The Social Consequences of Defensive Physiological States

Submitted by Megan Christina Barnsley to the University of Exeter
as a thesis for the degree of
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degree by this or any other University.

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Abstract

This thesis examines the validity of polyvagal theory as a model of normal socio-emotional responding (Porges, 1995, 2001, 2003a). Polyvagal theory makes several claims, and to date many of its predictions lack empirical testing. In the current research, five main hypotheses stemming from polyvagal theory were identified and tested using healthy participants. The initial empirical study examined the influence of laboratory stressors on autonomic function. The findings revealed that social evaluative threat increases activation of the sympathetic nervous system more than a virtual reality maze, and that arousal remains elevated for longer during anticipation of social evaluative threat in comparison to recovery from social evaluative threat. The second study investigated the effects of emotion regulation strategies on autonomic function, and highlighted the effectiveness of two meditation practices in reducing defensive physiological arousal and increasing subjective positive emotion. These studies were followed with a set of studies designed to evaluate the effects of defensive physiological arousal on socio-emotional functioning, as a direct test of polyvagal theory. The first study examined the effects of a laboratory stressor on facial expressivity, revealing that social evaluative threat had little impact on expressive regulation. A second study investigated the effects of a laboratory stressor on emotional sensitivity and spontaneous facial mimicry. Some limited support was found for polyvagal theory, although neither emotional sensitivity nor facial mimicry was significantly affected by laboratory stress. A final empirical study investigated the effects of a laboratory stressor on affiliation tendencies. The laboratory stressor did not influence participants’ willingness to spend time with others, however the experiment did reveal significant relationships between markers of social safeness and affiliation. The overall conclusion of this thesis is that polyvagal may not be a representative model of socio-emotional functioning in healthy participants. The implications of these findings are discussed in relation to the validity of polyvagal theory as a universal model of socio-emotional responding.
Acknowledgements

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My friends and colleagues at the Mood Disorders Centre have always offered invaluable support and advice, and my experience of the PhD wouldn’t have been the same without them. Same goes for my friends outside of academia, who have often been a welcome distraction from the PhD.

The encouragement from my family has been unwavering, and I would like to express my love and gratitude to them for their patience and support.

Finally, to Olly, who was there at the beginning, and there at the end – I couldn’t have done it without you.
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<tbody>
<tr>
<td>AAQ-II</td>
<td>Acceptance and Action Questionnaire – Revised</td>
</tr>
<tr>
<td>AN</td>
<td>anorexia nervosa</td>
</tr>
<tr>
<td>ANS</td>
<td>autonomic nervous system</td>
</tr>
<tr>
<td>BAS</td>
<td>behavioural activation system</td>
</tr>
<tr>
<td>BDI-II</td>
<td>Beck Depression Inventory – Second Edition</td>
</tr>
<tr>
<td>BEQ</td>
<td>Berkeley Expressivity Questionnaire</td>
</tr>
<tr>
<td>BIS</td>
<td>behavioural inhibition system</td>
</tr>
<tr>
<td>BPD</td>
<td>borderline personality disorder</td>
</tr>
<tr>
<td>bpm</td>
<td>beats per minute</td>
</tr>
<tr>
<td>DERS</td>
<td>Difficulties in Emotion Regulation Questionnaire</td>
</tr>
<tr>
<td>DSM-IV-TR</td>
<td>The Diagnostic and Statistical Manual of Mental Disorders (4th ed. Text revised)</td>
</tr>
<tr>
<td>DVC</td>
<td>dorsal vagal complex</td>
</tr>
<tr>
<td>ECG</td>
<td>electrocardiogram</td>
</tr>
<tr>
<td>EMG</td>
<td>electromyography</td>
</tr>
<tr>
<td>ER</td>
<td>Emotion Recognition Task</td>
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<td>FFFS</td>
<td>fight–flight–freeze system</td>
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<tr>
<td>GAD</td>
<td>generalised anxiety disorder</td>
</tr>
<tr>
<td>GAD-7</td>
<td>Generalised Anxiety Disorder Scale</td>
</tr>
<tr>
<td>HF</td>
<td>high-frequency</td>
</tr>
<tr>
<td>HF-HRV</td>
<td>high-frequency heart rate variability</td>
</tr>
<tr>
<td>HR</td>
<td>heart rate</td>
</tr>
<tr>
<td>HRV</td>
<td>heart rate variability</td>
</tr>
<tr>
<td>Hz</td>
<td>hertz</td>
</tr>
<tr>
<td>IBI</td>
<td>interbeat interval</td>
</tr>
<tr>
<td>LF</td>
<td>low frequency</td>
</tr>
<tr>
<td>MDD</td>
<td>major depressive disorder</td>
</tr>
<tr>
<td>ms²</td>
<td>milliseconds squared</td>
</tr>
<tr>
<td>PD</td>
<td>panic disorder</td>
</tr>
<tr>
<td>PNS</td>
<td>parasympathetic nervous system</td>
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<tr>
<td>POMS-SF</td>
<td>Profile of Mood States – Short Form</td>
</tr>
<tr>
<td>PTSD</td>
<td>post-traumatic stress disorder</td>
</tr>
<tr>
<td>RSA</td>
<td>respiratory sinus arrhythmia</td>
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<tr>
<td>SCL</td>
<td>skin conductance level</td>
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<tr>
<td>SNS</td>
<td>sympathetic nervous system</td>
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<tr>
<td>SSPS</td>
<td>Social Safeness and Pleasure Scale</td>
</tr>
<tr>
<td>T1</td>
<td>time 1</td>
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<tr>
<td>T2</td>
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</tr>
<tr>
<td>TRD</td>
<td>treatment-resistant depression</td>
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<tr>
<td>VNS</td>
<td>vagal nerve stimulation</td>
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<tr>
<td>VR</td>
<td>virtual reality</td>
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<tr>
<td>VVC</td>
<td>ventral vagal complex</td>
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<tr>
<td>µS</td>
<td>micro-Siemens</td>
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Chapter 1: The Social Consequences of Defensive Physiological States: 
An Introduction

It has long been established that the body plays an important role in socio-emotional responding. Emotions are multifaceted phenomena, and how we feel is intrinsically linked to our bodily state and how we behave (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Models linking physiological states with emotional responding have been emerging for several years, and one of the most recent models to be put forward is polyvagal theory (Porges, 1995, 2001, 2003a). Polyvagal theory suggests that complex cognitions and behaviours are contingent on the functioning of the autonomic nervous system. Polyvagal theory conjectures that the parasympathetic pathways that control the viscera are neuroanatomically connected to the neural pathways that regulate the muscles of the face head. The result of this connection is that social engagement behaviours (such as facial expressivity and social awareness) are withdrawn in environmental situations that signal threat or challenge. Although polyvagal theory has some promising hypotheses, much of the supporting arguments are theoretical and the theory has limited empirical support. This thesis set out to explore the social consequences of defensive physiological states.

The literature review in Chapter 2 commences with an overview of mind–body interaction models, and polyvagal theory (Porges, 1995, 2001, 2003a) is singled out as a peripheral model of emotional responding that requires verification. The review highlights that there are theoretical issues surrounding polyvagal theory’s three modes of responding (calm and self-soothing, mobilisation with fear, and immobilisation). The review also identifies that Porges’ claims regarding the links between social engagement and physiological state are lacking empirical support. Chapter 3 discusses and addresses methodological issues that are relevant to the empirical testing of polyvagal theory. The chapter considers several facets of emotional responding, and emphasises the need for a broad range of methodologies in the following empirical chapters.

To empirically test polyvagal theory we used experimental designs to measure different forms of social responding whilst manipulating the physiological state of healthy participants. Chapter 4 presents an initial set of experiments that explored and addressed the methodological issues relevant to manipulating physiological state.
Experiment 1 compared two stressor manipulations and found a speech task to be a superior stressor manipulation compared to a virtual reality maze. This was followed by a second experiment, which demonstrated the utility of using the anticipation period of speech tasks to prolong defensive physiological arousal (Feldman, Cohen, Hamrick, & Lepore, 2004; Gregg, James, Matyas, & Thorsteinsson, 1999). Specifically, defensive arousal was found to remain elevated for longer during a five-minute rest period before the speech (anticipation) compared to a five-minute rest period after the speech (recovery). After confirming that the speech task was able to increase defensive physiological arousal, it was reasoned that the speech task could be used to investigate hypotheses arising from polyvagal theory; namely that increased activation of the parasympathetic nervous system should be associated with down-regulation of the sympathetic nervous system, whilst increased activation of the sympathetic nervous system should be associated with deficits in socio-emotional responding.

To investigate the role of the parasympathetic nervous system in emotional responding, Chapter 5 was designed to investigate the effects of emotion regulation strategies on physiological return to baseline and self-reported distress. The focus of the study is on strategies that target the activation of the parasympathetic nervous system as well as positive affect. Participants prepared a three-minute speech to induce defensive physiological arousal before carrying out an emotion regulation strategy in response. Participants were given one of five strategies: smiling, mindful breathing, a loving-kindness meditation, neutral listening, or resting quietly (control condition). The most notable physiological effects were observed in the mindful breathing and neutral listening groups, whilst the loving-kindness meditation was associated with the highest level of positive affect.

Having demonstrated the utility of anticipatory stress and the effects of regulatory strategies in facilitating down-regulation of arousal, exploration of the social consequences of defensive arousal commences in Chapter 6. This chapter describes the findings of an investigation into the influence of defensive physiological arousal on expressive ability. Participants were filmed whilst being instructed to modulate their facial expressions (enhance, maintain, or suppress instructions; Bonanno, Papa, Lalande, Westphal, & Coifman, 2004). One block of the expressive regulation task was presented at baseline, whilst the other block was presented after a stressor manipulation: Half of the participants completed the speech task manipulation; the other half
completed a reading (control) manipulation. Analyses were conducted to investigate the
effects of defensive physiological arousal on expressive ability, however no significant
differences were found within or across the groups. It was concluded that increased
activation of the sympathetic nervous system might affect spontaneous expression of
emotion, but not consciously mediated displays of emotion.

Chapter 7 examined the influence of defensive physiological arousal on
emotional sensitivity (i.e., the ability to recognise emotions in others). The chapter
reports the findings of two experiments. In Experiment 1 a similar design to the study in
Chapter 6 was utilised: Participants carried out an emotion recognition task in two
blocks with the speech task used as a mid-point stressor manipulation. In this chapter,
electromyography was used to measure spontaneous facial mimicry (Dimberg, 1982).
No significant differences were found within or across the groups, possibly due to
limitations of the stressor manipulation. Experiment 2 adapted the design of the speech
task by splitting the stressor manipulation into three one-minute blocks that were
interspersed with the second half of the emotion recognition task. The adapted speech
task was able to maintain arousal in the stressor group for longer during the second
administration of the emotion recognition task, however this did not result in any
measurable changes in emotional sensitivity or facial expressivity.

A final empirical chapter (Chapter 8) explored the influence of facial
expressivity on others’ willingness to affiliate. Experiment 1 found evidence to support
Porges’ (2003a) claim that facial expressivity is important in interpersonal interactions.
Participants viewed videos of individuals regulating their facial expressions and
indicated that they would be more willing to spend time with regulators displaying
greater levels of emotion, particularly if they expressed positive emotion. As well as
affecting subjective ratings, displays of emotion were also associated with significant
changes in the observers’ heart rate. Interestingly, individuals’ willingness to affiliate
with others was found to be related to their self-reported social safeness as well as their
baseline high-frequency heart variability. These relationships provide some support for
Porges’ (2009a) argument that social interactions are dependent on environments being
perceived as safe. Experiment 2 investigated whether observers’ willingness to affiliate
could be influenced by defensive physiological arousal. Participants again viewed
videos of individuals regulating their facial expressions, with the adapted speech task
used as a stressor manipulation during the second half of the task. The findings did not
support the hypothesis that increased activation of the sympathetic nervous system is associated with decreased affiliation.

Several conclusions can be drawn from the empirical chapters of this thesis. Most importantly there is not a clear link between defensive physiological arousal and socio-emotional functioning in healthy control participants. The final chapter (Chapter 9) provides a general overview of the main findings of the thesis. It discusses the wider implications of the current findings in the context of emotion research. The chapter also considers the implications of the empirical findings in terms of the validity of polyvagal theory.
Chapter 2: The Autonomic Nervous System and Emotional Responding

Broadly defined, emotions guide action and organise behaviour towards salient goals (Davidson & Irwin, 1999). The presence of an emotion is linked to changes in subjective experience, appraisal, expression, physiological arousal, and goal-directed behaviour and involves several processes: The perception of an emotional stimulus, the production of an affective state and emotional behaviour, and the regulation of the affective state and emotional behaviour (Phillips, Drevets, Rauch, & Lane, 2003). When emotional stimuli are perceived, messages are sent from the central nervous system to the rest of the body, either by nerve cells or chemical hormones, generating changes in autonomic, neuroendocrine, and somatomotor systems. Importantly, these routes of communication are bidirectional creating a feedback loop between the central nervous system and the rest of the body allowing the organism to continuously adapt to the changing demands of the environment (Hugdahl, 1996; Porges, 2003a). The presence of an emotion is generally a private experience unless behavioural (e.g., facial, vocal, postural) changes associated with the experience of the emotion are expressed (Gross & John, 1995). Behaviourally expressing an emotion signals to others that an emotion is occurring, making emotional expression an important part of social interactions (Darwin, 1872/2009). In this chapter I provide an overview of how the mind and body interact to shape how we experience and express our emotions.

2.1. Integration of the Mind and Body

It has long been debated as to whether emotional experiences are shaped by the mind influencing the body, or the body influencing the mind (see Dalgleish, Dunn, & Mobbs, 2009, for a review). William James (1884) put forward the controversial argument that emotional experiences arise from direct perceptions of bodily change; we do not run because we are frightened, we are frightened because we run. The idea that an emotional experience does not start with a conscious experience but our experience of bodily changes was shared by Carl Lange (1885). Both James and Lange believed that bodily and behavioural responses precede the conscious experience of emotion, resulting in what it today known as the James–Lange theory of emotion. Walter B. Cannon (1927) critiqued the James–Lange theory arguing that visceral changes do not always result in the presence of an emotion. Cannon put forward evidence that contrasted with the claims of James and Lange, for example surgical separation of the
viscera from the body was shown not to attenuate emotional behaviour in animals, and Cannon reasoned that the range of visceral changes in the body resulted in too little differentiation to explain the range and variety of emotions experienced. As reviewed in Dalgleish et al. (2009), recent work has questioned Cannon’s claims with evidence suggesting that emotional responses may at least in part be distinguished on the basis of patterns of autonomic activity (Ekman, Levenson, & Friesen, 1983), that separation of the body from the brain can in fact reduce the intensity of emotional experience (e.g., following spinal injury, Montoya & Schandry, 1994), and that artificial stimulation of the viscera (e.g., via intravenous injection of peptides) can induce emotions (Harro & Vasar, 1991). The dissonance between the James–Lange theory of emotion and Cannon’s critique raised important questions in the field, and this longstanding debate is still relevant to our current understanding of how we experience emotions. I would argue that a major contribution of James, Lange, and Cannon, is that it is now generally accepted that both the mind and the body play important roles in shaping the emotional experience.

Mind–body research is inherently complicated because even when researchers attempt to isolate the brain from the body using separation designs, the emotional experience may still being driven by remaining peripheral feedback mechanisms (Heims, Critchley, Dolan, Mathias, & Cipolotti, 2004). Current researchers are aware of this limitation, so rather than isolate the origins of an emotional response to just the brain or body, a hybrid position is taken. The body is thought to signal a basic sense of emotional intensity to the brain, which is cognitively appraised to lead to a more nuanced experience (Dalgleish et al., 2009). Schachter and Singer (1962) provided key evidence for this two-factor theory of emotion by demonstrating that the emotional experience is dependent not only on the presence of bodily arousal, but also the cognitive expectation of why the arousal has occurred. Consequently when conducting emotion research it is essential to consider the how the body communicates with the brain to produce an emotional experience. Emotion is not a purely cognitive phenomena and the role of the body in generating the experience of an emotion needs to be emphasised.
2.2. The Autonomic Nervous System

The mind and body are linked by a complex network of neurons and synapses. Conventional models of the human nervous system group the neural networks of the brain and spinal cord together to form the central nervous system (CNS; Jessell, 1995). This system is responsible for processing signals sent to and from the rest of the body. The brain is often considered the central organ of behaviour as it can exert a top-down influence on many aspects of behaviour, allowing many of the body’s functions to be consciously controlled (Cameron, 2009; Jessell, 1995). Any nervous tissue lying outside of the brain and spinal cord is referred to as the peripheral nervous system. This second system carries motor and sensory information from the brain to the body and then relays information back to the CNS. Although the peripheral nervous system and CNS are theorised as being anatomically separate, they are functionally interconnected (Jessell, 1995). The peripheral nervous system has been further divided into the somatic system, which controls muscular activities, and the autonomic nervous system (ANS), which controls the viscera, glands, and sensory systems of the body (Sequeira, Hot, Silvert, & Delplanque, 2009). Most of the divisions alluded to have been established on the basis of anatomy, neuropharmacology, and function, which has led to some contention over the years (Blessing, 1997; Jänig, 2006).

Distinctions between the ANS and CNS are mainly due to historical conceptions about their anatomy and function; in reality the ANS and CNS interact to such an extent it is difficult to separate them from one another, although many still talk of them as though they are independent (van Toller, 1979). Current understanding suggests that the CNS and ANS are constituted of the same neural elements, however the arbitrary divisions made between “central” and “peripheral” physiology has diverted attention away from the role of the ANS in understanding emotional behaviour (Blessing, 1997). It is important to establish how the ANS controls the viscera of the body because the physiological changes that occur during an emotional response are mostly generated by this system. It has been argued that all physiological and behavioural events, and their associated affective experiences, can be explained in terms of neural circuitry, making the ANS a suitable focus for emotion research (Blessing, 1997).

The role of the ANS is to regulate and coordinate bodily activities such as respiration rate, heart rate, and digestion, many of which are usually without conscious
control. The CNS however, can control, inhibit, or bypass lower reflex mechanisms of the ANS via activity in areas such as the hypothalamus, amygdala, and prefrontal cortex (Jessell, 1995). On this basis, Blessing (1997) has criticised the used of the term “autonomic nervous system”, as this implies that the ANS acts independently of the CNS. In actual fact, specific patterns of neural outflow to the cardiovascular and visceral organs originate almost exclusively within the brain. To resolve this issue it has been suggested that the term ‘autonomic’ should be abandoned, and that instead researchers should refer terms such as efferent and afferent visceral neurons (Blessing, 1997). Although this argument has its merits, for ease of communication I will retain the use of the term autonomic nervous system in this thesis.

Because of the ANS, the human body is able to quickly alter its internal state to meet the demands of the external environment. This is extremely important when survival relies on the ability to quickly identify and appropriately respond to environmental threats and rewards (Darwin, 1872/2009; Porges, 2009b). The ANS innervates every organ in the body and has two divisions, the sympathetic branch and the parasympathetic branch, as can be seen in figure 2.1. The sympathetic nervous system (SNS) is generally a catabolic system that expends energy and prepares the body for fight–flight behaviours, resulting in changes such as increased heart rate, increased sweating, and increased blood flow to skeletal muscle, as well as the inhibition of the digestive system (Cannon, 1929). This system is often activated when a threat/stressor is encountered. Most of the nerve fibres for the SNS originate in the thoracic and lumbar segments of the spinal cord (i.e., the middle and lower back regions of the spine) and project a short way from the spinal cord into bundles of fibres that along the spinal vertebrae known as the sympathetic chain ganglia. The preganglionic fibres of the SNS are relatively short whereas the postganglionic fibres that project from the sympathetic chain to the effector organs are much longer. Innervation of the adrenal medulla by the SNS releases the catecholamines adrenaline and noradrenaline into the bloodstream (Parkinson, 1990), therefore the SNS is often conceptualised as having a diffuse effect throughout the body (as noted by Cannon, 1929; but see Gebber, 1990; and Morrison, 2001).

**Figure 2.1.** The autonomic nervous system: Parasympathetic and sympathetic divisions (from Morris & Maisto, 2001, p. 72).

The *parasympathetic nervous system* (PNS) which makes up the other half of the ANS is generally anabolic, promoting the conservation of energy and a state of rest and digest (Cannon, 1929). For example this system tends to activate the digestive tract whilst reducing heart rate. Most of the PNS nerve fibres originate in the cranial or sacral regions of the spinal cord (i.e., the very top and bottom regions of the spine), including the vagus nerve which originates from nuclei in the brainstem and plays a major role in the PNS. Unlike in the SNS, parasympathetic ganglia tend to be found in or near the innervated regions, allowing the activity of the PNS to be localised and specific. Also the PNS is characterised by the chemical acetylcholine rather than the catecholamines adrenaline and noradrenaline.

At first, the division of the SNS and PNS appears straightforward, however the systems interact in highly complex ways to produce resultant changes in physiology. Whilst the SNS tends to have an activating effect and the PNS tends to have an inhibitory effect, the two systems are not simply antagonistic as is often reported. The two systems can be co-activated, uncoupled, or reciprocal in their effects (Berntson, Cacioppo, & Quigley, 1991; Jänig, 2003). Following this understanding, it is recognised that both systems will function to adjust the body’s physiological response to meet the specific demands of the environment (Blessing, 1997). It should also be emphasised that the two systems can be very specific in their effects. Cannon's (1929) original conception of the SNS suggested that the system was functionally homogeneous in its
effects (i.e., components exhibited uniform increases or decreases in activity). This line of reasoning arose because the SNS was shown to innervate targets organs that are widely distributed, such as the blood vessels in skeletal muscle, skin, and viscera, as well as sweat glands (Jänig, 2006). However, contemporary research has established that sympathetic nerves innervating target organs can be non-uniformly changed, leading to complex visceral response patterns (Gebber, 1990; Morrison, 2001). The same specificity can also be applied the PNS (Jänig, 2006). Due to the existence of the sympathetic and parasympathetic nervous systems and their various modes of control, there is great flexibility and precision in the body’s ability to adjust internal states to meet external demands. This flexibility and precision can be easily demonstrated by looking at how the ANS controls the function of the heart.

2.3. The Heart and Emotional Responding

The heart is considered to be a sensitive index of emotional responding because of its role in regulating the somatic and visceral functions of the body. The heart is intimately involved in all aspects of emotional responding and behaviour, as most bodily functions are likely to cause adjustments in the cardiovascular system (Obrist, Webb, Sutterer, & Howard, 1970). The reason that the heart is so responsive to changes in internal and external demands is that it facilitates the preparation and performance of most behavioural responses (Schwartz, 1982). In addition to this, unlike most other visceral organs, the heart is also innervated by both the SNS and PNS, making it responsive to both branches of the ANS (Jänig, 2006).

The heart supplies the cells of the body with oxygen and nutrients via the blood. The rate at which the heart pumps blood around the body is generated by the sinoatrial node, the natural pacemaker of the heart. The rate of contraction is highly responsive to signals from both the ANS and the CNS. As mentioned previously the SNS acts to increase the amount of blood pumped around the body via the release of noradrenaline, which increases the excitability of the heart tissue and increases the force of contraction (Andreassi, 1989). Conversely the PNS inhibits the activity of the sinoatrial node, slowing heart rate. Under non-challenging conditions the heart is predominantly controlled by the PNS, so the heart beats at a slower rate than the pace of the sinoatrial node. Withdrawal of the vagus nerve (influence of the PNS) from the heart causes the heart’s rate of contraction to rapidly increase allowing for a rapid shift in behaviour.
Research has shown that parasympathetic-mediated alterations in heart rate occur much faster than those mediated by the sympathetic branch of the nervous system, so withdrawing the vagus nerve has a quicker effect on the heart than activating the SNS (Berntson, Cacioppo, & Quigley, 1993a). Such withdrawal is likely to occur if a threat is encountered, allowing the body to prepare for a fight–flight response.

Although measures of heart rate (e.g., beats per minute) can be useful indices of cardiovascular reactivity, changes in the activity of the heart occur on a beat-to-beat basis. Measures of heart rate average out the variability of the heart’s activity over a period of time, and this means that heart rate cannot be used to differentiate between the influences of the SNS and the PNS. To illustrate this point, increases detected in heart rate can be attributed to decreased PNS activity, increased SNS activity, or a combination of the two, however it is impossible to identify the source of the changes in activity from heart rate alone. Alternative methodologies can be used to resolve this issue, for example heart rate variability (HRV) and pre-ejection period (PEP) monitor changes in the heart’s activity on a beat-to-beat basis and respectively index activation of the PNS and SNS (Cacioppo, Uchino, & Berntson, 1994). Deconstruction of these measures allows for the isolation of changes in the activity of the heart due to parasympathetic effects, sympathetic effects, or a combination of both.

In recent times, HRV has been identified as a marker of psychological (Thayer, Hansen, Saus-Rose, & Johnsen, 2009) and physiological health (Thayer & Lane, 2007). Activation of the vagus nerve – a major determinant of HRV – has been shown to influence physiological and emotional responses to acute stressors (Appelhans & Luecken, 2006). As mentioned previously, external demands on the ANS can elicit a state of fight or flight (characterised by the SNS), or a state of rest and digest (characterised by the PNS). When stressors are encountered and a fight–flight response is adopted, the power in the low-frequency domain of HRV tends to increase whilst the high-frequency power decreases (Berntson & Cacioppo, 2004). In contrast, increases in the high-frequency component of HRV represent an increase in parasympathetic activity, which is linked with the promotion of rest and digest behaviours. In the longer term, overall reductions in HRV and autonomic imbalance (when one branch of the ANS dominates over the other) are both thought to contribute to premature ageing and disease. Low HRV has been identified as a prognostic marker for cardiovascular diseases, such as hypertension and myocardial infarction, as well as psychiatric...
disorders such as depression and anxiety (Berntson & Cacioppo, 2004; Thayer & Friedman, 1997). Consequently patterns in HRV and their autonomic counterparts have significance not just for short-term emotional responding, but also longer-term health and disease.

The contribution of the heart to emotional responding, and the ability for researchers to determine the influences of the SNS and PNS on the heart, has made the function of the heart a popular component of psychophysiological research. Models have begun to integrate the functions of the mind and the body using the heart as their main measure of autonomic functioning. Although these models are commendable, some researchers criticise this focus: The heart is only one component of the ANS, and it is not necessarily the most important or central one (Berntson, Cacioppo, & Grossman, 2007). Associations between the heart and other measures of psychophysiological responding also do not imply causation, and it is important to recognise this when interpreting models that integrate the mind and body.

2.4. Models of Mind–Body Interaction

For the purpose of this review I am going to focus on two models of mind–body interactions: The model of neurovisceral integration (Thayer & Lane, 2000) and polyvagal theory (Porges, 1995, 2001, 2003, 2007). Both models highlight the interactions that occur between the CNS and ANS, and as they are neurobiologically grounded models, they emphasise the influence of the body’s neural systems on emotional responding. The models acknowledge that dysfunction of the pathways between the brain, autonomic, neuroendocrine, and somatomotor systems, plays a key role in the development and maintenance of psychiatric disorders. The research that has resulted from both models has provided a better understanding of the deficits caused by the dysfunction of such systems, and has also aided the development of intervention and treatment strategies to help return the systems to levels of optimal functioning.

2.4.1. Neurovisceral Integration

Researchers are increasingly emphasising the importance of neurobiologically based systems that help coordinate emotional responding and social behaviour. Thayer and Lane (2000, 2009) proposed a general model of neurovisceral integration, which
recognises the interactions between the brain, visceromotor, neuroendocrine, and behavioural responses, that function to allow the body to respond rapidly to environmental challenges. Thayer and Lane’s (2000, 2009) model is centred on the function of the central autonomic network (CAN); a network of brain structures implicated in goal-directed behaviour and adaptability (Benarroch, 1993; Thayer & Brosschot, 2005). The CAN is comprised of the anterior cingulate, the insula, the ventromedial prefrontal cortices, the amygdala, the hypothalamus, the periaqueductal gray, the parabrachial nucleus, the nucleus of solitary tract, the nucleus ambiguus, the ventrolateral medulla, the ventromedial medulla, and the medullary tegmental field. These brain areas are intimately linked to emotional responding, and together they allow for tonic and reflexive control of autonomic functions (Benarroch, 1993). The prefrontal cortex is proposed to exert a top-down effect on the subcortical structures and play an important role integrating signals from the brain and the body.

There are two structures within the CAN that specifically relate to the current research: The ventromedial prefrontal cortex (vmPFC) and the dorsal anterior cingulate cortex (dACC). The vmPFC has been found to modulate the vagal efferent outflow to the heart (Wong, Massé, Kimmerly, Menon, & Shoemaker, 2007), whilst research in fear conditioning has shown that activation of the dACC and the vmPFC mediates fear expression in humans. Activation of the dACC promotes fear responses whilst activation of the vmPFC promotes safety (Milad, Quirk, et al., 2007; Milad, Wright, et al., 2007). In the neurovisceral integration model, activation in these regions signals the need for either a danger or safety response, which is communicated to the body and expressed via the autonomic nervous system (Thayer & Lane, 2000). The prefrontal cortices modulate the activation of the subcortical motivation circuits, with sympathoexcitatory circuits normally being under tonic inhibitory control (Thayer & Brosschot, 2005). This prefrontal inhibition is taken “offline” in the presence of uncertainty, novelty, and threat to let automatic, prepotent processes regulate behaviour (Thayer & Lane, 2000, 2009). A reduction in prefrontal activation results in a withdrawal of parasympathetic activation and an increase in sympathetic activation, which is consistent with defensive responding. Dysregulation of these cortical pathways may result in prolonged increases in sympathetic activation, which in the long term could result in potential autonomic imbalance. Prolonged action readiness and SNS over-activity have been linked to deficits in self-regulation and psychopathology (Thayer & Brosschot, 2005).
A positive correlation has been found between activation of the prefrontal cortices and vagally mediated HRV (Lane et al., 2009). The vagal component of HRV has also been related to the efficiency of physiological, cognitive, and emotional self-regulation (Segerstrom & Solberg Nes, 2007; Thayer & Brosschot, 2005; Thayer & Lane, 2009). Due to the direct links between the CAN and the vagal efferents to the heart, the model of neurovisceral integration suggests that HRV is a suitable marker of central–peripheral neural feedback and CNS–ANS integration (Thayer & Brosschot, 2005; Thayer & Lane, 2000, 2009).

2.4.2. Polyvagal Theory

Porges’ (1995, 2001, 2003a, 2007a) polyvagal theory emphasises how emotional responding has its roots based in the evolution of the mammalian ANS. The theory attempts to integrate physiology, behaviour, and psychosocial processes in a unified framework (Berntson et al., 2007). As discussed previously, the ANS is not a unified structure. Polyvagal theory suggests that different substrates of the ANS can provide an organising principle that illuminates the adaptive significance of affective processes (Porges, 1995). According to the theory, phylogenetic developments have resulted in neuroanatomical and neurophysiological links between the vagal regulation of the heart and the neural regulation of the striated muscles of the face and head (Porges, 1995, 2003a, 2007b). In other words the influence of the PNS on the heart is also related to the control of motor behaviours that are involved in social engagement (Porges, 2003a). Porges argues that social engagement is not a learned behaviour; it is an emergent behaviour of our neurophysiology. As a consequence socialisation is dependent on the body’s neuroregulatory state, which either inhibits or promotes the expression of social behaviours (Porges, 2009a).

The vagus, also known as the 10th cranial nerve, can be traced back to two origins within the brainstem, the nucleus ambiguus (NA) and the dorsal vagal motor nucleus (DVNX; Loewy & Spyer, 1990). These form parts of the ventral vagal complex (VVC) and dorsal vagal complex (DVC) respectively (Porges, 1995). Activation of the vagus nerve from either of these locations is parasympathetic, inhibiting the sinoatrial node to slow heart rate, however the effects of the two vagal branches are qualitatively and quantitatively different (Porges, 1995, 2001). It is proposed that in environments that are perceived as ‘safe’, the physiological state is largely dominated by activation of
the myelinated vagus nerve originating from the NA. This branch of the vagus nerve is the most recent evolutionary component of the polyvagal hierarchy, and is often referred to as the ‘newer’ vagus nerve. NA-mediated states occur through the VVC and are characterised by bradycardia, increased digestion, and are considered calming and self-soothing. This physiological state is linked to pro-social behaviours, with activation of a neural network defined as the social engagement system (Porges, 1998, 2001, 2003a).

![Figure 2.2](image)

Figure 2.2. The social engagement system (from Porges, 2003a).

The social engagement system (SES) consists of structures innervated by the cranial motor nerves V, VII, IX, X, and XI (see figure 2.2). During development these nerves originate in the embryonic branchial arches and project to and from the striated muscles of the face and head (Patestas & Gartner, 2006). Collectively they are known as special visceral efferent fibres and their function is to regulate the facial muscles (for facial expressivity), muscles of mastication (for ingestion), neck muscles (for looking behaviour), laryngeal and pharyngeal muscles (for vocalisation and intonation), as well as the middle ear muscles (for listening to human voice; Porges, 1995). These muscles are all involved in social engagement behaviours, such as maintaining eye contact, listening to speech, and making appropriate facial expressions. The source nuclei of these cranial motor nerves are located in the special visceral efferent column of the brainstem and are anatomically linked to the cardiac vagal fibres projecting from the NA (see figure 2.3). Consequently the motor nerves that control the muscles of the face and head communicate directly with the inhibitory neural system that slows heart rate, lowers blood pressure, and reduces arousal in order to produce calm states (Porges, 2003a). Porges hypothesises that due to this heart–face link in the brainstem, successful
social engagement is contingent on the calm and self-soothing physiological states, which are determined by activation of the myelinated vagus nerve (Porges, 2003b, 2009a; Porges & Lewis, 2010). According to polyvagal theory, if activation of the myelinated vagus is reduced or withdrawn the SES is down-regulated, and the individual’s ability to effectively socialise is compromised. This defensive physiological state is likely to arise if the individual is in a situation where they perceive their environment as ‘unsafe’.

Porges (2007b, 2009a) uses the term neuroception to describe how individuals evaluate the level of threat in their surrounding environment. To effectively move between autonomic substrates and alter one’s behaviour to meet the demands of the environment two processes need to occur: Firstly risk must be assessed; secondly if the environment is perceived as safe then structures that promote defensive behaviours such as fight, flight, or freeze must be inhibited. Neuroception occurs without conscious awareness and initiates a sequence of neural processes that facilitate adaptive defence behaviours when required. Consequently an individual may exhibit defensive behaviours without conscious awareness of the perceived threat.
In the presence of threatening stimuli vagal control of the heart is withdrawn. This withdrawal allows the organism to orient to the environment and assess the level of threat without having to initiate the SNS, which is metabolically demanding for the body (Alboni, Alboni, & Bertorelle, 2008). If the threat is only transitory vagal control of the heart can be restored. However if the threat is significant or fails to diminish, the SNS activates and promotes fight–flight behaviours. To recapitulate, an SNS response is one of mobilisation, characterised by increased heart rate, increased sweating, and decreased digestion (Cannon, 1929). During defensive states the body is driven mainly by sympathetic activation and vagal activity is reduced. This state prepares the body to either challenge the threat (fight) or attempt to escape from the threat (flight). Although adaptive in allowing the body to deal with potential threats, the reduction of vagal activity is accompanied by deactivation of the SES, which limits the individual’s ability to socially engage (Porges, 2001, 2003a).

In situations of extreme threat, when environmental stimuli may be perceived as ‘life threatening’, the older branch of the vagus nerve that originates from the DVMX is up-regulated. This DVC pathway is unmyelinated and usually maintains tone to the gut and regulates digestion (Chang, Mashimo, & Goyal, 2003). Heightened activation of the DVC is usually inhibited by the VVC and the SNS. When an extreme threat is present however the higher neural circuits are withdrawn, resulting in increased DVC activation, which causes bradycardia, apnea, increased gastric motility, and increased pain thresholds (Porges, 2007b). This form of autonomic activation also deactivates the SES. In the absence of SNS activation, activation of the older unmyelinated vagus nerve is exhibited as immobilisation, a primitive form of defence. In inescapable threat situations this autonomic state may result in syncope or even fear induced death (C. P. Richter, 1957).

From a polyvagal perspective, the ANS should be able to shift autonomic states to meet the demands of the environment when needed (Porges, 1995, 2003a, 2007b). Problems will arise if one’s autonomic state alters in excess to situational demands, or if an individual fails to successfully return to a state of calm and self soothing after a stressor (Friedman, 2007; Gilbert, 2001). According to polyvagal theory, the flexibility of the ANS depends on the dynamic control of the myelinated vagus nerve. The role of the myelinated vagus nerve is emphasised because polyvagal theory follows the principles of evolution and dissolution proposed by John Hughlings Jackson (1884);
that is, newer components of the ANS will dominate function until the demands of the environment overwhelm higher-level systems, causing lower neural substrates to assume regulation of the ANS. Porges’ claims that the myelinated vagus nerve is phylogenetically newer than the SNS and the unmyelinated vagus nerve, which prioritises its function over and above the other neural substrates (Porges, 1995, 2001).

It has been reported that the myelinated vagus nerve is largely responsible for vagal control of the heart; the role of the unmyelinated vagus nerve in cardiac control is less certain (Loewy & Spyer, 1990; D. W. Richter & Spyer, 1990; Stauss, 2003). Polyvagal theory assumes that the flexibility of the ANS can be indexed using HRV, as this is thought to reflect the efferent activity of the myelinated vagus nerve (Porges, 1995, 2001, 2003a). The myelinated vagus nerve usually exerts tonic inhibitory control over the rest of the ANS, hence why the myelinated vagus is sometimes termed the vagal “brake” (Porges, 1995, 2001, 2003a). The myelinated vagus nerve functions rapidly to mobilise or calm individuals by inhibiting or disinhibiting vagal tone to the heart. Consequently, the high-frequency component of HRV is an indicator of the efficiency of the vagal efferents involved in the coordination of visceral states. In support of Porges, appropriate regulation of the vagal brake has been associated with self-soothing in infants (Huffman et al., 1998), and has been shown to reflect higher self-regulation in adults (Fabes & Eisenberg, 1997). According to polyvagal theory, HRV should also index the neural regulation of the cranial nerves involved in emotional expression and social communication (Porges, 2007b). Despite this claim, research by Demaree and colleagues has so far failed to find a positive relationship between cardiac vagal control and emotional expressivity (Demaree, Pu, Robinson, Schmeichel, & Everhart, 2006; Demaree, Robinson, Everhart, & Schmeichel, 2004; Pu, Schmeichel, & Demaree, 2010).

2.4.3. Neurovisceral Integration vs. Polyvagal Theory

The model of neurovisceral integration (Thayer & Lane, 2000, 2009) and polyvagal theory (Porges, 1995, 2001, 2003a, 2007a) both highlight interactions that occur between the CNS and ANS. Both use HRV as a measure to index the level of inhibitory control exerted by the CNS on lower neuroregulatory systems, and posit that HRV is a biological marker of self-regulatory control and autonomic health. The two theories are similar and yet there are clear differences in their conceptualisation. The
model of neurovisceral integration is a centralised model, focusing on the top-down influence of the CAN on the ANS. Polyvagal theory on the other hand is a peripheral model, focusing on the influence of the vagal efferents that originate in the brainstem.

Polyvagal theory is unique in its recognition of the two sources of vagal outflow identified within the brainstem (Porges, 1995, 2001, 2003a). This differentiation from the model of neurovisceral integration offers an explanation as to why sometimes too much vagal activity can be detrimental; often it is assumed that greater vagal activity relates to greater health, however several areas of research suggest that this is not the case, for example in sudden infant death syndrome, stress-induced asthma, stress-induced gastric ulcerations, and vasovagal syncope (see Ritz, 2009). A second distinctive component of polyvagal theory is Porges’ conceptualisation of the heart–face link (Porges, 2003b, 2009a; Porges & Lewis, 2010). The cranial nerves controlling the face and head are anatomically and functionally linked with the vagal nuclei of the brainstem. This connection reinforces the notion that emotional and physiological responding are intimately linked, and that both forms of responding modulate interpersonal functioning. For these reasons I have chosen to focus on polyvagal theory as the predominant model of emotional responding in this thesis; however polyvagal theory is not without its deficiencies.

2.4.4. A Critique of Polyvagal Theory

Polyvagal theory makes bold assumptions about the function of the autonomic nervous system. There are several challenges to Porges’ theory that must be recognised. First of all, the term polyvagal is a misnomer, as polyvagal theory divides the vagal system into two efferent systems not many; Ritz (2009) has suggested that “bivagal theory” would be a more accurate description. Secondly, questions have been raised about the utility of dividing the vagal efferents originating from the NA and the DVMNX. Early research neglected the role of the brainstem in autonomic functioning because it contains large numbers of neurons that have no obvious connections to easily recognised and defined nuclei (Blessing, 1997): The term nucleus ambiguus perfectly represents the perplexity of the brainstem, as the name reflects the ill-defined borders of this region (Loewy & Spyer, 1990). Current techniques are unable to ascertain whether vagal outflow originates from the NA or the DMNX (Berntson et al., 2007; Grossman & Taylor, 2007), and this has made it impossible to verify Porges’ claims regarding
potential functional differences between the vagal efferent systems, at both psychophysiological and behavioural levels (Berntson et al., 2007; Ritz, 2009).

A further consideration is that much of the research used to substantiate polyvagal theory has been carried out with juvenile rather than adult populations. For example, vagal regulation has been associated with self-soothing in neonates (Huffman et al., 1998), facial expressivity in infants (Stifter, Fox, & Porges, 1989), and emotion regulation in infants and pre-school aged children (Hastings et al., 2008; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996; Stifter & Jain, 1996). Less empirical support has been provided using adult samples. Lower resting (tonic) high-frequency HRV has been linked to decreased regulation of negative affect and maladaptive coping (Fabes & Eisenberg, 1997; Pu et al., 2010), as well as poorer romantic attachment and marital quality (Diamond & Hicks, 2005; Smith et al., 2010). Further to this, smaller changes in high-frequency HRV in response to laboratory stressors have been associated with emotion regulation difficulties (Austin, Riniolo, & Porges, 2007; Hughes & Stoney, 2000; Sahar, Shalev, & Porges, 2001), and inferior social functioning (Egizio et al., 2008). Despite this support, there is also evidence that refutes some of Porges’ claims, for example Gyurak and Ayduk (2008) did not find a direct relationship between resting HRV and emotion control, and Demaree and colleagues have reported that cardiac vagal control does not predict emotional expressivity in response to film clips (Demaree, Robinson, et al., 2004; Demaree, Pu, et al., 2006). One explanation for the lack of positive findings in these studies relates to the methodology used: Supporting evidence in healthy adult populations tends to emerge during highly emotional situations (e.g., increased levels of daily stress, Fabes & Eisenberg, 1997), and not situations that do not warrant substantial emotional responses (e.g., passively viewing film clips, Demaree, Pu, et al., 2006). It may only be under conditions of challenge or threat that relationships between ANS function and socio-emotional behaviours clearly emerge in healthy adults.

The accumulating evidence is beginning to explore the utility of polyvagal theory as an integrative framework that can be used to simultaneously interpret physiology, behaviour, and psychosocial processes. Polyvagal theory has generated and stimulated new lines of research, but some have found theory to be too simplistic. Since its conception two important limitations have been addressed: Beauchaine (2001) has refined the repertoire of the SNS within the polyvagal hierarchy, whilst other
researchers have strived to differentiate between different forms of immobilisation response outside of the polyvagal framework (Bracha, 2004; Schauer & Elbert, 2010). These developments are addressed in sections 2.4.4.1 and 2.4.4.2.

### 2.4.4.1. Integrating polyvagal theory with BIS/BAS.

A caveat of polyvagal theory is that the basic hierarchy proceeds from a calm and self-soothing state, to mobilisation with fear, then to immobilisation (Porges, 1995, 2001, 2007b). The fundamental polyvagal framework neglects the occurrence of situations where mobilisation occurs without fear, for example during play and exercise. Only more recently has Porges’ considered this limitation: Porges (2009b) attributes playful behaviours to defensive fight–flight arousal coupled with dynamic VVC activation to insure safe interactions. Beauchaine (2001) was in fact first to address this issue, and instead of co-opting the neural substrates of the polyvagal hierarchy, Beauchaine combined Porges’ polyvagal theory with Gray’s reinforcement sensitivity theory. Gray’s (1987) theory hypothesises that in addition to fight–flight behaviours, individual responding is determined by two motivational systems – the behavioural activation system (BAS) and the behavioural inhibition system (BIS) – which independently respond to appetitive and aversive stimuli. Incorporating the behavioural inhibition and activation systems into the polyvagal hierarchy allows for a greater distinction between behaviours determined mainly by SNS activation, for example risky and impulsive behaviours (indicative of BAS activation), and hypervigilance and social avoidance/withdrawal (indicative of BIS activation).

Beauchaine’s (2001) integration of the BIS and BAS into the polyvagal hierarchy is useful, but Gray has since revised his original reinforcement sensitivity theory and altered the structure of the motivational systems outlined above (Gray & McNaughton, 2000). The role of the has BAS remained unchanged, responding to all positively valenced stimuli and mediating appetitive motivational functions, including both approach and active avoidance behaviours. Conversely the revised reinforcement sensitivity theory posits that the fight–flight system (FFS), not the BIS, is the system which is responsive to aversive stimuli and mediates aversive motivational functions, such as defensive aggression (fight) or escape (flight) behaviours. In the revised theory the BIS takes the role of inhibiting prepotent behaviours and controls the analysis of risk. The BIS is important in resolving conflict that can emerge in situations that contain elements of both reward and threat (i.e., conflicting activation of the BAS and the fight–
flight system). Although the BIS and BAS motivational systems have been altered the basic structure is still consistent with the polyvagal hierarchy.

One aspect of Gray and McNaughton’s (2000) revised theory which is not completely in line with the polyvagal hierarchy is the reinterpretation of the FFS, which is now defined as a fight–flight–freeze system. Temporary freezing, where the myelinated vagus nerve is withdrawn without SNS activation, is a response congruent with polyvagal theory, however this response is typically interpreted as an orienting response not a freeze response. A freeze response according to the polyvagal hierarchy is resultant from the absence of SNS activation with increased DVC activation, placing the freeze response outside the control of the SNS and FFS. Despite the divergence between Porges’ (1995, 2001, 2007b) polyvagal theory and Gray and McNaughton’s (2000) revised theory, the integration of the motivational systems into the polyvagal hierarchy is advantageous because it expands the range of behaviours that can be explained as a function of the ANS.

