performed - how well you counted and what other behaviours you might have exhibited. You should speak about how you think these factors might relate to other aspects of your life – for example, the kind of person you are, your university performance and your future life success. You might also speak about how you think your

¹¹ Participants were in fact given two minutes to prepare, due to time constraints.

performance compared to other participants'. You will need to talk for three minutes, so it's important that you use the preparation time to think carefully about your performance.

"I'll come back in after three minutes and you'll do the speech to me and to the video camera. After that, you'll review the video recordings of the previous participant and make some ratings on their performance. The next participant will do the same with your recordings. Remember to keep as still as possible and please keep facing the video camera.

"OK? You can start preparing now."

Appendix B

Mean Emotional Distraction (with Standard Deviations) for Different Distractor Frequencies
from Previous Research in Our Laboratory

Distractor Frequency ^a	n ^b	Emotional Distraction (ms) ^c
Experiment 1		
50%	36 (positive), 40 (negative)	29 (78)
Experiment 2		
25%	48	40 (60)
75%	48	14 (37)
Experiment 3		
25%	40	21 (50)
75%	41	2 (38)

Note. Effects were calculated from data submitted for publication (Grimshaw, Kranz, Carmel, Moody, & Devue, 2016).

^aDistractor frequency was manipulated between subjects.

^bSample size for each between-subjects condition.

^cEmotional distraction is the mean difference (and standard deviation of the paired difference) between emotional distraction (collapsed across positive and negative blocks) and neutral distraction difference scores. Difference scores were calculated by subtracting the distractor-present RTs from distractor-absent RTs.

Appendix C

Depression, Anxiety, and Stress Symptom Questionnaire (DASS; Lovibond & Lovibond, 1995)

Please read each statement and click 0, 1, 2 or 3, whichever indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 = Did not apply to me at all

1 = Applied to me to some degree, or some of the time

2 = Applied to me to a considerable degree, or a good part of the time

3 = Applied to me very much, or most of the time

1. I found myself getting upset by quite trivial things

2. I was aware of dryness of my mouth

3. I couldn't seem to experience any positive feeling at all

4. I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)

5. I just couldn't seem to get going

6. I tended to over-react to situations

7. I had a feeling of shakiness (e.g., legs going to give way)

8. I found it difficult to relax

9. I found myself in situations that made me so anxious I was most relieved when they ended

10. I felt that I had nothing to look forward to

11. I found myself getting upset rather easily

12. I felt that I was using a lot of nervous energy

13. I felt sad and depressed

14. I found myself getting impatient when I was delayed in any way (e.g., lifts, traffic lights, being kept waiting)

15. I had a feeling of faintness

16. I felt that I had lost interest in just about everything

17. I felt I wasn't worth much as a person

18. I felt that I was rather touchy

19. I perspired noticeably (e.g., hands sweaty) in the absence of high temperatures or physical exertion

20. I felt scared without any good reason

21. I felt that life wasn't worthwhile

22. I found it hard to wind down

23. I had difficulty in swallowing

24. I couldn't seem to get any enjoyment out of the things I did

25. I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)

26. I felt down-hearted and blue

27. I found that I was very irritable

28. I felt I was close to panic

29. I found it hard to calm down after something upset me

30. I feared that I would be "thrown" by some trivial but unfamiliar task

31. I was unable to become enthusiastic about anything

32. I found it difficult to tolerate interruptions to what I was doing

33. I was in a state of nervous tension

34. I felt I was pretty worthless

35. I was intolerant of anything that kept me from getting on with what I was doing

36. I felt terrified

37. I could see nothing in the future to be hopeful about

38. I felt that life was meaningless

39. I found myself getting agitated

40. I was worried about situations in which I might panic and make a fool of myself

41. I experienced trembling (e.g., in the hands)

42. I found it difficult to work up the initiative to do things

Note. Depression subscale items are in bold.

Appendix D

Mini Mood and Anxiety Symptom Questionnaire (Mini-MASQ; Clark & Watson, 1995)

You will now see a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then click on the number that best describes how much you have felt or experienced things this way during the past week, including today. Use this scale when answering:

- 1 = not at all
- 2 = a little bit
- 3 = moderately
- 4 = quite a bit
- 5 = extremely

- 1. Felt really happy
- 2. Felt tense or “high strung”
- 3. Felt depressed
- 4. Was short of breath**
- 5. Felt withdrawn from other people
- 6. Felt dizzy or lightheaded**
- 7. Felt hopeless
- 8. Hands were cold or sweaty**
- 9. Felt like I had a lot to look forward to
- 10. Hands were shaky**
- 11. Felt like nothing was very enjoyable
- 12. Felt keyed up, “on edge”
- 13. Felt worthless
- 14. Had trouble swallowing**
- 15. Felt like I had a lot of interesting things to do
- 16. Had hot or cold spells**
- 17. Felt like a failure
- 18. Felt like I was choking**
- 19. Felt really lively, “up”
- 20. Felt uneasy

21. Felt discouraged

22. Muscles twitched or trembled

23. Felt like I had a lot of energy

24. Was trembling or shaking

25. Felt like I was having a lot of fun

26. Had a very dry mouth

Note: Anxious Arousal subscale items are in bold.

Appendix E

State-Trait Anxiety Inventory – Trait Form Y-2 (STAI-T; Spielberger & Gorsuch, Lushene, Vagg, & Jacobs, 1983)

You will now read a number of statements that people have used to describe themselves. Read each statement and then click the appropriate number to indicate how you *generally* feel.

There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

1 = almost never

2 = sometimes

3 = often

4 = almost always

I feel pleasant

I feel nervous and restless

I feel satisfied with myself

I wish I could be as happy as others seem to be

I feel like a failure

I feel rested

I am “calm, cool, and collected”

I feel that difficulties are piling up so that I cannot overcome them

I worry too much over something that really doesn't matter

I am happy

I have disturbing thoughts

I lack self-confidence

I feel secure

I make decisions easily

I feel inadequate

I am content

Some unimportant thought runs through my mind and bothers me

I take disappointments so keenly that I can't put them out of my mind

I am a steady person

I get in a state of tension or turmoil as I think over my recent concerns and interests