2.4.4.2. Beyond fight–flight–freeze responses. It is still possible to argue that Beauchaine’s (2001) reinterpretation of the polyvagal theory is somewhat limited. The fight–flight–freeze responses are only a proportion of the defences that can be initiated in the presence of threat. Bracha (2004) added the terms fright and faint to the acute stress response spectrum, making a distinction between tonic immobility (fright) and flaccid immobility (faint). Bracha’s (2004) model also defined the role of the freeze response as one that occurs fairly early in the hierarchy. Porges (1995, 2004a, 2007b) and Gray (1987; Gray & McNaughton, 2000) both use the term freeze response, but there is confusion as to what point in the defence repertoire “freezing” should refer to; in Porges’ work, freezing refers to refers to the DVC being up-regulated and resulting in vasovagal syncope, whilst Gray encapsulated freezing as being analogous to the orienting response (which occurs higher up the hierarchy). Bracha (2004) made a clear distinction between these responses by proposing a freeze–flight–fight–fright–faint hierarchy. The three initial responses in the hierarchy reflect normal responses to acute stress. The initial orienting response involves “stop, look, and listen” behaviours and Bracha termed this as the freeze response (in the polyvagal hierarchy orienting occurs when the vagal brake is removed, prior to activation of the SNS). This is followed by flight and fight responses (commonly attributed to SNS activation). The next response according to Bracha is fright, also known as tonic immobility, which is less common as
a response to acute stress. This response evolved as an alternative to fight–flight
tendencies (Alboni et al., 2008). During fright the body is immobile, but should the
chance to escape arise the body will be able to rapidly initiate a mobilisation response.
This form of immobility can be distinguished from the last stage of the hierarchy which
involves flaccid immobility. This last stage Bracha termed faint, and it corresponds to
Porges’ description of the body when the DVC is up-regulated, resulting in vasovagal
syncope (i.e., a temporary loss of consciousness). This final stage in the response
hierarchy is the least common form of reaction to acute stress and is usually only
initiated in times of severe life threat, with the exception of some clinical disorders.

Recent work in the field of post-traumatic stress disorder (PTSD) has
highlighted the need to examine other possible defence repertoires that can be initiated
by the autonomic nervous system. Schauer and Elbert (2010) have extended the work of
Bracha (2004) to increase the cascade of fear responses to cover freeze–flight–fight–
fright–flag–faint. This hierarchy suggests that there is a further nuance in the defence
cascade than previously discussed. Schauer and Elbert reinforce the hierarchy proposed
by Bracha, but distinguish between “uproar reactions” and “shut-down reactions”.
Uproar reactions are the initial defences in the cascade. Freeze corresponds to the
orienting response, which is attentive immobility. This state is characterised by an initial
bradycardia, followed by cardiac acceleration. Psychologically this stage is one of
hypervigilance whilst the level of threat is evaluated. If a threat is detected then freeze is
followed by the stages of flight and fight which are driven by sympathetic activation.
Flight and fight are states of active mobilisation with tachycardia, peripheral
vasoconstriction, and increased perspiration. According to Schauer and Elbert,
sympathetic activation is accompanied by feelings of irreality. In this model of fear
responding, arousal levels continue to rise whilst threats are imminent, however the
increasing activation will eventually climax, leading to the initiation of shut-down
responses.

The first shut-down response to appear in the defence cascade is fright, which
occurs at the apex of arousal when the SNS and PNS become co-activated, resulting in
tonic immobility. The physiology at this stage is characterised by tachycardia,
vasoconstriction, and hypertension. Although during fright the body is immobile, the
underlying physiology is prepared for mobilisation if given the opportunity (i.e., flight
or fight). Psychologically this stage is associated with hyperalertness and high
emotional arousal. It should be noted that it is not clear from the authors if the PNS activation during fright is driven by the VVC or the DVC, although one would presume that at this stage in the hierarchy it should be caused by up-regulation of the DVC. Combined activation of the SNS and the DVC seems most appropriate, as this pattern of activation plausibly leads into the next shut-down response, which is characterised by decreasing SNS activation with increasing PNS (DVC) activation; a stage which is not well elucidated in the polyvagal hierarchy. Schauer and Elbert (2010) term this stage the flag response, which is the penultimate stage in the hierarchy eventually leading to faint (flaccid immobility). Flag is associated with bradycardia, vasodilation, and hypotension, with corresponding cognitive failure and emotional numbing. As the flag state progresses further emotional involvement is thought to decrease, which is consistent with a dissociative shut-down response (Schauer & Elbert, 2010). The final stage of the hierarchy, faint, occurs when the PNS (DVC) is up-regulated without SNS activation, which can result in vasovagal syncope (what Bracha, 2004, terms flaccid immobility). It has been hypothesised that this physiological reaction evolved to protect the heart from stressful/dangerous conditions, however it appears that this physiological reflex is not just limited to physical threat, it can also be induced during emotional stress (Alboni et al., 2008). Vasovagal syncope is characterised by brief cardiac acceleration and elevation in blood pressure, followed by cardiac deceleration and a drop in blood pressure (Bracha, 2004). The hypotension and bradycardia that result are responsible for a temporary loss of consciousness. The main distinction between tonic and flaccid immobility, is that in the latter the body is immobile, but this time it is not prepared for mobilisation should the opportunity arise (Schauer & Elbert, 2010).

The main limitation of Bracha’s (2004) work and Schauer and Elbert’s (2010) hierarchy is that they focus on the systems that respond to threat, and not the safety systems that can alleviate distress and return the body to a state of calm and self-soothing. Consequently the best way forward in this thesis is not to try and replace one hierarchy with another, but to integrate them into one overall hierarchy of autonomic responding. An integrated model combining Porges’ (1995, 2004a, 2007b) polyvagal hierarchy integrated with Gray and McNaughton’s (2000) revised reinforcement sensitivity theory and Schauer and Elbert’s (2010) defence cascade is represented in figure 2.4. Although this model conceptualises the full range of responses that may occur in response to a challenge/threat, to keep things simple I will mostly refer to just two broad classifications of defensive response: mobilisation responses, which are
mostly driven by the SNS and include fight–flight behaviours; and *immobilisation responses*, which are mostly driven by the PNS and include fright, freeze, and faint behaviours (Lang, Davis, & Öhman, 2000).

2.4.5. Polyvagal Theory and Psychopathology

Polyvagal theory claims that emotion dysregulation and psychopathology may result from abnormal ANS function, as opposed to structural abnormalities within the ANS (Porges, 2001, 2003a). This is in line with the functional approach to psychopathology, which focuses on dimensions of dysfunction rather than classifications of disorder (van Praag et al., 1980). The links made between the polyvagal hierarchy and emotion dysregulation in this thesis hitherto have focused mostly on fear responses relevant to PTSD; this focus is an artefact of the literature reviewed thus far. Polyvagal theory is relevant to several, if not all areas of psychopathology, but it is often overlooked in clinically-oriented research because many aspects of the theory lack empirical testing. In the following sections I provide a brief overview of how polyvagal theory can be applied to several psychiatric disorders: Anxiety disorders (section 2.4.5.1), treatment-resistant depression (section 2.4.5.2), autistic disorders (section 2.4.5.3), borderline personality disorder (section 2.4.5.4), and anorexia nervosa (section 2.4.5.5).

2.4.5.1. Anxiety disorders. Anxiety is usually associated with the basic emotion of fear (Barlow, 1988). At low levels anxiety can be seen as an adaptive behaviour, however intense anxiety can be disabling, particularly when associated with disorders such as generalised anxiety disorder (GAD), panic disorder (PD), and post-traumatic stress disorder (PTSD). Worry is a form of recurrent negative thinking found in anxiety disorders and is associated with increased arousal caused by SNS activation (Brosschot & Thayer, 2004). The Diagnostic and Statistical Manual of Mental Disorders (4th ed. text rev.; DSM-IV-TR, American Psychiatric Association, 2000) recognises that anxiety disorders are commonly associated with abnormal visceral activity. Physiological correlates of anxiety such as rapid heart rate, shortness of breath, and sweating have predominantly been viewed as signs of increased SNS activation, which has negated the role of the PNS in these disorders (Friedman & Thayer, 1998; Thayer, Friedman, & Borkovec, 1996).

Recent research has investigated PNS function in anxiety disorders with some illuminating findings. It has been shown that clinically anxious individuals exhibit lower resting vagal tone compared to controls (Friedman & Thayer, 1998; Lyonfields, Borkovec, & Thayer, 1995; Thayer & Lane, 2000). Yeragani et al. (1990) also found
that individuals with PD demonstrated decreased HRV at rest compared to non-anxious control participants, whilst Cohen et al. (1998) found that individuals with PTSD at rest exhibited reduced high-frequency power and overall HRV compared to control participants. It should be noted that in healthy populations increased high-frequency HRV at rest has been associated with higher state anxiety (perhaps reflecting increased attention and vigilance), but not higher trait anxiety (Jönsson, 2007). This finding suggests that patterns of autonomic dysregulation in clinical populations may not be present or detectable in healthy populations.

Decreased PNS activation is not only seen at rest in clinically anxious individuals, but is also apparent when anxious individuals are confronted with anxiety-inducing stimuli. Anxious individuals have a low threshold to threatening stimuli, which is behaviourally characterised by hypervigilance and high levels of anticipatory stress (Cacioppo et al., 1992). Lyonfields et al. (1995) experimentally assessed PNS functioning in GAD by assessing vagal tone during baseline, an aversive imagery task, and a worrisome thinking task. Compared to non-anxious controls, participants with GAD showed lower levels of vagal tone during the initial baseline and little change in vagal tone over the experimental tasks. Taken together, these findings suggest that anxious individuals are more rigid and inflexible when responding to their environment. This has led to a reinterpretation of the aetiology of hyperarousal in anxiety disorders: Instead of up-regulation of the SNS being the sole cause of physiological arousal, symptoms are now being attributed to decreased activation of the PNS which would normally inhibit SNS activation (Friedman, 2007).

From a polyvagal perspective, the resting physiological state of anxious individuals makes it more likely that they will initiate defensive behavioural strategies when presented with threatening stimuli; their autonomic inflexibility means that the PNS is more readily switched off, meaning SNS activation is not adequately inhibited (Friedman, 2007). The nature of the stimuli present will determine whether threats result in SNS activation characterised by the FFS or a combination of the FFS and BAS (Gray & McNaughton, 2000). Aversive threat stimuli is theorised to initiate FFS responses, resulting in hypervigilance, phobic avoidance, and active escape behaviours, as seen in phobias and panic (Corr, 2008). However if aversive stimuli are accompanied by appetitive rewards, goal conflict between the FFS and BAS may occur resulting in activation of the BIS. If the BIS cannot successfully resolve the conflict by favouring
approach or avoidance behaviours then hypervigilance and hyperarousal may simultaneously occur, as seen when people with GAD worry (Corr, 2008).

2.4.5.2. Treatment-resistant depression. Major Depressive Disorder (MDD) is characterised by persistent low mood and/or loss of interest in pleasure in daily activities, accompanied by symptoms such as changes in appetite, sleep disturbances, fatigue, and feelings of guilt (DSM-IV-TR, American Psychiatric Association, 2000). Depressive episodes are associated with changes in social functioning, including social withdrawal (Rottenberg & Gotlib, 2004), reduced involuntary facial expression (Gaebel & Wölwer, 1992), and reduced gaze behaviour (Segrin, 1992). Although a wide range of treatments are available for MDD, the initial treatment for many is antidepressant medication. Despite medication’s popularity as the first mode of treatment, Fava and Davidson (1996) reported that up to one third of patients only partially recover from initial treatment with antidepressant medication, and up to one fifth are considered to be “non-responders”. MDD is often classed as treatment-resistant when symptoms fail to improve after at least two courses of antidepressants (Thase & Rush, 1995, as cited in Trivedi, 2003). The definition of treatment-resistant depression (TRD) is increasingly being extended from failure to show symptomatic improvement after treatment to failure to reach symptomatic and functional remission, meaning treatment is only considered successful if individuals no longer meet the criteria for MDD (Wijeratne & Sachdev, 2008). The nature of TRD means depressive episodes are not only chronic, but individuals are also more likely to relapse after they recover from a depressive episode.

Research investigating autonomic functioning in depression suggests that the depression is associated with lower resting levels of HRV: Depressed individuals have been shown to have lower resting vagal tone than non-depressed controls (Carney et al., 2001; Rechlin, Weis, Spitzer, & Kaschka, 1994). Depression severity has also been linked to vagal indices, with greater severity correlated with lower levels of vagal tone (Agelink, Boz, Ullrich, & Andrich, 2002), and increases in the high-frequency component of HRV have been linked to successful reductions in self-reported depressive symptoms (Schultz, Anderson, & van de Borne, 1997). Findings such as these add weight to the idea that vagus nerve stimulation (VNS) may be a suitable treatment for managing TRD. VNS is an invasive procedure analogous to a cardiac pace-maker: A device is implanted into the chest that is designed to innervate the vagus nerve (Rush & Siefert, 2009). Research investigating the effects of VNS on TRD has
shown promising results in terms of symptom reduction and rates of remission, although the procedure is costly and has notable side effects (Rush & Siefert, 2009).

Although the majority of research suggests that depression is linked to changes in autonomic function (see Rottenberg, 2007, for a review), the findings are mixed with some studies finding no differences in HRV between depressed and non-depressed controls (e.g., Moser et al., 1998). Findings linking depression and HRV are likely to be mixed due to methodological differences across studies. Individual differences in depressive symptoms are likely to affect findings; whilst some symptoms are linked to increased vagal tone (e.g., sadness), others have been linked to decreased vagal tone (e.g., suicidal impulses; Rottenberg, Wilhelm, Gross, & Gotlib, 2002). It should also be noted that studies have used diverse samples in which to study depression and HRV, for example in some studies data has been collected using cardiovascular patients (e.g., Carney et al., 2001). The potential influence of antidepressant medication is another well-known confound in the study of depression and HRV (Rottenberg, 2007).

Differing symptoms of depression can be linked to specific neural substrates within the polyvagal framework. There is evidence to suggest to depression is associated with low levels of BAS functioning, which is predictive of poorer depression outcome at 8-month follow up (Kasch, Rottenberg, Arnow, & Gotlib, 2002). Similar to worry in anxiety, rumination in depression is associated with heightened arousal caused by decreased PNS activation and relative SNS dominance (Brosschot, van Dijk, & Thayer, 2007; Ray, Wilhelm, & Gross, 2008). Reduced PNS activation (as indicated by low HRV) may account for the some of the social difficulties seen in depression, as this should be paralleled by inhibition of the social engagement system (Brosschot et al., 2007). Social withdrawal and depressed mood may even be attributable to increased activity of the DVC. The behavioural repertoire of sickness is mediated by the ANS, and research is increasingly linking the pathophysiology of sickness to the psychopathology of depression (Eyre & Baune, 2012; Raison, Capuron, & Miller, 2006). Animal research investigating the role of the DVC in sickness-induced behaviours has suggested that activation of the DVC may underlie behaviours which parallel some of the symptoms seen in clinical depression. For example loss of interest, lethargy, and social withdrawal have all been linked to DVC activation (Marvel, Chen, Badr, Gaykema, & Goehler, 2004).
2.4.5.3. Autistic disorders. Autistic disorders are pervasive developmental disorders defined by impairments in social interaction, communication, and atypical behaviour patterns (DSM-IV-TR, American Psychiatric Association, 2000). Social deficiencies associated with autistic disorders include a reduction in the ability to interpret emotional states, a lack of emotional reciprocity, and impaired nonverbal communicative behaviour (e.g., reduced eye-gaze, facial expression, and non-verbal gestures; DSM-IV-TR, American Psychiatric Association, 2000). Research has shown that individuals with autistic disorders tend to spend less time looking at core features of faces (such as the eyes, nose, and mouth) than controls, and are also impaired at recognising facial emotional expressions (Pelphrey et al., 2002).

Autonomic functioning has been investigated in individuals diagnosed with autistic disorders. Toichi and Kamio (2003) found that unlike healthy control subjects, half of their autistic sample did not suppress PNS functioning during mental tasks that required sustained attention. They concluded that this finding was attributable to some of the autistic participants being more ‘stressed’ under resting conditions, although no significant differences in cardiac autonomic function were found between the groups at baseline. Goodwin et al. (2006) found that autistic children only showed significant physiological responses to a stressor 22% of the time, compared to the healthy control group which showed significant physiological responses 60% of the time. Ming, Julu, Brimacombe, Connor, and Daniels (2005) also found that children with autism exhibited significantly lower resting levels of cardiac vagal tone compared with healthy controls. It has been suggested that individuals with autistic disorders show diminished cardiovascular reactivity to the environment because they are normally in a general state of autonomic defensiveness (Goodwin et al., 2006).

Porges (2004b) directly links his theory to autistic disorders and assumes that impaired social communication is associated with functional deficits of the social engagement system, as opposed to neuroanatomical or neurophysiological abnormalities. Individuals with autistic disorder are hypothesised as having insufficient activation of the newer vagus nerve (VVC activation) and therefore have compromised activation of the social engagement system. On this basis Porges and colleagues have developed biologically-based behavioural interventions to attempt to activate the VVC and improve social behaviour (Porges, 2001). One of these interventions is the ‘Listening Project’, which uses acoustic stimuli to increase neural innervation to the
muscles of the middle ear (a component of the social engagement system). This acoustic intervention has been reported to improve social behaviour in children with autism, for example increasing eye gaze, facial expressivity, and social interaction with others (Porges, 2001).

2.4.5.4. Borderline personality disorder. Borderline personality disorder (BPD) is characterised by a pattern of instability in interpersonal relationships, self-image, and affects, and marked impulsivity (DSM-IV-TR, American Psychiatric Association, 2000). Research has shown that BPD is associated with high sensitivity to emotional stimuli, high emotional reactivity, and a slow return to baseline (Linehan, 1993a). This pattern of reactivity is thought to be linked to a core component of emotion dysregulation. The clinical correlates of this disorder are rapidly changing mood states, unstable and intense relationships, and recurrent suicidal or self-mutilating behaviours (DSM-IV-TR, American Psychiatric Association, 2000). BPD is also often associated with chronic feelings of emptiness and severe dissociative symptoms (DSM-IV-TR, American Psychiatric Association, 2000).

Two studies have investigated BPD in the context of the polyvagal framework. Austin et al. (2007) found that individuals with BPD had similar resting levels of vagal tone compared to control participants, however the groups demonstrated different physiological responses to a laboratory task. Austin et al. reported that after watching emotional film clips individuals with a BPD diagnosis expressed decreasing vagal influences on the heart, whilst individuals in a control group expressed increasing vagal influences. In contrast, Weinberg, Klonsky, and Hajcak (2009) found that individuals with BPD did demonstrate decreased PNS function at rest compared to control participants, although Ebner-Priemer et al. (2007) only reported significant differences in HRV between healthy control participants and medicated BPD participants (there was no significant difference between the controls and non-medicated BPD participants). In addition to this, Weinberg et al. (2009) found that individuals with BPD responded to a social stressor with increased SNS activation, compared to control participants who exhibited decreased SNS activation. In Austin et al. and Weinberg et al. the proposed mechanism for the divergence seen between the groups during the experimental tasks was that the BPD group may have been detecting invalid risk from their environment, either from their interaction with the experimenter or from the experimental stimuli. These studies concluded that BPD individuals are more likely to
express defensive physiological states that support fight–flight behaviours in response to stressors due to invalid neuroception.

From a polyvagal perspective, high sensitivity to emotional stimuli increases the likelihood of defensive behavioural strategies being initiated. Dysfunctional neuroception will mean states of calm and self-soothing are likely to give way to SNS activation even in the presence of only mildly threatening stimuli (Porges, 2004b). The risky and impulsive behaviours seen in individuals with BPD can be attributed to the SNS and high levels of BIS/BAS activation (Claes, Vertommen, Smits, & Bijttebier, 2009). In addition to this, the dissociative states seen in BPD may result from increased activation of the DVC. Up-regulation of the DVC would lead to the flag response proposed by Schauer and Elbert (2010), which is a dissociative shut-down response characterised by emotional numbing and feelings of irreality. The self-mutilating behaviours seen in BPD have been hypothesised to provide relief from the flag state by initiating vasovagal reactions that continue to up-regulate the DVC, resulting in greater reductions of emotional involvement (Schauer & Elbert, 2010).

2.4.5.5. Anorexia nervosa. Anorexia nervosa (AN) is predominantly considered to be an eating disorder; individuals refuse to maintain a minimally normal body weight, are afraid of gaining weight, and have a significant disturbance in perceptions about their body shape or size (DSM-IV-TR, American Psychiatric Association, 2000). Recently it has been recognised that a substantial number of individuals with AN exhibit symptoms characteristic of personality disorders. One of the personality subtypes often linked to AN is emotionally constricted personality disorder (Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001). Deficits in emotional functioning seen in AN include impaired recognition of emotion in others and reduced emotional expression, particularly the expression of negative emotions (Geller, Cockell, Hewitt, Goldner, & Flett, 2000). Individuals with AN have also been shown to have increased pain thresholds (Papezová, Yamamotová, & Uher, 2005).

Dysregulation of PNS function has been reported in infantile anorexia, with anorexic infants demonstrating decreases in HRV during social interaction paradigms, whilst control infants exhibited increases in HRV (Chatoor, Ganiban, Surles, & Doussard-Roosevelt, 2004). Research in anorexic populations tends to be complex, as both short term and chronic starvation are related to increases in HRV (Galetta et al.,
Individuals with AN tend to show signs of decreased SNS responsiveness, along with increased PNS responsiveness (Ishizawa, Yoshiuchi, Takimoto, Yamamoto, & Akabayashi, 2008). Zonnevylle-Bender et al. (2005) conducted a study comparing anorexic adolescents’ and healthy controls’ subjective and physiological responses to a public speaking task. Both groups reported increases in negative emotional arousal, with the control group also showing parallel increases in heart rate and cortisol. The anorexic group however showed a blunted physiological response, with significantly lower increases in heart rate and no significant changes in cortisol in relation to the stressor. These results suggest that autonomic dysregulation is characteristic of this sample.

There is no clear aetiology of AN, however some of the symptoms can be explained using the polyvagal framework. The physiological dysregulation associated with anorexia nervosa may mean that anorexic individuals feel the need to carry out behaviours, such as restricting their food intake, to help them regulate their physiology in order to feel “normal”. Behaviours such as reduced food intake could be interpreted by the body as life threatening. When a threat to survival is detected the body increases the activation of the DVC, resulting in inhibition of the SNS and the social engagement system. Clinically, individuals with increased DVC activation would present with limited emotional expressivity as well as numbing, both of which are seen in anorexia nervosa.

2.4.6. The Current Status of Polyvagal Theory

Polyvagal theory provides a novel explanation for some of the physiological, behavioural, and socio-emotional deficits seen in psychiatric disorders (Porges, 1995, 2001, 2003a). However, theories should not just be innovative frameworks for organising existing data; new theories must also generate and stimulate original research, and provide testable hypotheses (Popper, 1959/2002). The ability for polyvagal theory to stimulate and direct research is not refuted, however questions have arisen regarding some of the central tenets of polyvagal theory and their testability (Berntson et al., 2007). Evaluating Porges’ representation of the ANS is currently limited by methodological constraints: Researchers are unable to distinguish the vagal efferents originating from the NA and the DVNX (Berntson et al., 2007; Grossman & Taylor, 2007). This means it is currently impossible to falsify Porges’ claims regarding the different qualitative functions of the vagal efferent systems.
Although issues revolve around the biological components of polyvagal theory, this does not mean that the theory needs to be rejected outright. Lakatos (1970) noted that complex theories can be conceptualised as research programs. Research programs consist of central “hard core” assumptions that define a problem, and auxiliary hypotheses that derive from the hard core and make testable predictions. This line of reasoning suggests that even though Porges’ claims about the vagal systems cannot be explicitly tested, there are other aspects of polyvagal theory that are open to confirmation/falsification. This thesis aims to investigate the utility of polyvagal theory by identifying and bringing to the fore testable hypotheses that arise from the theory.

2.4.6.1. Hypotheses arising from polyvagal theory. Polyvagal theory suggests that complex behaviours, such as emotion regulation and social communication, depend on our physiological state (Porges, 1995, 2001, 2003a). If this premise is valid, our physiological, psychological, and social functioning is all dependent on how effectively the ANS is regulated. Although current methods cannot differentiate between VVC and DVC activation, it is possible to assess reciprocal influences of the PNS and SNS on the body using psychophysiology (see section 3.4 for further detail). It is also possible to measure socio-emotional functioning using self-report measures and behavioural paradigms (see sections 3.5 and 3.6 for further detail). The following hypotheses will be directed at investigating associations between ANS function and facets of emotional and behavioural responding to test the validity of polyvagal theory’s predictions.

2.4.6.1.1. Psychophysiological responses to laboratory stressors. According to polyvagal theory, during calm and self-soothing states the PNS promotes growth and restoration by minimising metabolic demands (Porges, 2001). Polyvagal theory clearly states that under normal circumstances the PNS should inhibit activation of the SNS, however when a threat or challenge is encountered the PNS should be withdrawn and the SNS up-regulated (Porges, 1995, 2001, 2003a). SNS activation is energy expending and is associated with increased heart rate and sweat response (Cannon, 1929). It is therefore hypothesised that:

Hypothesis 1. Laboratory stressors will be associated with decreased PNS activation, increased SNS activation, and increased negative affect. In response to a laboratory stressor, it is expected that individuals will demonstrate measurable increases in heart rate and sweat response (inferring increased SNS activation), coupled with a decrease in
high-frequency HRV (inferring decreased PNS activation). It is also expected that increased SNS activation will be coupled with increases in self-reported negative affect (Feldman et al., 1999). In addition to confirming the effects of laboratory stressors on physiological arousal, the aim of this line of research is to identify a stressor that can be used to investigate the subsequent hypotheses: As discussed in section 2.4.4, links between HRV and socio-emotional functioning may only be apparent during situations of challenge or stress (e.g., Fabes & Eisenberg, 1997). Although laboratory stressors consistently result in measurable physiological changes, these changes rapidly return to baseline as the body resumes its natural balance of PNS and SNS function (Jänig, 2006). As a result, there is a need to identify a laboratory stressor that maintains arousal beyond the duration of the task itself, to enable the investigation of the effects of defensive physiological arousal on socio-emotional functioning.

2.4.6.1.2. Psychophysiological responses to emotion regulation strategies. A second tenet of polyvagal theory is that up-regulation of the PNS increasingly inhibits activation of the SNS (Porges, 1995, 2001, 2003a). Changes in emotional state are said to parallel changes in physiological state, with higher levels of PNS activation being associated with increased positive affect (Bazhenova & Porges, 1997). Bidirectional links between higher brain structures and the brain stem suggest that not only do our visceral states affect how we feel, but our feelings in turn can influence our physiological state (Porges, 2009b). Consequently, it is conjectured that interventions targeting neural regulation may enhance the activation of the PNS (i.e., increase high-frequency HRV), accelerate down-regulation of the SNS, and improve affect (Porges, 2007a). As a result the following hypothesis is proposed:

**Hypothesis 2.** Emotion regulation strategies will be associated with increased activation of the PNS, and accelerate the down-regulation of physiological and psychological arousal after a stressor. Researchers have begun to investigate the usefulness of strategies that enhance cardiac vagal control and self-regulation. Interestingly, emotion regulation strategies that generate positive emotion are often associated with enhanced vagal control. For example, increases in vagal indices have been reported when participants have been instructed to consciously focus on feelings of care, appreciation, and social connectedness (Kok & Fredrickson, 2010; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995), as well as when carrying out structured meditation and relaxation.
exercises (Ditto, Eclache, & Goldman, 2006; Lehrer, Sasaki, & Saito, 1999; Sakakibara, Takeuchi, & Hayano, 1994; Tang et al., 2009).

2.4.6.1.3. Socio-emotional functioning during defensive physiological arousal.
Polyvagal theory asserts that activation of the neurophysiological systems that drive defensive behavioural repertoires will down-regulate the neural accessibility of the social engagement system, thus limiting the effectiveness of social engagement behaviours during times of threat (Porges, 2001, 2003a). Although links have been made between vagal tone and socio-emotional functioning in infants (for example, Bazhenova & Porges, 1997; Huffman et al., 1998; Stifter & Fox, 1990; Stifter et al., 1989; Stifter & Jain, 1996), fewer studies have been attempted with adults.

Porges has suggested that calm and self-soothing states should result in facial expressivity, physiological and behavioural flexibility, and social awareness (Porges, 2003a, 2009b). Similar to this, Scherer (2007) has proposed the existence of three emotional competencies that parallel the theorised outputs of the social engagement system: Emotion production competence (the ability to adaptively respond to an event, both physiologically and behaviourally); emotion regulation competence (the ability to monitor and manipulate one’s emotional state and its motor expression); and emotion perception competence (the ability to accurately perceive and interpret the emotional states of others). The outputs of the social engagement system also resemble the three nonverbal social skills identified by Riggio (1986): Emotional expressivity (i.e., encoding ability); emotional control (i.e., regulation ability); and emotional sensitivity (i.e., decoding ability). All of these frameworks suggest that deficiencies in any one of these competencies will impact the success of social engagement interactions. One aspect of social engagement that is often addressed in emotion research is affiliation (i.e., one’s willingness to spend time with others; Gump & Kulik, 1997; Taylor et al., 2000; Taylor, 2006). As a result, three hypotheses have been formulated regarding the effects of defensive physiological arousal on socio-emotional functioning:

Hypothesis 3. Increased activation of the SNS in response to a laboratory stressor will be associated with decreased facial expressivity. Vagal tone has been linked to emotional expressivity in infants (Stifter et al., 1989), however less convincing evidence has been reported with adults. Demaree and colleagues have repeatedly reported that indices of vagal tone do not predict greater emotional expressivity to film clips
(Demaree, Robinson, et al., 2004); if anything increased vagal tone has been linked to reduced expression of negative affect (Demaree, Pu, et al., 2006; Pu et al., 2010). A potential limitation of the work by Demaree and colleagues is that emotional expressivity has been measured during passive viewing tasks, and not during challenge or threat situations. It is hypothesised that manipulating physiological arousal with a laboratory stressor may enhance the relationship between ANS function and facial expressivity, making it more accessible to measurement.

In addition to this, it was hypothesised that reduced facial expressivity may impact on one’s ability to infer emotions from others’ facial expressions. Hypothesis 4 is as follows:

**Hypothesis 4.** Increased activation of the SNS in response to a laboratory stressor will be associated with decreased emotional sensitivity (i.e., the ability to recognise emotions in others). Facial mimicry is a proposed mechanism by which we are able to infer the emotional states of others (Stel & van Knippenberg, 2008; Stel & van den Bos, 2010). Following on from hypothesis 3, it is logical to suggest that changes in ANS function may affect emotional sensitivity as well as emotional expressivity; this is possibly what Porges (2003a, 2009b) means when he theorises that defensive physiological arousal reduces “social awareness”. Surprisingly few studies have investigated the effect of stressors on emotion recognition, although Hänggi (2004) reported that stress has a negative effect on emotional sensitivity.

The final hypothesis of this thesis aims to explore the relationship between ANS and one’s willingness to spend time with others:

**Hypothesis 5.** Increased activation of the SNS in response to a laboratory stressor will be associated with decreased affiliation tendencies. Polyvagal theory proposes that defensive physiological states are not compatible with social engagement behaviours (Porges, 2001, 2003a). In support of this, studies have suggested that increased cardiac vagal control is related to greater sociability in infants (Fox, 1989; Stifter & Corey, 2001; Stifter et al., 1989), and in adults’ indices of vagal tone have been positively associated with measures of romantic attachment (Diamond & Hicks, 2005), marital quality (Smith et al., 2010), and social functioning (Egizio et al., 2008). One mechanism that may be affected by defensive physiological arousal is one’s willingness to spend
time with others. There is some evidence to suggest that stress may actually increase affiliation tendencies (Gump & Kulik, 1997; Taylor et al., 2000; Taylor, 2006), however most of these studies to date have focused on self-report, behavioural, and neuroendocrine measures of arousal. Although neuroendocrine measures reflect ANS activation, they are qualitatively different from autonomic measures such as heart rate and sweat activity. As a result, it is proposed that a negative pattern of coherence between ANS function and affiliation tendencies may emerge when measures specifically reflect activation of the SNS.

2.5. Summary and Aims of the Thesis

This review has highlighted the role physiology plays in socio-emotional responding. Polyvagal theory (Porges, 1995, 2001, 2003a) is acknowledged as a suitable model of emotional responding that integrates physiological, behavioural, and social functioning, with autonomic regulation recognised as the “linchpin” of physical, psychological, and social development (Porges, 2009b). Although some aspects of polyvagal theory have been verified by empirical work, many of Porges’ propositions are theoretical and lack supporting evidence. Although current methods are unable to address the distinction made between the vagal efferent systems in polyvagal theory, Porges’ claims regarding links between ANS function and social engagement behaviours can be empirically tested. The review identified five key hypotheses that will be addressed in this thesis:

Hypothesis 1. Laboratory stressors will be associated with decreased PNS activation, increased SNS activation, and increased negative affect.

Hypothesis 2. Emotion regulation strategies will be associated with increased activation of the PNS, and accelerate the down-regulation of physiological and psychological arousal after a stressor.

Hypothesis 3. Increased activation of the SNS in response to a laboratory stressor will be associated with decreased facial expressivity.

Hypothesis 4. Increased activation of the SNS in response to a laboratory stressor will be associated with decreased emotional sensitivity (i.e., the ability to recognise emotions in others).

Hypothesis 5. Increased activation of the SNS in response to a laboratory stressor will be associated with decreased affiliation tendencies.
These hypotheses will be examined in healthy adult participants. However, before these hypotheses are addressed, it is necessary to clarify the methodology that will be used in this thesis. The next chapter will establish theoretically-relevant measures that can be used to index autonomic function, as well as a set of dependent measures to assess emotional expressivity, emotional sensitivity, and affiliation tendencies.
Chapter 3: Measures of Socio-Emotional Responding

Emotions are associated with changes in cognitions, feelings, behaviours, and physiology (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). To appreciate the widespread changes that occur throughout the emotion generation process, psychophysiological researchers have attempted to measure changes at subjective, behavioural, and physiological levels. Numerous techniques have been developed to capture the activation of the autonomic nervous system (ANS) to elucidate the role of the body in the generation and maintenance of emotions. This chapter discusses the rationale for the measures and methodologies used in this thesis; for a more general review of measures of emotional responding see Mauss and Robinson (2009).

3.1. Coherence between Measures of Socio-Emotional Responding

Polyvagal theory (Porges, 1995, 2001, 2003a) suggests that three broad categories of neurophysiological state are sufficient to organise the range of physiological responses to different classes of environmental stimuli and their associated behaviours (safe stimuli: calm and self-soothing; threatening stimuli: mobilisation with fear; life threatening stimuli: immobilisation). As discussed in Chapter 2, it has been contended that three states fail to capture the nuances of emotional responding, and additional physiological stages have been hypothesised: sympathetic nervous system (SNS) responses have been split into mobilisation with fear and mobilisation without fear (see section 2.4.4.1; Beauchaine, 2001); and tonic immobility as a parasympathetic nervous system (PNS) response has been distinguished from flaccid mobility (see section 2.4.4.2; Bracha, 2004; Schauer & Elbert, 2010).

Although in general broad classifications of sympathetic (mobilisation) and parasympathetic (immobilisation) responses can be useful, this distinction can also be an over-simplification. Unfortunately, due to methodological constraints it is impossible to differentiate between several of the proposed neurophysiological states using established measures of autonomic function (Berntson, Cacioppo, & Grossman, 2007; Grossman & Taylor, 2007). For example PNS activation from the ventral vagal complex (VVC; arising from the nucleus ambiguus) cannot be accurately differentiated from the activation of dorsal ventral complex (DVC; arising from the dorsal motor nucleus of the vagus nerve). Consequently, this thesis will use measures of autonomic
functioning to distinguish between activation of the PNS and SNS, without attributing function to specific levels of the polyvagal hierarchy.

In the following experiments a range of measures were used in order to capture the influence of physiological state on emotional responding and social behaviours. The first objective of using psychophysiology was to obtain objective indices of arousal, to ensure that participants were in a defensive physiological state after the stressor manipulations. It was expected that the stressor manipulations would result in increased SNS activation and decreased PNS activation: Increases in SNS activation should result in increased self-reported negative affect, along with increases in heart rate and sweat response, and decreased PNS activation should result in a decrease in high-frequency heart rate variability (Tuvblad et al., 2010). The second purpose of measuring psychophysiology was to examine the effects of emotion regulation strategies and behavioural laboratory tasks on participants’ physiological return to baseline. Finally, measuring psychophysiology would allow links to be drawn between ANS function and behavioural measures of social functioning.

It is important to note that although experimental manipulations often result in observable patterns of physiological activation, there is not always a clear coupling between response systems. Emotional responses are multifaceted, and response systems can show incoherent patterns of activation when emotions are experienced. There is some evidence for autonomic specificity in relation to basic emotions (see Ekman, Levenson, & Friesen, 1983), but there is also a great deal of evidence to the contrary: For example self-reports of emotion do not always link to behaviours (Bonanno & Keltner, 2004), and self-reports and behaviours do not always link to patterns of autonomic activation (Mauss et al., 2005). At best emotional response systems are thought to be “loosely coupled” (Lang & Cuthbert, 1984). It is convenient at this point to mention that a similar critique arises for measures of social functioning. Like emotion, the construct of “social function” has been operationalised through self-report measures, observer ratings, and behavioural assessments; however these measures also tend to show poor correspondence to one another (Segrin, 1998). Consequently researchers are often required to use a variety of physiological, self-report, and behavioural measures in order to capture the nuances of both emotional responding and social functioning.
A critique of using physiology along with other measures of emotion and social function is how much can be inferred from them: Researchers can be prone to suggest that a specific pattern of ANS activity relates to a particular response or function, however this view neglects the general-purpose nature of the ANS and its role in homeostasis, and other functions such as effort and attention (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Mauss & Robinson, 2009). Indices of ANS function are not end-points, and singular physiological measures cannot be used to infer psychological states or processes (Berntson et al., 2007).

Whilst the difficulties of measuring independent aspects of socio-emotional responding are recognised, investigating patterns of ANS function and socio-emotional behaviours are important when evaluating the polyvagal framework. Polyvagal theory (Porges, 1998, 2003a) suggests that there is a direct coupling between ANS function and the expression of social engagement behaviours. Consequently if the findings indicate that increased SNS activation results in measurable changes in facial expressivity, social awareness, or affiliation tendencies, this will provide empirical support for polyvagal theory. On the other hand, the absence of a link between autonomic indices and behavioural measures of social engagement may suggest that polyvagal theory is not a valid model of emotional responding.

**3.2. Research Design**

To put the measures in context, first I will give an overview of the general design of the current experiments. To investigate potential relationships between physiology and social engagement behaviours, a series of laboratory-based experiments were devised. Experimental designs were chosen as they allowed the participants’ physiology to be manipulated using laboratory-based stressors (see section 3.3). The effects of the stressor manipulations were assessed using autonomic indices, self-report questionnaires, and measures of behaviour. The participants recruited in the current experiments were healthy undergraduate students with no history of psychological or psychiatric disorders. Porges (2001, 2003a) claims that many of the socio-emotional deficits seen in psychiatric disorders are due to functional abnormalities; associations between ANS function and social engagement behaviours should therefore be observable in healthy populations. As discussed in Chapter 2 it was hypothesised that the relationship between ANS function and socio-emotional behaviours should be
particularly salient during conditions of challenge or threat, hence the use of stressor manipulations. Although it may have been advantageous to use a clinical population in this research to establish the clinical utility of polyvagal theory, it would be impossible to verify that any observed deficits in the expression of socio-emotional behaviours were due to functional and not disorder-specific anatomical abnormalities. The ability to generalise the findings to other populations would also be limited. The purpose of using healthy populations and experimental designs means that the findings can be used to evaluate polyvagal theory as a universal model of emotional responding.

3.3. Stressor Manipulations

Acute stressors are commonly used in emotion research to induce perceived threat and increase negative affect (Feldman et al., 1999). Difficult cognitive, social, or psychomotor tasks are often considered to be psychological stressors, however they also contribute to changes in physiology. The changes elicited depend on the nature of the stressor task, and a notable distinction has been made between active tasks and passive tasks (Obrist, 1981; Tomaka, Blascovich, Kelsey, & Leitten, 1993): Active stressors such as mental arithmetic and public speaking require motivated responses, and generally initiate physiological responses driven by activation of the SNS. Passive stressors such as viewing films or listening to music do not require instrumental action, and tend to initiate physiological responses that are milder in intensity. It has been suggested that active stressors produce more meaningful changes in physiology than passive stressors, because changes in ANS function are more likely to correspond to an individual’s psychological state when they are actively attempting to cope with the demands of the environment (Obrist, 1981).

Eliciting stress in laboratory settings is advantageous as it allows for the control of extraneous variables, however laboratory stressors are generally less aggravating than the types of stressor encountered in real life (Dimsdale, 1984). Because of this, researchers have continually questioned the ecological validity of laboratory stressors and how well they reflect real-world responding (Dimsdale, 1984; Kamarck & Lovallo, 2003; van Egeren & Sparrow, 1989). Two approaches can be used to increase the ecological validity of laboratory experiments. The first approach is to use tasks that revolve around social functioning; this is because daily stressors are often social in nature (Kamarck & Lovallo, 2003; Linden, Rutledge, & Con, 1998). A second approach
is to use virtual reality. Virtual reality tasks are increasing in popularity as virtual reality tools can be used to simulate real-world scenarios, whilst maintaining experimental conditions (Riva et al., 2007).

In the current experiments effective stressor manipulations were required to induce perceived challenge and/or threat in order to up-regulate the SNS. Activation of the SNS is hypothesised to limit the accessibility of the social engagement system (Porges, 2001, 2003a), and it is proposed that increased SNS activation will reveal greater coherence between ANS function and measures of socio-emotional behaviour. To ensure the stressor manipulations employed in the current research were effective at increasing the activation of the SNS, the first empirical chapter investigated the effects of two active social stressors: A virtual reality maze and a speech task (see Chapter 4).

### 3.4. Physiological Measures of Socio-Emotional Responding

To determine the effectiveness of the stressor manipulations and evaluate their effects on socio-emotional responding, several physiological measures were recorded during the experiments. Measures of physiology primarily index ANS function, but they are also thought to provide objective, external representations of the private emotional experience. Physiological changes occur during emotionally salient situations because emotions are inherently linked to underlying motivational states that guide behaviour (Bradley, Codispoti, Cuthbert, & Lang, 2001). It has been suggested that opposing appetitive and defensive motivational systems evolved to respond to environmental demands that promoted or threatened survival (Lang, Bradley, & Cuthbert, 1998). The physiological response elicited in any given situation will depend on the valence of the situation (i.e., pleasant–appetitive vs. unpleasant–defensive), and its associated arousal level (i.e., the strength of motivational activation). Changes in physiology facilitate the processing of the environment and prepare the body for an appropriate response.

Polyvagal theory suggests that the physiological determinants of the motivational systems index whether or not the body is in a state of calm and self soothing (i.e., safety) or a state of defence (Porges, 1995, 2001, 2003a, 2007b). If an individual is presented with an acute challenge that exceeds a critical level with respect to intensity and/or duration they will exhibit a defensive stress response (Boucsein, 1992). Autonomic defensive physiological states are often thought to be driven by SNS
activation, however, as emphasised by Berntson, Cacioppo, and Quigley (1991) physiological responses can result from the SNS and PNS being reciprocally activated, coactivated, coinhibited, or uncoupled. As a consequence, experiments measuring physiological responses to stress need to assess both SNS and PNS activation to capture the dynamic function of the ANS. In the current experiments skin conductance level was chosen as a marker of SNS activation (see section 3.4.1). Although heart rate can also be used to infer SNS activation, the heart is influenced by both the SNS and PNS. Heart rate was measured in the following experiments, but the data is interpreted with some caution (see section 3.4.2). In contrast to skin conductance level, high-frequency heart rate variability was used to index PNS activation (see section 3.4.3). The separate physiological indices and their methodological considerations are presented in the following sections.

3.4.1. Skin Conductance Level

One of the most simplistic indexes of autonomic responding is the measurement of electrodermal activity (i.e., the measurement of sweat gland activity on the surface of the skin). The most common type of sweat gland is the eccrine sweat gland, which is distributed across most regions of the body, especially the forehead, palms, and soles. The main function of eccrine sweat glands is to help regulate body temperature through evaporation (Boucsein, 1992). It is generally accepted that eccrine sweat glands are innervated by sympathetic fibres, although there is some debate regarding the role of the PNS in the regulation of sweat gland activity; decreases in electrodermal activity could arguably occur from a decrease in sympathetic activation and/or an increase in parasympathetic activation (Boucsein, 1992). Despite this caveat, increases in sweat activity are generally regarded to primarily index SNS activation (Dawson, Schell, & Filion, 2000; Jänig, 2006; Venables, 1991).

Electrodermal activity (EDA) can be measured by calculating changes in skin potential or skin resistance using electrodes placed on the surface of the skin (Jänig, 2006); this form of recording is termed *exosomatic*. Passing direct current across the surface of the skin results in tonic (skin conductance level) as well as phasic (skin conductance response) measures of EDA. Emotion researchers are interested in EDA due to the role it plays in emotional responding. Increased sweat gland activity is a concomitant of psychological and emotional states; a response that has been coined as
“emotional sweating” (Boucsein, 1992). EDA is considered to be a sensitive indicator of general arousal, demonstrating linear increases with indices of motivational activation strength. For example, Lang, Greenwald, Bradley, and Hamm (1993) reported a correlation of \( r = .81 \) between skin conductance and arousal rankings of affective pictures. The components of EDA tend to exhibit moderate test–retest reliability (generally between .50-.70), but these effects are task-dependent, with reliabilities across tasks usually reported as much lower (Dawson et al., 2000; Schell, Dawson, & Filion, 1988).

In the following studies, tonic EDA (i.e., skin conductance level [SCL]) was continuously measured with a sampling rate of 125 Hz, using a BIOPAC™ MP150 system connected to a computer running AcqKnowledge 4.1 software (BIOPAC Systems; Goleta, CA). Two grounded Ag/AgCl electrodes were attached to the medial phalanx of the index and middle fingers of the non-dominant hand. The second phalanges were used for recording because they are less prone to movement artefacts and tend to have less scaring than the fingertips (Boucsein, 1992).

### 3.4.2. Heart Rate

Heart rate is another indicator of ANS activity that is easily measured using non-invasive techniques. As mentioned in Chapter 2, the heart is intimately involved in all aspects of emotional responding and behaviour because of its role in preparing the body for behavioural responses (Schwartz, 1982). Heart rate has been shown to be a sensitive and specific index of valence (Boucsein, 1992), but it also plays in a role in more general emotional responding. The heart is a muscle located in the upper region of the torso which consists of four chambers: The atria (the two superior chambers) and the ventricles (the two inferior chambers). Its function is to supply oxygen and nutrients to the cells of the body. It does this by pumping deoxygenated blood from the right side of the heart, through the right atrium and right ventricle, to the lungs where gaseous exchange occurs (carbon dioxide is unloaded and passed through the alveoli to be exhaled, and is replaced with inhaled oxygen). The freshly oxygenated blood is then passed back through the heart, through the left atrium and left ventricles to be pumped to the rest of the body. Valves within the heart open and close to prevent a backflow of blood, and the opening and closing of these structures is what causes the lub-dub sound associated with the beating of the heart.
The natural rhythm of the heart arises because the heart is made up of autorhythmic fibres. These fibres intrinsically generate action potentials and trigger the contraction of the heart muscle (Tortora & Derrickson, 2006). To keep this intrinsic rhythm under control the heart has a pacemaker, called the sinoatrial node, which regulates the rate of contraction of the heart’s fibres. Despite its autonomy, the heart is not detached from the rest of the body; nerve impulses from the brain stem and hormones released by the adrenal medullae can efficiently change the timing and strength of each heartbeat in order to meet the demands of the viscera and somatomotor systems (Somsen, Jennings, & van der Molen, 2004).

The heart is one of the few organs that is innervated by both the PNS and SNS (Jänig, 2006). The PNS influences the heart via the vagus nerve. Vagal axons terminating in the heart release acetylcholine, which decreases heart rate by slowing the rate of depolarisation in the autorhythmic fibres. Under conditions of rest the vagus nerve has an inhibitory effect on heart rate, with the heart beating at a slower rate than the pace of the sinoatrial node. This neurophysiological arrangement has been termed the _vagal brake_ (Porges, 1995, 1999). Withdrawal of the vagal brake allows for a rapid increase in heart rate, without relying on the activation of the SNS (Berntson, Cacioppo, & Quigley, 1993a). In the short term the heart is able to adjust its output solely via parasympathetic influences in order to align with rapid shifts in behaviour. Behaviours that require more prolonged increases in heart rate are normally sustained by increases in SNS activation. The SNS triggers cardiac accelerator nerves to release noradrenaline, which speeds up the rate of depolarisation and increases the heart’s force of contraction, allowing a greater volume of blood to be pumped around the body (Andreassi, 1989).

The speed at which the heart contracts (i.e., heart rate) can be a useful index of cardiovascular reactivity, although dual innervation from the SNS and PNS makes it difficult to determine the source of changes using heart rate alone (for example increases in heart rate can result from decreased PNS activation, increased SNS activation, or a combination of both). Despite this drawback, heart rate is popular measure of autonomic function that has received a lot of attention over the years. Research has established that heart rate is generally a stable marker of cardiovascular reactivity, for example Cohen et al. (2000) reported a test–retest reliability of .64 when heart rate was measured in response to public speaking tasks carried out two weeks apart. It must be noted however, that although heart rate tends to demonstrate moderate
to high correlations within laboratory tasks, correlations are more modest across laboratory tasks (Kelsey, Ornduff, & Alpert, 2007). Cardiovascular reactivity is particularly unstable across laboratory and natural settings, suggesting that the ability to generalise reactivity from laboratory to real-life stressors may be limited (Abel & Larkin, 1991).

Despite the limitations of using heart rate as a marker of autonomic function, due to the relative ease of measuring heart rate, it is a popular outcome measure in psychophysiological research. It is possible to measure the activity of the heart using an electrocardiogram (ECG). Electrical currents are generated as action potentials propagate through the heart muscle, and it is possible to detect these on the surface of the body. The pattern of these signals can be decomposed to reflect the cardiac cycle and establish the function of the heart (see figure 3.1). An ECG wave contains three typical signals: The P wave, small upward deflection of the ECG which represents atrial depolarisation; the QRS complex, which begins as a small downward reflection, continues as a large, upright, triangular wave and ends as a downward wave, which represents rapid ventricular depolarization; and the T wave, a dome-shaped upward deflection towards the end of the cycle which indicates repolarisation (Tortora & Derrickson, 2006).

In the following experiments heart rate was continuously sampled at 512 Hz using surface electrodes arranged in a type II lead configuration with a BIOPAC™ MP150 system. Heart rate (HR) was determined using AcqKnowledge 4.1 (BIOPAC Systems; Goleta, CA). Offline a 0.5-35 Hz bandpass filter was applied to the heart rate
data, using 4000 co-efficients. Heart rate was then derived by detecting the R-peaks in the ECG wave and calculating the corresponding rate (in beats per minute).

3.4.3. Heart Rate Variability

Heart rate is a sensitive measure of emotional responding, but it only reflects the mechanical functioning of the heart and not the autonomic mechanisms driving cardiac function (Appelhans & Luecken, 2006). In Chapter 2 it was highlighted that changes in the activity of the heart can occur on a beat-to-beat basis. Measures of heart rate average out the changeability of the heart’s activity over a period of time, and fail to provide information about variations in the influence of the PNS and SNS on the heart. This information can be sourced from the ECG by using the variation in R-R intervals (i.e., the time between two consecutive R-peaks) to calculate heart rate variability (HRV).

Because the heart is intrinsically connected with other aspects of bodily functioning, oscillating rhythms within the ECG can be identified and linked to other physiological processes (Berntson et al., 1997). One of the most prominent relationships that can be found is between heart rate and respiration. The respiratory rhythm that can be identified in HRV is known as respiratory sinus arrhythmia (RSA; Berntson et al., 1993a). Stretch receptors in the torso activate on inspiration and are linked to a gating effect of the PNS. Subsequently RSA corresponds to an acceleration of the heart during inspiration and a deceleration of the heart during expiration (Jänig, 2006). Identifying the respiratory rhythms driven by the PNS can therefore be used as an index of cardiac vagal control (Porges, 1986). There is some limited evidence to suggest that RSA indexes the activation of the myelinated nerve arising from the nucleus ambiguus, rather than the unmyelinated vagus nerve originating from the dorsal motor nucleus of the vagus nerve (for example Rentero et al., 2002; Richter & Spyer, 1990), however this premise is still widely debated (Berntson et al., 2007; Blessing, 1997; Grossman & Taylor, 2007; Ritz, 2009). In the current thesis, vagal control of the heart will be interpreted as activation of the PNS without specifically attributing activation to the VVC or DVC. It should be noted at this point that there are several terms that can be used to describe measures of vagal activity, including HRV, RSA, and vagal tone. Differences in terminology do not always imply differences in measurement, although more than one methodology can be used to calculate vagal activity.
HRV can be calculated by generating a time series of the R-R intervals from an ECG to capture the variation in R-peaks. R-R intervals decrease when the heart is beating faster and increase when the heart is beating slower. The variation of the R-R intervals is what determines HRV (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). There are two main approaches that can be used to quantify HRV: time-domain methods and frequency-domain methods. There is no consensus on which methodology is most appropriate for measuring HRV (see Grossman & Taylor, 2007; Grossman, van Beek, & Wientjes, 1990; and Porges, 1986), and most measures of HRV seem to be relatively comparable (often correlating above $r = .90$, Grossman et al., 1990; Pumprla, Howorka, Groves, Chester, & Nolan, 2002). More often than not it is the research design that will determine the methodology for quantifying HRV. In the following experiments the frequency domain method was used to calculate HRV, as the Task Force (1996) recommended using frequency-domain methods rather than time-domain methods for short-term recordings.

![Figure 3.2](image)

**Figure 3.2.** Example of a heart rate (HR) power spectrum. Hz = hertz. LF = low-frequency. HF = high-frequency. RSA = respiratory sinus arrhythmia.

The frequency-domain method used in the following experiments involved the application of a fast Fourier transformation to the cardiovascular time series, which resulted in power spectral analyses (CARSPAN program, Groningen, The Netherlands; Mulder et al., 2007). An example of a heart rate power spectrum is shown in figure 3.2. Once a power spectrum has been generated it is possible to identify different peaks in the spectral analysis that fall into different frequency domains. These different frequency domains correspond to one or more physiological processes. The power
represented in the different frequency bands can be used to assess short-term cardiovascular functioning (Berntson et al., 1997). The high-frequency (HF) band of HRV (0.15 to 0.4 Hz) largely reflects the variations in sinoatrial control associated with respiration. The power in the HF power band consequently corresponds to RSA and is generally considered a marker of parasympathetic input. Conversely the low-frequency (LF) band (0.05 to 0.15 Hz) contains variability attributable to both sympathetic and parasympathetic rhythms. Due to the lack of clarity about this bandwidth, the LF domain has received less attention in the psychophysiological literature (Sokolov & Cacioppo, 1997).

The power in the HF band is considered the most important component of the HRV power spectrum in our research, because as well as being an index of cardiac vagal control it is also suggested that HF-HRV is a biological marker for the accessibility of the social engagement system (Porges, 1998, 2003a). The use of HRV as a marker for generalised vagal efferent activity is not without criticism however. HF-HRV is only a marker of cardiac vagal tone, and if one is being conservative then inferences made using HRV should only relate to the function of the heart and not the wider ANS (Ritz, 2009). Despite this caveat, cardiac vagal tone is still a known marker of PNS function, and HF-HRV has been linked to the self-regulation of cognition, emotion, and physiology (Segerstrom & Solberg Nes, 2007; Thayer & Brosschot, 2005; Thayer & Lane, 2009). This makes HF-HRV an informative measure with or without consideration of the social engagement system.

Research suggests that short-term measures of HRV tend to be fairly stable within individuals. For example Sinnreich, Kark, Friedlander, Sapoznikov, and Luria (1998) reported a test–retest correlation of .76 for the high-frequency component of HRV when short-term recordings were carried out two months apart. It should be noted that a review by Sandercock, Bromley, and Brodie (2005) emphasised that short-term measures of HRV tend to be more reliable at rest than during interventions such as orthostatic tilt and pharmacological blockade, and that HRV tends to be more stable in healthy populations rather than clinical populations.

In the following studies, HRV was calculated by extracting the interbeat intervals (IBI) of the ECG from AcqKnowledge 4.1 (BIOPAC Systems; Goleta, CA), which resulted in an IBI time series. Before subjecting the IBI time series to a fast
Fourier transformation the R-R data were scrutinized for artefacts. This process covered two criteria: (1) if an interbeat interval deviated more than five standard deviations from the running mean, or (2) the difference between two consecutive interbeat intervals was larger than five standard deviations, then a new interbeat value (based on R-peak times) was calculated using an interpolation between two preceding and two succeeding correct values (CARSPAN program, Groningen, The Netherlands; Mulder, Hofstetter, & van Roon, 2007). Once the time series had been corrected for artefacts a discrete fast Fourier transformation was applied, based on the non-equidistant sampling of the R-wave incidences, which resulted in an HRV power spectrum (CARSPAN program, Groningen, The Netherlands; Task Force, 1996). HRV power (ms$^2$) was calculated for the high-frequency band (HF-HRV: 0.15-0.4Hz) only.

3.4.4. Summary of Physiological Recordings

In summary, the physiological measures used in the current experiments were set up using the following protocol: Continuous measurements were made of heart rate (HR) and skin conductance level (SCL) using a BIOPAC™ MP150 system connected to a computer running AcqKnowledge 4.1 software (BIOPAC Systems; Goleta, CA). HR was recorded using two disposable Ag/AgCl electrodes positioned in a type II configuration and was sampled at 512Hz. SCL was measured using two grounded Ag/AgCl electrodes attached to the medial phalanx of the index and ring fingers of the non-dominant hand and was sampled at 125Hz. Analysis of the HR and SCL data was done using AcqKnowledge 4.1 (BIOPAC Systems; Goleta, CA). To assess heart rate variability (HRV), interbeat intervals (IBI) of the ECG were calculated using R-top detection in AcqKnowledge, which resulted in an IBI time series. The IBI time series was subjected to a discrete Fourier transform, based on non-equidistant sampling of the R-wave incidences, which resulted in an HRV power spectrum (CARSPAN program, Groningen, The Netherlands; Task Force, 1996). HRV power (ms$^2$) was calculated for the high-frequency band (HF-HRV: 0.15-0.4Hz) only. Note that in the experiments evaluating the physical presentation of the speech, HF-HRV was not calculated for the presentation period itself. Talking aloud is known to affect the reliability of HRV indices making it an unsuitable measure during tasks that involve speech (Beda, Jandre, Phillips, Giannella-Neto, & Simpson, 2007; Tininenko, Measelle, Ablow, High, 2012).
3.5. Self-Report Measures of Socio-Emotional Responding

Porges claims that the function of the ANS should influence one’s emotional state and vice versa (Porges, 1999, 2009a). One of the main predictions of this thesis is that up-regulation of the SNS should increase negative affect, whilst down-regulation of the SNS should decrease negative affect. The subjective experience of emotion is one of the most salient aspects of emotional responding, and it can be measured by asking participants to report how they are feeling. This is often achieved using standardised self-report measures that assess several domains of the emotional experience. Measures of subjective emotion can be categorical i.e., they assess a broad range of discrete emotions that tend to cluster together to form higher order factors (e.g. see Ekman, 1992), or they can be dimensional i.e., they assess a narrow range of fundamental dimensions that organise into emotional responses (dimensions such as valence and arousal; Bradley & Lang, 1994; Russell, 1994).

Measures of subjective emotion can measure either trait emotionality (i.e., one’s tendency to respond positivity or negatively to stimuli) or state emotionality (i.e., the moment-to-moment experience of emotion). The latter is more useful when researching short-term changes in mood, particularly when an emotional response is directed at a particular event/stimulus. The timing of state measures is important, as it has been argued that self-reports of recent emotional responding (particularly if they target the current moment) are more valid than self-reports concerning past, future, or trait emotional responding (Robinson & Clore, 2002). A challenge of using subjective measures of emotion, is that some participants can find it difficult to articulate how they are feeling (e.g., individuals with Alexithymia; Pollatos, Schubö, Herbert, Matthias, & Schandry, 2008) whereas others can report being disconnected from the experience of emotion altogether (e.g., individuals in dissociative states; Bremner et al., 1998). Although a stimulus may affect changes in behaviour, they may not be directly attributed to changes in mood (Bonanno & Keltner, 2004). Even when participants are able to report on emotional states, it is possible that self-report measures may not be sensitive enough to measure changes in emotional responding. Moreover, social desirability can play a role in how people respond to self-report measures of emotion, which again affects their validity (Feldman Barrett, 1996).
Despite the methodological limitations inherent in using self-report measures, the experiential component of an emotion is integral to an emotional response. Failure to account for subjective experience when studying emotion may mitigate or bias results. Thus, in the current experiments several established self-report measures of emotion were used: The Profile of Mood States – Short Form (Shacham, 1983) was used to capture short-term changes in emotion; to control for trait emotionality the depression scale from the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) or the Beck Depression Inventory – II (Beck, Steer, & Brown, 1996) were used to measure depression symptoms, and the anxiety scale from the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) or the 7-item Generalized Anxiety Disorder Scale (Spitzer, Kroenke, Williams, & Löwe, 2006) were used to measure anxiety symptoms; finally additional measures were used to capture self-reported behavioural expressivity (the Berkeley Expressivity Questionnaire; Gross & John, 1995), emotion regulation difficulties (the Difficulties in Emotion Regulation Scale; Gratz & Roemer, 2004), psychological flexibility (the Acceptance and Action Questionnaire-II; Bond et al., 2011), and social safeness (the Social Safeness and Pleasure Questionnaire; Gilbert et al., 2009). The measures used in the current experiments and their psychometric properties are discussed in the following sections:

3.5.1. Demographic Screening Questionnaire (see appendix 1)

To capture sociobiographic information, all participants completed a demographic screening questionnaire at the beginning of the testing session. Participants reported their age, gender, education status, ethnicity, smoking status, coffee consumption, and past or current treatment for psychological or psychiatric problems. Participants who reported past or current treatment for psychological or psychiatric reasons were excluded from taking part in the experiments. The other factors reported in the questionnaire were analysed as possible covariates.

3.5.2. Profile of Mood States – Short Form Questionnaire (see appendix 2)

The Profile of Mood States – Short Form (POMS-SF; Shacham, 1983) is an abbreviated version of the Profile of Mood States (POMS) scale developed by McNair, Lorr, and Droppleman (1971). The POMS-SF measures six domains using 37 items (Fatigue–Inertia, Vigour–Activity, Tension–Anxiety, Depression–Dejection,
Anger–Hostility, and Confusion–Bewilderment). The subscales can also be used to calculate a total distress score (often referred to as total mood disturbance). Respondents indicate the degree to which each adjective describes themselves right now using a five-point Likert scale (0 not at all, 4 extremely). In the following studies the POMS-SF was used to assess current mood state by applying the how are you feeling right now variant of the questionnaire. This is because using short-term instructions amplifies the questionnaire’s sensitivity to change (Rossi & Pourtois, 2011).

An advantage of using the shortened version of the POMS is that it takes less time to complete than the original version, and there is some evidence to suggest that the Tension–Anxiety subscale of the 37-item version is psychometrically superior to that of the original POMS (Curran, Andrykowski, & Studts, 1995; Rossi & Pourtois, 2011). Shacham (1983) initially reported that the internal consistency estimates of the six subscales of the POMS-SF ranged from .80-.91. Similar levels of internal consistency have been reported in studies sampling both healthy (Curran, Andrykowski, & Studts, 1995) and clinical populations (Baker, Denniston, Zabora, Polland, & Dudley, 2002). A further consideration of using the POMS-SF is its construct validity. Baker et al. (2002) carried out a confirmatory factor analysis that supported the six-factor structure of the POMS-SF. By maintaining the six subscales of the original POMS, the POMS-SF maintains a good level of specificity whilst also being able to provide a total distress score. The main rationale for using the POMS-SF in the current experiments is that the questionnaire is able to capture changes in state anxiety (an indicator of negative affect), as well as other changes in other mood states (Rossi & Pourtois, 2011).

3.5.3. Hospital Anxiety and Depression Scale (see appendix 3)

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is 14-item self-report measure of depression and anxiety symptoms. Each item is rated from 0 to 3 according to severity. The measure results in two scores: A depression score (HADS-D) and an anxiety score (HADS-A). Each subscale score can range from 0 to 21, with higher scores indicating higher levels of difficulty. Both factors of the HADS have demonstrated acceptable internal consistency in clinical populations (HADS-D alpha 0.84, Cameron, Crawford, Lawton, & Reid, 2008; HADS-A alpha 0.81, Spinhoven et al., 1997) and healthy populations (HADS-D alpha 0.82, HADS-A alpha 0.77; Crawford, Henry, Crombie, & Taylor, 2001). In addition to this, the HADS
subscales have demonstrated high test–retest reliabilities (HADS-D alpha 0.86, HADS-A alpha 0.89; Spinhoven et al., 1997).

Research has supported the two-factor structure of the HADS, but the scales are heavily correlated (for a review see Bjelland, Dahl, Haug, & Neckelmann, 2002). Both scales not only correlate with each other, but they also correlate interchangeably with other measures of depression and anxiety (Savard, Laberge, Gauthier, Ivers, & Bergeron, 1998). This may suggest that the HADS is more suited as a measure of symptoms that co-vary across depression and anxiety, rather than a measure that is able to distinguish depression from anxiety (Sphoven et al., 1997). Most of the contention seems to lie with the anxiety scale. To avoid overlap with general medical conditions, the HADS-A subscale minimises the significance of physical arousal, meaning it demonstrates poor specificity (Sphoven et al., 1997). The HADS-A also tends to overestimate the extent of anxiety symptoms in student populations (Andrews, Hejdenberg, & Wilding, 2006). The HADS was used in the first two empirical studies as a trait measure of depression and anxiety, but was later replaced with the Beck Depression Inventory – II (see section 3.5.4) and the Generalised Anxiety Disorder Scale (see section 3.5.5).

3.5.4. Beck Depression Inventory-II (see appendix 4)

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item measure of depressive symptoms. Scores can range from 0-63, with higher scores representing greater depression severity. The BDI has been found to correlate well with other measures of depression (such as the HADS-D; Savard et al., 1998; Tedman, Young, & Williams, 1997). An advantage of the BDI-II is that it has very robust psychometric properties, and is a useful measure of depressive symptoms even in student populations. Storch, Roberti, and Roth (2004) used confirmatory factor analysis with a student sample and identified a two-factor structure for the BDI-II: A cognitive-affective factor and a somatic factor. Both factors demonstrated good internal consistency (cognitive-affective alpha .87; somatic alpha .74), as did the total score (alpha .90). In addition to this, Sprinkle et al. (2002) examined the utility of the BDI-II in a sample of students receiving treatment at a university counselling centre. They found that BDI-II scores correlated highly with the number of depression items endorsed from the major depressive episode section of the Structured Clinical Interview
for the DSM-IV \( (r = .83) \). In a second sample of students, Sprinkle et al. also found a test–retest reliability of .96 for the BDI-II when it was completed over two separate sessions (intervals ranged from 1-12 days apart). In the current experiments participants with self-reported depression were excluded prior to testing, so this measure was used to infer trait levels of depression symptomatology only.

### 3.5.5. Generalized Anxiety Disorder Scale (see appendix 5)

The seven-item Generalized Anxiety Disorder Scale (GAD-7; Spitzer, Kroenke, Williams, & Löwe, 2006) is designed to assess general anxiety symptoms. Scores can range from 0-21, with higher scores representing higher anxiety symptoms. Although the scale was designed to detect symptoms of general anxiety disorder, the domains measured are not exclusive to this disorder, and the scale has been shown to also be a sensitive index of panic, post-traumatic stress disorder, and social phobia (Kroenke, Spitzer, Williams, Monahan, & Löwe, 2007). A strength of the GAD-7 is its focus on general anxiety symptoms rather than worry symptoms (cf. the Penn State Worry Questionnaire; see Dear et al., 2011). The GAD-7 is also a pure measure of anxiety, unlike other measures that have been shown to inadvertently assess depression symptoms as well (cf. the State–Trait Anxiety Inventory, see Bieling, Antony, & Swinson, 1998; and the HADS-A, see Savard et al., 1998). Spitzer et al. (2006) initially reported that the GAD-7 had high internal consistency (alpha .92) and a test–retest reliability of .83. Dear et al. (2011) confirmed the single factor structure of the GAD-7 using factor analysis, and verified that the internal consistency of the GAD-7 is acceptable in clinical populations (pre- and post-treatment alphas ranged from .79-.91). In the following experiments participants with self-reported anxiety disorders were excluded prior to testing, so this measure was used to infer trait levels of anxiety only.

### 3.5.6. Berkeley Expressivity Questionnaire (see appendix 6)

The Berkeley Expressivity Questionnaire (BEQ; Gross & John, 1995) is a 16-item measure of individual differences in emotional expressivity. Respondents indicate the degree to which each item describes themselves using Likert scales ranging from 1 \( \text{it does not apply to me at all} \) to 7 \( \text{it applies to me completely} \). The questionnaire assesses three facets of emotional expressivity: The \textit{negative expressivity} subscale consists of six items referring to the degree to which a person tends to express negative emotions (e.g.,
"I've learned it is better to suppress my anger than to show it"); the positive expressivity subscale consists of four items indicating the degree to which a person tends to express positive emotions (e.g., "Whenever I feel positive emotions, people can easily see exactly what I am feeling"); and the impulse strength subscale consists of six items which indicate the general strength of emotion (e.g., "I have strong emotions"). The subscales can also be used to calculate a general expressivity score.

The psychometric properties of the BEQ have been explored in several studies. In the original study, Gross and John (1995) reported that the Cronbach’s alphas for the subscales ranged from .68-.78, and that after a 2 month interval the scales demonstrated a test–retest reliability of .71 and above. In a follow up study Gross and John (1997) reported similar Cronbach’s alphas for the subscales using both self-report and observer versions of the questionnaire. They also found significant correlations between scores on the BEQ and participant’s emotionally expressive behaviour whilst they watched emotion-inducing film clips. The three factor structure of the BEQ has been confirmed using confirmatory factor analysis (Gross & John, 1997), although not everyone has replicated these findings, for example Dobbs, Sloan, and Karpinski (2007) did not find support for the three factor structure of the BEQ. A notable criticism of the BEQ is that the positive and negative expressivity scales represent some emotions better than others, for example Trierweiler, Eid, and Lischetzke (2002) suggest that the negative expressivity scale does not address the emotions of anger and shame. Although the BEQ has these limitations, Gross and John (1998) have found substantial correlations between the BEQ and other expressivity scales, suggesting that self-report measures of emotional expressivity share a common understanding of what constitutes the core domain of emotional expressivity. It has been suggested that to maximise the accuracy of self-report measures of emotional expressivity they should be combined with other methodologies, such as observation (Dobbs et al., 2007).

3.5.7. Difficulties in Emotion Regulation Scale (see appendix 7)

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item measure used to assess six facets of difficulties in regulating emotion: Non-acceptance of emotional responses, difficulties engaging in goal-directed behaviour, impulse control difficulties, lack of emotional awareness, limited access to effective emotion regulation strategies, and lack of emotional clarity. Participants rate each item
on a scale from 1 (*almost never, 0–10%*) to 5 (*almost always, 90-100%). The subscales can be used individually, or can be summed to calculate a total score, with higher scores being indicative of greater difficulties in emotion regulation.

Gratz and Roemer (2004) originally explored the psychometric properties of the DERS using a sample of undergraduate students. The DERS demonstrated high internal consistency for the total score (Cronbach’s alpha .93), adequate internal consistency for the subscales (Cronbach’s alphas above .80), and good test–retest reliabilities over a four-week period (\(\rho_i = .88, p < .01\)). Although the DERS has since been used in several studies using clinical populations (for example anxiety samples Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006; and depression samples Ehring, Fischer, Schnu, Bo, & Tuschen-Caffier, 2008), few studies have questioned the psychometric properties of the DERS. Data cited by Ehring et al. (2008) suggests that a German translation of the DERS has demonstrated similar psychometric properties to the original: Principle components analysis confirmed the six factor structure of the DERS, the subscales were reported to show good internal consistency (alpha’s ranging from .81-.95), and the subscales showed good stability over a two week period (alpha’s ranging from .72-.87). The DERS was also reported to correlate with other measures of emotion regulation.

3.5.8. Acceptance and Action Questionnaire-II (see appendix 8)

The Acceptance and Action Questionnaire – II (AAQ-II; Bond et al., 2011) is a brief seven-item scale designed to measure experiential avoidance and psychological inflexibility. Respondents indicate the degree to which each item describes themselves using a seven-point Likert scale (1 *always true*, 7 *never true*). Scores can range from 7-49, and in the current experiments the items were reverse-scored so that higher scores reflected greater psychological flexibility.

The original version of the Acceptance and Action Questionnaire had 16 items and was a predictive measure of outcomes such as depression, anxiety, and general mental health (for a review see Hayes, Luoma, Bond, Masuda, & Lillis, 2006). However its psychometric properties were modest; the scale had a Cronbach’s alpha of .70 and a test–retest reliability of .64 after a four-month interval (Hayes et al., 2004). Reducing the number of items in the AAQ–II improved the reliability of the questionnaire. Bond
et al. (2011) evaluated the AAQ-II as a measure of psychological flexibility using six samples, and reported superior internal consistencies for the revised scale (Cronbach’s alphas ranged from .78-.88), and a test–retest reliability of .81 at three months and .79 at 12 months. Confirmatory factor analysis with three of the samples also confirmed the single-factor structure of the AAQ–II.

3.5.9. Social Safeness and Pleasure Scale (see appendix 9)

The Social Safeness and Pleasure Scale (SSPS; Gilbert et al., 2009) is a scale designed to measure the extent to which people experience their social worlds as safe, warm, and soothing. The eleven items relate to feelings of belonging, acceptance, and feelings of warmth from others (e.g., “I feel content within my relationships” and “I feel secure and wanted”). Responses are made on a five-point Likert scale ranging from 0 almost never to 4 almost all the time. Scores can range from 0-44, with higher scores indicating higher social safeness.

The SSPS has not yet been psychometrically evaluated in full, and has so far only been used with student populations. In their original study, Gilbert et al. (2009) reported that the scale had a Cronbach’s alpha of .91, and verified the single factor structure of the scale using exploratory factor analysis. In terms of test–retest reliability, a more recent study by A. C. Kelly, Zuroff, Leybman, and Gilbert (2012) used the SSPS as a daily measure of social safeness over a seven day period. Scores for the SSPS demonstrated little variability during the study suggesting that the SSPS is a trait measure of social safeness (A. C. Kelly et al., 2012). Despite being a relatively stable measure, social safeness was reported as higher on days when participants also reported more received social support. Consequently the SSPS is an affective measure that relates to affiliative behaviour (although the direction of the relationship between social safeness and affiliative responding has yet to be determined).

3.6. Behavioural Measures of Socio-Emotional Responding

Several of the main hypotheses in this thesis are targeted at investigating the relationship between physiology and socio-emotional behaviours. This chapter has already introduced methodologies that can be used to infer activation of the SNS and PNS, as well as measures that can be used to assess the subjective component of
emotional responding. This last section addresses several methods that can be used to measure socio-emotional behaviour. Socio-emotional behaviours facilitate communication and social engagement, and are usually measured in terms of two core competencies: The successful *production* of emotional facial expressions, gestures and actions (known as *encoding ability*), and the successful *perception* and interpretation of such gestures (known as *decoding ability*; Riggio, 1986; Scherer, 2007). Deficits in these competencies have been linked to impaired communication and reduced success in social interactions, which can have implications for health. For example, deficits in encoding and decoding emotions have been associated with depression (Gehricke & Shapiro, 2000; Segrin, 2000), autism (Travis & Sigman, 1998), and schizophrenia (Gaebel & Wölwer, 1992). Impaired social functioning has also been shown to influence one’s willingness to spend time with others (e.g., decreased facial expressivity reduces feelings of rapport and affiliation; Butler et al., 2003). Consequently, the current research needs to consider both the sending and receiving aspects of socio-emotional behaviours, and how they affect interpersonal functioning. This section will review behavioural measures of emotional expressivity (section 3.6.1), followed by behavioural measures of emotional sensitivity (section 3.6.2), and will conclude with behavioural measures of affiliation (section 3.6.3).

### 3.6.1. Emotional Expressivity

The experience of an emotion is often associated with a display of emotion; this is most apparent through changes in facial expression. Emotions can result in changes in facial activity that are observable (Ekman, Friesen, & Ancoli, 1980), as well as changes that are invisible to the naked eye (Cacioppo, Petty, Losch, & Kim, 1986; Dimberg, 1990; Dimberg, Thunberg, & Elmehed, 2000). Distinct patterns of facial expression have been found to correspond with discrete emotions (e.g., Ekman, Freisen, & Ancoli, 1980; Weiss, Salloum, & Schneider, 1999), although observable displays of emotion do not always correspond with the felt emotional experience (see Bonanno & Keltner, 2004). This is particularly noticeable when one considers display rules, the cultural norms that dictate whether or not it is acceptable to display certain emotions in particular contexts (Ekman & Friesen, 1969).

Facial expressions are not only a concomitant of experiencing an emotion, but also play a role in regulating emotion. This concept goes back to as far as Darwin
(1872/2009), and was later taken up by Gellhorn (1964) and Izard (1971). Afferent signals from facial muscles can elicit ANS changes as well as changes in the emotional experience (Hennenlotter et al., 2009; Levenson, Ekman, & Friesen, 1990). This corresponds to the notion that facial expressions are not only a readout of our emotional experience, but they also have a feedback role which shapes how the emotional experience changes over time (see Buck, 1980). For further discussion on the function of facial expressions in this thesis see Chapter 6.

Many have suggested that emotional expressions have a biological basis. This notion is reinforced when one looks at the universality of emotional facial expressions. Ekman and colleagues (Ekman, 1972; 1992; Ekman & Friesen, 1969, 1971; Ekman et al., 1987; Ekman, Sorenson, & Friesen, 1969) have found evidence of six basic emotions that are inherent across cultural and geographic divides (anger, fear, happiness, disgust, sadness, and surprise); other emotional categories are often marked as variants or blends of the basic prototypes (Ekman et al., 1987; Ekman, 1992b). There is some debate as to whether or not emotions are categorical and therefore have discrete facial expressions (Ekman et al., 1980; Etcoff & Magee, 1992; Young et al., 1997), or whether emotions and their corresponding facial expressions can be considered dimensional (e.g., expressed in terms of valence and arousal; Russell, 1994). Despite a lack of consensus regarding the organisation and primary function of facial expressions, the measurement of facial motor activity is popular in emotion research.

It is generally accepted that certain anatomical facial movements (known as action units) are coupled with certain emotional responses (e.g., genuine smiles are associated with positive affect; Ekman & Friesen, 1982; Soussignan, 2002). Specific patterns of facial movements can be captured using a variety of techniques to infer the presence of an emotion. The current studies used emotion recognition software and electromyography as measures of behavioural expressivity. The rationale for these measures will be provided in the following sections.

3.6.1.1. Emotion recognition software. Emotion recognition software is a useful measure of facial activity when more invasive measures of facial expression may be inappropriate, for example when experimental designs do not allow for obstruction of the face, making the use of electrodes inappropriate. Emotion recognition software tends to use algorithms that replicate facial coding systems used by human observers,
such as Ekman and Friesen’s Facial Action Coding System (FACS; Ekman & Friesen, 1978; Ekman, Friesen, & Hager, 2002). By mapping the contours of the face, software can be used to ‘read’ emotions. Often software will adhere to the basic classifications of anger, fear, sadness, happiness, surprise, and disgust. An advantage of emotion recognition software is that it is more objective than subjective observers, however such technology can be costly and it relies on good quality video (for example faces need to be clearly visible and can be affected by artefacts such as movement). Although software can be very good at detecting facial movements, arguably most software is not very adept at detecting other emotional gestures (such as head nodding or shrugging shoulders), and they do not tend to interpret gaze direction.

In Chapter 6 participants were filmed modulating their facial expressions, and the videos were analysed for emotional expressivity using Visual Recognition’s eMotion software (version 1.21), developed at the University of Amsterdam (Gevers, 2008; see also Sebe et al., 2007). This software is able to categorise how fully six basic emotions (happiness, sadness, disgust, surprise, fear, and anger) are expressed in videos of facial activity. The absence of emotion is also categorised as neutral expression. Once a file has been targeted in the eMotion software, the user is able to map a mesh computer model onto the features of the face in the video (see figure 3.3). eMotion calculates the level of facial expressivity in the video by evaluating the moving vectors of the facial points, identifying movements such as curvature of the lips, raising of the eyebrows, and cheek contraction (in accordance with Ekman et al.’s, 2002, FACS). The output of eMotion is a text file that contains the percentage of each of the six emotions and the percentage of neutral expression shown within each frame of the video.
Figure 3.3. Screenshot from the eMotion software. The green line shows the markers of the computer model that can be adjusted to align the model to the contours of the face.

The videos in the current experiments were digitalised with a frame rate of 25 frames per second, resulting in 250 frames of eMotion output per 10-second video. Indices of positive and negative emotion were created by combining the percentages of the basic emotion scales: The positive emotion index consisted of the happy and surprise scales; the negative emotion index consisted of the sadness, disgust, fear, and anger scales. The positive and negative indices of emotional expressivity were used to calculate an expressive enhancement ability score, whilst the neutral scale was used to calculate an expressive suppression ability score (see Chapter 6 for more details).

3.6.1.2. Electromyography. Electromyography (EMG) is a physiological measure of facial muscle activity. This measure has been placed in the behavioural section of this chapter, because in the current experiments emotional expressivity is conceptualised as a behavioural outcome rather than an index of ANS function. EMG is a useful method for measuring emotional expressivity because the EMG signal is instantaneously recorded, meaning that the data does not require a large amount of visual inspection or coding from observers which can be costly and time consuming. EMG is extremely sensitive to changes in muscle activity. EMG amplitude has been shown to provide a reliable and valid index of muscle action, even when there are no perceptible muscle contractions (Cacioppo et al., 1986). This is important, as rapid automatic and unconscious facial movements can occur in response to emotive stimuli, which can be easily missed using observation methods (Dimberg, 1990).
Three recording sites on the face have been identified as valid and reliable indices of specific muscle actions that are able to differentiate between emotional processes (Fridlund & Cacioppo, 1986; Tassinary, Cacioppo, & Green, 1989): The *corrugator supercilii* (CS) is the muscle responsible for brow furrowing and is typically related to negative affect (Tassinary & Cacioppo, 1992); the *zygomaticus major* (ZM) is the cheek muscle which retracts the corner of the mouth and is typically related to positive affect (Tassinary & Cacioppo, 1992); the third muscle is the *levator labii* (LL) which pulls up the top lip and is seen in prototypical facial expressions of disgust (Vrana, 1993). In Chapter 7, EMG was used to measure the level of mimicry shown by participants in response to facial displays of emotion.

### 3.6.2. Emotional Sensitivity

Emotional sensitivity refers to the threshold at which emotions can accurately be recognised (Lynch et al., 2006). Although emotional sensitivity can be measured in a range of modalities, most research has focused on the recognition of emotion from facial expressions. Arguably facial displays are the most salient signals of emotion and they are the easiest to decode (Wallbott, 1998; Wallbott & Scherer, 1986). A common measure of emotion recognition using facial displays involves presenting participants with photographs of prototypical facial expressions, which participants are then asked to categorise (e.g., the Pictures of Facial Affect, Ekman & Friesen, 1976). The use of static photographs in emotion research has been heavily criticised however (see Adolphs, 2006; Blairy, Herrera, & Hess, 1999; Hess & Blairy, 2001). First of all, photographs of facial displays tend to depict intense, prototypical emotional expressions, and these are often presented for several seconds at a time (Hess & Blairy, 2001). This has been contrasted to real life displays of emotion, where faces are dynamic and display a large range of expressions that are often low in intensity and short in duration (Blairy et al., 1999). Static pictures therefore have questionable ecologically valid. A second criticism of asking participants to categorise prototypes of emotional expressions is that this method measures *emotional accuracy*, and not emotional sensitivity (Wehrle, Kaiser, Schmidt, & Scherer, 2000). As most batteries of facial displays are typical emotional expressions that are easily categorised, this can lead to ceiling effects in healthy populations (Coupland et al., 2004).
To overcome these limitations several approaches have been adopted. One approach has been to modify static images to make recognising the emotions more difficult, such as isolating parts of the face (e.g., the Reading the Mind in the Eyes Task; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). These techniques still lack ecological validity however, as in real life situations faces are normally perceived whole (K. J. Kelly & Metcalfe, 2011). Another technique has been to morph or blend photographs of different emotions to produce graded levels of expression (Calder, Young, Perrett, Etcoff, & Duncan, 1996). Blended emotions are more difficult to categorise than pure emotions; the closer a face is to the prototype emotion, the easier it is to correctly identify (Calder et al., 1996; Young et al., 1997). Although blended facial expressions are often more true to life than prototypical expressions, this methodology still lacks ecological validity if the blended emotions are presented as static images. The role of motion is increasingly being emphasised in emotion recognition research, as facial action units have been shown to be important emotional cues (Kilts, Egan, Gideon, Ely, & Hoffman, 2003). Facial action units are visible in dynamic presentations of emotional expressions, and using moving facial stimuli has been shown to have a facilitative effect on emotion recognition (Wehrle et al., 2000). A major advantage of using dynamic presentations is that facial displays of increasing emotional intensity can be used to measure the threshold at which an emotion is recognised i.e., these tasks can be used to measure emotional sensitivity as well as emotional accuracy (Blair, Colledge, Murray, & Mitchell, 2001; Wehrle et al., 2000).

3.6.2.1. Multimorph Facial Affect Recognition Task. In the current experiments the Multimorph Facial Affect Recognition Task (Blair et al., 2001) was identified as an appropriate measure of emotional sensitivity. The Multimorph Facial Affect Recognition Task (Blair, Colledge, Murray, & Mitchell, 2001) is based on the Pictures of Facial Affect (Ekman & Friesen, 1976). The Pictures of Facial Affect consist of 110 still photographs of emotional facial expressions. All of the photographs are black and white, and show actors producing facial configurations that relate to six basic emotions: Happiness, sadness, fear, anger, disgust, and surprise. The photographs have been validated in numerous studies, with research showing high agreement across cultures in the assignment of the six basic emotions to the corresponding facial expressions (see Ekman, 1989). The Pictures of Facial Affect are often used as a basic measure of emotion recognition; participants simply have to identify the emotion shown in the photograph. Consequently the principle measure of this battery in its original
form is the percentage of correct judgments made for each emotion. As previously mentioned, a drawback of the Pictures of Facial Affect is that they are prototypical, high intensity, still photographs that lack ecological validity (Hess & Blairy, 2001).

The Multimorph Task (Blair et al., 2001) overcomes the limitation of the Pictures of Facial Affect battery by using collections of the original stimuli to create dynamic presentations of emotional facial expressions. This effect is achieved by blending the photographs of the prototypical emotional facial expressions (i.e., 100% emotion) with the corresponding individual’s demonstration of a neutral expression (0% emotion). In practice, the Multimorph Task requires participants to identify the emotional expression shown whilst the facial expression changes from a neutral to an emotional expression. Each trial begins with a neutral face, which gradually morphs through 39 stages of 450 ms each into one of the six prototypic emotional expressions (happiness, sadness, fear, anger, disgust, and surprise). In total there are 36 stimuli; the six distinct emotional expressions are each portrayed by three male and three female actors. The advantage of the design of the Multimorph Task is that the tool assesses speed of recognition (i.e., the mean number of stages required to achieve the correct classification of emotion), as well as the level of accuracy for the emotion at 100% expression.

3.6.3. Affiliation Tendencies

The term affiliation describes enjoying and valuing close interpersonal bonds, and being warm and affectionate (Depue & Collins, 1999). A distinction has been made between affiliation as a disposition (Depue & Morrone-Strupinsky, 2005; Hill, 1991), and affiliation as a set of behaviours that emerge during social interactions (Gonzaga, Keltner, Londahl, & Smith, 2001; Gump & Kulik, 1997; Park & Maner, 2009). A challenge of this distinction is that although participants may have an affiliative disposition, they may not have the opportunity to express affiliative behaviours during experimental conditions (Depue & Morrone-Strupinsky, 2005). This is important to consider when choosing affiliation measures.

The current experiments were designed to evaluate the relationship between ANS function and state affiliation. As a result the current experiments required short-term indices of affiliation tendencies. One method that can be used to measure
affiliation is to observe the frequency and duration of affiliation cues during social interactions, such as looking behaviours, smiling, and nodding (Gonzaga et al., 2001; Gump & Kulik, 1997). A caveat of this method is that it is open to bias, as observers often have to make subjective inferences whilst coding behaviours (Luxen, 2005). Another method that can be used to measure affiliation is affiliate–choice paradigms; this is where participants are asked if they would prefer to be alone or with others during a phase of the experiment (for example Rofé, 1984; Schachter, 1959). A criticism of this technique is that the paradigm involves a forced choice (i.e., alone vs. with others), and any given response does not capture the strength of the willingness to spend time with others. To overcome this, the outcome of affiliate–choice paradigms can be made more complex. For example participants can be given a choice of more than one affiliation partner, or can be asked to indicate their preference for the available interactions (Hill, 1991; Li, Halterman, Cason, Knight, & Maner, 2008). Although this method is more reliable, the number of times participants can be presented with plausible interaction scenarios within one experiment is limited.

An alternative measure of affiliation is to ask participants to rate how much they want to spend time with others, or how willing they would be to form a friendship with others (Butler et al., 2003; Cheng & Chartrand, 2003; Park & Maner, 2009). Ratings are a quick and easy way of capturing the intensity of participants’ willingness to interact with others, and are a useful indication of affiliation tendencies when numerous measurements are required. In Chapter 8 participants were presented with videos of individuals responding to affective pictures (the Rating Faces Task, see section 8.2.2.1). Participants were asked to complete several rating scales, including how much time they would like to spend with the respondents on a seven-point scale (1 not much time to 7 a lot of time). This rating was averaged over a number of trials to indicate a general willingness to spend time with others.

3.7. Overview of the Empirical Chapters

To investigate the relationship between ANS function and socio-emotional responding in healthy adults, a series of laboratory-based experiments was conducted. Chapter 4 describes the effects of two stressor manipulations on ANS function and mood state using measures of heart rate, sweat response, high-frequency heart rate variability, and the Profile of Mood States – Short Form (Shacham, 1983). In Chapter 5
the influence of positive emotion regulation strategies on the down-regulation of arousal after a stressor manipulation was explored using the same measures.

The subsequent empirical chapters evaluate the effect of increased SNS activation on socio-emotional responding. Psychophysiology was measured during all of the experiments, and various combinations of the self-report measures were used to obtain state and trait indices of emotional responding. The experiments mainly differ in their use of behavioural outcome measures, which were repeated in each of the experiments before and after a stressor manipulation designed to increase SNS activation. In Chapter 6 eMotion (Gevers, 2008) was used to calculate facial expressivity in response to an expressive regulation task (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004); this experiment aimed to investigate the relationship between ANS function and expressive ability (i.e., encoding ability). In Chapter 7 the Multimorph Facial Affect Recognition Task (Blair et al., 2001) was used to investigate the relationship between increased SNS activation and emotional sensitivity (i.e., decoding ability). In Chapter 7 electromyography was also used to measure facial mimicry to further explore the relationship between SNS activation and facial expressivity. As a final measure of socio-emotional behaviour, in Chapter 8 participants completed a Rating Faces Task, which involved rating how much time they would be willing to spend with others. This was designed to investigate whether ANS function influences affiliation tendencies. As a conclusion to the thesis, a summary of the results and conclusions is presented in Chapter 9.
Chapter 4: The Psychophysiological Effects of Laboratory Stressors

Environmental stimuli elicit psychological and physiological changes in the body. This is because the body will automatically adapt and respond to external demands in order to promote survival (Porges, 2009b). More often than not, challenge will result in down-regulation of the parasympathetic nervous system (PNS) and up-regulation of the sympathetic nervous system (SNS), eliciting detectable changes in functions such as heart rate and sweat response. Laboratory stressors have been shown to be reliable tools for inducing arousal, a product of SNS activation (Hughes & Stoney, 2000; Schubert et al., 2009). As well as causing physical changes, stressors are also able to consistently increase negative emotion (Feldman et al., 1999). The psychophysiological changes that arise from being stressed in a laboratory are often thought to parallel the changes that occur when we encounter threats in the real world (but see Dimsdale, 1984). Common tasks used in laboratory settings to induce arousal include the passive viewing of film clips and affective pictures (e.g., Gross & Levenson, 1995; Lang, Bradley, & Cuthbert, 1999), as well as more active tasks such as mental arithmetic and speech tasks (Feldman, Cohen, Hamrick, & Lepore, 2004; Kirschbaum, Pirke, & Hellhammer, 1993).

When investigating the psychophysiological sequelae of stressors, it is important to select an appropriate stressor manipulation. As discussed in Chapter 2, there are two main opposing domains of stress response: mobilisation (e.g., fight–flight behaviours involving the SNS) and immobilisation (e.g., fright, freeze, and faint behaviours involving the SNS and/or the PNS). Most laboratory stressors are associated with mobilisation responses; human immobilisation responses in the laboratory have received less attention (Schmidt, Richey, Zvolensky, & Maner, 2008). This is possibly because techniques have precluded the measurement of freezing responses, but may also be a factor of most laboratory stressors being too insignificant to initiate pure defensive immobilisation, particularly as the strength of laboratory stressors are limited by ethical constraints. As an aside, it is worth mentioning that researchers are increasingly investigating freeze responses in laboratory settings (Roelofs, Hagenaars, & Stins, 2010; Schmidt et al., 2008).

The focus of the current research was to identify a stressor manipulation that can be used to effectively induce a mobilisation response (i.e., increase SNS activation).
Although both active and passive stressors have been shown to induce mobilisation responses in the laboratory, active stressors are more likely to elicit physiological changes that are stronger in intensity because they require motivated action (Obrist, 1981; Tomaka, Blascovich, Kelsey, & Leitten, 1993). This has been demonstrated empirically, with active stressors resulting in more myocardial reactivity (i.e., detectable changes in the activity of the heart), and passive stressors eliciting more vascular reactivity (i.e., peripheral changes indexed by measures such as total peripheral resistance; Gregg, James, Matyas, & Thorsteinsson, 1999; Hartley, Ginsburg, & Heffner, 1999).

The aim of this study was to identify a stressor manipulation that would not only increase SNS activation during the task, but would also cause arousal to remain elevated after the task. This was so the stressor manipulation could be used in the following empirical studies to identify strategies that target the PNS, and to investigate the social consequences of defensive physiological states. As mentioned in Chapter 3, laboratory stressors that are ecologically valid are more likely to elicit changes in physiology that parallel real-world responding (Dimsdale, 1984). Virtual reality and social stressors have both been identified as ways of increasing ecological validity in laboratory-based experiments (Kamarck & Lovallo, 2003; Riva et al., 2007). As a result the current research investigated the effects of virtual reality and social stress on changes in physiology. In Experiment 1, two active stressors were identified and developed: A virtual reality maze and a speech task. The experiment used a within subjects design to test the following hypothesis:

**Hypothesis 1. Laboratory stressors will be associated with decreased PNS activation, increased SNS activation, and increased negative affect.**

For a task to be considered an effective laboratory stressor, participants needed to demonstrate decreases in PNS activation (i.e., suppressed high-frequency heart rate variability), increases in SNS activation (i.e., increased heart rate and sweat response) and increases in negative affect (i.e., self-reported changes in mood state). Although both tasks elicited measurable changes in physiology and self-reported mood state, only the speech task elicited changes that were clearly consistent with a mobilisation response. Experiment 2 focused on refining the speech task to increase its effectiveness in maintaining arousal after the completion of the task.
4.1. Experiment 1: The Psychophysiological Effects of Active Stressors

The active stressors investigated in this experiment were identified as ecologically valid ways of inducing stress in laboratory settings (Kamarck & Lovallo, 2003; Riva et al., 2007). Virtual reality (VR) environments are being increasingly used in laboratory-based research, as they allow researchers to simulate real-life environments as a means of enhancing mood inductions (Riva et al., 2007). Simulated environments can be tailored to promote different emotional experiences, and VR has been utilised to induce both positive and negative emotion (Riva et al., 2007). VR has also been used to create stressful environments that result in defensive physiological arousal (Meehan, Insko, Whitton, & Brooks, 2002). Because of its success, VR is also being increasingly used in therapeutic practice as a component of exposure-based treatments, for example VR has been used to treat fear of flying (Rothbaum, Hodges, Smith, & Price, 2000), substance abuse (Bordnick et al., 2008; Saladin, Brady, Graap, & Rothbaum, 2006), and spider phobias (Garcia-Palacios, Hoffman, Carlin, Furness, & Botella, 2002). An advantage of VR is that the software has strict control parameters, resulting in standardised presentations across participants, which increases the internal validity of experiments (Botella, Perpiña, Baños, & García-Palacios, 1998). Combining VR with psychophysiological measures enables researchers to look at how the properties of an environment can influence participants’ subjective experiences as well as their physiology.

In contrast to VR, speech tasks are a well-established method used to induce stress in laboratory settings. Speech tasks became popular laboratory stressors after their utility was demonstrated as part of the Trier Social Stress Test (TSST; Kirschbaum et al., 1993). Humans are sensitive to social challenge (Gilbert, 2001) and are influenced by feelings of control (Isowa, Ohira, & Murashima, 2006). The social-evaluative threat and uncontrollability factors inherent in public speaking are thought to drive the psychological and physiological responses induced by speech tasks (Dickerson & Kemeny, 2004). Several formats of speech task exist, for example the task has been successfully adapted for youths, allowing the speech to be prepared beforehand (Westenberg et al., 2009), whilst another format of the task allow for group administrations (von Dawans, Kirschbaum, & Heinrichs, 2011). The task’s ability to produce reliable changes in arousal and affect, even when the format is modified, explains why it is such a popular manipulation in stress-related research.
Both VR (Meehan et al., 2002) and speech tasks (Schubert et al., 2009) have previously been shown to elicit immediate physiological stress responses in laboratory settings. However, whether residual arousal remains once the tasks are terminated has yet to be explored. Research has shown that prior exposure to a stressor can have cardiovascular carryover effects (Kelsey et al., 1999), but there is less clarity on how residual arousal from the current stressors could affect subsequent performance on secondary tasks. The aims of Experiment 1 were to investigate the psychophysiological changes associated with each stressor task, and to evaluate arousal levels once the tasks had been terminated. The recovery period after the tasks was set as five minutes; this is because elevations in SNS activation over this duration would be sufficient to investigate the consequences of defensive physiological arousal in the subsequent studies.

4.2. Method

4.2.1. Participants

Thirty-six undergraduate psychology students volunteered to participate in the study and were awarded course credits as part of their undergraduate course requirements. Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders, current psychological or pharmacological treatment and a history of epilepsy or migraines. Five participants were excluded from taking part in the study: Three due to current antidepressant use, one due to past depression, and one due to a history of migraines; one participant withdrew from the study due to cybersickness during the VR maze. The final dataset comprises data from thirty participants (3 males, 27 females). They ranged in age from 18–30 with a mean age of 19.77 years ($SD = 2.54$). 90.0% of these participants identified themselves as Caucasian, 3.3% as Mixed, 3.3% as Asian, and 3.3% as Other.
4.2.2. Stressor Manipulations

4.2.2.1. The virtual reality maze. The VR maze was designed for this study using VR Worlds 2 (Psychology Software Tools Inc.). The program was used to simulate a series of corridors, which constituted an unsolvable maze. The maze was designed so participants could interact with the environment by walking around, moving their view to change their perspective, and were able to control opening and closing doors. Within the maze a guard dog was simulated and programmed to growl and follow the participants to encourage them to move around the maze quicker (see figure 4.1).

![Screen shot of the virtual reality maze with the guard dog.](image)

The VR maze was presented on a 17” monitor (screen size, 1024 x 768 pixels) connected to a desktop computer. Participants were told that they were going to experience a VR maze that they needed to try and solve by finding the exit. Instructions were provided on how to move around the maze using the mouse and keyboard. Once familiar with the controls, participants were given three minutes to solve the maze and were told that most people managed to find the exit within this time. Participants were not warned in advance of the guard dog and were given no further instructions.

4.2.2.2. The speech task. The speech task was based on the procedure used by Schubert et al. (2009). Participants were informed that they would have to prepare and present a three-minute speech in which they were to argue for and against the legality of euthanasia (Lepore, Allen, & Evans, 1993). During a three-minute preparation period the participants were given a list of bullet points on the topic of euthanasia and a pen and paper to help prepare their speech. A video camera was prominently displayed in
the room and the participants were informed that they would be filmed during the presentation of their speech so they could be evaluated later on several criteria, e.g., speaking clearly. After the preparation period the participants were instructed to present their speech to the video camera for three minutes. If the participants stopped talking before the end of the three minutes, they were asked to continue talking by summarizing the main points.

4.2.3. Performance Ratings

4.2.3.1. Post-task questionnaire. At the end of each stressor task participants were asked to rate their performance on three nine-point Likert scales (1 not at all true to 9 very true). For the VR maze participants were asked to rate themselves on the following statements: I felt fully engrossed in the virtual reality environment, I thought the virtual reality environment was realistic, and I felt nervous during the maze. For the speech task participants rated themselves on the following statements: I felt as though I had enough time to prepare my speech, I felt my performance was satisfactory, and I felt nervous during the speech.

4.2.4. Procedure

Participants attended a single testing session in an air-conditioned, sound attenuated room. After obtaining written consent participants completed the demographic screening questionnaire (see section 3.5.1) and the Hospital Anxiety and Depression Scales (HADS; Zigmond & Snaith, 1983, see section 3.5.3). After the questionnaires were completed electrodes for recording heart rate (HR) and skin conductance levels (SCL) were applied following standard procedures (see section 3.4.4). Recording HR allowed for the calculation of heart rate variability (HRV, see section 3.4.3). A five-minute baseline recording was carried out during which the participants were asked to sit quietly. After these five minutes participants completed the Profile of Mood States – Short Form (POMS-SF; Shacham, 1983, see section 3.5.2) to assess their current mood state. Instructions were then given for one of the stressors: the virtual reality maze or the speech task. The order of the virtual reality task and speech task was counterbalanced across participants. Each stressor manipulation was followed by a five-minute recovery period and re-administration of the POMS-SF. For each stressor task participants also completed a post-task questionnaire rating their
performance. Once the participants had completed the post-task questionnaire the second stressor manipulation was carried out, with the POMS-SF administered pre- and post-stressor again. A flowchart diagram of the laboratory stressors procedure can be found in appendix 10.

4.3. Results

4.3.1. Statistical Analyses

For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. To identify potential covariates, the relationships between potential confounds (i.e., age, ethnicity, sex, anxiety and depression scores) and the dependent variables (i.e., HR, SCL, HF-HRV, and the POMS-SF) were examined. Correlations revealed no significant associations between age, ethnicity, sex, self-reported anxiety ($M = 7.87$, $SD = 3.93$) or self-reported depression symptoms ($M = 3.87$, $SD = 3.56$) with any of the dependent variables (all $p_s < .10$). Thus, no demographic covariates were included in analyses.

Mean HR and SCL values were calculated for the baseline periods and each minute of the stressor manipulations. High-frequency heart rate variability (HF-HRV) was also calculated using the HR data (see section 3.4.3); as HRV is more reliable when calculated over larger windows of time (i.e., at least 10 cycles of the target rhythm; Berntson et al., 1997), HF-HRV was calculated using data from three minutes of the baseline periods, as well as the three-minute stressor manipulation tasks. It should be noted that HF-HRV during the presentation period of the speech task was not calculated due to the known effects of respiratory changes on HRV indices (Beda, Jandre, Phillips, Giannella-Neto, & Simpson, 2007; Tininenko, Measelle, Ablow, High, 2012). Analyses were performed to establish if there were any baseline differences between the stressor manipulations. Analyses of variance (ANOVAs) with the physiological data from the baseline periods (HR, SCL, and HF-HRV) as the dependent variables revealed no differences in physiology across the baseline periods (all $p_s > .10$). A separate ANOVA with the POMS-SF data for the baseline periods also failed to reveal any significant baseline differences between the stressor manipulations (all $p_s > .10$).
The first main group of analyses investigated the physiological changes associated with each of the stressor manipulations. To evaluate reactivity within each stressor manipulation, repeated-measures ANOVAs (with the repeated factor: Time) were carried out using the raw HR, SCL, and HF-HRV data. Then, as the baseline physiological variables did not differ across the stressor manipulations, reactivity scores (differences from baseline) were used to evaluate differences in reactivity between the stressor manipulations. Reactivity scores were calculated for HR and SCL by subtracting the data for the last minute of each pre-task baseline period from the data for each minute of the corresponding stressor manipulation period; reactivity for HF-HRV was calculated using the complete three-minute data for these periods (Kamarck et al., 1992; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). Repeated-measures ANOVAs (with the repeated factor: Task) were performed on the reactivity scores for HR, SCL, and HF-HRV with the Huynh-Feldt degrees of freedom correction applied where necessary (i.e., when factors violated sphericity assumptions, as confirmed by Mauchly’s tests). Significant main effects were followed up by pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05. To investigate whether changes in ANS function were related to changes in self-reported emotion, correlations were carried out between the mean HR, SCL, and HF-HRV reactivity scores and the mean POMS-SF reactivity scores. No significant relationships were revealed within the virtual reality maze data or the speech task data.

A second main group of analyses investigated the effects of each stressor manipulation on self-reported affect. Repeated-measures ANOVAs (with the repeated factors: Time and Scale) were used to identify significant changes in the POMS-SF subscales within each of the stressor manipulations. As a further investigation into the effects of each stressor manipulation, descriptive analyses were also carried out on the post-task questionnaire data. Similar to the physiological data, a repeated-measures ANOVA (with the repeated factors: Task and Scale) was carried out using POMS-SF reactivity scores (differences from baseline) to evaluate differences in reactivity between the stressor manipulations.

The third main group of analyses evaluated physiological return to baseline after the stressor manipulations. Recovery scores were calculated for HR and SCL by subtracting the data for each minute of the recovery period from the last minute of each
stressor manipulation period (some researchers prefer to use residualised change scores for recovery, however Llabre et al., 1991 suggest this is not necessary in many cases). Repeated-measures ANOVAs (with the repeated factor: Time) were performed on the recovery scores for HR and SCL to investigate how the physiological variables changed over the course of the recovery period. Again the Huynh-Feldt correction was applied where necessary. Paired $t$-tests using the raw physiological data were also used to compare each minute of the recovery period with the baseline period to establish if participants returned to physiological baseline after each task. To investigate recovery effects on HRV, HF-HRV power during the recovery period was compared to baseline HF-HRV power for each stressor manipulation using repeated-measures ANOVAs (with the repeated factor: Task); the baseline period was used as the comparison period rather than the stressor manipulation because HF-HRV was not calculated during the presentation period of the speech.

4.3.2. Physiological Reactivity to the Stressor Manipulations

The first set of analyses was conducted to test the hypothesis that the stressor manipulations would be associated with decreased PNS activation and increased SNS activation:

4.3.2.1. The virtual reality maze. As shown in table 4.1, the VR maze resulted in initial changes in HR and SCL, but these were not sustained for the duration of the task. Repeated-measures ANOVAs for HR and SCL were performed for each minute of the VR maze to determine the effects of the stressor over time. A repeated-measures ANOVA comparing baseline and the three one-minute sections of the VR maze for the HR data found a significant effect of Time, $F(2.60, 75.32) = 3.85, p = .017$. Linear contrasts revealed that the decrease in HR from baseline was only significant for the first minute, $F(1, 29) = 4.84, p = .036$. A second repeated-measures ANOVA was performed on the SCL data which also revealed a significant effect of Time, $F(1.26, 36.59) = 6.85, p = .009$. A linear contrast revealed that the increase in SCL from baseline was again only significant for the first minute, $F(1, 29) = 13.43, p = .001$. HF-HRV power was calculated for baseline and the full duration of the VR maze. A repeated-measures ANOVA revealed that HF-HRV power was significantly lower during the VR maze than during the baseline period, $F(1, 29) = 4.69, p = .039$. 
### Table 4.1

Effects of the stressor manipulations on heart rate, skin conductance level, and high-frequency heart rate variability.

<table>
<thead>
<tr>
<th></th>
<th>Pre-task Baseline</th>
<th>Stressor Manipulation Period</th>
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<tbody>
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<td>1st min</td>
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<td>3rd min</td>
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<td>6th min</td>
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<td>VR Maze Task</td>
<td></td>
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<tr>
<td>HR (bpm)</td>
<td>76.11 (10.20)</td>
<td>74.00 (9.89)*</td>
<td>74.68 (8.77)</td>
<td>76.15 (8.89)</td>
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</tr>
<tr>
<td>SCL (µS)</td>
<td>2.71 (3.06)</td>
<td>3.62 (2.51)*</td>
<td>3.20 (2.17)</td>
<td>3.03 (2.06)</td>
<td>---</td>
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</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.24 (1.18)</td>
<td>6.91 (0.74)*</td>
<td></td>
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<tr>
<td>Speech Task</td>
<td></td>
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<tr>
<td>HR (bpm)</td>
<td>77.05 (9.71)</td>
<td>87.25 (11.15)**</td>
<td>86.68 (13.28)**</td>
<td>87.04 (11.19)**</td>
<td>96.47 (11.30)**</td>
<td>87.62 (9.21)**</td>
<td>85.44 (9.43)**</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>1.78 (1.26)</td>
<td>3.98 (2.32)**</td>
<td>3.69 (1.98)**</td>
<td>3.64 (2.07)**</td>
<td>4.87 (3.03)**</td>
<td>4.57 (2.89)**</td>
<td>4.51 (2.95)**</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>6.98 (1.02)</td>
<td>6.71 (1.02)**</td>
<td>---</td>
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*Note. Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from baseline * p < .05, ** p < .01, ***p < .001
4.3.2.2. The speech task. The speech task resulted in noticeable increases in HR and SCL (as shown in table 4.1). A repeated-measures ANOVA comparing baseline and the one-minute sections of the speech task for HR found a significant effect of Time, \(F(3.35, 93.69) = 29.56, p < .001\). Linear contrasts revealed that HR was significantly higher than baseline throughout all time periods of the speech task (significant at the \(p < .001\) level). A repeated-measures ANOVA was performed on the SCL data which also revealed a significant effect of Time, \(F(1.92, 55.64) = 23.63, p < .001\). Linear contrasts revealed that SCL was also significantly higher than baseline throughout all time periods of the speech task (significant at the \(p < .001\) level). In addition to the changes in HR and SCL, a repeated-measures ANOVA revealed a significant decrease in the HF-HRV power band during the preparation period of the speech task, \(F(1, 29) = 7.60, p = .010\) (HF-HRV during the speech was not analysed). It is worth mentioning that although arousal peaked during the presentation period of the speech, the HR and SCL data both show gradual declines across the final three minutes of the task. This is an important factor to consider when evaluating recovery after the task.

4.3.2.3. Comparison of the stressor manipulations. Repeated-measures ANOVAs using HR and SCL reactivity scores were used to compare the effects of the stressor manipulations on increases in physiology from baseline. To equate for differences in the duration of the stressor manipulations, the VR maze was only compared to the preparation period of the speech task. For HR reactivity, there were significant main effects of Task, \(F(1, 29) = 20.22, p < .001\), and Time, \(F(1, 29) = 21.84, p < .001\), and a significant interaction effect of Task x Time, \(F(1, 29) = 43.86, p < .001\). Bonferroni pairwise comparisons confirmed that HR significantly increased during the preparation phase of the speech task, but significantly decreased during the VR maze task (significant at \(p < .05\)). For SCL reactivity, there was also a significant main effect of Time, \(F(1, 29) = 71.91, p < .001\), and a significant interaction effect of Task x Time, \(F(1, 29) = 10.37, p = .003\). Bonferroni pairwise comparisons revealed that although SCL increased during both stressor manipulations, the increase in SCL was significantly higher during the preparation phase of the speech task than during the VR maze task (significant at \(p < .05\)). Taken together the HR and SCL reactivity scores indicate that the speech task was more arousing than the VR maze. Despite these effects, a repeated-measures ANOVA failed to reveal any significant differences in HF-HRV reactivity across the stressor manipulations; both tasks resulted in significant decreases in HF-HRV.
Table 4.2.

Mean scores for the POMS-SF subscales before and after each stressor manipulation

<table>
<thead>
<tr>
<th></th>
<th>VR Maze Before</th>
<th>VR Maze After</th>
<th>Speech Task Before</th>
<th>Speech Task After</th>
<th>F (time x task)</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression–Dejection</td>
<td>1.60 (3.16)</td>
<td>1.67 (3.30)</td>
<td>1.67 (3.80)</td>
<td>2.57 (4.12)</td>
<td>1.82</td>
<td>1, 29</td>
<td>.188</td>
</tr>
<tr>
<td>Vigour–Activity</td>
<td>6.00 (3.44)</td>
<td>4.50 (3.13)*</td>
<td>5.30 (3.97)</td>
<td>4.60 (3.18)</td>
<td>0.84</td>
<td>1, 29</td>
<td>.367</td>
</tr>
<tr>
<td>Anger–Hostility</td>
<td>1.03 (2.33)</td>
<td>1.07 (1.91)</td>
<td>0.97 (1.90)</td>
<td>1.83 (3.66)</td>
<td>1.49</td>
<td>1, 29</td>
<td>.232</td>
</tr>
<tr>
<td>Tension–Anxiety</td>
<td>3.23 (2.19)</td>
<td>3.37 (3.33)</td>
<td>2.70 (2.52)</td>
<td>5.10 (3.67)***</td>
<td>7.03</td>
<td>1, 29</td>
<td>.013</td>
</tr>
<tr>
<td>Confusion–Bewilderment</td>
<td>1.93 (1.64)</td>
<td>2.43 (2.27)</td>
<td>2.10 (1.90)</td>
<td>2.70 (2.35)</td>
<td>0.75</td>
<td>1, 29</td>
<td>.859</td>
</tr>
<tr>
<td>Fatigue–Inertia</td>
<td>3.27 (3.32)</td>
<td>3.70 (3.14)</td>
<td>4.07 (3.22)</td>
<td>3.47 (3.33)</td>
<td>2.41</td>
<td>1, 29</td>
<td>.131</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Significant change from baseline * p < .05, ***p < .001

Table 4.3.

Mean recovery scores (recovery period – stressor manipulation) for heart rate and skin conductance level during the five-minute recovery period after each stressor manipulation

<table>
<thead>
<tr>
<th></th>
<th>Recovery Period</th>
<th>F (time)</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maze Task</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR Recovery</td>
<td>1.03 (4.08)</td>
<td>-0.24 (3.62)</td>
<td>0.43 (4.44)</td>
<td>-0.62 (3.92)</td>
</tr>
<tr>
<td>SCL Recovery</td>
<td>0.27 (0.50)</td>
<td>0.02 (0.64)</td>
<td>-0.12 (0.61)</td>
<td>-0.20 (0.62)</td>
</tr>
<tr>
<td>Speech Presentation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR Recovery</td>
<td>-5.87 (7.38)</td>
<td>-9.84 (7.16)</td>
<td>-9.31 (6.44)</td>
<td>-9.19 (7.29)</td>
</tr>
<tr>
<td>SCL Recovery</td>
<td>-0.27 (0.36)</td>
<td>-0.75 (0.69)</td>
<td>-1.07 (1.04)</td>
<td>-1.24 (1.13)</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level.
4.3.3. Mood Effects of the Stressor Manipulations

The second set of analyses was carried out to evaluate the hypothesis that the stressor manipulations would be associated with increased self-reported negative affect:

4.3.3.1. The virtual reality maze. A repeated-measures ANOVA with the POMS-SF data for the VR maze revealed a significant main effect of Scale, $F(4.10, 118.78) = 15.93, p < .001$, and a significant Scale x Time interaction, $F(3.52, 343.28) = 3.41, p = .015$. Paired $t$-tests revealed that the only significant change related to the VR maze was a decrease in the vigour–activity scale, $t(29) = 2.37, p = .025$ (see table 4.2). Descriptive analyses on the post-task questionnaire data (scales ranging from 1 not at all true, to 9 very true) revealed that although participants felt moderately engrossed in the virtual reality environment ($M = 4.59, SD = 2.16$), the maze did not make participants feel overly nervous ($M = 3.86, SD = 2.24$) and did not come across as realistic ($M = 2.83, SD = 1.85$).

4.3.3.2. The speech task. A repeated-measures ANOVA with the POMS-SF data for the speech task revealed a significant main effect of Scale, $F(3.93, 114.04) = 8.03, p < .001$, and a significant Scale x Time interaction, $F(3.76, 108.89) = 4.45, p = .003$. Paired $t$-tests revealed a significant increase in the POMS-SF tension–anxiety subscale after the speech task, $t(29) = -4.19, p < .001$ (see table 4.2). In addition to this, the post-task questionnaire (scales ranging from 1 not at all true, to 9 very true) revealed that the speech task made the participants feel reasonably nervous ($M = 6.13, SD = 2.37$). The other post-task ratings demonstrated that the participants felt like they did not have enough time to prepare for the speech ($M = 3.83, SD = 2.38$), however they were moderately satisfied with their performance ($M = 4.47, SD = 2.08$).

4.3.3.3. Comparison of stressor manipulations. A repeated-measures ANOVA using reactivity scores for the POMS-SF data was used to compare the effect of the stressor manipulations on changes in self-reported mood. The analysis found a significant main effect of Scale, $F(3.43, 99.56) = 5.23, p < .001$, and a significant Scale x Task interaction, $F(3.25, 94.32) = 3.24, p = .023$. Paired $t$-tests revealed that the only significant difference in the POMS-SF reactivity scores between the stressor manipulations was for the tension–anxiety scale, with the speech task demonstrating greater change than the VR maze, $t(29) = 2.65, p = .013$. In addition to this, a paired $t$-
test on the post-task questionnaire ratings confirmed that participants reported feeling significantly more nervous during the speech task \((M = 6.13, SD = 2.37)\) than the VR maze \((M = 3.86, SD = 2.24)\), \(t(29) = 3.49, p = .002\).

4.3.4. Recovery from the Stressor Manipulations

The final set of analyses was carried out to investigate the levels of arousal remaining after the completion of each stressor task. Recovery scores were used to investigate physiological changes within each of the stressor manipulations. Comparisons were not made across the tasks, as the stressor manipulations did not result in equivalent changes in physiological arousal.

4.3.4.1. The virtual reality maze. At the end of the VR maze, the physiological indices were not significantly different from baseline; however the five-minute recovery period was still investigated for recovery effects (see table 4.3). A repeated-measures ANOVA on the HR recovery scores for the VR maze did not find a significant effect of Time, suggesting that HR remained stable during the recovery period. Paired \(t\)-tests on the raw HR data confirmed that HR was not significantly different from baseline during the five minutes following the VR maze. In contrast to this, a significant effect of Time was found for the SCL recovery scores, \(F(2.17, 63.06) = 15.00, p < .001\). Paired \(t\)-tests using the raw SCL data confirmed that SCL was significantly higher than baseline during the first minute of the recovery period, \(t(29) = -2.16, p = .039\), but only during this epoch. A repeated-measures ANOVA failed to find any differences between HF-HRV power during the recovery period \((M = 7.18, SD = 0.84)\) and the baseline period for the VR maze \((M = 7.24, SD = 1.18)\).

4.3.4.2. The speech task. As mentioned previously, the HR and SCL data were already exhibiting reductions during the presentation period of the speech, indicating that arousal levels were falling in advance of the recovery period. A repeated-measures ANOVA using the HR recovery scores for the speech task revealed a significant effect of Time, \(F(2.54, 73.54) = 9.28, p < .001\) (see table 4.3). Paired \(t\)-tests using the raw HR data confirmed that HR was significantly higher than baseline during the first minute of the recovery period, \(t(29) = -2.37, p = .025\), but was not significantly different during the remaining four minutes. The recovery for SCL was more linear, with SCL showing a general decline across the whole five-minute recovery period. A repeated-measures
ANOVA confirmed that the effect of Time was significant for SCL recovery, $F(1.43, 41.57) = 23.80, p < .001$. Paired $t$-tests using the raw SCL data revealed that SCL remained significantly higher than baseline during the whole five-minute recovery period (smallest $t = 3.77$, largest $p = .001$). In addition to the HR and SCL effects, HF-HRV was significantly higher during the recovery period ($M = 7.44$, $SD = 0.93$) than it had been at baseline for the speech task ($M = 6.98$, $SD = 1.02$), $F(1, 29) = 29.30, p < .001$.

4.4. Discussion

Experiment 1 was designed to demonstrate the psychophysiological effects of two active stressors. The stressor manipulations were evaluated for their effects on physiological reactivity, changes in mood state, and physiological recovery.

4.4.1. Physiological Reactivity

Rather than inducing a mobilisation response, the VR maze was found to initiate a response associated with orienting and sustained attention (reduced HR, increased SCL, and reduced HF-HRV; Bradley, Codispoti, Cuthbert, & Lang, 2001). This profile is more consistent with a response that is represented by co-activation of the SNS and PNS. Contrastingly, the speech task induced increases in HR and SCL, accompanied by a decrease in HF-HRV. These physiological changes were maintained throughout both the preparation and presentation periods of the speech task. This profile of response is consistent with previous research evaluating the effects of speech task paradigms, and suggests that speech tasks are able to induce up-regulation of the SNS coupled with down-regulation of the PNS (i.e., induce a defensive mobilisation response; Feldman et al., 2004; Kirschbaum et al., 1993). Consequently only the speech task upheld the hypothesis that laboratory stressors would be associated with decreased PNS activation and increased SNS activation.

4.4.2. Changes in Mood State

Both tasks initiated changes in mood state that are normally indicative of increased negative affect (McNair, Lorr, & Droppleman, 1971; Shacham, 1983). Whilst the VR task was associated with a significant reduction in the vigour–activity subscale
of the POMS-SF, the speech task was associated with a significant increase in the
tension–anxiety subscale. Analyses comparing the effects of the stressor manipulations
revealed that the speech task was associated with significantly larger changes in mood
state than the VR maze. Taken together, the findings of this experiment suggest that the
speech task is a valid laboratory stressor, as it initiates changes in both physiology and
subjective mood state.

4.4.3. Physiological Recovery

As the VR maze did not result in sustained changes in physiology during the
stressor manipulation period, it was expected that the physiological indices would not
be significantly different from baseline during the recovery period. This was supported
by the analyses of the recovery data. A disparate pattern of physiological reactivity and
recovery was seen with the speech task. The speech task induced physiological changes
associated with the fight–flight response (withdrawal of the PNS and activation of the
SNS). The observed increases in physiological arousal were accompanied by increased
self-reports of tension and anxiety (as measured by the POMS-SF). Although the speech
task was successful at inducing an initial mobilisation response, further analyses
revealed that the increase in HR caused by the speech task did not remain significantly
above baseline once the task was terminated. SCL increases were maintained, but
showed a steady decline over the five-minute recovery period; this suggests that the
SNS was less active during this time. HF-HRV also demonstrated a significant increase
during the recovery period; this is indicative of increased PNS activation.

A potential motivator behind the return to baseline after the speech task could be
the body simply adapting to the stressor. Reactivity to acute stressors tends to peak early
during the initial presentation of a task, when novelty and uncertainty are greatest
(Kelsey et al., 1999). As mentioned in Chapter 2, shifts away from the body’s normal
physiological balance will initiate processes to return the body to a state of homeostasis
(Jänig, 2006); hence up-regulation of the SNS during a stressor will usually be followed
by increased activation of the PNS. A second factor that may have prompted the
physiological return to baseline is inherent to the presentation period of the speech task;
the majority of participants rated that they were moderately satisfied with their
performance, an appraisal that may have helped them to down-regulate their response to
the task. In addition to this, the completion of the speech may have prompted
participants to actively engage in self-regulation to reduce any remaining arousal from
the task. The increase observed in HF-HRV would be consistent with this inference, as
this measure has been linked to self-regulatory effort (Segerstrom & Solberg Nes,
2007). Regardless of the mechanism behind the return to baseline, the decrease in
arousal seen during the presentation period and subsequent recovery period limits the
use of the speech task as a stressor in the remaining studies.

4.5. Experiment 2: Speech Task Anticipation

Experiment 1 demonstrated the utility of a speech task in initiating a
mobilisation response. As mentioned in the previous experiment, the speech task was
limited in its ability to maintain arousal levels significantly above baseline once the task
had ended. The speech task in its current format is therefore limited in its use as a
manipulation to investigate how arousal influences socio-emotional responding. In the
first instance, the speech task cannot be used to investigate the effectiveness of emotion
regulation strategies in reducing arousal (Chapter 5): Administering interventions
designed to facilitate recovery from stress need to have residual arousal to down-
regulate; otherwise their effects may be confounded with the body’s normal return to
baseline. In the second instance, the current thesis is interested in the effect of defensive
physiological states on facial expressivity (Chapter 6), emotional sensitivity (Chapter 7),
and social affiliation tendencies (Chapter 8). In order to elucidate the effects of SNS
activation on socio-emotional responding participants need to remain in defensive
physiological states during measures of these competencies; the proposed designs for
the subsequent experiments require a stressor that has carryover effects in terms of
arousal.

One way to overcome the limitations of the speech task could be to use the
preparation period of the task more effectively. It has been documented that the
preparation period before giving a speech can significantly increase arousal (as shown
in Experiment 1, see also Feldman, Cohen, Hamrick, & Lepore, 2004; and Waugh et al.,
2010). Research has shown that anticipation of a stressor can elicit a similar stress
type to that caused by an actual stressor (Brosschot, Pieper, & Thayer, 2005;
Waugh et al., 2010). It has also been shown that the physiological changes that arise
during anticipation of a stressor may persist longer than the stressor itself (Gregg et al.,
1999). Studies have used the preparation component of speech tasks when investigating
differences between high and low anxious individuals, as these populations only tend to show measurable differences when under threat/challenge (for example Garner, Mogg, & Bradley, 2006; and Mansell, Clark, Ehlers, & Chen, 1999). Despite using the preparation component of speech tasks as a stressor manipulation, only behavioural outcomes have been reported in these studies; investigators have yet to report on how well defensive arousal is maintained during the secondary tasks in these populations.

The aim of Experiment 2 was to explore the potential use of a short anticipation period before the speech presentation. A between subjects design was used to compare the physiological recovery of a group of participants anticipating the speech presentation \((n = 20)\), to the physiological recovery of the speech group \((n = 30)\) from Experiment 1. It was hypothesised that arousal levels would remain elevated for longer in the group where the five-minute rest period was undertaken before presenting the speech (anticipation), in comparison to the group where the five-minute rest period was taken after presenting the speech (recovery: as in Experiment 1). The assumption underlying this hypothesis is that participants’ initial arousal levels are linked to the anticipation of the speech, not the physical presentation of the speech.

### 4.6. Method

#### 4.6.1. Participants

Twenty different undergraduate students (6 males, 14 females) participated in Experiment 2 (anticipation group). They ranged in age from 18–35 with a mean age of 21.55 years \((SD = 5.37)\). Participants were awarded course credits as part of their undergraduate course requirements. 95% of these participants identified themselves as Caucasian and 5% as Other. Exclusion criteria were the same as Experiment 1. The data collected from the original speech task in Experiment 1 (recovery group, \(n = 30\): 3 males, 27 females; mean age = 19.77, \(SD = 2.54\)) was used as comparison data.

#### 4.6.2. Procedure

The procedure was similar to that in Experiment 1, except for a change in the design of the speech task. After completing the consent form, the demographic screening questionnaire (see section 3.5.1) and the HADS (Zigmond & Snaith, 1983,
see section 3.5.3), electrodes for recording HR and SCL were applied (see section 3.4.4) and a five-minute baseline recording was carried out. Participants then completed the POMS-SF (Shacham, 1983; see section 3.5.2) and were given instructions for the speech task. The three-minute preparation period of the speech task was carried out as before (as in Schubert et al., 2009; see section 4.2.2.2), but after the preparation period the pen and paper were taken away and the participants were asked to rest quietly for five minutes before presenting their speech (five-minute anticipation period). After the anticipation period the participants completed a second POMS-SF and were then instructed to present their speech to the video camera for three minutes. A flowchart diagram of the speech task anticipation procedure can be found in appendix 11.

4.7. Results

4.7.1. Statistical Analyses

The analyses are similar to those from Experiment 1. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. In addition to this the relationship between potential confounds and the dependent variables were examined. Bivariate correlations revealed a significant relationship between self-reported depression symptoms ($M = 3.75, SD = 2.94$) and baseline SCL ($r = -.47, p = .035$). Correlations revealed no other significant associations between age, ethnicity, sex, or self-reported anxiety symptoms ($M = 8.25, SD = 3.40$) with any of the dependent variables (all $ps < .10$). Due to the relationship between the HADS depression score and baseline SCL, analyses with the SCL data were repeated with the HADS depression score (HADS-D) as a covariate.

As in Experiment 1 mean HR and SCL values were calculated for the baseline period and each minute of the speech task. HF-HRV was also calculated for the baseline period and speech task preparation period (see section 3.4.3). Basic analyses were first performed to establish if there were any baseline differences between the anticipation group and the recovery group. The groups did not significantly differ in terms of sex, ethnicity, self-reported anxiety or self-reported depression symptoms, however the anticipation group were significantly older (mean age 21.55 years) than the recovery group (mean age 19.77 years), $t(48) = -1.58, p = .007$. Due to significant differences in age between the two samples, analyses that compared the groups were repeated with
Age entered as a covariate. Univariate ANOVAs did not reveal any significant differences between the groups for HR, SCL, HF-HRV, the POMS-SF subscales, or the HADS subscales at baseline. The raw physiological data for the anticipation group is shown in table 4.4; the POMS-SF data can be found in table 4.5.

The first main set of analyses carried out in the current experiment was to investigate whether the groups exhibited equivalent responses to the speech task preparation period. Repeated-measures ANOVAs were carried out using the raw HR, SCL, HF-HRV, and POMS-SF data from the anticipation group to evaluate within group changes. Then, as in Experiment 1, reactivity scores were calculated for the HR, SCL, HF-HRV, and the POMS-SF data. Mixed-factorial ANOVAs (with the repeated-measures factor Time; and between subjects factor Group) were carried out to compare the anticipation group’s reactivity scores with the reactivity scores of the recovery group. The analyses were repeated with Age as a covariate; however inclusion of this factor did not affect the reported findings. To investigate whether changes in ANS function were related to changes in self-reported emotion, correlations were carried out between the mean HR, SCL, and HF-HRV reactivity scores and the mean POMS-SF reactivity scores. Positive relationships were identified between HR reactivity and changes in the POMS-SF confusion–bewilderment subscale \( r = .48, p = .031 \) and SCL reactivity and the changes in POMS-SF vigour–activity subscale \( r = .58, p = .008 \). The directions of these relationships suggest that larger increases in SNS activation were associated with greater negative affect. In addition to this, negative relationships were revealed between HF-HRV reactivity and the POMS-SF tension–anxiety subscale \( r = -.65, p = .008 \) and the POMS-SF confusion–bewilderment subscale \( r = -.52, p = .018 \), suggesting that larger decreases in PNS activation were also associated with larger increases in indices of negative affect.

The second set of main analyses was carried out to evaluate group differences in physiological recovery. Recovery scores for each minute of the recovery period were calculated for HR and SCL; for the anticipation group difference scores were calculated using the last minute of the preparation period, as opposed to the presentation period as in Experiment 1. Recovery scores for HF-HRV were also calculated by subtracting baseline HF-HRV from the recovery period HF-HRV. Mixed-factorial ANOVAs (with the repeated-measures factor Time; and between subjects factor Group) were carried out to compare the groups’ recovery from the speech tasks.
4.7.2. Reactivity to the Speech Tasks

4.7.2.1. Reactivity in the anticipation group. First, analyses were carried out to confirm that the participants in the anticipation group reacted to the speech task (see table 4.4). A repeated-measures ANOVA on the anticipation group’s HR data, comparing the three one-minute sections of the preparation period with baseline found a significant effect of Time, $F(1.81, 86.77) = 41.44, p < .001$. Linear contrasts confirmed that HR was significantly higher than baseline throughout the preparation period (significant at the $p = .003$ level). A repeated-measures ANOVA was also performed on the SCL data revealing a significant effect of Time, $F(1.79, 34.08) = 54.07, p < .001$. Linear contrasts confirmed that SCL was also significantly higher than baseline throughout the preparation period (significant at the $p < .001$ level). These effects remained significant when the HADS-D was entered as a covariate. In addition to these changes, a repeated-measures ANOVA revealed a significant decrease in the HF-HRV power band during the preparation period of the adapted speech task, $F(1, 19) = 11.52, p = .003$.

In addition to the physiological measures, a repeated-measures ANOVA on the POMS-SF data revealed significant main effects of Time, $F(1, 19) = 4.58, p = .046$, and Scale, $F(2.86, 54.23) = 7.42, p < .001$, along with a significant Scale x Time interaction, $F(4.35, 82.64) = 7.91, p < .001$. Paired $t$-tests confirmed that the anticipation group only exhibited a significant change in the tension–anxiety subscale, $t(19) = -4.42, p < .001$; see table 4.5.
Table 4.4.
Mean effects of the adapted speech task on heart rate, skin conductance level, and high-frequency heart rate variability.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Speech Preparation period</th>
<th></th>
<th>df</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1st min</td>
<td>2nd min</td>
<td>3rd min</td>
<td>(time)</td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>78.74 (9.85)</td>
<td>91.78 (12.43)**</td>
<td>90.26 (14.16)**</td>
<td>89.30 (13.23)**</td>
<td>41.44</td>
<td>1.81, 86.77</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>1.43 (1.04)</td>
<td>3.46 (1.89)**</td>
<td>3.26 (1.58)**</td>
<td>3.24 (1.56)**</td>
<td>54.07</td>
<td>1.79, 34.08</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.27 (1.07)</td>
<td>6.52 (1.08)</td>
<td></td>
<td></td>
<td>11.52</td>
<td>1, 19</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from baseline ** p < .01, *** p < .001

Table 4.5.
Mean scores for the POMS-SF subscales before and after the anticipation period

<table>
<thead>
<tr>
<th></th>
<th>Speech Task Anticipation</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>t</td>
<td>df</td>
<td></td>
</tr>
<tr>
<td>Depression–Dejection</td>
<td>2.40 (4.33)</td>
<td>4.20 (5.20)</td>
<td>-1.49</td>
<td>19</td>
<td>.154</td>
</tr>
<tr>
<td>Vigour–Activity</td>
<td>5.80 (3.69)</td>
<td>5.50 (3.33)</td>
<td>0.55</td>
<td>19</td>
<td>.587</td>
</tr>
<tr>
<td>Anger–Hostility</td>
<td>1.45 (1.82)</td>
<td>2.90 (4.72)</td>
<td>-1.39</td>
<td>19</td>
<td>.180</td>
</tr>
<tr>
<td>Tension–Anxiety</td>
<td>3.80 (2.78)</td>
<td>8.90 (5.96)**</td>
<td>-4.44</td>
<td>19</td>
<td>.001</td>
</tr>
<tr>
<td>Confusion–Bewilderment</td>
<td>2.90 (2.83)</td>
<td>4.25 (4.40)</td>
<td>-1.50</td>
<td>19</td>
<td>.151</td>
</tr>
<tr>
<td>Fatigue–Inertia</td>
<td>4.25 (2.83)</td>
<td>3.30 (3.01)</td>
<td>1.30</td>
<td>19</td>
<td>.209</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Significant difference from baseline *** p < .001
4.7.2.2. **Group differences in reactivity.** To establish whether the speech tasks induced a similar level of arousal during the preparation period in the anticipation and recovery groups, mixed-factorial ANOVAs were performed on the reactivity scores for HR and SCL. A univariate ANOVA was also performed using the HF-HRV reactivity score. No significant differences were found suggesting that both groups found the preparation period equally stressful. These analyses remained insignificant when Age and the HADS-D scores were entered as covariates. In addition to the physiological indices, a repeated-measures ANOVA using reactivity scores for the POMS-SF data was used to evaluate group differences in changes in self-reported mood. The analysis found a significant main effect of Scale, \( F(2.90, 138.96) = 12.30, p < .001 \). Both groups demonstrated significant increases in the POMS-SF anxiety–tension subscale (significant at the \( p < .001 \) level), but not the other scales. No main effects or interactions were found involving Group suggesting that the groups experienced equivalent changes in mood state.

4.7.3. **Physiological Recovery from the Speech Tasks**

Comparisons were made between the five-minute rest period of the adapted speech task (anticipation: completed before the presentation period) and the five-minute rest period of the original speech task (recovery: completed after the presentation period). As in Experiment 1, recovery scores were used to investigate the changes in arousal during the rest periods. No adjustments were made for baselines in these analyses, because univariate ANOVAs revealed comparable physiological baselines for each of the speech tasks: The physiological indices for the last minute of preparation in the anticipation group were not significantly different from the physiological indices for the last minute of the presentation in the recovery group (all \( p_s > .05 \)).

A repeated-measures ANOVA on the HR recovery scores revealed significant main effects of Time \( F(2.81, 134.82) = 16.86, p < .001 \), and Group, \( F(1, 48) = 4.08, p = .049 \). Both groups demonstrated decreases in HR across the rest periods (as shown in figure 4.2), but the recovery group demonstrated a significantly larger decrease than the anticipation group (confirmed by Bonferroni pairwise comparisons, \( p < .05 \)). Paired \( t \)-tests with the raw HR data revealed that HR in the recovery group only remained significantly above baseline for the first minute of the rest period (\( p = .023 \)), whereas HR in the anticipation group remained significantly above baseline throughout the
whole rest period (significant to at least \( p = 0.13 \)). When the ANOVA was repeated with Age as a covariate the main effects remained significant.

**Figure 4.2.** Mean heart rate recovery scores for each minute of the anticipation/recovery period for the recovery group (original speech design) and the anticipation group (adapted speech design). Error bars represent the standard error.

For SCL recovery, significant main effects were found for Time, \( F(1.49, 71.45) = 21.96, p < .001 \), and Group, \( F(1, 48) = 43.16, p < .001 \), as well as a significant Time x Group interaction, \( F(1.49, 71.45) = 6.11, p = .008 \). Paired \( t \)-tests with the raw SCL means confirmed that SCL remained significantly higher than baseline throughout the full five minutes of the rest period in both groups (at least \( p = .001 \)). Further analysis revealed that the slope of SCL recovery for the anticipation group was significantly different from the recovery group (Linear contrast, \( F(1, 48) = 26.19, p < .001 \)), with the anticipation group exhibiting a slower decrease in SCL over the five minutes (shown in figure 4.3). The effects remained significant when HADS-D scores were entered as a covariate, but when Age was entered into the ANCOVA there was no longer a significant main effect of Time, although the Group and Group x Time effects remained significant.
CHAPTER 4: LABORATORY STRESSORS

Figure 4.3. Mean skin conductance level recovery scores for each minute of the anticipation/recovery period for the recovery group (original speech design) and the anticipation group (adapted speech design). Error bars represent the standard error.

A final univariate ANOVA with the HF-HRV recovery scores revealed a significant effect of Group on recovery, $F(1, 49) = 9.66, p = .003$. Whilst the recovery group demonstrated a significant increase in HF-HRV during their rest period (significant at the $p < .001$ level), the anticipation group exhibited a non-significant decrease in HF-HRV. When the ANOVA was repeated with Age as a covariate the effect of Group remained significant.

4.8. Discussion

Experiment 1 established that completing a speech task initiates a mobilisation response. A complication of the task however, is that presenting the speech is associated with a physiological return to baseline. This means that the task has limited carryover effects (i.e., by the end of the task participants are no longer in a defensive physiological state). The main aim of Experiment 2 was to investigate whether the preparation period of the speech task could be utilised to produce arousal that would remain above baseline for a longer period of time.

The design of the speech task was adapted in Experiment 2 to introduce a five-minute rest period after the preparation period. It was expected that during this time anticipation of the speech would prevent arousal levels from returning to baseline (Gregg et al., 1999; Waugh et al., 2010). Analyses comparing data from the speech task recovery period in Experiment 1 (original speech task design) and the anticipation
period from the current experiment (adapted speech task design) revealed that HR remained significantly higher in the anticipation group. In addition to this, SCL levels remained significantly elevated in both groups, but the anticipation group demonstrated a slower rate of decline during the five minutes of the rest period. In Experiment 2 baseline SCL was significantly associated with self-reported depression symptoms, although controlling for the HADS-D scores did not affect any of the observed differences in reactivity or recovery between the groups. The residual arousal seen in the anticipation group is indicative of SNS activation (Jessell, 1995).

In Experiment 1 it was revealed that the decrease in SNS activation during the recovery period was also accompanied by a significant increase in HF-HRV, suggesting up-regulation of the PNS (Cacioppo, Uchino, & Berntson, 1994). The anticipation group in the current experiment did not exhibit a significant difference in HF-HRV during the rest period, suggesting that the anticipation group were not engaging the activation of the PNS during this time. Taken together, the physiological findings support the hypothesis that a five-minute anticipation period before the presentation of a speech can be used to maintain arousal levels above baseline.

### 4.9. General Discussion

The aim of the current study was to identify an active stressor than not only increased arousal during the task but caused arousal levels to remain above baseline after the task was completed. Healthy undergraduate students completed a VR maze and a speech task in Experiment 1. The experiment highlighted the usefulness of speech tasks in increasing arousal, but the suitability of the task was limited by a rapid return to physiological baseline once the speech was presented. Experiment 2 found that introducing a five-minute anticipation period before the presentation of the speech was a suitable way of maintaining elevations in arousal. This finding is consistent with previous studies that have highlighted the role of anticipation in initiating and maintaining defensive arousal: Anticipation of a threat or challenge is a powerful way of inducing arousal that is sustained for a short period of time (Feldman et al., 2004; Gregg et al., 1999; Waugh et al., 2010).

For the subsequent studies, it is hypothesised that the residual arousal from the anticipation period may remain elevated long enough for secondary tasks to be
administered whilst the participants remain in a defensive physiological state. A limitation of this study is that it is unknown how strong the carryover effects from the preparation period will be once a secondary task is undertaken during this time. Although uninterrupted anticipation is associated with maintained arousal, secondary tasks carried out during the anticipation period may cause a distraction effect, and also have physiological effects of their own. It is beyond the scope of this study to establish the effects of such secondary tasks, as each individual task is likely to initiate a unique emotional and physiological response. The effects of secondary tasks during the anticipation period will need to be investigated in the subsequent experiments.

To conclude, the current experiments identified an active stressor that can be used to induce and maintain arousal. The anticipation of a speech task causes significant changes in HR, SCL, and HF-HRV, as well as self-reported tension and anxiety. This change is consistent with a mobilisation response, and reflects activation of the SNS. The defensive arousal associated with presenting a speech can be sustained for a few minutes if an anticipation period is employed, as demonstrated by increased HR and SCL. The anticipation period is a window of opportunity when the participant is still in a defensive physiological state for secondary tasks to be administered (as in Garner et al., 2006; and Mansell et al., 1999). Based on the findings from this study, anticipation of a speech task was used as a stressor manipulation in the following chapters.
Chapter 5: The Psychophysiological Effects of Regulatory Strategies

The aim of Chapter 4 was to identify an active stressor that could be used to activate the sympathetic nervous system (SNS). The experiments in Chapter 4 demonstrated that a defensive physiological state could be induced in healthy participants by engaging them in a public speaking task. Anticipation of the speech task caused reliable increases in heart rate and sweat response, with a decrease in high-frequency heart rate variability. Increased activation of the SNS plays an important role in the following studies, of equal importance however, is the role of the parasympathetic nervous system (PNS) in being able to dampen the effects of the SNS. There is a need for understanding how the body’s physiology can be manipulated to down-regulate defensive physiological arousal to facilitate the promotion of calm and self-soothing states.

This chapter provides an overview of emotion regulation processes, and discusses the limitations of cognitive and behavioural regulation strategies. In place of these approaches, it is argued that strategies which directly target functioning of the autonomic nervous system (ANS) may be more appropriate for regulating emotion. The current experiment was designed to investigate the effects of three positive emotion regulation strategies: deliberate smiling (testing the facial feedback hypothesis), mindful breathing, and a loving-kindness meditation. These strategies were hypothesised to be able to reduce arousal levels by enhancing the activation of the myelinated vagus nerve (i.e., the PNS). Activation of the PNS should inhibit the SNS, causing a reduction in sympathetic arousal.

5.1. Emotion Regulation

To be able to change the course of an emotional response, we must first understand how emotions are generated. Emotional responses are multifaceted and produce changes in cognitions, feelings, behaviours, and physiology (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Whilst emotions help to shape behaviours, influence decision making, and facilitate interpersonal interactions, at times emotions can be more of a hindrance than a help. According to polyvagal theory defensive physiological states triggered by emotions can affect our ability to further regulate complex behaviours such as attention and social engagement (Porges, 1995, 2001).
When an emotion is generated in an inappropriate context or at an inappropriate intensity level it can cause problems. Inappropriate emotional responses are intrinsic to psychopathology, social difficulties, and even physical illness (Gross & Muñoz, 1995; Sapolsky, 1997). Being able to successfully regulate emotions is essential for everyday functioning.

According to the modal model of emotion, emotion generation involves a four-stage process (see figure 5.1, Gross & Thompson, 2009). The process begins with a psychologically relevant situation. To generate an emotion the situation must be attended to, and then appraised by the individual. Appraisal of the situation leads to changes in experiential, behavioural, and physiological response systems. Although this process may seem prescriptive there is actually a great deal of flexibility in generating an emotional response. Firstly, generating an emotion is a constant process of change and modification which involves feedback loops; a salient situation demands attention and appraisal resulting in a response, and as soon as a response occurs, it automatically alters the demands of the original situation so the situation needs to be re-appraised and the process begins again. This process will occur repeatedly during the generation of an emotion. Secondly, humans are not passive spectators when emotions are generated; an individual can intervene at any stage to modify an aspect of the emotional response, which in turn will affect the subsequent stages of the process.

![Figure 5.1. A process model of emotion regulation that highlights five types of emotion regulation strategies (from Gross & Thompson, 2009).](image)

The modal model of emotion conceptualises how emotions are generated, however the model suggests that a situation or event must be appraised before a response is initiated (Gross & Thompson, 2009). The role of cognition in the generation
of emotions is a hotly contested topic (see arguments from Lazarus, 1991, and Zajonc, 1980, 1984). I will begin by giving a brief overview of emotion regulation approaches that are consistent with the modal model, before considering alternative emotion regulation techniques that are more consistent with the reasoning of polyvagal theory.

5.1.1. Emotion Regulation Approaches

Emotion regulation has been defined as “the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions” (Gross, 1998a, p. 275). The strategies used to regulate emotions can be consciously or unconsciously driven, and some efforts to regulate emotions are more adaptive than others. Because emotional responses do not develop in a synchronised fashion there are several components of the emotion generation process that can be regulated. Antecedent-focused strategies are those that are initiated before an emotional response is fully elicited (Gross, 1998b). In anticipation of a situation eliciting an emotion individuals can choose to engage in or avoid the situation (situation selection) or can undertake efforts to modify the situation to alter its emotional impact (situation modification). It is also possible to regulate emotions without changing the environment: individuals can redirect their attention away from the stimuli (attentional deployment) or can reappraise the situation and give it a different meaning (cognitive change). Response-focused strategies on the other hand are those that attempt to alter the physiological, experiential, or behavioural aspects of emotional responding once a response tendency has been elicited (response modulation) (Gross, 1998b).

Teaching people how to regulate their emotions appropriately is central to several types of psychotherapy. There are currently two mainstream approaches to psychotherapeutic practice: cognitive therapy and behaviour therapy. Cognitive and behavioural strategies focus on different aspects of the emotion generation process. As examples of each approach, I will briefly consider the roles cognitive reappraisal and behavioural exposure can play in emotion regulation.

5.1.1.1. Reappraisal. Cognitive restructuring (i.e., altering faulty thoughts) is a popular tenet of several psychotherapies, most notably cognitive behavioural therapy (CBT; Beck et al., 1979; Campbell-Sills & Barlow, 2009). Maladaptive appraisals are thought to be at the core of several psychological disorders (Aldao, Nolen-Hoeksema, &
Schweizer, 2010). Cognitive techniques focus on teaching clients to recognise and alter inappropriate appraisals, with the aim of helping clients to develop more realistic and adaptive appraisals, which should in turn facilitate the regulation of their emotional state. Barber and DeRubeis (1989) label the strategies taught in cognitive therapy as *compensatory skills*. This is because cognitive strategies such as reappraisal do not change the content of primary appraisals; instead, cognitive strategies introduce dissonance into the belief system, allowing the opportunity for the development of more adaptive cognitions that can substitute the primary appraisal i.e., compensate for the original thought.

There are two salient limitations that can influence the effectiveness of cognitive strategies. Firstly, not all cognitions and behaviours are under conscious control. In fact, our nervous system is hypothesised to constantly evaluate and respond to risk outside of conscious control (Porges, 2004b). It may therefore be challenging to try and regulate unconscious responses using strategies that are consciously driven. Secondly, cognitive strategies minimise the role of the body in initiating and maintaining emotional responses. If the target of a strategy (i.e., thoughts) is not the main determinant of an emotional response, the strategy is likely to have a limited effect.

5.1.1.2. Behavioural exposure. Behavioural strategies can be used as alternatives to cognitive interventions, or can be used to complement them (as in CBT, Beck et al., 1979). From a behavioural perspective individuals respond to defined stimulus conditions in consistent ways; these patterns of behaviour are termed *habits*. Habits are formed via the processes of conditioning and associative learning. Because habits are learned, they should decline in strength if they fail to continue to be adaptive. With emotional disorders, maladaptive behaviours are assumed to be learned behaviours that have failed to decline in strength even though the behaviours are debilitating (such as social withdrawal) or harmful (such as substance abuse).

Behavioural techniques focus on the process of *relearning*; their aim is to weaken the association between stimulus conditions and habits. Wolpe's (1968, 1969) theory of *reciprocal inhibition* proposed that competing behavioural responses to anxiety-evoking stimuli could be enacted to inhibit the expression of unwanted behaviours. For example relaxation is thought to be incompatible with anxiety; in anxiety provoking situations relaxation could be used to inhibit and displace the
previous behavioural habits associated with the stimulus conditions. A similar technique to this is taught in dialectical behavioural therapy (DBT; Linehan, 1993a): **Opposite action** involves encouraging clients to act in a manner opposite to an emotion’s action tendency; such as deep, slow breathing to generate physiological relaxation when a client is feeling anxious (McMain, Korman, & Dimeff, 2001). Clients are encouraged to adopt facial expressions, body postures, movements, and thoughts that oppose the behaviour prompted by the emotion. Behaving in an opposite manner to the emotion’s action tendency may allow for the meaning of the emotional event to be altered automatically and without conscious effort (Linehan, Bohus, & Lynch, 2009).

A major limitation of exposure-based treatments is that learning new associations does not destroy old learning (Kehoe & Macrae, 1998). This means that stimulus–habit pairings can be rapidly reinstated, either through spontaneous recovery or reacquisition (Bouton, 1988). Several factors that contribute to renewal effects (i.e., the return of unwanted behaviours after extinction) have been identified; these include the role of contextual cues in maintaining conditioned change (Bouton & Nelson, 1998; Vansteenwegen et al., 2005) and the ability for interoceptive cues (i.e., the perception of internal states) to cue unwanted behaviours (Bouton & Swartzentruber, 1991). Behavioural techniques may therefore limited by both external and internal cues.

### 5.1.2. Emotion Regulation: A Third Way?

Both cognitive and behavioural techniques are hypothesised to work by using compensatory strategies: cognitive reappraisal aims to replace inappropriate negative cognitions, whilst behaviour therapy aims to replace inappropriate stimulus–habit pairings. In neither case is the original response completely extinguished (primary appraisals remain present, and original stimulus–habit pairings remains to compete with newer associations). The existence of original responses is a limitation of these approaches, because there is always the possibility that they will become reactivated. Activation of an original response is likely to trigger the neural circuits that respond to challenge and threat (SNS or DVC activation; Porges, 2004b). Being in a state of physiological defensiveness may reduce the effectiveness of cognitive or behavioural strategies, because individuals will be less successful at modulating their thoughts and behaviours.
Taking an optimistic stance, we can use our understanding of cognitive and behavioural strategies to inform a third approach to emotion regulation. Rather than trying to target cognitions or behaviours directly, polyvagal theory suggests that we can take advantage of the bidirectional pathways between the peripheral nervous system and the brain, and use biologically-based strategies to target the body’s physiology as a means of targeting unwanted cognitions and behaviours (Porges, 1995, 2001, 2003a). Research has demonstrated that lower level neural circuits can regulate the processing of higher neural structures; this means that our physiology can influence cognitive, attentional, and affective processes (Berntson, Cacioppo, & Sarter, 2003). If polyvagal theory is a valid model of emotional responding, instead of using cognitive and behavioural techniques to regulate emotions, therapists should be teaching strategies that influence the function of the ventral vagal complex (i.e., the PNS). Stimulating pathways that activate the ventral vagal complex should not only reduce the expression of unwanted behaviours, but should also promote calming and self-soothing states and increase the accessibility of higher level cognitive structures (Porges, 2009b). Once clients have learned to regain control of their physiological state, it may then be more effective to use cognitive or behavioural strategies if deemed necessary. Although this approach sounds promising, despite a large theoretical basis, polyvagal theory does not explicitly identify strategies that are able to up-regulate the ventral vagal complex (with the exception of an acoustic intervention that has not been empirically tested; Porges, 2003a).

5.1.3. Positive Emotions and Emotion Regulation

Our emotional state is thought to parallel our physiological state. Positive emotions have been associated with activation of the ventral vagal complex and the PNS (Bazhenova & Porges, 1997), whilst negative emotions have been linked to activation of the SNS (Fredrickson, Mancuso, Branigan, & Tugade, 2000). Polyvagal theory is based on the assumption that our visceral states affect how we feel, and in turn our feelings affect our physiological state (Porges, 2009a). It is conjectured that interventions that target the body’s physiology may be able to enhance the activation of the PNS and accelerate down-regulation of the SNS (Porges, 2007a). These physiological changes should be accompanied by reduced levels of subjective distress and increased social engagement (Porges, 2001). These are important aims in clinical
practice for clients who have difficulties regulating their emotions and sustaining interpersonal relationships.

Behavioural interventions that target biological systems (i.e., biobehavioural strategies) are increasingly being incorporated into psychotherapeutic practices. For example DBT has a distinct set of skills that are specifically aimed at down-regulating physiological arousal (Linehan et al., 2009). The advantage of biobehavioural skills is that they do not require a high level of cognitive processing to complete, yet they have a high impact on physiological arousal (Linehan et al., 2009). Based on polyvagal theory, it is conjectured that combining biobehavioural strategies with techniques that promote positive emotion may enhance their overall effectiveness. Several strategies aimed at increasing positive emotion have been associated with enhanced cardiac vagal control and self-regulation. In the current research, three positive emotion regulation strategies were identified as potential techniques that may be used to increase activation of the PNS: smiling, mindful breathing, and a loving-kindness meditation. These techniques are discussed individually in the following sections.

### 5.1.3.1. Smiling

Teaching clients to smile when feeling distressed is part of the opposite action repertoire of emotion regulation skills (Linehan, 1993a). The ability for smiling to induce positive affect and changes in physiology relies on feed-back rather than feed-forward processes; in this sense smiling is a bottom-up strategy, rather than a top-down strategy (Berntson et al., 2003; Taylor, Goehler, Galper, Innes, & Bourguignon, 2010). Smiling targets the afferent pathways of the face, making it plausible that this strategy may influence the body’s physiology by stimulating lower order neural pathways. Afferent pathways from the face have source nuclei in the brainstem near to where the cardiac vagal motor neurons originate (Porges, 1995; Sawchenko, 1983). Activating these source nuclei may result in up-regulation of the cardiac vagal motor neurons, resulting in increased activation of the PNS.

Several emotion theorists believe facial expressions play a key role in the experience of emotions (Izard, 1971; Tomkins, 1962). Darwin (1872/2009) was one of the first to recognise that enhancing or inhibiting the expression of an emotion can alter the intensity of the emotional experience. It has been proposed that facial expressions can affect emotional experience because of bidirectional links between the motor cortex and the brain regions involved in generating emotional responses (Hennenlotter et al.,
2009; Levenson, Ekman, & Friesen, 1990). Posed facial expressions involve different neuronal pathways from spontaneous facial expressions (Matsumoto & Lee, 1993), but despite the different efferent pathways both forms of expression result in afferent feedback from the facial muscles. Afferent feedback from the face is presumed to be able to modulate the emotional experience: This theory is known as the facial feedback hypothesis (Buck, 1980; Strack, Martin, & Stepper, 1988; Tomkins, 1962; Zuckerman, Klorman, Larrance, & Spiegel, 1981). It is important to note that there is contrasting evidence refuting the influence of facial feedback (Matsumoto, 1987; Tourangeau & Ellsworth, 1979), however the theory has received a great deal of attention over the last three decades.

The facial feedback hypothesis suggests that positive facial expressions (e.g., smiling) should not only be accompanied by greater self-reports of positive affect, but also their psychophysiological counterparts (Zuckerman et al., 1981). Positive emotion coupled with spontaneous smiling has been associated with faster cardiovascular recovery from negative mood inductions (Fredrickson & Levenson, 1998). Positive emotional expression has also been linked with psychological flexibility and increased attention (Johnson, Waugh, & Fredrickson, 2010). Consequently encouraging individuals to produce smiles when experiencing negative emotions may help to reduce the experience of negative emotions, and may also reduce their psychophysiological arousal.

To produce a smile involves contraction of the zygomaticus major muscle (the cheek muscle which pulls up the corner of the mouth; Fridlund & Cacioppo, 1986). This muscle is controlled by cranial nerves that originate in the brainstem proximal to the source nuclei of the myelinated vagus nerve. Porges (1995, 2003a, 2003b) has theorized that the anatomical overlap between the cranial nerves controlling the face and head and the myelinated vagus nerve has resulted in a heart–face link that also has direct connections to the prefrontal cortex. Porges’ terms this network the social engagement system (SES; Porges, 1998, 2003a). Utilising the bidirectional pathways between the heart and face through smiling may directly stimulate the SES and increase the activation of the myelinated vagus nerve. Up-regulation of the SES should ultimately result in decreased defensive arousal due to the dampening effects of the myelinated vagus nerve on SNS activation.
5.1.3.2. Mindful breathing. A second technique that targets the body’s physiology and is associated with positive emotion is mindful breathing. A basic definition of mindfulness is “moment-by-moment awareness” (Germer, 2005, p. 6). The primary goal of mindfulness is not to relax, but to observe arising stimuli from a non-judgemental perspective (Baer, 2003). Mindfulness is a state of mind that is accessible in all situations, and its practice has been linked to increases in psychological functioning as well as increases in well-being (Carmody & Baer, 2008; Segal, Williams, & Teasdale, 2002). Due to the psychological benefits associated with mindfulness it is increasingly being included in psychotherapeutic treatment programs, for example mindfulness-based cognitive therapy (MBCT; Segal et al., 2002) and dialectical behavioural therapy (DBT; Linehan, 1993a).

Mindfulness does not involve consciously directing one’s attention to positive experiences, however there is a wide range of evidence to suggest that mindfulness can increase positive affect by creating conditions for positive emotions to develop (Fredrickson, 2000). Awareness itself has qualities that are similar to positive emotions (Fredrickson, 2003), and it has been suggested that mindfulness practices may create a positively valenced state of mind (Garland, Gaylord, & Park, 2009). Mindfulness may increase engagement with positive emotions, as the act of mindfulness can foster awareness and attention to positive events and feelings (Erisman & Roemer, 2010). Indeed greater mindfulness training has been shown to correspond to decreased negative affect and increased positive affect (Jha, Stanley, Kiyonaga, Wong, & Gelfand, 2010), and has also been found to activate areas of brain involved in the experience of positive emotion (Davidson et al., 2003).

Several behavioural techniques have been developed to enhance mindfulness skills, including body scan techniques, mindfulness meditations, and breathing practices (Kabat-Zinn, 1994; Linehan, 1993a). Using the breath as a focus of attention is a basic mindfulness practice where the breath itself is not manipulated. The focus is on the physical sensations of the breath, and other thoughts, feelings, and sensations are simply observed without controlling attention (Segal et al., 2002). Focusing attention on the breath reminds individuals to recognise their experience in the present moment. When the mind wanders from the breath, individuals are gently encouraged to bring their attention back to their breathing. Being aware that the mind has wandered and bringing attention back to the breath is an important aspect of the practice. Mindful breathing
practices are highly accessible because the breath is a neutral object of attention for most people, and individuals can tune into the sensations of their breath at any given moment (Teasdale, Segal, & Williams, 1995).

In terms of physiology, breathing is a bodily function that is regulated by the autonomic nervous system. Breathing in is associated with an increase in heart rate, whilst breathing out is associated with a decrease in heart rate. The coupling between respiration and the heart is a determinant of heart rate variability (HRV; Berntson, Cacioppo, & Quigley, 1993b). Even though mindfulness practices do not actively manipulate bodily functions such as the rate or depth of breathing, participants practicing mindfulness techniques have been found to show increases in HRV (Ditto, Eclache, & Goldman, 2006; Lehrer, Sasaki, & Saito, 1999; Wu & Lo, 2008). Recently it has been shown that even short-term meditation training (five days of 20 minutes) can improve HRV (Tang et al., 2009). The changes in HRV have been attributed to increases in vagal activity, suggesting a link between mindfulness and the PNS.

5.1.3.3. Loving-kindness meditations. A second closely related meditation practice to mindfulness is loving-kindness. Loving-kindness meditations are used to consciously increase feelings of warmth and care for the self and others (Salzberg, 1995). Fredrickson et al. (2008) demonstrated that the practice of loving-kindness can lead to increases in a wide range of positive emotions, including love, joy, contentment, interest, and amusement. Techniques that promote positive emotions have been shown to help people regulate their emotions and maintain calm states (Fredrickson et al., 2008; Gilbert & Procter, 2006; Linehan, 1993a). Loving-kindness meditation practices have also been linked to increases in personal resources, for example increased social support and decreased illness symptoms (Fredrickson et al., 2008). Further to this loving-kindness meditations have been shown to reduce psychological distress and chronic pain, as well as increase feelings of social connectedness (Carson et al., 2005; Hutcherson, Seppala, & Gross, 2008). Calling into mind feelings of support and connectedness in the absence of meditation has itself been linked to attenuated cardiovascular activity (Ratnasingam & Bishop, 2007).

From a physiological point of view, the positive emotions generated by loving-kindness meditations are thought to be able to ‘undo’ the after-effects of negative emotions i.e., they can down-regulate the psychophysiological effects of negative
emotions that activate fight–flight behaviours (Fredrickson & Levenson, 1998; Fredrickson et al., 2000). Little empirical research has been carried out to explore the link between loving-kindness meditations and the autonomic nervous system, although increased PNS activation is a possible mechanism by which positive emotions may down-regulate the psychophysiological effects of negative emotions. Rockliff, Gilbert, McEwan, Lightman, and Glover (2008) found that compassion-focused imagery could increase HRV and stimulate self-soothing in some individuals, however individuals who were more self-critical with insecure attachment styles responded defensively to the imagery and showed a reduction in HRV. Increases in vagal indices have also been reported when participants consciously focus on feelings of care, appreciation, and social connectedness (Kok & Fredrickson, 2010; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995).

Porges (1998) has explored the relationship between love and the autonomic nervous system from a polyvagal perspective. Feelings of love are important in facilitating social interactions and reproduction. To increase the possibility of social interactions and reproduction occurring proximity must be increased. Consequently the social behaviours associated with love (e.g., social engagement and courtship) rely on feelings of safety and activation of the myelinated vagus nerve. Activation of the myelinated vagus nerve may help to explain why increased compassion is linked to increases in HRV, and why generating emotions such as love can down-regulate the psychophysiological effects of negative emotions (Fredrickson & Levenson, 1998; Rockliff et al., 2008).

5.2. Current Study Aim and Hypothesis

The current study used a between subjects design to investigate the psychophysiological effects of three emotion regulation strategies in response to a stressor manipulation (the adapted speech task from Chapter 4). The strategies evaluated were smiling, mindful breathing, and a loving-kindness meditation. These were compared to a neutral listening task and resting quietly. The aim of the current experiment was to establish whether or not any of the strategies of interest could influence the activation of the autonomic nervous system. The following hypothesis was tested:
Hypothesis 2. Emotion regulation strategies will be associated with increased activation of the PNS, and accelerate the down-regulation of physiological and psychological arousal after a stressor.

Targeting the PNS should result in increases in high-frequency heart rate variability (HF-HRV) and cause quicker physiological returns to baseline during acute stress. As well as focusing on the ‘undoing’ effects of the emotion regulation strategies, due to the design of the speech task used it was also possible to investigate whether the strategies had a buffering effect on impending stress.

5.3. Methodology

5.3.1. Participants

One hundred undergraduate psychology students (14 males, 86 females) volunteered to participate in the study and were awarded course credits as part of their undergraduate course requirements. Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders, and current psychological or pharmacological treatment. The participants ranged in age from 18–35 with a mean age of 19.80 years ($SD = 3.37$). 93.0% of these participants identified themselves as Caucasian, 2.0% as Asian, 1.0% as Mixed, and 4.0% as Other. Participants were allocated into five intervention groups: smiling ($n = 20$: 2 males, 18 females; mean age = 19.05, $SD = 1.82$), mindful breathing ($n = 20$: 2 males, 18 females; mean age = 20.10, $SD = 4.17$), loving-kindness ($n = 20$: 2 males, 18 females; mean age = 18.80, $SD = 0.83$), neutral listening ($n = 20$: 4 males, 16 females; mean age = 19.50, $SD = 1.96$), and a control group ($n = 20$: 4 males, 16 females; mean age = 20.57, $SD = 3.32$).

5.3.2. Regulatory Strategies

5.3.2.1. Smiling. Smiling activates afferent pathways from the facial muscles. “Duchenne” smiles are often induced in laboratory studies by getting participants to grip pencils between the teeth and pull the corners of their lips upward (i.e., contracting the zygomaticus major muscle), but this technique has little ecological validity (Soussignan, 2002). An alternative technique that activates the muscles of the face is taught in DBT:
the half-smile (Linehan, 1993a). The half-smile involves turning attention to the face. Clients are encouraged to relax the face, neck, and shoulders, and then turn up the corners of their lips (Linehan, 1993b). The smile is not an exaggerated smile, as tense smiling will signal hiding or masking; instead the half-smile is a soft smile like the smile of the Mona Lisa. In this experiment participants were shown a picture of the Mona Lisa for five minutes and were asked to imitate the Mona Lisa’s facial expression.

5.3.2.2. Mindful breathing. Participants listened to a five-minute audio recording that instructed them to breathe normally whilst focusing their entire attention on their breath. The recording was an abbreviated version of a mindful breathing exercise by Kabat-Zinn (2005). Each time they exhaled they were also asked to press a computer key that recorded the action. The script for the mindful breathing practice can be found in appendix 12.

5.3.2.3. Loving-kindness meditation. Participants listened to a five-minute audio recording of a loving-kindness meditation that paralleled the seven-minute visualization procedure outlined in Hutcherson et al. (2008). To try and increase the feelings of love and warmth participants were asked to bring a photograph of a loved one with them to the experiment (Master et al., 2009). The audio recording instructed the participants to generate feelings of love and kindness whilst viewing their photograph, and silently repeat a series of phrases that brought attention to the self and wished themselves health, happiness, and well being. The script for the loving-kindness meditation can be found in appendix 13.

5.3.2.4. Neutral listening. Participants listened to a five-minute audio recording that replicated the unfocused attention condition used by Arch and Craske (2006). This involved participants listening to instructions which allowed their mind to wander, for example “Simply think about whatever comes to mind. Let your mind wander freely without trying to focus on anything in particular.” Variants of these instructions were repeated every 30–60 seconds for five minutes. The script for the neutral listening task can be found in appendix 14.

5.3.2.5. Control. To establish the effects of the regulatory strategies over and above the effect of time, a control task of resting quietly for five minutes was included.
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5.3.3. Performance Ratings

5.3.3.1. Post-task questionnaire. At the end of the experiment participants were asked to think about the strategy they had used and to rate the following statements on a nine-point Likert scale (1 not at all true to 9 very true): I put a lot of effort into carrying out the task, I found the task easy to do, and I enjoyed doing the task. They were also asked to complete dichotomous rating scales as to whether the strategy made them feel happy or sad (1 happy to 9 sad), tense or relaxed (1 tense to 9 relaxed), and aroused or not aroused (1 aroused to 9 not aroused). Participants were also asked to think about the performance of their speech and to rate the following statements on a nine-point Likert scale (1 not at all true to 9 very true): I felt as though I had enough time to prepare my speech, I felt my performance was satisfactory, and I felt nervous during the speech.

5.3.4. Procedure

Participants attended a single testing session in an air-conditioned, sound attenuated room. After completing the consent form, the demographic screening questionnaire (see section 3.5.1), and the Hospital Anxiety and Depression Scales (HADS; Zigmond & Snaith, 1983, see section 3.5.3), electrodes for recording heart rate (HR) and skin conductance level (SCL) were applied (see section 3.4.4). Recording HR allowed for the calculation of heart rate variability (HRV, see section 3.4.3). A five-minute baseline recording was carried out during which the participants were asked to sit quietly. Participants then completed the Profile of Mood States – Short Form (POMS-SF; Shacham, 1983, see section 3.5.2). After this the participants were informed that they would have to prepare and present a three-minute speech (see section 4.6.2). After the three-minute preparation period the participants were told they had a few minutes before they would have to present their speech (five-minute intervention period). During the five-minute intervention period the participants were asked to carry out one of five strategies: smiling, mindful breathing, a loving-kindness meditation, neutral listening, or a control strategy (resting quietly). After the intervention period all of the participants completed a second POMS-SF questionnaire and were then instructed to present their speech in front of the video camera for three minutes. If the participants stopped talking before the end of the three minutes, they were asked to continue talking by summarizing the main points. A flowchart diagram of the emotion regulation strategies procedure can be found in appendix 15.
5.4. Results

5.4.1. Statistical Analyses

For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. To identify potential covariates, the relationships between potential confounds (i.e., age, ethnicity, sex, self-reported anxiety and depression symptoms) and the dependent variables (i.e., HR, SCL, HF-HRV, and the POMS-SF) were examined. Bivariate correlations revealed significant associations between sex and the SCL data (baseline SCL: $r = .20, p = .045$; preparation SCL: $r = .23, p = .020$; intervention SCL: $r = .26, p = .10$). The direction of the relationship suggests that females exhibited higher SCL than males. Consequently Sex was included as a covariate in all analyses of SCL. Correlations revealed no significant associations between age, ethnicity, or self-reported depression and anxiety symptoms with any of the other dependent variables (all $p$s < .10).

Mean HR and SCL values were calculated for the baseline periods and each minute of the speech task and intervention periods. High-frequency heart rate variability (HF-HRV) was also calculated using the HR data for three minutes of the baseline period, the three-minute preparation period, and the five-minute intervention period. HF-HRV was not calculated for the presentation period of the speech task due to the known effects of respiratory changes on HRV indices (Beda, Jandre, Phillips, Giannella-Neto, & Simpson, 2007; Tininenko, Measelle, Ablow, High, 2012).

Analyses were performed to establish if there were any baseline differences between the intervention groups. Chi-squared tests confirmed that the groups did not significantly differ in terms of sex, smoking status, coffee consumption, or self-reported anxiety and depression symptoms (see table 5.1 for the self-report means for each group, all $p$s > .05). A univariate analysis of variance (ANOVA) also revealed that the intervention groups did not significantly differ in terms of age ($p > .05$). Univariate ANOVAs with the physiological data from the baseline periods (HR, SCL, and HF-HRV) as the dependent variables revealed no differences in physiology across the baseline periods (all $p$s > .10). The SCL analysis was repeated with Sex as a covariate, but still no significant group differences were revealed. A separate ANOVA with the
POMS-SF data for the baseline periods also failed to reveal any significant baseline differences between the intervention groups (all $p > .10$). The raw HR, SCL, and HF-HRV scores for the interventions groups are shown in Table 5.2.

### Table 5.1.

<table>
<thead>
<tr>
<th>Intervention Group</th>
<th>HADS-A Total</th>
<th>HADS-D Total</th>
<th>POMS-SF Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smiling</td>
<td>6.65 (4.61)</td>
<td>1.90 (1.59)</td>
<td>5.50 (11.93)</td>
</tr>
<tr>
<td>Mindful Breathing</td>
<td>6.45 (3.32)</td>
<td>2.70 (2.16)</td>
<td>8.00 (10.13)</td>
</tr>
<tr>
<td>Loving-Kindness</td>
<td>6.20 (3.68)</td>
<td>2.15 (1.73)</td>
<td>4.70 (8.83)</td>
</tr>
<tr>
<td>Neutral Listening</td>
<td>7.10 (3.40)</td>
<td>2.45 (2.14)</td>
<td>3.90 (10.55)</td>
</tr>
<tr>
<td>Control</td>
<td>7.45 (3.68)</td>
<td>2.72 (2.59)</td>
<td>7.72 (10.65)</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HADS-A = Hospital Anxiety and Depression Scale – Anxiety subscale, HADS-D = Hospital Anxiety and Depression Scale – Depression subscale, POMS-SF = Profile of Mood States – Short Form.

As in Chapter 4, *reactivity scores* (differences from baseline) were used to establish differences in physiological reactivity across the intervention groups (Tomaka, Blascovich, Kelsey, & Leitten, 1993). Reactivity scores were calculated for HR, SCL, and HF-HRV by subtracting the speech preparation data from the corresponding baseline data (Kamarck et al., 1992; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). To establish that the preparation period induced a similar level of arousal for each of the groups ANOVAs were performed on the reactivity scores for HR, SCL, and HF-HRV. The SCL ANOVA was repeated with Sex as a covariate. No significant differences were found suggesting that the groups found the preparation period comparatively stressful (all $F > 1$, all $p > .05$).

The main set of analyses reported in this chapter investigated the effects of the interventions on physiological arousal over time. To allow for clearer comparisons across the groups, HR and SCL recovery scores were calculated for each minute of the intervention period. *Recovery scores* were calculated for HR and SCL by subtracting the data for each minute of the recovery period from the last minute of the preparation period (Llabre et al., 1991). HF-HRV recovery was calculated by subtracting the HF-HRV power during the preparation period from the intervention period. Repeated-measures ANOVAs were performed on the recovery scores for HR, SCL, and HF-HRV with the Huynh-Feldt degrees of freedom correction applied where necessary (i.e., when factors violated sphericity assumptions, as confirmed by Mauchly’s tests). No adjustments were made for baselines in these analyses, because comparable
physiological baselines were revealed across the intervention groups; the SCL analyses were however repeated with Sex as a covariate. Significant main effects for all analyses were followed up with pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05. Post hoc paired t-tests were also used to examine differences within each intervention group (for each minute of the recovery period raw HR and SCL were compared with baseline).

5.4.2. Psychophysiological Recovery during the Interventions

The HR recovery data is shown in figure 5.2. All of the intervention groups exhibited reductions in HR during the intervention period. A repeated-measures ANOVA on the HR recovery data revealed significant main effects of Time, \( F(3.14, 298.56) = 53.20, p < .001 \), and Group, \( F(4, 95) = 2.52, p = .046 \). A significant Time x Group interaction was also revealed, \( F(12.57, 298.56) = 2.86, p = .001 \). On average, HR return to baseline was lowest in the control group (mean change = -4.38 beats per minute) and largest in the mindful breathing group (mean change = -10.20 beats per minute). Univariate ANOVAs investigating each minute of the recovery period revealed that the largest changes in HR were seen during the first two minutes of the intervention period: the smiling, mindful breathing, and neutral listening groups demonstrated significantly larger decreases in HR than the control group during the first minute; although only the mindful breathing and neutral listening groups maintained this advantage during the second minute (as confirmed by Bonferroni pairwise comparisons, \( p < .05 \)). After the third minute there were no significant differences between the intervention groups.

Paired t-tests were used to examine within group differences in HR recovery: By the fifth minute of the intervention period HR in the control group was still significantly above baseline, \( t(19) -2.92, p = .009 \). In contrast, HR in the smiling, mindful breathing, and loving kindness groups was no longer significantly different from baseline (all \( ts < 0.65 \), all \( ps > .05 \)). Only the neutral listening group exhibited significantly lower HR than baseline at the end of the intervention period, \( t(19) 2.53, p = .020 \).
### Table 5.2.
Mean heart rate, skin conductance level, and high-frequency heart rate variability by group during baseline and the phases of the speech task.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Speech Task</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preparation</td>
<td>Intervention</td>
<td>Presentation</td>
<td>F</td>
<td>df</td>
</tr>
<tr>
<td>Smiling</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>82.48 (12.99)</td>
<td>91.63 (12.97)</td>
<td>83.72 (11.35)</td>
<td>91.75 (10.35)</td>
<td>18.49</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.22 (1.35)</td>
<td>4.45 (1.97)</td>
<td>4.56 (1.94)</td>
<td>5.93 (2.52)</td>
<td>54.09</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.14 (1.13)</td>
<td>6.88 (1.12)</td>
<td>7.40 (1.04)</td>
<td>--</td>
<td>4.85</td>
</tr>
<tr>
<td>Mindful Breathing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>80.95 (11.81)</td>
<td>90.25 (13.21)</td>
<td>80.80 (12.52)</td>
<td>92.76 (15.35)</td>
<td>37.63</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.27 (1.33)</td>
<td>4.77 (4.16)</td>
<td>4.45 (3.43)</td>
<td>6.05 (4.28)</td>
<td>18.31</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.18 (0.75)</td>
<td>6.80 (0.71)</td>
<td>7.60 (0.93)</td>
<td>--</td>
<td>18.35</td>
</tr>
<tr>
<td>Loving-Kindness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>84.25 (11.31)</td>
<td>92.02 (11.56)</td>
<td>82.80 (11.25)</td>
<td>94.93 (12.86)</td>
<td>27.87</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.02 (1.52)</td>
<td>3.84 (2.16)</td>
<td>3.78 (2.37)</td>
<td>5.31 (2.55)</td>
<td>82.12</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.15 (0.87)</td>
<td>6.85 (0.81)</td>
<td>7.34 (0.72)</td>
<td>--</td>
<td>4.91</td>
</tr>
<tr>
<td>Neutral Listening</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>79.90 (13.94)</td>
<td>89.17 (12.00)</td>
<td>79.29 (12.93)</td>
<td>95.91 (12.84)</td>
<td>36.14</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.10 (1.45)</td>
<td>3.97 (2.33)</td>
<td>3.84 (2.46)</td>
<td>4.99 (3.10)</td>
<td>37.74</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>6.99 (1.13)</td>
<td>6.67 (1.19)</td>
<td>7.30 (1.08)</td>
<td>--</td>
<td>11.25</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>79.27 (10.08)</td>
<td>90.44 (12.79)</td>
<td>84.92 (11.19)</td>
<td>94.97 (13.78)</td>
<td>16.88</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>1.53 (1.34)</td>
<td>3.32 (1.65)</td>
<td>3.73 (1.77)</td>
<td>4.41 (2.26)</td>
<td>58.71</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.23 (0.92)</td>
<td>6.47 (1.04)</td>
<td>7.16 (1.00)</td>
<td>--</td>
<td>10.80</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
In contrast to the HR data, SCL initially increased for all of the groups following the preparation period, and then exhibited a slow decline across the intervention period (see figure 5.3). A repeated-measures ANOVA revealed significant main effects of Time, $F(1.48, 140.20) = 50.01, p < .001$, and Group, $F(4, 95) = 3.28, p = .015$, as well as a Time x Group interaction, $F(5.90, 140.20) = 4.47, p < .001$. The effect of Group and the Time x Group interaction remained significant when Sex was entered into the analysis as a covariate. A linear contrast confirmed that the slope of the SCL recovery scores differed as a function of Group, $F(4, 94) = 4.94, p < .001$. Post hoc analyses revealed that the mindful breathing group’s SCL recovery was significantly steeper than the smiling, control, and neutral listening groups (all $p$s < .05), and Bonferroni pairwise comparisons confirmed that the mindful breathing group was significantly lower than the control group at minute five of the intervention period ($p = .006$). Despite the Time x Group interaction, at the end of the intervention period SCL remained significantly higher than baseline in all of the groups (all $t$s > 3.57, all $p$s < .002).

5.4.2.1. Heart rate variability. All of the intervention groups demonstrated increases in HF-HRV from the preparation period to the intervention period, see figure 5.4. Despite notable differences in HF-HRV across the time periods, a repeated-measures ANOVA on the HF-HRV recovery scores indicated that there was no significant effect of Group on HF-HRV. Paired $t$-tests examining within group effects revealed that HF-HRV during the intervention period was significantly higher than baseline for the smiling ($p = .022$), mindful breathing ($p = .002$), and neutral listening ($p = .008$) groups, but not the loving kindness or control groups ($p > .05$).
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Figure 5.2. Mean heart rate recovery scores for each minute of the intervention period as a function of group. Error bars represent the standard error.

Figure 5.3. Mean skin conductance level recovery scores for each minute of the intervention period as a function of group. Sex was entered into the model as a covariate (variable coded as male = 1, female = 2; covariate value = 1.86). Error bars represent the standard error.

Figure 5.4. Mean high-frequency heart rate variability (HF-HRV) in milliseconds square (ms^2) for as a function of group during the preparation period and the intervention period. Error bars represent the standard error.
5.4.3. Effects of the Interventions on Mood

5.4.3.1. POMS-SF. Compared to baseline, all of the groups demonstrated an increase in the POMS-SF tension–anxiety subscale after the intervention period, as shown in table 5.3. A repeated-measures ANOVA confirmed that there was a significant effect of Time on self-reported tension and anxiety, $F(1, 95) = 91.79, p < .001$. No significant group differences were revealed, suggesting that the groups experienced equivalent changes in mood state. Paired $t$-tests examining within group effects revealed that the tension–anxiety subscale was significantly higher after the intervention period in all of the intervention groups (at least significant to $p = .001$).

Table 5.3.

Mean scores for the POMS-SF tension–anxiety subscale by intervention group

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Before Mean (SD)</th>
<th>After Mean (SD)</th>
<th>$t$</th>
<th>df</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smiling</td>
<td>2.60 (3.47)</td>
<td>6.90 (6.56)**</td>
<td>-3.92</td>
<td>19</td>
<td>.001</td>
</tr>
<tr>
<td>Mindful Breathing</td>
<td>3.30 (2.25)</td>
<td>7.25 (4.52)**</td>
<td>-3.49</td>
<td>19</td>
<td>.002</td>
</tr>
<tr>
<td>Loving-Kindness</td>
<td>3.50 (2.89)</td>
<td>7.15 (3.91)***</td>
<td>-4.55</td>
<td>19</td>
<td>.001</td>
</tr>
<tr>
<td>Neutral Listening</td>
<td>2.85 (2.56)</td>
<td>8.20 (5.49)***</td>
<td>-5.39</td>
<td>19</td>
<td>.001</td>
</tr>
<tr>
<td>Control</td>
<td>3.73 (3.21)</td>
<td>8.63 (5.32)***</td>
<td>-4.42</td>
<td>19</td>
<td>.001</td>
</tr>
</tbody>
</table>

*Note. Standard deviations are reported in parentheses. Significant change from baseline ** $p < .01$, ***$p < .001$

5.4.3.2. Relationships between mood and recovery. To investigate whether changes in ANS function during the intervention period were related to changes in self-reported emotion, correlations were carried out between the mean HR, SCL, and HF-HRV recovery scores and the mean POMS-SF reactivity scores. Significant correlations were only found in the control group. Larger HR recovery in the control group was associated with larger decreases in the POMS-SF confusion–bewilderment subscale ($r = .73, p = .027$), the POMS-SF tension–anxiety subscale ($r = .52, p = .020$), and the POMS-SF anger–hostility subscale ($r = .57, p = .009$). The directions of these relationships suggest that larger decreases in SNS activation during the intervention period in the control group during were associated with greater decreases in self-reported negative affect. In addition to this, negative relationships were revealed between HF-HRV recovery in the control group and the POMS-SF tension–anxiety subscale ($r = -.71, p < .001$) and the POMS-SF confusion–bewilderment subscale ($r = -.68, p = .001$), suggesting that larger increases in PNS activation during the intervention period in the control group were also associated with larger increases in
indices of negative affect. Although this latter relationship is counter-intuitive, increased negative affect may result in increased self-regulation, hence an increase in PNS activation being coupled with indices of negative affect.

5.4.3.3. Post-task questionnaire. Only the active intervention groups completed the post-task questionnaire (the control group who rested quietly did not complete the post-task questionnaire as they did not have an active task to evaluate); the active intervention groups’ responses were compared using a univariate ANOVA. The post-task questionnaire indicated a significant difference between the groups on the statement: “I put a lot of effort into carrying out the task”, \( F(3, 76) = 7.00, p < .001 \). Bonferroni pairwise comparisons revealed that the smiling group \((M = 4.50, SD = 2.07)\) reported putting significantly less effort into the task than the other groups (mindful breathing \(M = 7.00, SD = 1.84\); loving kindness \(M = 6.60, SD = 1.27\); neutral listening \(M = 6.50, SD = 2.26\)). Significant effects of Group were also found for the statement “I enjoyed the task”, \( F(3, 76) = 3.12, p = .031 \), and the nine-point happy–sad rating scale, \( F(3, 76) = 3.60, p = .017 \). In both instances the loving-kindness group reported significantly greater scores than the smiling group (enjoyment \(Ms = 6.15\ vs. 4.05\); happy–sad \(Ms = 2.60\ vs. 4.00\), suggesting that the loving-kindness task resulted in greater positive affect than the smiling task.

5.4.4. Psychophysiological Reactivity to the Speech Presentation

As shown in table 5.2, all of the groups demonstrated significant increases in HR and SCL during the presentation of the speech \((p = .001)\); HF-HRV was not calculated for the presentation period so this change was not analysed. Univariate ANOVAs failed to reveal any group differences in terms of physiological reactivity to the speech presentation. Consequently the interventions evaluated had little effect in buffering individuals from future stress. Univariate ANOVAs were also carried out on the self-report ratings evaluating the speech task performance. A significant effect of Group was found for the rating “I felt as though I had enough time to prepare my speech”, \( F(4, 95) = 7.78, p < .001 \). Bonferroni pairwise comparisons revealed that the control group felt as though they had more time to prepare their speech \((M = 5.90, SD = 2.90)\) compared to the other groups (loving kindness \(M = 2.60, SD = 1.47\); smiling \(M = 2.90, SD = 1.86\); neutral listening \(M = 3.00, SD = 2.20\); mindful breathing \(M = 3.90, SD = 2.08\)). A second significant effect of Group was found for the rating “I felt my performance was
satisfactory”, $F(4, 95) = 4.07, p = .004$. The control group ($M = 4.90, SD = 2.25$) reported the greatest satisfaction with their performance (loving kindness $M = 2.90, SD = 1.45$; neutral listening $M = 2.95, SD = 1.64$; smiling $M = 3.75, SD = 2.10$; mindful breathing $M = 4.45, SD = 2.28$), although only the differences between the control group and the loving kindness and neutral listening groups were significant ($p < .05$).

5.5. Discussion

The aim of this study was to evaluate the effects of emotion regulation strategies on psychophysiological recovery from an acute stressor. The chosen strategies have previously been identified as techniques that can increase positive emotion, as well as increase activation of the PNS. It was hypothesised that interventions targeting physiology may be able to enhance the down-regulation of SNS arousal and subjective distress associated with laboratory stress.

5.5.1. Psychophysiological Effects of Emotion Regulation

The current experiment compared the effects of resting quietly and neutral listening to three emotion regulation strategies: smiling, mindful breathing, and a loving-kindness meditation. The psychophysiological effects of the strategies will be evaluated in turn.

5.5.1.1. Resting quietly. Similar to the anticipation group in Chapter 4, the group that rested quietly in the current experiment demonstrated a limited return to physiological baseline. By the end of the five-minute intervention period, participants who had rested quietly still exhibited significantly higher HR and SCL than baseline. No significant changes were identified in HF-HRV. Interestingly, greater SNS activation whilst resting quietly was associated with greater increases in self-reported negative affect. The results of this group may suggest that in times of acute stress, doing something active may be better than simply resting quietly.

5.5.1.2. Smiling. In the smiling group HR generally declined towards baseline (particularly in the first minute), whilst SCL levels remained high. By the end of the five-minute intervention period, HR was no longer significantly different from baseline although SCL remained elevated. The HR and SCL responses were combined with a
significant increase in HF-HRV during the intervention period, although this was not significantly different from the other groups. In summary, the smiling group did not tend to show greater reductions in defensive physiological arousal than the other groups, nor did the smiling group report feeling significantly happier than the other groups during the intervention period (this is in contrast to previous research on the facial feedback hypothesis; Strack et al., 1988; Zuckerman et al., 1981). The effects of smiling on down-regulating physiological arousal and increasing positive affect are therefore considered to be limited. The effects may have been limited by the instructions for the task, as the facial feedback hypothesis is reported to be more powerful when the facial configuration is a representative visual analogue of enjoyment smiles (Soussignan, 2002).

5.5.1.3. Neutral listening. Letting one’s mind wander had a discernible effect on recovery during the intervention period. Although SCL remained significantly higher than baseline, the neutral listening group was the only group to demonstrate a decrease in HR that was significantly lower than baseline at the end of the intervention period. In addition to the decreases in HR and SCL, HF-HRV demonstrated a significant increase above baseline during the intervention period, suggesting that the task increased activation of the PNS. It is possible that these effects were caused by distraction: Distraction involves directing one’s attention away from the self and current problems, and is often used as an emotion regulation technique (McRae et al., 2009). Short-term distraction can be an adaptive way to regulate emotion, however chronic use of distraction can lead to avoidance and is thought to be maladaptive (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). A potential flaw with this conclusion is that it ignores the fact that encouraging participants to let their mind wander is analogous to mindfulness. This caveat is considered to be a limitation of this experiment. Despite this, listening to neutral audio during acute stress may be a useful strategy that can be employed to help to down-regulate physiological arousal during acute stress.

5.5.1.4. Mindful breathing. The mindful breathing group exhibited a rapid return to baseline for HR, and despite an initial increase in SCL demonstrated a notable return to baseline for SCL (the rate of change for SCL recovery was significantly greater than for the smiling, control, and neutral listening groups, $p < .05$). The HF-HRV data also indicated that the mindful breathing group showed an increase in HF-HRV during the intervention period, however this was not significantly different from
the other groups. It is possible that the mindful breathing group may have shown a greater return to baseline, over and above the other groups, had the intervention period been longer. It is important to take into account the fact that the individuals in the mindful breathing condition had no prior practice of the task, which may have limited the effects of the intervention (practice time is often linked to better outcomes in meditation studies; e.g., J. W. Carson et al., 2005; Pace et al., 2009). Had the effects been greater, mindful breathing would have elicited changes consistent with the hypothesis (i.e., decreased HR and SCL, with increased HF-HRV).

5.5.1.5. Loving-kindness meditation. The loving-kindness meditation group demonstrated a similar response to the neutral listening group. HR showed a rapid return to baseline along with a decline in SCL, which demonstrated a steady reduction but did not return to baseline during the five minutes. HF-HRV showed an increase in the loving-kindness group, however this was not significantly different from baseline. Although the loving-kindness meditation did not show a unique physiological response to the stressor, the group did report notable positive affect in the post-task questionnaire (in terms of enjoyment, and ratings on the happy–sad scale). The ability for loving-kindness meditations to increase positive affect is consistent with previous research (Fredrickson et al., 2008). It may be that brief loving-kindness meditations are limited in their ability to influence physiological responding, but can increase positive emotion. Similar to the mindful breathing group, it should be recognised that participants carrying out the loving-kindness meditation had no prior practice of the task, which may have dampened its effectiveness.

5.5.2. Buffering Against Impending Stress

In addition to evaluating the down-regulatory effects of the interventions, analyses were also conducted to investigate whether the regulatory strategies provided any protection against future stress. Comparisons were made across the groups’ reactivity to the speech presentation, which was carried out after the intervention period. All of the groups demonstrated equivalent changes in HR, SCL, and HF-HRV suggesting that the emotion regulation strategies did not buffer against impending stress. If anything, the strategies were associated with poorer performance ratings for the speech presentation: Participants in the intervention groups felt like they had less time
to prepare their speech and were consequently less satisfied with their performance than
the control group which rested quietly.

5.5.3. Underlying Mechanisms

The findings of the current experiment suggest that the doing something may be
better than doing nothing during times of acute stress, although this may impact on
future performance. The mechanisms underlying the observed physiological effects are
unclear. Although the strategies were hypothesised to activate the PNS and increase
positive affect, limited effects were observed across the strategies. A proposed
mechanism for the observed changes in physiology may be that engaging in emotion
regulation serves as a distraction from the stressor. In the short term distraction can be a
useful way of regulating emotions, however over time distraction simply serves as an
avoidance technique (Nolen-Hoeksema et al., 2008). To partial out distraction effects
from other regulatory mechanisms, future research needs to investigate the longer term
effects of these emotion regulation strategies.

It is notable that the largest effects were observed in the neutral listening group,
which demonstrated a significant reduction in HR at the end of the intervention period,
and the mindful breathing group, which exhibited the largest recovery in SCL. It is
possible that these effects were driven by listening to the human voice. Porges and
colleagues (2001, 2003a; Porges & Lewis, 2010) have argued that the human voice
itself can be a soothing stimulus that can promote feelings of safety. If this is the case,
then the audio recordings in these tasks may have elicited reductions in arousal by
engaging the PNS via neural activation of the middle ear muscles. Although this is a
possibility, it does not explain why the loving-kindness group did not demonstrate
similar reductions in arousal.

One prominent difference between the mindful breathing intervention and the
other interventions that may underlie the differences in SCL recovery is the focus of the
mindful breathing task on the breath. Although the task did not instruct participants to
actively manipulate their breathing, participants often alter their breathing patterns when
focusing their attention on the breath (Grossman, 2010). Breathing has been highlighted
as one of the most direct ways to influence the autonomic nervous system, and
breathing practices are therefore an appealing interventions for therapies which wish to target both the mind and body (Jerath, Edry, Barnes, & Jerath, 2006).

5.5.4. Limitations

The results suggest that the effects of the interventions were fairly limited. Dramatic divergences in HR recovery were notable for the first two minutes of the intervention period, but over time the control group demonstrated a similar pattern of responding, and all of the groups maintained increased SCL over the five minutes. Response-focused tasks that require ‘active coping’ have previously been shown to induce SNS activation (Gross, 1998b; Tomaka et al., 1993), which may explain why the chosen strategies only demonstrated limited effects on down-regulating SNS arousal. It may seem counterintuitive that tasks aimed at reducing SNS arousal should actually activate the SNS, but this may be a confound of investigating the effects of novel emotion regulation strategies in a laboratory environment. There are two main shortcomings associated with the current design. First of all, participants were observed practising the interventions for the first time. The novelty and uncertainty associated with the tasks is likely to have increased SNS arousal (Kelsey et al., 1999). These effects would most likely diminish had the participants been able to practice the techniques beforehand. A second consideration is that the current experiment used brief variants of well-established interventions, which may limit the ecological validity of the findings. The interventions employed in this experiment do not accurately represent the how these treatment interventions are used clinically, for example mindful breathing is often not taught as a stand-alone technique, it tends to be incorporated within mindfulness approaches and requires training and a large amount of practice. Indeed, as clients become more familiar with a technique they may habituate to the demands of the task, thus resulting in greater efficacy of the task.

Further methodological limitations should also be considered. Each of the intervention groups had a relatively small number of participants, which resulted in limited power to demonstrate consistently significant findings. In addition to this, a homogenous sample was used (undergraduate students), which will limit the generalisability of the findings to samples representing the wider population. It is assumed that the current sample is representative of the wider student population, although several participants scored fairly high on the HADS anxiety subscale ($M = \ldots$
6.77, \( SD = 3.74 \), cut off for mild anxiety >8; Zigmond & Snaith, 1983). Research has highlighted that the HADS anxiety subscale psychopathologises non-clinical and student populations, suggesting that another measure of depression may be more suitable in the subsequent experiments (Andrews, Hejdenberg, & Wilding, 2006; Crawford, Henry, Crombie, & Taylor, 2001). As a final critique, the emotion regulation strategies evaluated were not equated in their design (for example different sensory modalities were targeted across the different tasks). To identify the mechanisms driving the observed physiological and subjective changes, research needs to equate strategies and compare their effects over a larger window of time.

5.5.5. Conclusion

The current experiment focused on a narrow range of emotion regulation strategies. The regulatory strategies of interest were chosen because it was hypothesised that they may be able regulate arousal and emotions by activating the PNS and enhancing positive affect. The interventions that demonstrated responses most consistent with the hypothesis (i.e. reduced SNS activation and increased PNS activation) were the neutral listening task and the mindful breathing task, although as five-minute interventions their effects were limited. The most notable shortcoming of the interventions was that none of the tasks were associated with a return to baseline for the SCL data. The maintenance of elevated SCL was unexpected as all of the groups exhibited increases in HF-HRV during the intervention period, which suggests that the PNS was being up-regulated during this time. According to polyvagal theory up-regulation of the PNS should inhibit activation of the SNS (Porges, 1995, 2001, 2003a), however the findings are more in line with the doctrine of autonomic space, which emphasises the potential for the SNS and PNS to be co-activated (Berntson, Cacioppo, & Quigley, 1991).

In conclusion, the findings of the current study do not suggest that emotion regulation strategies are ineffective at influencing the autonomic nervous system. In fact, all of the strategies were found to exhibit significant effects on HR and SCL, even if they were only minor changes that were sustained for a short period of time. With increased practice and sufficient fine-tuning, all of the strategies may be able to produce significant reductions in arousal over and above the effects observed here. Future
research should continue to identify strategies that target function of the autonomic nervous system to enhance self-regulation during times of stress.
Facial expressions are just one channel through which emotions manifest themselves; other channels include emotional feelings, physiological changes, and changes in cognition (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). However facial expressions are unique in that they turn the private emotional experience into a public display of feeling (Darwin, 1872/2009; Ekman, 1993). It is believed that emotional processes result in the generation of emotional facial expressions because the human body has been biologically prewired to co-opt the muscles of the face when emotions are experienced (Dimberg, 1990). This hardwiring is evolutionarily adaptive; humans are designed to decode and respond to the facial displays of others because communicative signalling helps our ability to survive and reproduce (Buck, 1994; Lakin, Jefferis, Cheng, & Chartrand, 2003).

Emotions are conveyed through a mixture of facial expressions, behaviours, and gestures, and it has been conjectured that displays of emotion serve more than one function (Hess, 2001). First of all, emotional displays, particularly facial expressions, initiate and maintain social interactions (Darwin, 1872/2009; Ekman, 1993). They do this by conveying messages about internal states, behavioural intentions, and action requests (Fridlund, 1991; Lynch et al., 2006). In conjunction with this, the facial muscles involved in displaying emotions are thought to play an active role in feeding back to the brain the intra-individual experience of emotion (Izard, 1990). Consequently facial muscle activity simultaneously acts as a “read-out system” for emotional reactions, and a “feedback system” for the emotional experience (Buck, 1994; Dimberg, 1990). This dual role means that facial displays serve a self-regulatory function as well as a social-communicative function (Lanzetta, Cartwright-Smith, & Kleck, 1976), thus facial displays are relevant to two emotional competencies: emotion production (i.e., encoding ability) and emotion modulation (i.e., regulation ability) (see Riggio, 1986; Scherer, 2007).

6.1. Expressive Regulation

Polyvagal theory conceptualises facial expressions as emergent behaviours driven by activation of the autonomic nervous system (Porges, 1995). However it is well known that top-down cortical control can influence facial expressions; facial
expressions can be up-regulated (enhanced) or down-regulated (suppressed) via conscious control (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Matsumoto & Lee, 1993). The modulation of facial expressions can have personal and social consequences. Firstly, changing the outward expression of an emotion can function to change the subjective experience of the emotion. Enhancing facial expressions has been shown to increase concomitant emotional experiences (Demaree, Robinson, Everhart, & Schmeichel, 2004), whilst expressive suppression has been associated with limited effects on subjective feelings (Gross & Levenson, 1993). In addition to this, expressive regulation can be used to hide feelings. Sometimes our emotions can be considered inappropriate (i.e., they do not match the context; Bonanno et al., 2007), or at other times we can choose to control our facial expressions in order to deceive others (Ekman, 2003). Controlling emotional expressions can therefore function to facilitate interpersonal interactions (e.g., by preserving social norms, Ekman & Friesen, 1969), but they can also function to protect the self (e.g., by preserving self-esteem, Ekman, 1997).

Individuals differ in their ability to up-regulate or down-regulate their facial expressions (Gross & John, 1997). Research suggests that people have tendencies towards either expressing or suppressing emotion. Everyday emotional expression has been associated with enhanced well-being and health (Gohm & Clore, 2002; Harker & Keltner, 2001), whilst emotional suppression has been linked to more negative outcomes such as increased sympathetic nervous system (SNS) activation and poorer well-being (Gross & John, 2003; Gross & Levenson, 1993). Interestingly, reduced facial expressiveness is often reported to be a core feature of several clinical disorders, for example Major Depressive Disorder (Gehricke & Shapiro, 2000), Autistic Spectrum Disorders (Travis & Sigman, 1998), and Schizophrenia (Gaebel & Wölwer, 1992).

As well as affecting health and well-being, our tendency to express or inhibit our emotional expressions has been shown to influence our social relationships. Butler et al. (2003) found that individuals who were instructed to suppress their facial expressions in response to a negative film were rated by their naive counterparts as being less friendly, and their counterparts were less willing to spend time with them in the future. Suppressors themselves also notice difficulties in interpersonal functioning: students who habitually suppress their emotional expressions have been found to report lower
levels of social support, feeling less close to others, and lower social satisfaction (Srivastava, Tamir, McGonigal, John, & Gross, 2009).

6.1.1. Expressive Flexibility

The relationship between emotional expression and well-being is not as straightforward as it may initially appear. Even the expression of emotion can be detrimental if prolonged or considered inappropriate. For example, although sad expressions can stimulate sympathy and helping responses, eventually they can lead to withdrawal and rejection by others (Consedine, Magai, & Bonanno, 2002). On the other hand, the suppression of emotion has been shown to adaptive in certain situations. Whilst suppression is linked to poorer psychological health in Western cultures, Eastern cultures which value emotional control and restraint do not show this suppression–health relationship (Butler, Lee, & Gross, 2007; Soto, Perez, Kim, Lee, & Minnick, 2011; but see Roberts, Levenson, & Gross, 2008). Recently researchers have been giving more consideration to the role of adaptive responding and the ability to change one’s expressions to meet the demands of the environment. This line of enquiry has stemmed from the coping literature where it has been emphasised that adaptability does not depend on which strategy is used, but whether coping is applied in a flexible manner (Cheng, 2001).

The ability to flexibly down- and up-regulate facial expression has been explored by Bonanno and colleagues (Bonanno et al., 2004; Westphal, Seivert, & Bonanno, 2010). Their argument is that different contexts can call for either the enhancement or the suppression of emotion, and that the most adaptive response is one that fits with the environment (Bonanno et al., 2007). Consequently individuals who are most adaptive are able to modulate their facial expressions in both directions (i.e., they have expressive flexibility). Expressive flexibility is considered to be a trait marker of flexible responding, and the ability to flexibly modulate emotional facial expressions has been linked to greater resilience from adversity (Westphal et al., 2010).

6.1.2. Autonomic Function and Facial Expressivity

Polyvagal theory conjectures that the ability to control our facial expressions is intrinsically linked to our physiological state (Porges, 1995, 2001, 2003a). The source
nuclei of the cranial motor nerves controlling the face and head are anatomically linked to the cardiac vagal fibres projecting from the nucleus ambiguus in the brainstem. Polyvagal theory posits that the cranial motor nerves communicate directly with the inhibitory neural system that promotes calm and self-soothing physiological states (Porges, 2003a). When the inhibitory effect of the vagus nerve is withdrawn to promote defensive physiological responding, it is theorised that the accessibility of the cranial motor nerves becomes restricted. Consequently, shifts in physiological state should cause observable changes in behavioural measures of emotional expression.

Contrasting theories have arisen about the relationship between expressive behaviour and autonomic responding. The most notable theories are the theory of psychophysiological arousal and emotional discharge theory (for a review see Cacioppo et al., 1992). The theory of psychophysiological arousal suggests that the intensity of an emotional response is manifested in general physiological arousal that can be observed in both internal and external responses (i.e., behavioural expressivity is hypothesised to covary with ANS activation; Lanzetta et al., 1976; Zuckerman, Klorman, Larrance, & Spiegel, 1981). Emotional discharge theory on the other hand suggests that there is an inverse relationship between ANS function and behavioural expressivity (Notarius & Levenson, 1979; Notarius, Wemple, Ingraham, Burns, & Kollar, 1982). This relationship is commonly symbolised by internalisers (individuals who demonstrate high SNS activation and low facial expressivity) and externalisers (individuals who demonstrate low SNS activation and high facial expressivity). Cacioppo et al. (1992) reached a compromise and suggested that both theories are valid models of expressive behaviour and autonomic function: the theory of psychophysiological arousal tends to explain intra-individual differences in patterns of responding, whilst the emotional discharge theory tends to explain inter-individual differences in patterns of responding. It is conjectured that individual differences in emotional reactivity and response styles means that autonomic functioning is not transparently linked to facial expressivity.

6.1.3. The Influence of Arousal on Facial Expressivity

Bonanno and colleagues claim that expressive flexibility is a trait measure of behavioural responding (Bonanno et al., 2004; Westphal et al., 2010). However, despite individual participants demonstrating consistent scores in expressive ability over time,
the test-retest reliability data has been collected in similar testing conditions; whether these abilities generalise to other contexts is unknown. Contextual demands have been shown to be an important factor in determining expressive ability. For example, Westphal et al. (2010) themselves found that the relationship between expressive flexibility and adjustment is moderated by the presence of immediate threat (i.e., differences in expressive ability emerged across threat primes vs. neutral primes). This suggests that there is a state component to this ability.

A major factor that may influence facial expressivity is physiology. After all, one’s physiological state is inherently linked to the subjective and behavioural aspects of emotion (James, 1884; Lange, 1885; Mauss et al., 2005), and modulating one’s facial expressions has been shown to influence autonomic indices such as heart rate and skin conductance (Demaree, Schmeichel, Robinson, & Everhart, 2004; Gross & Levenson, 1993, 1997). Although it is logical to suggest that the ability to produce facial displays of emotion may be inherently linked to one’s physiological state, polyvagal theory’s assertion that facial expressivity is linked to calm and self-soothing states has received limited empirical support. Some limited support has come from research investigating vagal tone and emotional expressivity in infants (Stifter & Fox, 1990; Stifter, Fox, & Porges, 1989), but research with adults has failed to find positive links between PNS function and facial expressivity (Demaree, Robinson, et al., 2004). In fact some evidence suggests that PNS function is associated with reduced expression of negative affect (Demaree, Pu, Robinson, Schmeichel, & Everhart, 2006; Pu, Schmeichel, & Demaree, 2010). In contrast to this, SNS activation has been associated with both increased and decreased facial expressivity (Cacioppo et al., 1992). The lack of positive findings in support of polyvagal theory may be due to methodological reasons: facial expressivity is often measured during passive viewing tasks, and not during challenge or threat situations (for an exception see Notarius & Levenson, 1979). Under normal viewing conditions it may be difficult to reveal associations between facial expressivity and the function of the autonomic nervous system.

6.2. Current Study Aims and Hypotheses

The current study used a between subjects design to investigate whether defensive physiological arousal influences facial expressivity. To achieve this, the expressive regulation (ER) task was replicated from Bonanno et al. (2004). This task
provides behavioural indices of expressive enhancement ability (i.e., the ability to up-regulate facial expressions) and expressive suppression ability (i.e., the ability to down-regulate facial expressions). The experiment tested the following hypothesis:

**Hypothesis 3.** Increased activation of the SNS in response to a laboratory stressor will be associated with decreased facial expressivity.

In Bonanno et al. (2004) the ER task was presented as a single task, however for the current experiment the task was split into two parts: The first half of the task was presented after baseline to measure typical expressivity, and the second half was presented after a stressor manipulation to measure expressivity during increased SNS activation: half of the participants were asked to prepare a short speech – a task that is associated with increased heart rate, increased sweat response, and decreased high-frequency heart rate variability (for a more detailed overview see Chapter 4); the other half of the participants completed a reading task (as a non-stressful control condition). It was hypothesised that individuals in the speech group would show a reduction in facial expressivity during the second administration of the ER task compared with the first half; this would be due to a decrease in expressive enhancement ability coupled with an increase in expressive suppression ability.

### 6.3. Method

#### 6.3.1. Participants

Eighty-two students volunteered to participate in the study for course credits or a small payment (£10). Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders (including fear of public speaking), and current psychological or pharmacological treatment. One participant was excluded from taking part in the study due to a self-reported diagnosis of schizophrenia. After reviewing the video recordings six further participants were excluded from analysis due to incorrectly following the instructions on the ER Task on one or more trials. The final dataset is comprised of data from 75 participants (21 males, 54 females). They ranged in age from 18–45 with a mean age of 20.27 years ($SD = 3.96$). 96.0% of these participants identified themselves as Caucasian, 1.3% as Mixed, 1.3% as Black, and 1.3% as Other. The participants were randomised into two groups
during the experiment: a speech group \( n = 38 \): 12 males, 26 females; mean age = 20.50, \( SD = 5.07 \) and a reading group \( n = 37 \): 9 males, 28 females; mean age = 20.03, \( SD = 2.40 \).

6.3.2. Behavioural Measure of Facial Expressivity

6.3.2.1. Expressive Regulation Task. The Expressive Regulation (ER) Task was replicated from Bonanno et al. (2004). Participants were seated before a desktop computer with a webcam positioned above their line of vision. E-Prime (Version 1.1; Psychology Software Tools, Pittsburgh, PA) was used to display blocked sequences of five digitized picture stimuli selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999). Stimuli were balanced for valence and arousal across blocks using the IAPS norms (Lang et al., 1999). Within each block, each stimulus was presented for 10 seconds, with 4 seconds between stimuli. For practice, participants viewed randomly presented blocks of positive or negative stimuli, and then for each block rated the degree to which they felt "negative emotion" (e.g., anger, revulsion, sadness, distress), by clicking a number between 1 (no negative emotion) and 7 (extreme negative emotion) on a visual analogue scale, and then the degree to which they felt "positive emotion" (e.g., happiness, joy, amusement, interest), using a similar scale.

Following the practice trials, participants were told that there was another participant in the adjacent room who was also taking part in the experiment (another participant was not actually present). They were told that they would not see the other person, but the other person would sometimes be able to view them on a video monitor; they were also told that they would always be informed when the monitor was on and when it was off; and that the other person would not hear them or see the picture stimuli but would have to guess their emotions for each block of stimuli. Participants were told that when the experiment began, the computer would (a) sometimes ask them to enhance their expression of emotion so the observer could more easily guess what they were feeling, (b) sometimes ask them to suppress their expression of emotion so the observer could not easily guess what they were feeling, and (c) sometimes inform them that the monitor was turned off and that the observer would be unable to see them, in which case they should behave as they would normally. Participants were then shown the three instruction paragraphs describing each condition (from Bonanno et al., 2004),
and were informed that one of the instructions would always precede each block of stimuli, and that each block of stimuli would always be followed by the emotion ratings. The instructions for the enhance condition were as follows:

_Shortly, you will be presented with a set of images. Please view each image carefully. While viewing the images, please do your best to EXPRESS AS FULLY AS POSSIBLE THE EMOTIONS you feel while viewing the images. Remember that the person viewing you on the monitor can only see your head and neck, and cannot hear you. It is important for the sake of this study that you do your best to communicate what you are feeling. So please do the best you can to BEHAVE IN SUCH A WAY THAT THE PERSON VIEWING YOU ON A MONITOR WILL BE ABLE TO GUESS FROM YOUR FACIAL EXPRESSIONS what you are feeling while viewing the images. Before each image, focus your attention on the ‘X’ in the middle of the screen. After viewing each set of images, you will be asked to rate the emotional reactions you had to the images._

The instructions for the suppression condition were:

_Shortly, you will be presented with a set of images. Please view each image carefully. While viewing the images, please do your best to SUPPRESS AS FULLY AS POSSIBLE ANY EXPRESSION OF THE EMOTIONS you feel while viewing the images. Remember that the person viewing you on the monitor can only see your head and neck, and cannot hear you. It is important for the sake of this study that you do your best to conceal what you are feeling. So please do the best you can to BEHAVE IN SUCH A WAY THAT THE PERSON VIEWING YOU ON A MONITOR WILL NOT BE ABLE TO GUESS FROM YOUR FACIAL EXPRESSIONS what you are feeling while viewing the images. Before each image, focus your attention on the ‘X’ in the middle of the screen. After viewing each set of images, you will be asked to rate the emotional reactions you had to the images._

The instructions for the control condition were:

_Shortly, you will be presented with a set of images. Please view each image carefully. NO ONE WILL BE VIEWING YOU FOR THIS SET OF IMAGES. Simply view the images and behave as you would naturally do so. Before each image, focus_
your attention on the ‘X’ in the middle of the screen. After viewing each set of images, you will be asked to rate the emotional reactions you had to the images.

The ER task had twelve presentation blocks in total (six positive, six negative). The first ER task (ER T1) randomly presented half of the blocks (three positive and three negative blocks paired with enhancement, suppression, or control instructions), whilst the participant was videoed modulating their facial expression (see figure 6.1 for examples of participants complying with the regulation instructions). The first administration of the ER task provided a baseline measure of expressive ability for each participant. The second ER task (ER T2) was completed after the three-minute speech/reading task, and randomly presented the remaining blocks (three positive and three negative blocks paired with enhancement, suppression, or control instructions). The second administration allowed us to determine if there were any changes in expressive ability during expressive regulation after the stressor manipulation.

To calculate the level of facial expression shown in each block, the video recordings of the participants’ modulating their facial expressions were rated for emotional expressivity using Visual Recognition’s eMotion software, developed at the University of Amsterdam (Gevers, 2008). This software is able to categorise how fully six basic emotions (happiness, sadness, disgust, surprise, fear, and anger) are expressed in photographs and videos of facial activity. The videos in the current experiment were digitalised with a frame rate of 25 frames per second, resulting in 250 frames of eMotion output per 10-second video. Indices of positive and negative emotion were created by combining the percentages of the basic emotion scales: The positive emotion index consisted of the happy and surprise scales; the negative emotion index consisted of the sadness, disgust, fear, and anger scales. The positive and negative indices of emotional expressivity were used to calculate an expressive enhancement ability score, whilst the neutral scale was used to calculate an expressive suppression ability score. For each participant the mean percentage of emotion shown during each block of stimuli was calculated for the enhancement, suppression, and control blocks. The mean positive or negative emotion scores for each congruent block (i.e., positive or negative block) were then used to calculate expressive ability scores for ER T1 and ER T2. Expressive ability scores were derived following the methodology of Bonanno et al. (2004). Expressive enhancement ability was obtained by subtracting the mean expression of emotion in the control condition from the mean expression of emotion in
the enhancement condition. Expressive suppression ability was obtained by subtracting the mean expression of emotion in the suppression condition from the mean expression of emotion in the control condition. As in Westphal et al. (2010), enhancement and suppression ability were significantly inversely correlated ($r = -.42, p < .001$).

In Westphal et al. (2010) the expressive enhancement scores and the expressive suppression scores were used to create a measure of balanced expressive flexibility (EF). According to Westphal et al. (2010) balanced EF is a clear marker of expressive flexibility because high balanced EF scores represent extreme but opposite response tendencies, whilst extreme scores in one form of regulation (i.e., enhancement or suppression) result in lower balanced EF scores. Although this argument is convincing, balanced EF scores are misleading; different participants who demonstrate even response tendencies will receive similar balanced EF scores, but these scores do not differentiate between the individuals who show equal levels of good enhancement and suppression, compared to those individuals who show equal levels of poor enhancement and suppression. For this reason, the current research will index facial expressivity by calculating enhancement and suppression abilities (as in Bonanno et al., 2004), but will not use these measures to calculate expressive flexibility scores (as in Westphal et al., 2010).
Enhance Condition

Control Condition

Suppress Condition

Figure 6.1. Examples of the eMotion software analysis with participants regulating their facial expressions in accordance with the Expressive Regulation instructions.
6.3.3. Procedure

Participants attended a single testing session in an air-conditioned, sound attenuated room. On arrival, participants completed a written consent form and the demographic screening questionnaire (see section 3.5.1). Participants then completed questionnaires to assess their levels of psychological flexibility (Acceptance and Action Questionnaire II, AAQ-II; Bond et al., 2011, see section 3.5.8), usual levels of emotional expressivity (Berkeley Expressivity Questionnaire, BEQ; Gross & John, 1995, see section 3.5.6), and how safe they feel in their social relationships (Social Safeness and Pleasure Scale, SSPS; Gilbert et al., 2009, see section 3.5.9). Participants also completed measures to assess trait mood (Beck Depression Inventory, BDI-II; Beck, Steer, & Brown, 1996, see section 3.5.4; and the Generalised Anxiety Disorder Scale, GAD-7; Spitzer, Kroenke, Williams, & Löwe, 2006, see section 3.5.6). The questionnaires were included to control for individual differences in factors that might influence the expressivity scores. Electrodes for recording heart rate (HR) and skin conductance level (SCL) were then applied following standard procedures (see section 3.4.4) and a five-minute baseline recording was carried out during which the participants were asked to sit quietly. Recording HR allowed for the calculation of heart rate variability (HRV, see section 3.4.3). After these five minutes participants completed a questionnaire to assess their current emotional state (Profile of Mood States – Short Form, POMS-SF; Shacham, 1983, see section 3.5.1). Instructions were then given for the first half of the Expressive Regulation (ER T1) Task (Bonanno et al., 2004).

Once the first half of the ER task was completed, participants were randomly assigned to one of two conditions: a speech task vs. a reading task. The speech task was adapted from the procedure used by Schubert et al. (2009; see section 4.6.2). Participants were informed that they would have to prepare and present a three-minute speech. Participants in the speech task group \((n = 38)\) were given a three-minute preparation period during which they were given a list of bullet points with arguments for and against euthanasia, and a pen and paper to help prepare their speech. The reading condition was employed as a non-stressful alternative to the speech task (Feldman, Cohen, Hamrick, & Lepore, 2004). Participants assigned the reading task \((n = 37)\) were given three minutes to quietly read through written passages containing arguments for and against euthanasia (these corresponded with the arguments outlined
in the material given to participants in the speech task condition). After the three-minute task, both groups recomplied the questionnaire to assess their mood state (POMS-SF) and then carried out the second half of the ER Task (ER T2). A flowchart diagram of the expressive regulation procedure can be found in appendix 16.

6.4. Results

6.4.1. Statistical Analyses

For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests.

To evaluate the influence of possible covariates in the planned analyses comparing the stressor manipulation groups, bivariate correlations were computed among the dependent variables (expressive enhancement ability, expressive suppression ability, and self-reported emotion ratings) and possible covariates (age, sex, trait mood [BDI-II and GAD-7], self-reported expressivity [BEQ], social safeness [SSPS], and psychological flexibility [AAQ-II]). No significant correlations were identified for expressive enhancement ability. Expressive suppression ability was significantly correlated with the AAQ-II \( r = -.23, p = .050 \). Higher self-reported psychological flexibility was associated with decreased expressive suppression. Subsequent analyses examining group differences in expressive suppression ability accounted for the influence of the AAQ-II by including the measure as a covariate. Further correlations revealed that self-reported emotion ratings were significantly correlated with sex \( r = .34, p = .003 \), the BEQ \( r = .44, p < .001 \), and the SSPS \( r = .30, p = .008 \): Females tended to report higher levels of felt emotion than males, individuals reporting greater behavioural tendencies to express emotion also reported higher levels of felt emotion, and individuals reporting greater social safeness expressed higher levels of felt emotion. Sex, the BEQ total scores, and the SSPS total scores were entered into the subsequent analyses of the self-report ratings as covariates.
Table 6.1.
Mean recordings of physiological arousal during the experimental tasks by stressor group.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>ER T1</th>
<th>Manipulation</th>
<th>ER T2</th>
<th>$F$</th>
<th>$df$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reading Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>74.45 (11.07)</td>
<td>74.06 (10.79)</td>
<td>72.42 (9.73)*</td>
<td>72.19 (9.48)**</td>
<td>4.63</td>
<td>2.74, 98.79</td>
<td>.006</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.13 (1.42)</td>
<td>3.98 (1.91)***</td>
<td>4.15 (2.17)***</td>
<td>4.07 (2.26)***</td>
<td>61.95</td>
<td>1.45, 52.15</td>
<td>.001</td>
</tr>
<tr>
<td>HF-HRV (ms$^2$)</td>
<td>7.64 (0.82)</td>
<td>7.62 (0.68)</td>
<td>7.64 (0.85)</td>
<td>7.62 (0.78)</td>
<td>0.06</td>
<td>2.62, 94.14</td>
<td>.974</td>
</tr>
<tr>
<td><strong>Speech Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>78.55 (9.90)</td>
<td>76.92 (8.43)*</td>
<td>83.34 (12.75)**</td>
<td>75.58 (9.14)***</td>
<td>22.42</td>
<td>1.69, 62.26</td>
<td>.001</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.93 (2.03)</td>
<td>5.10 (2.75)***</td>
<td>5.75 (2.74)***</td>
<td>4.99 (2.48)***</td>
<td>101.74</td>
<td>2.10, 77.78</td>
<td>.001</td>
</tr>
<tr>
<td>HF-HRV (ms$^2$)</td>
<td>7.28 (1.04)</td>
<td>7.44 (0.88)</td>
<td>7.11 (0.97)</td>
<td>7.41 (0.91)</td>
<td>2.97</td>
<td>2.59, 95.90</td>
<td>.043</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant change from baseline * $p < .05$, ** $p < .01$, *** $p < .001$

Table 6.2.
Stressor group characteristics

<table>
<thead>
<tr>
<th></th>
<th>Reading Group ($n = 37$)</th>
<th>Speech Group ($n = 38$)</th>
<th>$t$</th>
<th>$df$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$ (SD)</td>
<td>$M$ (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AAQ-II</td>
<td>39.38 (7.47)</td>
<td>37.50 (6.86)</td>
<td>1.14</td>
<td>73</td>
<td>.260</td>
</tr>
<tr>
<td>BDI-II</td>
<td>5.59 (6.27)</td>
<td>8.29 (9.05)</td>
<td>-1.50</td>
<td>73</td>
<td>.139</td>
</tr>
<tr>
<td>BEQ Mean</td>
<td>4.53 (0.87)</td>
<td>4.48 (0.81)</td>
<td>0.22</td>
<td>73</td>
<td>.830</td>
</tr>
<tr>
<td>GAD-7</td>
<td>4.68 (4.79)</td>
<td>4.92 (4.03)</td>
<td>-0.24</td>
<td>73</td>
<td>.811</td>
</tr>
<tr>
<td>POMS-SF Total</td>
<td>2.62 (9.52)</td>
<td>3.89 (10.17)</td>
<td>-0.56</td>
<td>73</td>
<td>.578</td>
</tr>
<tr>
<td>SSPS</td>
<td>34.51 (8.41)</td>
<td>31.89 (8.67)</td>
<td>1.33</td>
<td>73</td>
<td>.189</td>
</tr>
</tbody>
</table>

*Note.* AAQ-II = Acceptance and Action Questionnaire, BDI-II = Beck Depression Inventory, BEQ = Berkeley Expressivity Questionnaire, GAD-7 = Generalised Anxiety Disorder Scale, POMS-SF = Profile of Mood States – Short Form, SSPS = Social Safeness and Pleasure Scale.
Mean HR, SCL, and HF-HRV values were calculated for the baseline period, each block of the ER task, and the stressor manipulation period (see table 6.1). Analyses were then performed to establish if there were any baseline differences between the stressor manipulation groups (the group characteristics can be found in table 6.2). Chi-squared tests confirmed that there were no significant differences between the stressor groups in terms of sex and ethnicity. Univariate analyses of variance (ANOVAs) also failed to identify any significant differences between the groups in terms of age, self-reported psychological flexibility, social safeness, emotional expressivity, or mood at baseline. Further to this, a univariate ANOVA did not find any significant group differences for HR, SCL, or HF-HRV at baseline or during the ER task at T1.

The first main set of analyses reported in this chapter investigated the effects of the ER task on subjective ratings of felt emotion, as well as facial expressivity. To establish whether the subjective ratings were significantly affected by valence and condition, the subjective ratings at T1 were compared across the individual ER blocks using paired t-tests and a univariate repeated-measures analysis of covariance (ANCOVA: repeated factor Condition; Sex, BEQ and SSPS scores as covariates). Further to this, a repeated-measures ANOVA was carried out to evaluate the effect of condition on the eMotion data (i.e., Level of Expression). Significant main effects for all analyses were followed up with pairwise comparisons. All pairwise contrasts were evaluated using Bonferroni critical values of .05.

The second set of analyses evaluated the effects of the ER task on the physiological indices. Repeated-measures ANOVAs were carried out on the HR, SCL, and HF-HRV data from T1, with Condition (enhance, control, suppress) and Valence (positive, negative) as repeated factors. Significant main effects for all analyses were followed up with pairwise comparisons. All pairwise contrasts were evaluated using Bonferroni critical values of .05.

The third main set of analyses investigated the effects of the stressor manipulations on the physiological indices of arousal. Reactivity scores were calculated for HR, SCL, and HF-HRV by subtracting the stressor manipulation period from the corresponding baseline data (Kamarck et al., 1992; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). To establish if the stressor manipulations induced different levels of arousal across the groups, univariate ANOVAs were performed on the reactivity
scores with Group as the fixed factor. Mixed-factorial ANOVAs (with Time as the repeated factor, and Group as the between subjects factor) were also performed using the POMS-SF means and the raw physiological data to investigate if the groups demonstrated different physiological profiles across the two ER tasks.

Finally, the fourth set of analyses investigated whether there were any group differences in expressive enhancement ability and expressive suppression ability across the two administrations of the ER task. Mixed-factorial ANOVAs (with Time as the repeated factor, and Group as the between subjects factor) were performed on the separate expressive ability scores. As expressive suppression ability was significantly correlated with the AAQ-II, the suppression ability analysis was repeated with the AAQ-II as a covariate.

6.4.2. Manipulation Check

Analyses of the self-rated subjective emotion at T1 were consistent with the valence of the stimuli (see table 6.3). Participants rated the positive stimuli as significantly more positive than negative across all conditions: enhancement, \( t(74) = 23.27, p < .001 \); suppression, \( t(74) = 23.14, p < .001 \); and control, \( t(74) = 23.56, p < .001 \). Participants also rated negatively valenced stimuli as significantly more negative than positive across all conditions: enhancement, \( t(74) = 23.06, p < .001 \); suppression, \( t(74) = 30.64, p < .001 \); and control, \( t(74) = 20.83, p < .001 \). In line with Bonanno et al.’s (2004) findings, subjective emotion ratings not matching the valence of the stimuli were low across conditions (positive ratings for negative stimuli \( M = 1.30, SD = 0.61 \); negative ratings for positive stimuli \( M = 1.35, SD = 0.74 \)). As incongruent ratings from the ER task have not been linked to any meaningful effects, they were excluded from the subsequent analyses.

To investigate the effects of Condition on the subjective experience of emotion the congruent self-report emotion ratings (i.e., mean positive ratings for positive stimuli and mean negative ratings for negative stimuli) were analysed using repeated-measures ANCOVAs (repeated measure: Condition; with Sex, the BEQ scores and the SSPS scores entered as covariates). Condition had a significant effect on negative ratings, \( F(2, 142) = 3.59, p = .030 \), but not positive ratings. Although self-reported negative emotion was higher in the enhance and suppress conditions than the control condition,
Bonferroni pairwise comparisons revealed that negative ratings were not significantly different across the conditions.

Table 6.3.
Mean self-rated subjective emotion during the ER Task at T1 by Condition and Valence.

<table>
<thead>
<tr>
<th></th>
<th>Enhance</th>
<th>Control</th>
<th>Suppress</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive Stimuli</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive rating</td>
<td>5.29 (1.10)</td>
<td>5.09 (1.09)</td>
<td>4.93 (1.26)</td>
<td>3.72</td>
<td>2, 148</td>
<td>.027</td>
</tr>
<tr>
<td>Negative rating</td>
<td>1.29 (0.65)</td>
<td>1.47 (0.98)</td>
<td>1.29 (0.51)</td>
<td>1.71</td>
<td>2, 148</td>
<td>.187</td>
</tr>
<tr>
<td>Negative Stimuli</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive rating</td>
<td>1.32 (0.66)</td>
<td>1.37 (0.67)</td>
<td>1.20 (0.46)</td>
<td>3.53</td>
<td>2, 148</td>
<td>.032</td>
</tr>
<tr>
<td>Negative rating</td>
<td>5.56 (1.21)</td>
<td>5.47 (1.45)</td>
<td>5.59 (1.09)</td>
<td>0.39</td>
<td>2, 148</td>
<td>.676</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses.

Analyses of the eMotion output at T1 supported the manipulation of expressive regulation. Level of Expression was determined for each condition by calculating the mean percentage of congruent emotion displayed in each block (i.e., percentage of positive emotions expressed during positive blocks, and percentage of negative emotions expressed during negative blocks). A repeated-measures ANOVA revealed a significant effect of Condition (enhancement, suppression, control) on Level of Expression, $F(2, 146) = 62.18, p < .001$ (see figure 6.2). As expected, Bonferroni pairwise comparisons indicated that Level of Expression was significantly greater in the enhancement condition ($M = .48, SD = .17$) than the control condition ($M = .34, SD = .14$), and significantly lower in the suppression condition ($M = .24, SD = .15$) than the control condition (all significant at $p < .001$).
Figure 6.2. Mean percentage of congruent emotion exhibited (as rated by eMotion) across the three expressive-regulation conditions. Higher percentages represent greater level of expression. Error bars represent the standard error.

6.4.3. Physiological Effects of Expressive Regulation

Previously the ER Task has not been carried out whilst simultaneously measuring changes in psychophysiology. The physiological data are summarised in table 6.4. The physiological data recorded at T1 were analysed to elucidate the effects of expressive regulation on physiological arousal. Repeated-measures ANOVAs for Condition (enhance, control, suppress) and Valence (positive, negative) were carried out on the HR, SCL, and HF-HRV data. For HR a significant main effect was found for Condition, $F(2, 148) = 29.80, p < .001$. Bonferroni pairwise comparisons (all significant at $p = .002$) confirmed that HR was significantly higher in the enhancement condition ($M = 76.38, SD = 10.45$) than the control condition ($M = 73.81, SD = 10.83$), and significantly lower in the suppression condition ($M = 72.02, SD = 10.29$) than the control condition. A significant main effect of Condition was also found for SCL, $F(2, 148) = 38.74, p < .001$. Bonferroni pairwise comparisons (all significant $p < .05$) demonstrated that SCL was highest in the enhancement condition ($M = 4.75, SD = 2.49$), followed by the suppression condition ($M = 4.51, SD = 2.54$), with SCL lowest in the control condition ($M = 4.40, SD = 2.37$). Finally, a significant main effect of Condition was also found for HF-HRV, $F(2, 148) = 4.73, p = .010$. Bonferroni pairwise comparisons ($p = .002$) demonstrated that HF-HRV was significantly higher in the suppression condition ($M = 7.64, SD = .83$) compared to the control condition ($M = 7.45, SD = .85$).
Table 6.4.
Mean recordings of physiological arousal during the ER Task at T1

<table>
<thead>
<tr>
<th></th>
<th>Enhancement</th>
<th>Control</th>
<th>Suppression</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>76.38 (10.45)**</td>
<td>73.81 (10.83)</td>
<td>72.02 (10.29)**</td>
<td>29.80</td>
<td>2, 148</td>
<td>.001</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>4.75 (2.49)**</td>
<td>4.40 (2.37)</td>
<td>4.51 (2.54)*</td>
<td>38.74</td>
<td>2, 148</td>
<td>.001</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.58 (0.86)</td>
<td>7.45 (0.85)</td>
<td>7.64 (0.83)**</td>
<td>4.73</td>
<td>2, 148</td>
<td>.010</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from the control condition * = \( p < .05 \). ** = \( p < .01 \). *** = \( p < .001 \).

### 6.4.4. Physiological Reactivity to the Stressor Manipulations

Univariate ANOVAs were carried out on the reactivity scores for HR, SCL, and HF-HRV. A significant effect of Group was revealed for the HR reactivity scores, \( F(1, 73) = 17.21, p < .001 \), and the SCL reactivity scores, \( F(1, 73) = 6.32, p = .014 \), but not the HF-HRV reactivity scores (\( p > .05 \)). Bonferroni pairwise comparisons confirmed that the speech group demonstrated significantly higher HR and SCL reactivity than the reading group (significant at \( p < .05 \)), although it should be noted that both groups demonstrated an increase in SCL that was significantly above baseline.

To investigate the effect of the stressor manipulations on arousal during the ER tasks a series of mixed-factorial ANOVAs (with Time as the repeated factor, and Group as the between subjects factor) were carried out on the POMS-SF subscales and the physiological variables. As shown in figure 6.3, the speech group demonstrated a significant Group x Time interaction showing an increase in the POMS-SF tension–anxiety subscale from T1 to T2, which was not shown by the reading group, \( F(1,73) = 38.19, p < .001 \). Although the speech group showed greater increases in HR and SCL during the manipulation task, these changes were not maintained during the second ER task at T2. As a result there were no significant physiological differences between the groups at T2.
6.4.4.1. Relationships between mood and physiological reactivity. To investigate whether changes in ANS function during the stressor manipulation period were related to changes in self-reported emotion, correlations were carried out between the mean HR, SCL, and HF-HRV reactivity scores and the mean POMS-SF reactivity scores. Significant correlations revealed that greater HR reactivity was associated with larger increases in the POMS-SF tension–anxiety subscale \( r = .30, \ p = .006 \) and the POMS-SF confusion–bewilderment subscale \( r = .25, \ p = .026 \), whilst greater SCL reactivity was associated with larger increases in the POMS-SF tension–anxiety subscale \( r = .22, \ p = .046 \). The directions of these relationships suggest that larger increases in SNS activation were associated with greater self-reported negative affect.

6.4.5. Effect of the Stressor Manipulation on Facial Expressivity

It was hypothesised that the stressor manipulation would cause changes in arousal in the speech group that would influence expressive ability at T2. Mixed-factorial ANOVAs (with Time and as the repeated factor, and Group as the between subjects factor) revealed that there were no significant differences in expressive enhancement ability or expressive suppression ability between the groups at T1 or T2 (see table 6.5). The expressive suppression ability analysis was repeated with the AAQ-II as a covariate. The AAQ-II was significantly associated with the expressive suppression ability scores, \( F(1, 72) = 4.22, \ p = .044 \), but the main effects of Time and Group remained insignificant. Although the speech group expressed less emotion during
T2, as demonstrated by their reduced expressive enhancement ability and their increased expressive suppression ability, the changes were small and did not reach statistical significance.

Table 6.5.
Mean expressive ability scores at T1 and T2 by stressor group.

<table>
<thead>
<tr>
<th></th>
<th>Enhancement Ability</th>
<th>Suppression Ability</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
<td>T1</td>
<td>T2</td>
<td></td>
</tr>
<tr>
<td>Reading Group</td>
<td>0.12 (0.17)</td>
<td>0.14 (0.20)</td>
<td>0.09 (0.03)</td>
<td>0.11 (0.19)</td>
<td>0.00</td>
</tr>
<tr>
<td>Speech Group</td>
<td>0.15 (0.20)</td>
<td>0.12 (0.18)</td>
<td>0.12 (0.18)</td>
<td>0.16 (0.20)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses.

6.5. Discussion

The present study aimed to investigate the effects of defensive physiological arousal on facial expressivity. It has previously been suggested that individual differences in facial expressivity are trait-like, due to high correlations between expressive enhancement and expressive suppression ability scores appearing over time (Bonanno et al., 2004). However, since immediate threat contexts have been shown to influence expressive flexibility factors (Westphal et al., 2010), the current research decided to further explore the possibility that facial expressivity is also determined by state factors such as physiology and state anxiety.

The ER task developed by Bonanno et al. (2004) provides a behavioural measure of expressive regulation ability. In the present study participants completed half of the ER task followed by a stressor manipulation – half of the sample completed a reading task, and the other half completed a speech task – followed by a second version of the ER task. The aim of the stressor manipulation was to induce a defensive physiological state in the speech group in order to establish the effects of defensive arousal on expressive ability scores. No significant differences were found between the groups at T1 or T2 for any of the expressivity scores. This is despite the speech group showing a decrease in enhancement ability and an increase in suppression ability at T2.

Despite the stressor manipulation being successful at significantly increasing arousal in the speech group during the manipulation period, the physiology of the speech group was not significantly different from the reading group during the second
ER task. As discussed in Chapter 4, previous research has used the speech task to induce social evaluative threat during experimental tasks (e.g., Garner, Mogg, & Bradley, 2006; Mansell, Clark, Ehlers, & Chen, 1999), however these studies have not reported how well arousal is maintained during the secondary tasks. The findings of this study suggest that after stressor manipulations healthy individuals are likely to exhibit a physiological return to baseline, minimising the differences seen between groups assigned to different stressor manipulations.

Although the current experiment did not find significant differences in enhancement or suppression ability between the stressor groups at T2, there is still the possibility that defensive arousal affects emotional expressivity. Porges’ (1995, 2001, 2003a) polyvagal theory claims that facial expressions are emergent properties of calm and self-soothing states, and that defensive physiological states should limit the accessibility of neural pathways that automatically control facial expressions. What this premise fails to appreciate is that a reduction in spontaneous expression caused by defensive arousal does not prevent the regulation of expressions via consciously-mediated pathways (e.g., participants can voluntarily produce facial displays to influence social interactions). Thus participants in the speech group may have been compensating for a reduction in natural expressivity by exerting more conscious effort to regulate their facial expressions during the second ER task. This may explain why the changes in expressivity scores at T2 for the speech group went in the expected direction, but were not large enough to reach significance.

Evidence to support the distinction between spontaneous facial expressions and consciously driven facial expressions comes from two sources. First of all, research has shown that posed (voluntary) facial expressions involve different neuronal pathways from spontaneous (involuntary) facial expressions (Matsumoto & Lee, 1993; Rinn, 1984). Secondly, in the current experiment the Behavioural Expressivity Questionnaire (a measure that has been shown to index behavioural tendencies to express emotion; Gross & John, 1995) only correlated with the subjective self-report ratings of emotion and not the expressive enhancement or suppression scores. This finding supports the argument that instructionally manipulated displays of emotion may not be equated with natural expressions of emotion (Notarius & Levenson, 1979).
6.5.1. Limitations

A major limitation of this study was the inability to distinguish the stressor groups on the basis of physiology. It is possible that the ER task could have limited the effects of the stressor task. Participants in the speech group may have found the speech task less threatening than participants in the previous experiments because they were already being filmed during the ER blocks. Secondly, the physiological demands associated with the ER task may limit the emergence of group differences in arousal after the stressor manipulation: Both manipulation groups demonstrated increases in SCL from baseline at T1 and T2. Previous research has shown that engaging in expressive regulation increases arousal so this was not unexpected (Demaree, Schmeichel, et al., 2004; Gross & Levenson, 1993, 1997), however it was expected that the group differences in arousal after the stressor manipulation would be larger. A third consideration is that the speech task is simply not threatening enough to maintain heightened arousal during secondary tasks. Maintaining arousal during secondary tasks is a major methodological challenge for the current research. As a final consideration, the current sample was homogenous in terms of age, sex, and ethnicity, which means that the results may not be generalisable to the wider population. For example, previous research has demonstrated that females tend to be more expressive than males (Dimberg & Lundquist, 1990; Hess & Bourgeois, 2010), which was not evident in the current experiment. Using samples that are more diverse in age, sex, and ethnicity may reveal latent relationships between these factors and facial expressivity.

6.5.2. Conclusion

In conclusion, the current experiment failed to find a significant effect of defensive arousal on facial expressivity. Despite this, several interesting findings were observed. First of all, whilst the stressor manipulation was able to induce short-term increases in physiological arousal, the differences between the groups were not maintained during the secondary task. This finding could be attributable to various factors (e.g., distraction or habituation in the speech group, or excessive arousal in the reading group). Researchers need to be mindful of these effects when using stressor manipulations in conjunction with secondary tasks. The second finding that is worth noting, is that consistent with previous research, expressive regulation results in measurable changes in physiology: enhancement resulted in increased HR and SCL,
whilst suppression resulted in decreased HR coupled with increased SCL (Demaree, Schmeichel, et al., 2004; Gross & Levenson, 1993). These physiological concomitants support the premise that facial expressivity is linked to autonomic function.

Despite associations being identified between facial expressivity and autonomic function, the hypothesis that increased SNS activation would be associated with significant reductions in facial expressivity was not supported. Although the speech group exhibited changes in expressive ability at T2 that were in the direction of the hypothesis, the changes in expressivity scores were not significant. Future research should consider the possibility that defensive physiological arousal affects spontaneous facial expressivity, but not conscious regulation of facial displays. One way to test this hypothesis would be to covertly measure spontaneous facial expressions of emotion before and after a stressor manipulation.
Chapter 7: Defensive Physiological Arousal and Emotional Sensitivity

The previous studies have demonstrated that social evaluative threat is associated with reliable increases in defensive physiological arousal. Chapter 6 attempted to investigate the effects of defensive arousal on facial expressivity, but despite the stressor manipulation resulting in higher arousal levels in the speech group, the group differences in arousal were not maintained during the secondary task. The lack of findings in Chapter 6 may be attributable to the expressive regulation task being too arousing; the secondary task itself resulted in increases in arousal, hence both the stressor group and the control group demonstrated increases in arousal during the secondary task, masking the effects of the stressor manipulation. Using a secondary task that is less arousing could initiate greater group differences in physiology after the stressor manipulation. Keeping arousal levels higher in the speech group may enhance the detection of stress-related changes in socio-emotional responding during the secondary task.

The current chapter is designed to investigate the effect of defensive physiological states on the ability to recognise emotions in others. Research has suggested that when people are stressed and anxious their ability to recognise non-verbal expressions of emotion is impaired (Hänggi, 2004). Recognising emotions in others is an important component of social interactions, helping us to understand the emotional states of others as well as the intentions of our interaction partners (Blairy, Herrera, & Hess, 1999; Riggio, 1986; Scherer, 1995). An inability to decode emotional cues can lead to interpersonal difficulties such as empathic insensitivity and inappropriate expression of emotion.

7.1. Emotional Sensitivity

Successful communication involves two complex processes: Not only do we need to be able to encode the displays which convey our emotional states and our interpersonal intent, but we also need to be able to successfully decode the signals of our interaction partners (Blairy et al., 1999; Riggio, 1986; Scherer, 2007). The ability to recognise emotions is thought to be innate; an ability that co-evolved with the capacity to modulate our facial expressions to communicate internal states and behavioural intentions (Ekman, 1997; Izard, 1994). The processes involved in recognising and
discerning emotional expressions are thought to be automatic (Tracy & Robins, 2008); we can even perceive emotional expressions without awareness (Dimberg, Thunberg, & Elmehed, 2000). Despite emotion recognition being a universal ability, individuals differ in their ability to perceive emotions in others (Martin, Berry, Dobranski, Horne, & Dodgson, 1996; Rozin, Taylor, Ross, Bennett, & Hejmadi, 2003).

*Emotional sensitivity* refers to the threshold at which an individual can recognise emotional stimuli (Lynch et al., 2006; Riggio, 1986). Emotional sensitivity is a distinct measure from *emotional accuracy*, which relates to the ability to distinguish one emotion from another. Emotional sensitivity is about the level of cues needed for the emotion to be perceived. The most common methods used to test people’s ability to recognise emotions are with standardised batteries of emotive faces or vocal stimuli. The emotive stimuli can be presented statically, or techniques such as morphing the emotions (to form graded presentations, or blends of emotion) can be used to present the emotions dynamically (see section 3.6.2 for an overview).

Factors that can moderate the ability to accurately identify emotional expressions include age (Calder et al., 2003), gender (Hoffmann, Kessler, Eppel, Rukavina, & Traue, 2010; Rotter & Rotter, 1988), culture (see Elfenbein & Ambady, 2002, for a review), and psychopathology (Csukly, Czobor, Simon, & Takács, 2008; Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998). These factors can have differential effects on different emotions, and this has resulted in the suggestion that emotional sensitivity may not be a singular capability, but may be better conceptualised as a group of related abilities with individuals showing varied sensitivities to different emotions (Sprengelmeyer et al., 1998).

### 7.1.1. Facial Mimicry

Another factor that has been associated with emotional sensitivity is facial reactivity to stimuli (Stel & van Knippenberg, 2008). Individuals who facially express more emotion are more sensitive to self-produced cues of emotion as well as the emotions of others (Halberstadt, Dennis, & Hess, 2010; Laird et al., 1994); this has led to the belief that facial expressions may play a role in emotion recognition processes. It is not uncommon for people to respond to emotional stimuli with facial expressions, in particular facial displays of emotion are likely to induce reciprocal facial displays in the
observer. Dimberg (1982) found that when people were shown pictures of positive or negative facial displays, they produced distinct facial electromyographic reactions in muscles relevant to the valence of the display. Facial displays in response to others’ facial expressions are hypothesised to facilitate social interactions and exchanges, either by being similar, i.e., mimicking others’ expressions (Hatfield, Cacioppo, & Rapson, 1993; Lakin, Jefferis, Cheng, & Chartrand, 2003), or by being complementary (Keltner & Kring, 1998). Using emotional expressions can help to show emotional reciprocity and increase empathic understanding and rapport (Butler et al., 2003; Chartrand & Bargh, 1999; Gueguen, Jacob, & Martin, 2009; Lakin & Chartrand, 2003). Facial expressivity in response to others’ facial displays is therefore an important social signal (Adolphs, 2006).

In the same way that self-generated facial expressions can initiate afferent feedback, facial expressions generated through mimicry can also induce changes in affective state (Hess, Philippot, & Blairy, 1998). This process is known as emotional contagion (Hatfield et al., 1993). The reverse simulation model suggests that people recognize emotions by mimicking observed facial expressions, which in turn generates the corresponding emotion in the observer (Rives Bogart & Matsumoto, 2009). The mechanism for emotional contagion in this model is thought to be the same mechanism by which self-generated facial expressions can produce emotions (c.f. the facial feedback hypothesis; Buck, 1980; Dimberg, 1990). An alternative explanation is that simply observing others’ facial expressions may be enough to generate an emotional experience in the observer (Chartrand & Bargh, 1999; Hatfield et al., 1993). The production of corresponding facial expressions is something that is often unconscious and automatic (Dimberg, 1982; Dimberg et al., 2000). The unconscious mimicry of facial expressions, gestures, and mannerisms has been labelled the “chameleon effect” (Chartrand & Bargh, 1999).

**7.1.2. Linking Emotional Sensitivity and Facial Mimicry**

The links between emotional sensitivity and facial mimicry are not well-established. Action observation and action performance share the same neural substrates (Decety & Grèzes, 1999), which suggests that facial mimicry should improve perception of emotions, however research has demonstrated that facial mimicry and feedback is not necessary for emotion recognition. Rives Bogart and Matsumoto (2009)
found that people with Moebius Syndrome (i.e., bilateral facial paralysis) were no less accurate in recognising emotions than healthy control subjects. They concluded that peripheral muscle activity is not required for emotion recognition; however neural substrates in the motor cortex which would normally initiate the facial movement may provide sufficient feedback to induce the emotional experience. In line with this, Blairy et al. (1999) found no evidence to support the hypothesis that mimicry (either spontaneous or voluntary) increased emotion recognition accuracy. An interesting finding of their study however, was that voluntarily mimicking facial expression did result in lower perceived decoding difficulty.

Contrary to the findings of Rives Bogart and Matsumoto (2009), Oberman, Winkielman, and Ramachandran (2007) found that blocking facial mimicry (through behavioural manipulations) did affect recognition of specific facial expressions, suggesting that facial feedback does play a role emotional sensitivity. Complementary findings have been found in studies investigating the effects of botulinum toxin (commonly known as Botox), which suggests that blocking facial mimicry attenuates the activation of neural circuits involved in emotion (Hennenlotter et al., 2009). Schneider, Hempel, and Lynch (2012) also found that instructing participants to suppress their facial expressions when carrying out an emotion recognition task was associated with reduced emotional sensitivity, whilst instructing participants to actively mimic facial expressions had a facilitative effect. In addition to this, Stel and colleagues (Stel & van Knippenberg, 2008; Stel & van den Bos, 2010) have also demonstrated that facial mimicry can facilitate understanding the emotions of others. Stel and van Knippenberg (2008) found that mimicry was able to influence emotional sensitivity (particularly in females), but it had little effect on emotional accuracy.

7.2. Experiment 1: The Effects of Arousal on Emotional Sensitivity and Facial Mimicry

Polyvagal theory (Porges, 1995, 2001, 2003a) emphasises the role of facial expressions in social engagement, and suggests that facial expressivity is important during interpersonal interactions. As discussed in section 6.1.3, function of the autonomic nervous system may restrict facial expressivity due to withdrawal of the parasympathetic nervous system (PNS) and activation of the sympathetic nervous system (SNS; see Porges, 2001, 2003a). Reduced facial expressivity due to defensive
physiological arousal may reduce automatic mimicry of facial expressions, which in turn may lower emotional sensitivity. SNS activation is therefore likely to influence both sides of the communication process, with deficiencies in both the encoding and decoding of emotional signals. The present study is designed to investigate the effects of defensive physiological arousal on both emotional sensitivity and facial mimicry by using a simple behavioural measure of emotional sensitivity (the Multimorph Facial Affect Recognition Task; Blair, Colledge, Murray, & Mitchell, 2001) in conjunction with electromyography. Two hypotheses were proposed:

**Hypothesis 3.** Increased activation of the SNS in response to a laboratory stressor will be associated with decreased facial expressivity. This hypothesis was also tested in Chapter 6, but the behavioural measure of facial expressivity in the last chapter was based on the ability to voluntarily modulate facial expressions in response to instructions. In contrast, the current research aims to investigate the effects of increased SNS activation on spontaneous facial expressivity (i.e., involuntary mimicry in response to facial displays). In addition to this, the following hypothesis will be tested:

**Hypothesis 4.** Increased activation of the SNS in response to a laboratory stressor will be associated with decreased emotional sensitivity (i.e., the ability to recognise emotions in others). Surprisingly few studies have investigated the effects of stressors on emotion recognition, although Hänggi (2004) reported that stress has a negative effect on emotional sensitivity. Hänggi's (2004) findings result from an Internet-based experiment where participants carried out emotion recognition tasks during an online procedure designed to induce stress. During the experimental tasks participants in the stress condition experienced several stressor manipulations; these included negative feedback on performance, a form malfunction so that data entered during the experiment had to be repeated, and increased time pressure. Hänggi's results revealed that individuals in the stress condition demonstrated poorer recognition of emotional facial displays compared to those in a control condition. In the current experiment, it is proposed that participants in the speech group will exhibit decreased sensitivity (i.e., will be slower to recognise emotional expressions) after a stressor manipulation.

Two experiments using between-subjects designs were carried out to investigate these hypotheses. Experiment 1 replicated the design of the study in Chapter 6: two groups of participants carried out an emotion recognition task before and after a stressor manipulation.
manipulation (speech task vs. control condition). Although the emotion recognition task in the current research was found to be less arousing than the expressive regulation task from Chapter 6, there were no group differences in physiological arousal during the second administration of the emotion recognition task. Consequently, Experiment 2 further refined the speech task to increase its effectiveness in maintaining arousal in the speech group after the stressor manipulation. The modified speech task was able to maintain arousal for longer in the speech group during the secondary task allowing the effects of defensive physiological arousal on emotional sensitivity and facial mimicry to be evaluated.

7.3. Methodology

7.3.1. Participants

Eighty undergraduate students (16 males, 64 females) volunteered to participate in the experiment and were awarded course credits as part of their undergraduate course requirements. Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders, and current psychological or pharmacological treatment. The participants ranged in age from 18–30 with a mean age of 20.10 years ($SD = 2.37$). 96.3 % of these participants identified themselves as Caucasian and 3.8% as Mixed. During the experiment participants were randomly allocated one of two tasks: a speech task ($n = 40$: 6 males, 34 females; mean age = 21.05, $SD = 2.59$) or a control task ($n = 40$: 10 males, 30 females; mean age = 19.15, $SD = 1.67$). Due to recording errors two participants’ data were excluded from the skin conductance level analyses.

7.3.2. Behavioural Measures of Emotional Sensitivity and Facial Mimicry

7.3.2.1. The Multimorph Facial Affect Recognition Task. The Multimorph Facial Affect Recognition Task is a tool used to assess the speed and accuracy with which one identifies an emotion expression (see section 3.6.2.1; see also Blair et al., 2001). Six distinct emotional expressions (happiness, sadness, fear, anger, surprise, and disgust) feature in the task, each portrayed by three male and three female actors (36 stimuli in total). Each trial begins with a neutral face, which gradually morphs through 39 stages of 450 ms each into one of the six prototypic emotional expressions (see
Prior to completing the task, the participants were given the following instructions:

*You will be presented with a series of faces. These faces are initially neutral, that is, they have a blank expression. However, the faces will slowly change over many stages, to reveal one of the six target emotions listed on the screen. For each face, you will have to determine which expression is displayed as soon as possible in as few stages as possible, without merely guessing. So remember, the aim is to say which emotion is being shown as soon as you recognize it by choosing one of the six emotions: fear, sadness, disgust, surprise, happiness, or anger. Once you have given an answer, you can change your mind when you want to, and as often as you wish right up until the end of the expression. Finally, for each face, you will also be asked to give a final answer.*

*Figure 7.1. Example of anger stimulus presentation from the Multimorph Facial Affect Recognition Task. Stimuli taken from Pictures of Facial Affect (Ekman & Friesen, 1976).*

The principal measure of performance is the mean number of stages required to achieve the correct classification of emotion (maximum number of stages = 39); faster reaction times indicate a greater degree of emotional sensitivity. Secondary measures include the first stage at which any response is made and performance accuracy for the expressions at 100% expression. In addition to these measures facial electromyographic (EMG) activity was captured throughout the task to assess the degree of facial mimicry a person displays.

For this study the Multimorph Task procedure was replicated from the original task using e-Prime (Version 1.1; Psychology Software Tools, Pittsburgh, PA). E-Prime was used so that the stimuli could be randomised appropriately across the two halves of
the task, and so that the program could signal the onset of stimuli to the
psychophysiology hardware. The task was programmed so that each trial was preceded
by a two-second fixation cross to create a small baseline period for the EMG data.
During the first administration (T1), eighteen stimuli (three from each emotion
category) were presented in a random order using the procedure described above. Once
the intermediate stressor manipulation had been completed, the remaining eighteen
stimuli were presented in a random order (T2).

7.3.2.2. Electromyography. Facial muscle activity was recorded from the
zygomaticus major (cheek; positive emotion), corrugator supercillii (brow; negative
affect), and levator labii superioris (upper lip; disgust) muscles using pairs of 4mm
Ag/AgCl electrodes on the left side of the face (see section 3.6.1.2; see also Fridlund &
Cacioppo, 1986). Electromyographic activity was sampled at a frequency of 2000 Hz.
The raw EMG data were filtered online with a high pass filter at 10 Hz, a low pass filter
at 500 Hz, and were amplified by 1000 (BIOPAC Systems; Goleta, CA). To correct
for the positive skew inherent to EMG data, the EMG data was integrated with a 20ms
window. The EMG data was then standardised within participants and muscle sites to
allow meaningful comparisons to be made (Winkielman & Cacioppo, 2001). Difference
scores for the EMG data were calculated using the final 1000 ms of the fixation screen
as a baseline. The amplitude of integrated EMG data has been shown to be a valid and
reliable index of changes in muscle action potentials, making EMG a sensitive measure
of facial expressivity (Cacioppo, Petty, Losch, & Kim, 1986).

7.3.3. Procedure

Participants attended a single testing session in an air-conditioned, sound
attenuated room. After obtaining written consent participants completed the
demographic screening questionnaire (see section 3.5.1). Participants then completed
questionnaires to assess their usual levels of emotional expressivity (Berkeley
Expressivity Questionnaire, BEQ; Gross & John, 1995, see section 3.5.6), their ability
to regulate their emotions (Difficulties in Emotion Regulation Questionnaire, DERS;
Gratz & Roemer, 2004, see section 3.5.7), and how safe they feel in their social
relationships (Social Safeness and Pleasure Scale, SSPS; Gilbert et al., 2009, see section
3.5.9). Participants also completed measures to assess trait mood (Beck Depression
Inventory, BDI-II; Beck et al., 1996, see section 3.5.4; and the Generalised Anxiety
Disorder Scale, GAD-7; Spitzer et al., 2006, see section 3.5.6). The questionnaires were included to control for individual differences in factors that might influence emotional sensitivity and/or facial mimicry. Following the questionnaires, electrodes for recording heart rate (HR) and skin conductance level (SCL) were applied (see section 3.4.4) as well as electromyography (EMG) electrodes on the face to capture muscle activity (see section 7.3.2.2). Standard procedures for placing the electrodes were followed and a five-minute baseline recording was carried out during which the participants were asked to sit quietly. Recording HR allowed for the calculation of heart rate variability (HRV, see section 3.4.3). After the five minutes participants completed a questionnaire to assess their current emotional state (Profile of Mood States – Short Form, POMS-SF; Shacham, 1983, see section 3.5.1). Instructions were then given for the first half of the Multimorph Facial Affect Recognition Task (T1). Following this, the participants were randomised to either a speech task (speech group; see section 4.6.2) or a control task (reading group; see section 6.3.3). After the three-minute stressor manipulation, both groups recompleted the questionnaire to assess their mood state (POMS-SF) and then carried out the second half of the Multimorph Facial Affect Recognition Task (T2). A flowchart diagram of the emotion recognition procedure can be found in appendix 17.

7.4. Results

7.4.1. Statistical Analyses

For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. To evaluate the influence of possible covariates in the planned analyses comparing the stressor manipulation groups, bivariate correlations were computed among the dependent variables (emotional sensitivity, emotional accuracy, and EMG means) and possible covariates (age, sex, trait mood [BDI-II and GAD-7], self-reported expressivity [BEQ], social safeness [SSPS], and difficulties in emotion regulation [DERS]). The findings from the correlations are discussed in section 7.4.2.

Mean HR, SCL, and high-frequency heart rate variability (HF-HRV) values were calculated for the baseline period, each block of the Multimorph task, and the stressor manipulation period. Mean EMG amplitudes were also calculated for each
stimulus presentation from the Multimorph task. Analyses were then performed to establish if there were any baseline differences between the stressor manipulation groups (the group characteristics can be found in table 7.1). Chi-squared tests confirmed that there were no significant differences between the stressor groups in terms of sex and ethnicity. Univariate analyses of variance (ANOVAs) also failed to identify any significant differences between the groups in terms of age, self-reported difficulties in emotion regulation, social safeness, emotional expressivity, or mood at baseline. Further to this, univariate ANOVAs did not find any significant group differences for HR, SCL, or HF-HRV at baseline or during the Multimorph task at T1 (the physiological data are shown in table 7.2). It is worth noting however, that only the reading group exhibited a significant decrease in HF-HRV from baseline during the Multimorph task at T1, \( t(39) = 2.93, p = .006 \). Finally, mixed factorial ANOVAs (repeated measure: Emotion; between subjects factor: Group) did not find any significant differences between the groups for Emotional Sensitivity or Emotional Accuracy at T1 (the group means can be seen in table 7.1).

### Table 7.1.

Stressor group characteristics

<table>
<thead>
<tr>
<th></th>
<th>Reading Group (n = 40)</th>
<th>Speech Group (n = 40)</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI-II</td>
<td>6.80 (6.06)</td>
<td>6.23 (5.61)</td>
<td>-.44</td>
<td>78</td>
<td>.661</td>
</tr>
<tr>
<td>BEQ Mean</td>
<td>4.60 (0.73)</td>
<td>4.60 (0.89)</td>
<td>-.27</td>
<td>78</td>
<td>.979</td>
</tr>
<tr>
<td>DERS Total</td>
<td>79.97 (20.28)</td>
<td>83.13 (18.59)</td>
<td>.72</td>
<td>78</td>
<td>.471</td>
</tr>
<tr>
<td>GAD-7</td>
<td>4.23 (3.96)</td>
<td>3.80 (3.01)</td>
<td>-.54</td>
<td>78</td>
<td>.590</td>
</tr>
<tr>
<td>POMS-SF Total</td>
<td>4.25 (11.89)</td>
<td>3.43 (9.55)</td>
<td>-.34</td>
<td>78</td>
<td>.733</td>
</tr>
<tr>
<td>SSPS</td>
<td>32.95 (7.17)</td>
<td>33.13 (6.43)</td>
<td>.12</td>
<td>78</td>
<td>.909</td>
</tr>
</tbody>
</table>

#### Multimorph

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>22.35 (3.54)</td>
<td>23.87 (3.53)</td>
<td>0.63</td>
<td>78</td>
<td>.530</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>22.62 (3.06)</td>
<td>22.32 (3.38)</td>
<td>-0.42</td>
<td>78</td>
<td>.679</td>
</tr>
<tr>
<td>Accuracy T1 (%)</td>
<td>87.31 (7.65)</td>
<td>89.67 (8.03)</td>
<td>1.35</td>
<td>78</td>
<td>.182</td>
</tr>
<tr>
<td>Accuracy T2 (%)</td>
<td>90.39 (5.75)</td>
<td>91.53 (6.29)</td>
<td>0.85</td>
<td>78</td>
<td>.400</td>
</tr>
</tbody>
</table>

*Note. BDI-II = Beck Depression Inventory, BEQ = Berkeley Expressivity Questionnaire, DERS = Difficulties in Emotion Regulation Scale, GAD-7 = Generalised Anxiety Disorder Scale, POMS-SF = Profile of Mood States Questionnaire, SSPS = Social Safeness and Pleasure Scale.*

The first main set of analyses in this chapter evaluated the effects of the Multimorph task on facial mimicry at T1. A repeated-measures ANOVA was carried out on the EMG amplitude data with Muscle and Emotion as repeated factors. The Huynh-Feldt degrees of freedom correction was applied where necessary (i.e., when
factors violated sphericity assumptions, as confirmed by Mauchly’s test). Significant main effects for all analyses were followed up with pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05. To investigate the effects of facial mimicry bivariate correlations were conducted between the dependent variables (i.e., EMG amplitudes, emotional sensitivity scores, and emotional accuracy scores).

The second main set of analyses investigated the effects of the stressor manipulations on the physiological indices of arousal. Reactivity scores were calculated for HR, SCL, and HF-HRV by subtracting the stressor manipulation period from the corresponding baseline data (Kamarck et al., 1992; Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). To establish if the stressor manipulations induced different levels of arousal across the groups, univariate ANOVAs were performed on the reactivity scores with Group as the fixed factor. The physiological reactivity scores were also correlated with the POMS-SF reactivity data to investigate whether changes in ANS function during the stressor manipulation period were related to changes in self-reported emotion. Significant correlations revealed that greater HR reactivity was associated with larger increases in the POMS-SF tension–anxiety subscale \( r = .35, p = .001 \) and the POMS-SF confusion–bewilderment subscale \( r = .35, p = .002 \), whilst greater SCL reactivity was associated with larger increases in the POMS-SF tension–anxiety subscale \( r = .24, p = .032 \). Taken together, the directions of these relationships suggest that larger increases in SNS activation were associated with greater self-reported negative affect. To investigate if the changes in mood and physiology were carried over to the Multimorph tasks mixed-factorial ANOVAs (with Time as the repeated factor, and Group as the between subjects factor) were also performed using the POMS-SF means and the raw physiological data.

The third set of analyses investigated whether there were any group differences in emotional sensitivity and emotional accuracy across the two administrations of the Multimorph task. Mixed-factorial ANOVAs (with Time and Emotion as the repeated factors, and Group as the between subjects factor) were performed on the separate emotional sensitivity and accuracy scores. Significant main effects for all analyses were followed up with pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05.
CHAPTER 7: EMOTIONAL SENSITIVITY

Table 7.2.
Mean heart rate, skin conductance level, and high-frequency heart rate variability by stressor group

<table>
<thead>
<tr>
<th></th>
<th>Reading Group</th>
<th>Speech Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Multimorph (T1)</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>74.19 (9.28)</td>
<td>74.20 (9.56)</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>1.69 (1.18)</td>
<td>2.06 (1.40)**</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.58 (1.13)</td>
<td>7.34 (1.01)**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Reading Group</th>
<th>Speech Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Multimorph (T1)</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>71.65 (7.87)</td>
<td>71.25 (8.28)</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>1.55 (1.29)</td>
<td>1.95 (1.54)***</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.48 (0.93)</td>
<td>7.35 (0.98)</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant change from baseline *p < .05, **p < .01, ***p < .001

Table 7.3.
Mean scores for the POMS-SF subscales before the first (T1) and second (T2) administrations of the Multimorph task.

<table>
<thead>
<tr>
<th></th>
<th>Reading Group (n = 40)</th>
<th>Speech Group (n = 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Multimorph (T1)</td>
<td>Multimorph (T2)</td>
</tr>
<tr>
<td>Depression–Dejection</td>
<td>1.35 (1.35)</td>
<td>0.40 (0.93)**</td>
</tr>
<tr>
<td>Vigour–Activity</td>
<td>5.43 (3.88)</td>
<td>3.95 (3.27)**</td>
</tr>
<tr>
<td>Anger–Hostility</td>
<td>0.50 (1.28)</td>
<td>0.23 (0.66)</td>
</tr>
<tr>
<td>Tension–Anxiety</td>
<td>2.62 (3.76)</td>
<td>2.05 (2.41)</td>
</tr>
<tr>
<td>Confusion–Bewilderment</td>
<td>1.38 (1.78)</td>
<td>1.20 (1.59)</td>
</tr>
<tr>
<td>Fatigued–Inertia</td>
<td>3.82 (3.86)</td>
<td>3.83 (3.84)</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Significant change from T1 **p < .01 ***p < .001
Finally, to investigate the effects of the stressor manipulations on facial mimicry, analyses investigated whether there were any group differences in EMG reactivity across the two administrations of the Multimorph Task. To achieve this, a mixed-factorial ANOVA (repeated measures: Time, Muscle, Emotion; between subjects factor: Group) was carried out using the EMG amplitude data from T1 and T2.

7.4.2. Individual Differences in Emotional Sensitivity and Accuracy at T1

Data collected from the first half of the Multimorph (T1) were analysed to identify individual differences in Emotional Sensitivity and Emotional Accuracy.

7.4.2.1. Emotional sensitivity. At T1 the mean number of stages taken to correctly identify an emotion was 23.60 (SD = 3.52). Bivariate correlations revealed significant relationships between Emotional Sensitivity and Sex ($r = 2.80$, $p = .012$) and Emotional Sensitivity and the non-acceptance scale of the DERS ($r = -0.26$, $p = .021$). Emotional Sensitivity tended to be lower for men, with women correctly identifying the emotional expressions 2.5 stages quicker on average. The relationship between Emotional Sensitivity and the non-acceptance scale of the DERS was negative, suggesting that greater difficulties in accepting one’s own emotional responses is related to earlier correct responses on the Multimorph (i.e., increased sensitivity to the emotions of others). Due to their effects, Sex and the non-acceptance scale of the DERS were entered as covariates in subsequent analyses relating to Emotional Sensitivity.

7.4.2.2. Emotional accuracy. At T1 the mean percentage of emotions correctly identified by all participants at full expression was 88.48% (SD = 7.88). None of the baseline questionnaire measures were significantly correlated with Emotional Accuracy. Emotional Sensitivity however was found to be negatively correlated with Emotional Accuracy ($r = -0.24$, $p = .035$), suggesting that people who were quicker to identify the emotional expressions were more likely to correctly distinguish the emotions from one another by the time they reached full expression.

7.4.3. EMG Correlates of Emotional Sensitivity and Accuracy

To investigate the effects of facial mimicry on emotional sensitivity and emotional accuracy two analyses were carried out. First of all, to confirm that observing
facial displays results in facial mimicry, a 3 x 6 repeated-measures ANOVA was carried out on the EMG amplitude data from T1 with the repeated factors of Muscle (corrugator, zygomaticus, and levator) and Emotion (anger, disgust, sadness, fear, surprise, and happiness). A significant Muscle x Emotion interaction was found, $F(4.42, 58.43) = 5.98, p < .001$, which can be seen in figure 7.2. Generally corrugator activity was higher for negative emotions and zygomaticus activity was higher for positive emotions (although the zygomaticus also exhibited an increase for facial displays of anger). Contrary to expectations, activation of the levator labii muscle did not show increased activation for disgust stimuli. A second set of analyses was carried out to investigate the effects of facial mimicry on emotional sensitivity and emotion accuracy. Bivariate correlations were conducted using the EMG amplitude data and the emotional sensitivity and emotional accuracy scores for each emotion. No significant relationships were found between the mean EMG amplitude of each muscle site and Emotional Sensitivity, although Emotional Accuracy for anger was positively correlated with activation of the zygomaticus major ($r = .26, p = .020$), that is participants who exhibited higher levels of zygomaticus major activity during anger displays were more likely to correctly identify the emotion at 100%.

![Figure 7.2. Mean activation of the corrugator supercilii (CS), zygomaticus major (ZM) and levator labii (LL) muscle sites as a function of Emotion during the Multimorph at T1. Error bars represent the standard error.](image)

7.4.4. Physiological Reactivity to the Stressor Manipulations

To investigate the effects of the stressor manipulations, univariate ANOVAs (fixed factor: Group) were carried out separately for the HR, SCL, and HF-HRV
reactivity scores; the raw means for each group are shown in table 7.2. A significant effect of Group was revealed for all of the physiological variables: HR reactivity $F(1, 78) = 36.67, p < .001$; SCL reactivity $F(1, 78) = 6.03, p = .016$; and HF-HRV reactivity $F(1, 78) = 6.41, p = .012$. Bonferroni pairwise comparisons confirmed that the speech group demonstrated significantly higher reactivity to the stressor manipulation than the reading group across all of the physiological variables (larger increases in HR and SCL, and a larger decrease in HF-HRV; significant at $p < .05$), although it should be noted that both groups demonstrated increases in SCL that were significantly above baseline.

To investigate the effect of the stressor manipulations on arousal during the Multimorph tasks a series of mixed-factorial ANOVAs were carried out with the POMS-SF data and the physiological variables. A mixed-factorial ANOVA was carried out on the raw POMS-SF scores with Time and Scale (depression–dejection, vigour–activity, anger–hostility, tension–anxiety, confusion–bewilderment, and fatigue–inertia) as repeated factors, and with Group as the between subjects factor. A significant main effect was found for Scale, $F(2.60, 20.04) = 43.28, p < .001$, as well as significant two-way interactions for Time x Group, $F(1, 78) = 15.20, p < .001$, Scale x Group, $F(2.60, 20.04) = 5.52, p = .002$, and Time x Scale, $F(3.53, 274.95) = 23.93, p < .001$. These were superseded by a significant three-way interaction of Time x Scale x Group, $F(3.53, 274.95) = 21.88, p < .001$. Paired $t$-tests revealed that the reading group reported being significantly less depressed and vigorous at T2, whilst the speech group reported feeling significantly less vigorous and fatigued, but significantly more tense and confused (significance is marked in table 7.3).

Mixed-factorial ANOVAs (with Time as the repeated factor, and Group as the between subjects factor) were carried out using the raw physiological data. Although the speech group exhibited greater HR, SCL, and HF-HRV reactivity during the stressor manipulation (i.e., greater SNS activation), these changes were not maintained during the second ER task at T2. As a result there were no significant physiological differences between the groups at T2. Again, only the reading group demonstrating a significant decrease in HF-HRV from baseline during the Multimorph task at T2, $t(39) = 2.46, p = .018$. 
7.4.5. Effects of the Stressor Manipulations on Emotional Sensitivity and Accuracy

The mean number of stages to correctly identify each emotion can be seen in figure 7.3. Mixed-factorial ANCOVAs (repeated measures: Time, Emotion; between subjects factor: Group; covariates: Sex, DERS non-acceptance) were carried out on the emotional sensitivity scores to assess the impact of the stressor manipulation on performance during the Multimorph (T1 compared to T2). Consistent with the baseline analyses Sex was significantly associated with Emotional Sensitivity, $F(1, 73) = 9.22$, $p = .003$, with females being consistently faster at correctly identifying emotions than males. In contrast, the DERS non-acceptance scale was not significant, $F(1, 73) = 3.65$, $p = .060$. Emotional Sensitivity was also significantly influenced by a main effect of Emotion, $F(5, 365) = 4.60, p < .001$; happiness was the emotion quickest to be correctly identified overall, followed by surprise, disgust, anger, sadness, and fear. In addition to this, a significant two-way interaction was revealed for Time x Emotion, $F(4.70, 343.08) = 2.57, p = .030$, which was superseded by a Time x Emotion x Sex interaction, $F(4.70, 343.08) = 2.37, p = .043$. Participants were more sensitive to surprise and fear during the second administration of the Multimorph ($M = 3.5$ and $M = 1.5$ stages quicker respectively, as shown in figure 7.3); post hoc analyses revealed that this increase in sensitivity was more pronounced for male participants, although female participants were still quicker at identifying all emotions. The ANCOVA did not reveal any significant main effects or interaction effects involving Group.

![Figure 7.3](#)  
*Figure 7.3. Mean number of stages taken to correctly identify each emotion across the two administrations of the Multimorph Task. Covariates were controlled at the following values: Sex = 1.81, DERS non-acceptance = 13.21. Error bars represent the standard error.*
A second mixed-factorial ANOVA revealed that Emotional Accuracy was also significantly affected by Emotion, $F(4.23, 308.45) = 20.47, p < .001$; overall happiness was the emotion most likely to be correctly identified at 100% expression, followed by surprise, sadness, anger, fear, and disgust (as shown in Figure 7.4). No significant effects were revealed involving Time or Group.

7.4.6. Effects of the Stressor Manipulations on Facial Mimicry

A 2 x 3 x 6 x 2 mixed-factorial ANOVA (repeated measures: Time, Muscle, Emotion; between subjects factor: Group) was carried out to investigate the effects of the stressor manipulations on facial mimicry. A significant two-way interaction was found for Emotion x Muscle, $F(7.58, 590.93) = 9.43, p < .001$. Consistent with the EMG findings at T1, overall corrugator activity was higher for negative emotions and zygomaticus activity was higher for positive emotions (as in Figure 7.2). It was originally hypothesised that the speech group would exhibit less facial mimicry during the Multimorph task after the stressor manipulation, but there were no significant effects involving Time or Group meaning this hypothesis was not supported.

7.5. Discussion

The aim of this study was to investigate the effects of defensive physiological arousal on emotional sensitivity, as well as facial mimicry. In Experiment 1, despite several of the findings being consistent with the existing literature, the primary
hypotheses regarding the effects of the stressor manipulation on emotional sensitivity and facial mimicry were not supported.

7.5.1. Baseline Correlates of Emotional Sensitivity and Accuracy

Emotional Sensitivity at baseline was correlated with sex and the non-acceptance scale of the DERS. This is in line with previous research that has indicated that emotional sensitivity is moderated by factors such as gender (Hoffmann et al., 2010) and symptoms of psychopathology (Csukly et al., 2008; Sprengelmeyer et al., 1998). Female participants were quicker to identify all emotions during both administrations of the Multimorph tasks, whilst difficulties in emotion regulation were associated with increased sensitivity to emotional expressions.

The EMG data was consistent with previous research which has consistently found that increased activation of the corrugator supercili is associated with viewing negative stimuli, whilst increased zygomaticus major activity is associated with viewing positive stimuli (Cacioppo et al., 1986; Fridlund & Cacioppo, 1986; Larsen, Norris, & Cacioppo, 2003). Inconsistent with previous findings was the lack of correspondence between facial displays of disgust and activation of the levator labii (Lundqvist, 1995; Vrana, 1993). Disgust was the emotion most likely to be misidentified at full expression, which may explain why the observed patterns of EMG activity did not correspond to this emotion (on average disgust was only correctly identified on 79.5% of occasions, whereas the other emotions were correctly identified more than 88% of the time). A second inconsistent finding was the association between anger displays and increased activation of the zygomaticus major. Interestingly increased activation of the ZM muscle was correlated with emotional accuracy for anger displays. The observed pattern of EMG activation for anger may be the result of counter-mimicry effects (i.e., opposite facial displays produced in response to angry facial expressions; see Englis, Vaughan, & Lanzetta, 1982; Lanzetta & Englis, 1989), although this is not consistent with the increase in corrugator activation also seen in response to the anger displays.

The complementary patterns of EMG activity observed for most of the emotions during the Multimorph task does suggest that participants were mimicking the facial displays presented (Dimberg, 1982; Hatfield et al., 1993). Despite this, there was no evidence to suggest that facial mimicry was associated with emotional sensitivity. This
is parallel to the findings of Blairy et al. (1999) and Rives Bogart and Matsumoto (2009), but is at odds with the research by Stel and colleagues (Stel & van Knippenberg, 2008; Stel & van den Bos, 2010). It is possible that the current findings are disparate from Stel and colleagues because the current experiment used shorter displays of stimuli to elicit facial mimicry.

7.5.2. Effects of Defensive Arousal on Emotional Sensitivity and Accuracy

No significant differences were found between the groups in terms of emotional sensitivity or emotional accuracy at T2. Both groups demonstrated an increase in sensitivity, suggesting that the Multimorph task is vulnerable to practice effects. Male participants were more likely to show a greater increase in emotional sensitivity across the two tasks, but females remained quicker at correctly identifying all of the emotional categories (this is consistent with previous research, for example Hoffmann et al., 2010; Rotter & Rotter, 1988). There are two possible explanations for the current lack of findings. First of all, the Multimorph task may not be a sensitive enough measure of emotional sensitivity. Evidence opposing this conclusion comes from a range of studies, which have previously demonstrated the Multimorph’s capacity to discriminate between different populations (e.g., psychopaths, Blair et al., 2004; boys with autism spectrum disorder, Rogers, Viding, Blair, Frith, & Happé, 2006; borderline personality disorder, Lynch et al., 2006; and avoidant personality disorder, Rosenthal et al., 2011). A second, more likely possibility, is that the stressor manipulation used in the current research is not strong enough. The speech task is unable to induce changes in arousal that are sustained over the secondary task. This means that the stressor groups cannot be discriminated on the basis of arousal at T2, which may have contributed to the lack of differences in emotional sensitivity, emotional accuracy, and facial mimicry observed at T2.

7.5.3. Limitations

The most notable limitation is that the current stressor manipulation appears to be ineffective during secondary tasks. Previous studies have utilised social evaluative threat tasks that have resulted in observable differences in behavioural outcomes following the manipulation period. This could be because participants in these studies are often selected based on how they score on questionnaire measures (e.g., high
socially anxious vs. low socially anxious participants; Garner, Mogg, & Bradley, 2006; Mansell, Clark, Ehlers, & Chen, 1999). The current studies are interested in how socio-emotional responding is affected by changes in autonomic function in healthy control participants; this factor may be underpinning the physiological return to baseline seen in the speech group.

A simple way to overcome the limitation of the speech task may be to follow other researchers and select participants based on questionnaire measures (e.g., participants reporting high anxiety or depression symptoms), however I believe that using this does not provide an adequate test of polyvagal theory. Differences observed in analogue populations can be informative, but polyvagal theory suggests that the communicative deficits associated with defensive physiological responding should be applicable all mammalian organisms when they are under threat/challenge (Porges, 1995, 2001, 2003a). Polyvagal theory hypothesises that the some of the deficits in emotional responding seen in clinical and sub-clinical populations are due to functional abnormalities, as opposed to anatomical or biological deficiencies (Austin, Riniolo, & Porges, 2007; Porges, 2003a). Identifying and isolating populations who are vulnerable to the effects of defensive physiological arousal does not shed light on this argument. To evaluate the utility of polyvagal theory as a universal model of emotional and physiological responding requires healthy samples to demonstrate deficits in socio-emotional behaviours during challenging situations, even if only to a minor degree.

7.5.4. Conclusion

The current experiment failed to support the hypothesis that defensive physiological arousal is associated with reduced facial expressivity. Participants in the speech group did not exhibit less facial mimicry after the manipulation period, and in turn there were no differences between the stressor groups in emotional sensitivity or emotional accuracy at T2. A major limitation of this experiment was the inability of the stressor manipulation to induce a prolonged defensive physiological response in the speech group (i.e., increased SNS activation was not maintained during the Multimorph task at T2). To address this limitation the current design needs to be revised in order to ensure secondary tasks occur with concurrent defensive physiological arousal. Only then can conclusions be drawn about the influence of defensive physiological arousal on facial expressivity and emotional sensitivity.
7.6. Experiment 2: Emotional Sensitivity and Facial Mimicry in Response to a Repetitive Stressor Task

Experiment 1 was unable to find any supporting evidence to suggest that increased activation of the SNS is associated with decreases in facial expressivity; defensive physiological arousal was not linked to any changes in facial mimicry or emotional sensitivity. A limitation of Experiment 1 was the lack of residual arousal remaining after the stressor manipulation in the speech group. Although participants in the speech group demonstrated larger increases in physiological arousal during the manipulation period (i.e., increased SNS activation), along with increases in self-reported tension and anxiety, these increases were not maintained during the second Multimorph task. Although previous research has shown the utility of using social evaluative tasks to induce challenge/threat during secondary tasks, often these have used analogue populations (e.g., Garner, Mogg, & Bradley, 2006; Mansell, Clark, Ehlers, & Chen, 1999). The current studies are unique in that they purposefully sample a healthy population. A difficulty of using healthy control participants is that they are insusceptible to the effects of the speech task once a secondary task has been employed. In Experiment 2 the between-subjects design of Experiment 1 was repeated but with a modification to the speech task preparation period.

7.7. Repetitive Stress and the Speech Task Adaptation II

The findings from the previous studies in this thesis have emphasised the reliability of using a speech task preparation period to induce arousal. However, they have also highlighted that arousal is quickly down-regulated during the completion of a second, unrelated task. To enhance the utility of the speech task in this experiment the preparation period of the speech task was split into three blocks. The task was divided into smaller sections because individuals tend to habituate to stressors over long periods of time, and in contrast arousal levels will tend to remain high when stressors are consistently novel and involve uncertainty (Kelsey, Ornduff, & Alpert, 2007; Kelsey, Soderlund, & Arthur, 2004). The secondary task, the Multimorph Facial Affect Recognition Task (Blair et al., 2001), was modified to incorporate three one-minute breaks during the second administration of the task (T2), during which the stressor manipulation could be completed. Participants allocated to the speech group used the one minute blocks to prepare their speech, whilst participants in the reading group were
given time to silently read through material corresponding to the content of the speech task. It was hypothesised that alternating between the stressor manipulations and the Multimorph task would increase the amount of residual arousal during the secondary task. It was hoped that in turn, this would make the effects of the stressor manipulation more apparent in the behavioural measures at T2.

7.8. Method

7.8.1. Participants

Sixty-four undergraduate students (13 males, 51 females) volunteered to participate in the experiment and were awarded course credits as part of their undergraduate course requirements. Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders, and current psychological or pharmacological treatment. The participants ranged in age from 18–34 with a mean age of 19.53 years ($SD = 3.30$). 92.2% of these participants identified themselves as Caucasian, 3.1% as Asian, 3.1% as Mixed, and 1.6% as Other. During the experiment participants were randomly allocated one of two tasks: the speech task ($n = 32$: 4 males, 28 females; mean age = 19.56, $SD = 3.17$) or the control task ($n = 32$: 9 males, 23 females, mean age = 19.19, $SD = 2.89$). Three participants’ HR data were excluded from analyses: two due to excessive noise in the HR recording, and one due to the heart-rate monitor failing during data collection. Subsequently some of the HR and HF-HRV analyses have a sample size of 61 participants (speech group $n = 30$, reading group $n = 31$).

7.8.2. Procedure

The procedure was replicated from Experiment 1, however the second half of the Multimorph task was modified to incorporate the blocks of the stressor manipulation: The second half of the Multimorph Facial Affect Recognition Task (T2) was completed whilst alternating the task with one-minute blocks of the stressor manipulation (either preparing a speech [see section 4.6.2], or reading quietly [see section 6.3.3]). During the manipulation blocks the e-Prime program instructed participants to complete the task assigned to them. Participants were signalled to resume the Multimorph task at the end of the one-minute blocks by a beep and an alert on the
screen. A flowchart diagram of the adapted emotion recognition procedure can be found in appendix 18.

7.9. Results

7.9.1. Statistical Analyses

Similar analyses were conducted to Experiment 1 (see section 7.4.1). For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. Mean HR, SCL, and HF-HRV values were calculated for the baseline period, each block of the Multimorph task, and each block of the stressor manipulation. Mean EMG amplitudes were also calculated for each stimulus presentation from the Multimorph task. First, analyses were conducted to establish whether there were any differences between the stressor manipulation groups at baseline. Secondly, bivariate correlations were carried out to identify confounds that might affect emotional sensitivity, emotional accuracy, or facial mimicry. Thirdly, analyses were carried out to investigate the effects of the repetitive stressor manipulations on physiology and mood state. Fourthly, analyses were conducted to evaluate the effects of the repetitive stressor manipulations on emotional sensitivity and emotional accuracy. Finally, analyses were conducted to investigate the effects of the repetitive stressor manipulations on facial mimicry. As in Experiment 1, the Huynh-Feldt degrees of freedom correction was applied where necessary (i.e., when factors violated sphericity assumptions, as confirmed by Mauchly’s test). Significant main effects for all analyses were followed up with pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05.

7.9.2. Stressor Group Characteristics

As in Experiment 1, the two groups were compared using their demographic data and baseline self-report measures (see table 7.4 for group characteristics). The groups did not significantly differ in terms of age, sex, or baseline questionnaire measures. Univariate ANOVAs did not reveal any significant differences between the groups for HR, SCL, or HF-HRV at baseline or during the Multimorph at T1 (see table
7.5 for raw physiological means). Further to this, mixed-factorial ANOVAs (repeated measure: Emotion; between subjects factor: Group) did not find any significant differences between the groups for Emotional Sensitivity or Emotional Accuracy at T1.

Table 7.4.
Stressor group characteristics

<table>
<thead>
<tr>
<th></th>
<th>Reading Group (n = 32)</th>
<th>Speech Group (n = 32)</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>10.22 (9.27)</td>
<td>8.81 (7.20)</td>
<td>0.68</td>
<td>62</td>
<td>.501</td>
</tr>
<tr>
<td>DERS Total</td>
<td>81.41 (26.16)</td>
<td>78.91 (19.72)</td>
<td>0.43</td>
<td>62</td>
<td>.667</td>
</tr>
<tr>
<td>GAD-7</td>
<td>4.88 (4.69)</td>
<td>4.09 (3.08)</td>
<td>0.79</td>
<td>62</td>
<td>.434</td>
</tr>
<tr>
<td>POMS-SF Total</td>
<td>6.84 (14.71)</td>
<td>5.72 (11.90)</td>
<td>0.34</td>
<td>62</td>
<td>.738</td>
</tr>
<tr>
<td>SSPS</td>
<td>31.81 (8.81)</td>
<td>33.97 (7.84)</td>
<td>-1.04</td>
<td>62</td>
<td>.305</td>
</tr>
<tr>
<td>Multimorph</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitivity T1</td>
<td>23.99 (4.67)</td>
<td>23.65 (3.87)</td>
<td>0.31</td>
<td>62</td>
<td>.755</td>
</tr>
<tr>
<td>Sensitivity T2</td>
<td>22.19 (4.22)</td>
<td>22.54 (5.02)</td>
<td>-0.31</td>
<td>62</td>
<td>.762</td>
</tr>
<tr>
<td>Accuracy T1 (%)</td>
<td>89.72 (8.22)</td>
<td>90.24 (7.45)</td>
<td>-0.27</td>
<td>62</td>
<td>.791</td>
</tr>
<tr>
<td>Accuracy T2 (%)</td>
<td>92.92 (6.11)</td>
<td>91.14 (6.89)</td>
<td>1.09</td>
<td>62</td>
<td>.281</td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory, DERS = Difficulties in Emotion Regulation Scale, GAD-7 = Generalised Anxiety Disorder Scale, POMS-SF = Profile of Mood States Questionnaire, SSPS = Social Safeness and Pleasure Scale.

7.9.3. Individual Differences in Emotional Sensitivity and Accuracy at T1

Data collected from the first half of the Multimorph (T1) were analysed to identify individual differences in Emotional Sensitivity and Emotional Accuracy.

7.9.3.1. Emotional sensitivity. With the current sample none of the baseline questionnaire measures or baseline psychophysiology variables were correlated with Emotional Sensitivity at T1.

7.9.3.2. Emotional accuracy. In contrast to the previous experiment, Emotional Accuracy at T1 was correlated with the POMS-SF baseline total ($r = -.30, p = .017$). Higher self-reported distress at baseline was associated with decreased accuracy. A negative relationship was again found between Emotional Sensitivity and Emotional Accuracy, but this time the relationship did not reach significance ($r = -.23, p = .069$). In analyses relating to Emotional Accuracy the POMS-SF baseline total was included as a covariate.
7.9.4. EMG Correlates of Emotional Sensitivity and Emotional Accuracy

To investigate the effects of facial mimicry on emotional sensitivity and emotional accuracy two analyses were carried out. First of all, a 3 x 6 repeated-measures ANOVA was carried out on the EMG amplitude data from T1 with Muscle (corrugator, zygomaticus, and levator) and Emotion (anger, disgust, sadness, fear, surprise, and happiness) as repeated factors. Significant main effects were found for Muscle, $F(2, 126) = 6.03, p = .003$, and Emotion, $F(5, 315) = 4.00, p = .002$. These were superseded by a significant Muscle x Emotion interaction, $F(8.25, 519.60) = 10.03, p < .001$, which can be seen in figure 7.5. As in Experiment 1, corrugator activity was higher for negative emotions. In addition to this, zygomaticus activity was higher for happiness, and activation of the levator was increased for disgust stimuli. In the present experiment the zygomaticus and levator also demonstrated increased activation for facial displays of fear. Bivariate correlations were conducted using the EMG amplitude data and the emotional sensitivity and emotional accuracy scores for each emotion. A significant negative relationship was found between Emotional Sensitivity scores for disgust and activation of the levator labii ($r = -.28, p = .028$), that is participants who exhibited higher levels of levator labii activity during disgust displays were more likely to correctly identify the emotion at lower levels of intensity. No significant relationships were identified between the mean EMG amplitude data and Emotional Accuracy scores.

![Figure 7.5](image-url)
7.9.5. Reactivity to the Stressor Manipulations

Mixed-factorial ANOVAs (repeated measure: Time; between subjects factor: Group) were carried out separately for the HR, SCL, and HF-HRV data. Because the stressor manipulation was carried out over several blocks, the raw mean data for each block was entered into the analyses instead of reactivity scores to allow for more meaningful comparisons across the blocks; the raw means for each group are shown in table 7.5. For HR a significant main effect of Group was found, $F(1, 59) = 5.07, p = .028$. Bonferroni pairwise comparisons confirmed that HR in the speech group ($M = 77.22, SD = 12.43$) was consistently higher during the manipulation blocks than HR in the reading group ($M = 72.39, SD = 8.28$). The SCL analysis found a significant main effect of Time, $F(1.73, 107.13) = 30.40, p < .001$, which occurred because SCL was significantly higher in the first one-minute block for both groups ($p < .001$); SCL then exhibited a decline in both groups during the second and third stressor block. Although the speech group generally demonstrated higher SCLs during the one-minute blocks, the effect of Group was not significant, $F(1, 62) = 3.43, p = .069$. For HF-HRV there was a significant Time x Group interaction, $F(2, 118) = 4.12, p = .019$. During the manipulation blocks HF-HRV was consistently suppressed in the speech group, whereas the reading group demonstrated a significant increase in HF-HRV during the first block of the stressor manipulation that then returned to baseline.

The POMS-SF subscales also demonstrated that the speech group responded differently during the stressor manipulation period than the reading group (see table 7.6). A mixed-factorial ANOVA was carried out on the POMS-SF data with Time and Scale (depression–dejection, vigour–activity, anger–hostility, tension–anxiety, confusion–bewilderment, and fatigue–inertia) as repeated factors, and with Group as the between subjects factor. A significant main effect was found for Scale, $F(2.87, 178.09) = 29.42, p < .001$, as well as significant two-way interactions for Time x Group, $F(1, 62) = 8.92, p = .004$, and Time x Scale, $F(3.57, 221.27) = 21.58, p < .001$. These were superseded by a significant three-way Time x Scale x Group interaction, $F(3.57, 221.27) = 9.38, p < .001$. Paired $t$-tests revealed that both groups reported being significantly less vigorous at T2, but only the speech group reported feeling significantly more tense (significance is marked in table 7.6).
**Table 7.5.**
Mean heart rate, skin conductance level, and high-frequency heart rate variability during the manipulation periods by stressor group

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Stressor Manipulation</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Block 1</td>
<td>Block 2</td>
<td>Block 3</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td><strong>(time)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Reading Group</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>74.00 (4.49)</td>
<td>72.21 (9.30)</td>
<td>72.34 (8.32)</td>
<td>72.63 (8.93)</td>
<td>0.80</td>
<td>1.58, 47.35</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.79 (1.75)</td>
<td>3.72 (2.18)**</td>
<td>3.28 (2.16)*</td>
<td>3.36 (2.26)**</td>
<td>2130.35</td>
<td>1.01, 31.30</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.44 (0.83)</td>
<td>7.76 (0.82)*</td>
<td>7.36 (0.73)</td>
<td>7.47 (0.81)</td>
<td>5.16</td>
<td>3, 90</td>
</tr>
<tr>
<td><strong>Speech Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>71.88 (8.92)</td>
<td>78.78 (11.27)***</td>
<td>77.37 (10.51)***</td>
<td>77.16 (10.06)***</td>
<td>16.01</td>
<td>2.33, 67.50</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.86 (1.55)</td>
<td>4.67 (1.79)***</td>
<td>4.20 (1.87)***</td>
<td>4.27 (2.10)***</td>
<td>1999.84</td>
<td>1.01, 30.32</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.54 (1.00)</td>
<td>7.32 (0.82)</td>
<td>7.35 (0.96)</td>
<td>7.34 (0.89)</td>
<td>1.81</td>
<td>3, 90</td>
</tr>
</tbody>
</table>

**Note.** Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from Baseline * = p < .05 ** = p < .01 *** = p < .001

**Table 7.6.**
Mean scores for the POMS-SF subscales before the first (T1) and second (T2) administrations of the Multimorph task

<table>
<thead>
<tr>
<th></th>
<th>Reading Group (n = 32)</th>
<th>Speech Group (n = 32)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Multimorph (T1)</td>
<td>Multimorph (T2)</td>
<td>Multimorph (T1)</td>
<td>Multimorph (T2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(time x group)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression–Dejection</td>
<td>1.59 (2.75)</td>
<td>1.94 (2.98)</td>
<td>1.37 (3.05)</td>
<td>1.94 (3.39)</td>
<td>0.17</td>
<td>1, 62</td>
</tr>
<tr>
<td>Vigour–Activity</td>
<td>6.22 (3.94)</td>
<td>4.28 (3.42)**</td>
<td>7.13 (3.81)</td>
<td>5.50 (4.03)***</td>
<td>0.29</td>
<td>1, 62</td>
</tr>
<tr>
<td>Anger–Hostility</td>
<td>0.91 (2.18)</td>
<td>1.00 (2.65)</td>
<td>1.09 (3.31)</td>
<td>1.75 (3.93)</td>
<td>1.51</td>
<td>1, 62</td>
</tr>
<tr>
<td>Tension–Anxiety</td>
<td>3.13 (3.07)</td>
<td>3.12 (3.24)</td>
<td>2.84 (2.69)</td>
<td>7.16 (5.06)***</td>
<td>30.70</td>
<td>1, 62</td>
</tr>
<tr>
<td>Confusion–Bewilderment</td>
<td>2.38 (2.47)</td>
<td>2.50 (2.64)</td>
<td>2.59 (2.34)</td>
<td>2.91 (2.64)</td>
<td>0.28</td>
<td>1, 62</td>
</tr>
<tr>
<td>Fatigued–Inertia</td>
<td>5.06 (4.79)</td>
<td>4.78 (4.25)</td>
<td>4.94 (3.94)</td>
<td>4.50 (3.91)</td>
<td>0.08</td>
<td>1, 62</td>
</tr>
</tbody>
</table>

**Note.** Standard deviations are reported in parentheses. Significant difference from Multimorph T1 ** = p < .01 *** = p < .001
Table 7.7.
Mean heart rate, skin conductance level, and high-frequency heart rate variability by stressor group

<table>
<thead>
<tr>
<th></th>
<th>Multimorph (T1)</th>
<th>Multimorph (T2)</th>
<th>F (time)</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Block 1</td>
<td>Block 2</td>
<td>Block 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>72.61 (8.73)</td>
<td>72.13 (8.88)</td>
<td>72.99 (9.40)</td>
<td>73.06 (8.63)</td>
<td>2.30</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.95 (2.00)</td>
<td>3.42 (2.09)***</td>
<td>3.30 (2.25)**</td>
<td>3.36 (2.32)**</td>
<td>8.78</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.38 (0.80)</td>
<td>7.50 (0.78)*</td>
<td>7.34 (0.79)</td>
<td>7.45 (0.81)</td>
<td>3.21</td>
</tr>
<tr>
<td>Speech Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>71.54 (8.55)</td>
<td>71.29 (8.57)</td>
<td>71.61 (8.70)</td>
<td>72.20 (8.34)</td>
<td>1.19</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>3.06 (1.73)</td>
<td>4.09 (1.62)***</td>
<td>4.01 (1.71)***</td>
<td>4.11 (1.66)***</td>
<td>47.14</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.35 (0.92)</td>
<td>7.66 (0.83)***</td>
<td>7.60 (0.83)***</td>
<td>7.68 (0.73)***</td>
<td>15.26</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from Multimorph T1 * = p < .05 ** = p < .01 *** = p < .001
To investigate whether the changes in ANS function during the stressor manipulation period were related to changes in self-reported emotion, correlations were carried out between the physiological reactivity scores and the POMS-SF reactivity scores. Significant correlations revealed that greater HR reactivity was associated with larger decreases in the POMS-SF fatigue–inertia subscale ($r = -.26, p = .039$), whilst greater SCL reactivity was associated with larger increases in the POMS-SF vigour–activity subscale ($r = .28, p = .029$). Taken together, the directions of these relationships suggest that larger increases in SNS activation were associated with lower levels of tiredness (i.e., less fatigue coupled with more vigour).

Mixed-factorial ANOVAs were carried out on the blocks of the Multimorph task to establish if arousal was maintained during the secondary task (see table 7.7). Although HR did not show any significant effects involving group, Time x Group interactions were found for both SCL, $F(1.72, 106.33) = 8.73, p = .001$, and HF-HRV, $F(3, 183) = 5.48, p = .001$. Both groups demonstrated increases in SCL at T2, but this increase was greater in the speech group. Contrary to expectation, the speech group also demonstrated a larger increase in HF-HRV during T2 compared to T1 (all blocks significant at $p < .001$), although $t$-tests revealed that HF-HRV at T2 was not significantly different from baseline.

### 7.9.6. Effects of the Repetitive Stressor Manipulations on Emotional Sensitivity and Accuracy

Mixed-factorial ANOVAs (repeated measures: Time, Emotion; between subjects factor: Group) were carried out to assess the impact of the repetitive stressor manipulations on performance during the Multimorph (T1 compared to T2). Emotional Sensitivity was significantly influenced by main effects of Time, $F(1,60) = 12.32, p = .001$, and Emotion, $F(5, 300) = 75.43, p < .001$. In addition to this, a significant two-way Time x Emotion interaction was revealed, $F(5, 300) = 5.44, p < .001$. As in Experiment 1, happiness was the quickest emotion to be identified, followed by surprise, disgust, anger, sadness, and fear. During the second administration of the Multimorph both stressor manipulation groups were significantly more sensitive to fear ($M = 2.0$ stages quicker), sadness ($M = 3.5$ stages quicker), and disgust ($M = 3.5$ stages quicker), as shown in figure 7.6. No significant main effects or interaction effects were found involving Group.
CHAPTER 7: EMOTIONAL SENSITIVITY

Figure 7.6. Mean number of stages taken to correctly identify each emotion across the two administrations of the Multimorph Task. Error bars represent the standard error.

A mixed-factorial ANCOVA (with POMS-SF baseline total as a covariate) revealed that Emotional Accuracy was also significantly influenced by Emotion, $F(4.26, 242.68) = 24.07, p < .001$. A main effect for the POMS-SF baseline total was also found, $F(1, 57) = 4.40, p = .040$. These main effects were superseded by a Time x POMS-SF baseline total two-way interaction, $F(1.00, 57.00) = 4.48, p = .039$, and a Time x Emotion x POMS-SF baseline total three-way interaction, $F(4.51, 257.00) = 4.35, p = .001$. As for Emotional Sensitivity, participants were better at recognising sadness and disgust expressions at T2 (increases in accuracy of 6.5% and 7.0% respectively, as shown in figure 7.7), however only participants reporting higher distress on the POMS-SF at baseline improved at recognising fear expressions: a median split based on the POMS-SF baseline score revealed that participants with higher POMS-SF baseline totals demonstrated a 5% increase in accurately identifying anger at T2 compared to a 4% decrease for individuals with lower POMS-SF baseline totals. No significant effects were revealed involving Group.
CHAPTER 7: EMOTIONAL SENSITIVITY

7.9.7. Effects of the Repetitive Stressor Manipulations on Facial Mimicry

Finally, a 2 x 3 x 6 x 2 mixed-factorial ANOVA (repeated measures: Time, Muscle, Emotion; between subjects factor: Group) was carried out to investigate the effects of the repetitive stressor manipulations on facial mimicry. A significant main effect was found for Emotion, $F(5, 310) = 2.47, p = .033$, and significant two-way interactions were found for Emotion x Muscle, $F(8.14, 504.49) = 13.38, p < .001$, Time x Emotion, $F(5, 310) = 3.14, p = .009$, and Time x Muscle, $F(2, 214) = 6.00, p = .003$. Consistent with the EMG findings at T1, overall corrugator activity was higher for negative emotions and zygomaticus activity was higher for positive emotions (as in figure 7.5). Participants tended to exhibit less corrugator activity at T2, particularly for fear, sadness, and disgust. The decrease in corrugator activity for these emotions could be due to practice effects: Increased accuracy for these emotions suggests that less effort may have been needed to identify these emotions at T2. Reductions in effort can be indexed by corrugator activity as people tend to crease their forehead when confused or concentrating (Rozin & Cohen, 2003). As in Experiment 1 there were no significant effects involving Group.

7.10. Discussion

The aim of Experiment 2 was to replicate the design of Experiment 1, but with a further adaptation to the stressor manipulation. The modification made to the speech
task preparation period was more successful in maintaining increases in SCL arousal during the Multimorph task at T2, but again no significant differences were found between the stressor groups in terms of emotional sensitivity, emotional accuracy, or facial mimicry at T2.

7.10.1. Baseline Correlates of Emotional Sensitivity and Accuracy

In the current experiment, emotional sensitivity was not related to any of the baseline measures. This is in contrast to Experiment 1, which found that Emotional Sensitivity could be predicted by sex and the non-acceptance scale of the DERS. This discrepancy may be due to the smaller sample size in the this experiment, or could be a function of higher depression symptoms in the sample from Experiment 2 ($M = 9.52$, $SD = 8.30$ vs. Experiment 1 sample $M = 6.51$, $SD = 5.81$), $t(109.05) = -2.46$, $p = .015$. The former is a more likely explanation as there were no significant differences between the samples in Emotional Sensitivity at baseline, and the BDI-II was not a significant predictor of performance on the Multimorph.

Current distress (as indexed by the POMS-SF baseline total) was negatively with decreased Emotional Accuracy at T1 in the current experiment. This finding is in line with previously reported findings (e.g., Csukly et al., 2008, reported an inverse relationship between emotional accuracy and the Symptom Checklist-90). To add to this, the current findings suggest that emotional sensitivity is related to emotional accuracy, with individuals who are quicker at identifying emotions being more likely to correctly label them at full expression (although in Experiment 2 this relationship failed to reach significance, $p = .069$).

The EMG findings in this experiment complement the results from Experiment 1. Corrugator activity was associated with viewing negative facial displays, whilst zygomaticus and levator activation were indicative of happy and disgust stimuli respectively. The observed patterns of EMG activation for each emotion category is further confirmation that viewing facial displays results in facial mimicry (Dimberg, 1982; Hatfield et al., 1993). A notable finding in Experiment 2 was the relationship found between the levator labii amplitude and Emotional Sensitivity for the disgust stimuli. This relationship suggests that facial mimicry is associated with the recognition of emotion in others (in line with Stel & van Knippenberg, 2008; Stel & van den Bos,
2010), although in the current research this effect was confined to the recognition of disgust. As no impairments were identified in facial mimicry at T2, the relationship between facial mimicry and emotional sensitivity is still unresolved.

### 7.10.2. Effectiveness of the Repetitive Stressor Manipulation

The altered presentation of the stressor manipulation caused notable changes in the arousal levels of the stressor groups. As in Experiment 1, during the preparation period the speech group demonstrated larger increases in HR and SCL than the reading group, coupled with a decrease in HF-HRV. However in the current experiment, the speech group also maintained a larger increase in SCL across the blocks of the Multimorph task compared to the reading group. The increased SCL in the speech group was also accompanied by a significant increase in HF-HRV. Increased HF-HRV indicates higher levels of self-regulatory effort (Segerstrom & Solberg Nes, 2007), which may suggest that individuals in the speech group were working harder to self-regulate during the Multimorph task.

### 7.10.3. Effects of Defensive Arousal on Emotional Sensitivity and Accuracy

No significant differences were found between the groups in terms of emotional sensitivity, emotional accuracy, or facial mimicry at T2. As in the previous experiment, both groups demonstrated an increase in Emotional Sensitivity, particularly for fear, sadness, and disgust expressions. The absence of disparate findings between the stressor groups challenges the hypothesis that increased SNS activation after a stressor should be associated with a decrease in these competencies. In the current experiment the revision of the stressor manipulation task meant that the speech group continued to exhibit increases in arousal during the Multimorph task (as indexed by SCL). The ability to now discriminate between the stressor groups on the basis of their physiological arousal suggests that the current lack of findings may not be due to the stressor manipulation being ineffective. Instead, questions may begin to be raised about the validity of polyvagal theory.
7.10.4. Limitations

A limitation of the current experiment is the small sample size of the groups, which may have limited the ability to identify individual differences in emotional sensitivity and emotional accuracy at baseline. Further to this, the sample in Experiment 2 was higher in self-reported depression symptoms (BDI-II scores). This occurrence may be due to a sampling bias, with the second experiment recruiting participants later in the academic term. A methodological limitation of the current experiments is that they only focus on one form of emotional sensitivity: the ability to recognise emotions from facial expressions. Emotions can be inferred from other channels of communication, such as vocal expressions or gestures (Scherer, 1995; Wallbott, 1998). Further research is needed to explore the effects of defensive physiological arousal on emotional sensitivity in these channels, as the abilities needed to decode these signals may also be vulnerable to activation of the SNS.

In Experiment 1 it was argued that a strength of the current studies was the use of healthy populations, however the use of this sample may be obscuring the effects of defensive physiological arousal on socio-emotional responding. Analogue populations are useful because they often demonstrate distinctive responses during experimental tasks (e.g., increased reactivity to stimuli), and these responses are known to be diluted when such samples include individuals with only mild characteristics of the target population (Borkovec & Rachman, 1979). As the current research is specifically using samples without self-reported deficits in emotion regulation and psychopathology, this may be reducing the impact of the stressor manipulation. The ability of the samples to self-regulate when under challenge/threat is clear; for example in the second experiment the stressor group exhibited a larger increase in HF-HRV during the second administration of the Multimorph task suggesting that they were engaging self-regulatory effort (Segerstrom & Solberg Nes, 2007). Successful self-regulation in the samples, resulting in increased PNS activation, may be preventing the stressor manipulations from influencing the secondary tasks; perhaps it is only when self-regulation is unsuccessful (i.e., in clinical and sub-clinical populations) that deficits are likely to appear.
7.10.5. Conclusion

The findings of the current study were consistent with the findings from Chapter 6: the results did not support the hypothesis that defensive physiological arousal is associated with reduced facial expressivity. Participants in the speech group did not exhibit less facial mimicry after the stressor manipulation period, and in turn there were no differences between the stressor groups in emotional sensitivity or emotional accuracy at T2. Thus far the findings do not provide sufficient empirical support for the hypotheses stemming from polyvagal theory (Porges, 1995, 2001, 2003a). In the current study, the only finding that may provide limited indirect support for polyvagal theory is the association found between the levator labii activation and the speed at which participants recognised disgust. The direction of the relationship suggests that emotional sensitivity for this emotion may be contingent on afferent feedback from the face. Consequently, if SNS activation does decrease facial expressivity, this would suggest that emotional sensitivity for disgust (if not other emotions) may also be impaired as a result of SNS activation.

In conclusion, the use of a stressor manipulation has not been associated with deficits in facial expressivity or emotional sensitivity. It is possible that defensive physiological arousal does influence socio-emotional responding, but not via the mechanisms already examined in the current collection of studies. The final empirical chapter aims to investigate the effects of defensive physiological arousal on affiliation tendencies.
Chapter 8: Expressive Regulation and Willingness to Affiliate

Chapter 6 demonstrated the effects of regulating facial expressions on one’s own physiological state. Enhancing emotional expressions was associated with increases in heart rate and sweat response, whilst suppressing emotional expressions was associated with increased sweat response and high-frequency heart rate variability, but decreased heart rate (see also Demaree, Schmeichel, Robinson, & Everhart, 2004; Gross & Levenson, 1993, 1997). Studies researching the psychophysiological effects of expressive regulation often only focus on negative emotions (e.g., Demaree, Schmeichel, et al., 2006; Gross & Levenson, 1993), and tend to compare expressive suppression with antecedent-focused techniques such as reappraisal, as opposed to other response-focused techniques such as expressive exaggeration (for example Gross & Levenson, 1997, but see Demaree, Schmeichel, et al., 2004; and Jackson, Malmstadt, Larson, & Davidson, 2000). The focus also tends to be on the responses of the regulators who are modulating their emotions (i.e., the senders), rather than the observers who are decoding the expressions of emotion.

In the current chapter two experiments are described. The first experiment investigated how expressive regulation affects observers in terms of their psychophysiology and their willingness to spend time with others. The second experiment then tested the hypothesis that activation of the sympathetic nervous system (SNS) is associated with a decreased willingness to spend time with others.

8.1. Experiment 1: The Psychophysiological Effects of Observing Expressive Regulation

Previous research has shown that regulating facial expressions has effects on both observers and regulators. Butler et al. (2003) found that individuals who were instructed to suppress their facial expressions were rated by their naive counterparts as being less friendly, and the counterparts were less willing to spend time with the regulators in the future. Not only this, but observers had notable increases in blood pressure. Experiment 1 was designed to investigate the effects of expressive regulation on observers’ physiological states and their willingness to affiliate with regulators. The experiment used a within-subjects design to establish whether observing individuals either suppressing or exaggerating their facial expressions would cause different
physiological responses (indexed by heart rate and sweat response). As well as recording physiological responses, subjective ratings were also collected to establish if expressive regulation would affect the amount of time observers would be willing to spend with the regulators.

It was hypothesised that observers would be more willing to spend time with individuals showing more emotion, and that this effect would be particularly robust with regulators displaying positive affect. Positive emotions are theorised to facilitate pleasurable interactions (Harker & Keltner, 2001), and have been associated with feelings of social connectedness (Mauss et al., 2011), trustworthiness (Oosterhof & Todorov, 2009), and co-operation (Boone & Buck, 2003). Interestingly co-operation is associated with general expressivity, and is not confined to positive facial displays (Schug, Matsumoto, Horita, Yamagishi, & Bonnet, 2010). Evidence suggests that general expressivity is associated with engendering feelings of rapport and likability (Bernieri, Gillis, Davis, & Grahe, 1996; Riggio & Friedman, 1986). This is because in certain situations using negative facial expressions to signal aversion can be pro-social, for example expressing disgust in response to unfairness (Chapman, Kim, Susskind, & Anderson, 2009) or expressing embarrassment in response to violations of social convention (Feinberg, Willer, & Keltner, 2011). Consequently facial expressions of both positive and negative emotion can be adaptive; this means that overt emotional displays in general should increase others’ willingness to affiliate.

In addition to affecting affiliation, it was expected that viewing regulators modulating their facial expressions would also influence observers’ physiological responses. Viewing affective stimuli is known to elicit changes in physiology, with valence and arousal both able to effect changes in heart rate and sweat response (Lang, Greenwald, Bradley, & Hamm, 1993). Viewing positive and negative facial displays has also been shown to elicit consistent changes in heart rate and sweat response (Dimberg, 1982). Interestingly little research has been carried out to evaluate the effects of expressive regulation on observers. Butler et al. (2003) reported that expressive suppression resulted in higher SNS activation in observers (as indexed by blood pressure); however this experiment involved real-time interactions. To date, the effect of expressive regulation on the physiology of passive observers’ has not been reported.
8.2. Methodology

8.2.1. Participants

Forty-two undergraduate psychology students (10 males, 32 females) volunteered to participate in the study and were awarded course credits as part of their undergraduate course requirements. Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders, and current psychological or pharmacological treatment. The participants ranged in age from 18–24 with a mean age of 19.74 years ($SD = 1.50$). 90.5% of these participants identified themselves as Caucasian, 7.1% as Mixed, and 2.4% as Other. Due to movement artefacts two participants’ data were excluded from the heart rate analyses.

8.2.2. Behavioural Measure of Affiliation

8.2.2.1. Rating Faces Task. The Rating Faces Task involved participants viewing video clips, which showed individuals’ facial reactions to emotive pictures. The video stimuli were obtained from the experiment described in Chapter 6, where participants viewed emotive pictures from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999) whilst regulating their facial expressions (enhance, suppress, or maintain). All of the participants filmed gave written consent for their videos to be stored and used in future research, which resulted in a video library of people voluntarily controlling their facial expressions.

For this experiment, thirty-six videos were edited into 10-second clips of individuals enhancing, suppressing, or maintaining their facial expressions. The videos were presented using e-Prime (Version 2.0; Psychology Software Tools, Pittsburgh, PA). Participants in the current experiment were shown the IAPS picture seen by the regulator for 4 seconds, followed by the video clip of the regulator’s corresponding facial expression, and were asked to complete four Likert scales for each clip (as shown in figure 8.1): one rating the individual’s attractiveness (attractiveness: $1$ not at all attractive to $7$ very attractive), one rating the individual’s familiarity (familiarity: $1$ not at all familiar to $7$ very familiar), one rating the individual’s distinctiveness (distinctiveness: $1$ not at all distinctive to $7$ very distinctive), and one rating how much
time they would like to spend with the individual (affiliation: 1 *not much time* to 7 *a lot of time*). The current participants were not informed that the individuals in the videos were following instructions to control their facial expressions. At the end of the video clips, the participants were shown stills from the videos and were be asked to complete a further Likert scale rating the level of facial expression shown by the individual (expressivity: 1 *too little* to 7 *too much*). At the end of the experiment participants were asked to identify if they knew any of the individuals rated in the task.

![Image]

**Figure 8.1.** Example time rating from the Rating Faces Task.

### 8.2.3. Procedure

Participants attended a single testing session in an air-conditioned, sound attenuated room. After obtaining written consent participants completed the demographic screening questionnaire (see section 3.5.1). Participants then completed questionnaires to assess their current mood state (Profile of Mood States – Short Form, POMS-SF; Shacham, 1983, see section 3.5.1) and how safe they feel in their social relationships (Social Safeness and Pleasure Scale, SSPS; Gilbert et al., 2009, see section 3.5.9). Electrodes for recording heart rate (HR) and skin conductance level (SCL) were applied following standard procedures (see section 3.4.4) and a five-minute baseline recording was carried out during which the participants were asked to sit quietly. Recording HR allowed for the calculation of heart rate variability (HRV, see section
3.4.3). The participants then completed the Rating Faces Task, which involved viewing short videos and completing several rating scales. Once the Rating Faces Task was completed the session was terminated and the participant debriefed. A flowchart diagram of the rating faces task procedure can be found in appendix 19.

8.3. Results

8.3.1. Statistical Analyses

In total, 1512 sets of ratings were completed: 30 (1.98%) of the regulators in the videos were marked as acquaintances of the observers and this data was subsequently removed from the analyses, resulting in 1482 ratings in the final data set. Three participants were unable to complete the final expressivity ratings due to technical problems, resulting in 1378 ratings for this measure.

For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. Mean HR, SCL, and high-frequency heart rate variability (HF-HRV) values were calculated for the baseline period. Mean HR and SCL were also calculated for each 10-second video. To evaluate the influence of possible covariates in the planned analyses, bivariate correlations were computed among the dependent variables and possible covariates (age, sex, social safeness [SSPS], and mood state [POMS-SF]). Bivariate correlations revealed that the affiliation ratings (i.e., the amount of time observers would be willing to spend with regulators) were significantly correlated with the SSPS ($r = .08$, $p = .001$) and baseline HF-HRV ($r = .16$, $p < .001$). Individuals who reported higher levels of social safeness and higher levels of HF-HRV, a marker of parasympathetic nervous system (PNS) function, were more willing to spend time with the regulators. The SSPS scores and baseline HF-HRV were entered into the subsequent affiliation analyses as covariates.

The main analyses in this experiment were carried out to evaluate the psychophysiological effects of expressive regulation on observers. Separate analyses of variance (ANOVAs) were carried out on the dependent variables (expressivity ratings, physiological indices, and affiliation ratings), with the factors Condition (enhance,
maintain, suppress) and Valence (positive, negative). Significant main effects for all analyses were followed up with pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05.

### 8.3.2. Manipulation Check

Analyses of the expressivity ratings supported the manipulation of the expressive regulation strategies. A repeated-measures ANOVA revealed a significant effect of Condition (enhancement, suppression, maintain) on rated expressivity, $F(1.37, 51.85) = 246.95, p < .001$. As expected, Bonferroni pairwise comparisons indicated that the expressivity ratings were significantly greater in the enhancement condition ($M = 4.72, SD = 0.58$) than the maintain condition ($M = 3.25, SD = 0.55$), and were significantly lower in the suppress condition ($M = 2.63, SD = 0.66$) than the maintain condition (all significant at $p < .001$).

### 8.3.3. The Social Consequences of Expressive Regulation

To investigate the social consequences of expressive regulation, the affiliation ratings were evaluated. A univariate analysis of covariance (ANCOVA), with SSPS total scores and baseline HF-HRV as covariates, revealed that the time observers were willing to spend with regulators varied as a function of Condition, $F(2, 1474) = 99.37, p < .001$, Valence, $F(1, 1474) = 6.54, p = .011$, SSPS Total, $F(1, 1474) = 14.17, p < .001$, and baseline HF-HRV, $F(1, 1474) = 46.34, p < .001$. There was also a significant Condition x Valence interaction, $F(2, 1474) = 19.31, p < .001$. As shown in figure 8.2, observers were most willing to spend time with individuals in the enhance condition, followed by the maintain and suppress conditions (all differences significant at $p < .001$). Valence influenced the affiliation ratings, but the effect was only significant in the enhance condition, with observers being significantly more willing to spend time with individuals showing more positive emotion than individuals showing more negative emotion, $t(439) = 5.89, p < .001$. To investigate whether individual characteristics were driving these effects, bivariate correlation analyses were carried out using the main ratings data. The analyses revealed that the affiliation ratings were positively correlated with the ratings for attractiveness ($r = .61, p < .001$), familiarity ($r = .32, p < .001$), and distinctiveness ($r = .34, p < .001$). When the attractiveness,
familiarity, and distinctiveness ratings were included in the original ANCOVA all of the effects involving Condition and Valence remained significant ($p < .001$).

**Figure 8.2.** Mean willingness to spend time with regulators by Condition and Valence. Error bars represent the standard error. Covariates were controlled at the following values: Attractiveness = 3.54, Distinctiveness = 3.45, Familiarity = 2.38, SPSS total = 35.58, HF-HRV = 7.36.

### 8.3.4. The Effects on Expressive Regulation on Observers’ Physiology

Repeated-measures ANOVAs on the physiological indices with Condition and Valence as repeated factors revealed that expressive regulation did influence observers’ physiology. For HR a significant main effect of Condition was revealed, $F(2, 78) = 11.70$, $p < .001$; HR was lowest when viewing enhanced expressions and highest when viewing suppressed expressions, as shown in figure 8.3. Bonferroni pairwise comparisons confirmed that HR during the suppress condition was significantly higher than HR during the maintain and enhance conditions ($p < .05$). There were no significant effects of Condition or Valence on SCL.
Figure 8.3. Mean heart rate by Condition. Error bars represent the standard error.

8.4. Discussion

The results of Experiment 1 replicated the findings of previous research. In conjunction with the findings from Chapter 6, the current findings strengthen the evidence base that suggests that expressive regulation has personal and social consequences (Butler et al., 2003). Taken together, the findings from Chapter 6 and the current experiment indicate that both regulators and observers are affected by expressive regulation. Enhancing emotional expressions was shown to result in increased HR and SCL in regulators (Chapter 6; see also Demaree, Schmeichel, et al., 2006, Demaree, Schmeichel, et al., 2004). The increase in sympathetic functioning in regulators could be due to mobilisation effects, with the muscles of the face requiring more energy when facial expressions are up-regulated (Gross & Levenson, 1993).

In contrast to the effects of enhancement, regulators instructed to suppress emotional expressions are found to exhibit decreases in HR coupled with increased SCL and HF-HRV (Chapter 6). Decreases in HR are thought to occur during suppression because of reduced bodily movement and energy expenditure (Gross & Levenson, 1993), whilst increases in SCL and HF-HRV are indicative of increased self-regulatory effort (Butler, Wilhelm, & Gross, 2006; Gross, 1998b; Segerstrom & Solberg Nes, 2007). In observers, viewing individuals suppressing emotional expressions was associated with increased HR (this study). It is proposed that the increase in HR occurs because reduced facial expressivity may act as a danger signal. Facial displays are important signals of safety and danger that help to regulate social interactions (McHugo & Smith, 1996; Orr & Lanzetta, 1980). Whilst greater expression of emotion signals
safety, the absence of facial expressions may signal danger or deception, resulting in decreased likability and a reduction in the observers’ willingness to interact with the person signalling (see Riggio & Friedman, 1986). This association may explain why observers were less willing to affiliate with regulators following suppression instructions.

It is possible that the observers’ affiliation ratings were influenced by peripheral feedback from the ANS; that is, increased SNS activation in response to regulators suppressing their facial displays may have affected the observers’ willingness to spend time with the regulators. On the other hand, the positive social outcomes associated with emotional expression, such as increased social connectedness and trustworthiness (Boone & Buck, 2003; Mauss et al., 2011) are also plausible explanations as to why regulators enhancing their emotions were rated more favourably by observers than regulators in the maintain and suppress conditions.

An interesting finding of the current experiment was that affiliation ratings were positively associated with social safeness and baseline HF-HRV. The Social Safeness and Pleasure Scale indexes feelings of belonging, acceptance, and feelings of warmth from others (Gilbert et al., 2009), whilst HF-HRV is a marker of PNS function (Cacioppo, Uchino, & Berntson, 1994). Thayer and Lane (2000) have previously argued that individuals with low HRV are less able to experience ‘safety’ when it is present, suggesting that HF-HRV may in fact be a biological analogue of social safeness. The influence of these measures on one’s willingness to spend time with others provides some support for the emotional and behavioural components of polyvagal theory: Porges (2001, 2003a) claims that individuals are more likely to exhibit social engagement behaviours when they feel safe, which is contingent of the activation of the PNS. This suggests that withdrawal of the PNS during a stressor may be associated with reductions in affiliation tendencies.

8.4.1. Limitations

There are several limitations to Experiment 1. First of all, the experiment was not based on real-time interactions. It is possible that the physiological responses of the observers in the current study would be different if they were actively involved in an interaction with the regulators (similar arguments arise in studies on mimicry, for
example Blairy, Herrera, & Hess, 1999). More information is conveyed in face-to-face interactions, so it is interesting that the current experiment revealed such a strong effect of expressive regulation on affiliation ratings from visual presentations alone. A second limitation is that the current results suggest that higher levels of facial expressivity should result in a higher willingness to affiliate in observers: I would argue that this is not always the case.

Although open expression of emotion can signal pro-social intentions as well as safety signals, expressions are mediated by context (Bonanno et al., 2007). In the current experiment regulators were shown to produce expressions that were congruent with the valence of the stimuli presented. If the experiment had manipulated the congruence of the IAPS stimuli and the regulators reactions (i.e., falsely pairing negative stimuli with positive expressions and vice versa) this probably would have been seen as a violation of social norms, and observers would have been less willing to affiliate with individuals displaying inappropriate affect (Cole, Michel, & Teti, 1994; Keltner & Kring, 1998). Further to this, regulators were instructed to enhance their expressions and make it obvious what they were feeling; they were not instructed to exaggerate their responses. As the participants from Chapter 6 were expecting others to view them, they are likely to have conformed to display rules during the expressive regulation task (Ekman & Friesen, 1969). Future research should also consider the effect of exaggerated facial expressions on others’ willingness to spend time with regulators. Future findings may suggest that the relationship between expressivity and affiliation is in fact curvilinear, with too much or too little emotion being detrimental.

8.4.2. Conclusion

The current study was designed to investigate the effects of expressive regulation on observers. The current results replicated previous findings, and extended the literature by demonstrating that positive and negative emotional enhancement have disparate effects on observers’ willingness to affiliate with regulators. The results provided some support for polyvagal theory (Porges, 1995, 2001, 2003a), as decreased facial expressivity was associated with negative social consequences, and affiliation was influenced by perceived feelings of social safety and cardiac vagal tone (as indexed by HF-HRV). However there still remains the question as to whether or not there is a link between defensive physiological arousal and deficits in social functioning. Experiment
2 was therefore designed to evaluate whether defensive physiological arousal is associated with changes in affiliation.

8.5. Experiment 2: Defensive Physiological Arousal and Willingness to Affiliate

Experiment 1 confirmed that expressive regulation plays an important role in social interactions. Observers were less willing to spend time with individuals who suppressed emotional expressions. Conversely, individuals who expressed emotion were rated more favourably, particularly if they exhibited positive emotional expressions. The previous experiment revealed a significant relationship between the affiliation ratings and activation of the PNS (HF-HRV at baseline). Consequently it is proposed that one’s willingness to affiliate with others in contingent on physiological state. The current experiment was carried out to investigate the effects of social evaluative threat on individuals’ willingness to spend time with others. If polyvagal theory is valid, defensive physiological arousal should be associated with decreased affiliation tendencies, as individuals feel less safe (Porges, 1998, 2003a). Polyvagal theory suggests that during defensive physiological states individuals should be prioritising behaviours that promote survival not socialisation (Porges, 2007a). Interestingly, this argument is at odds with the tend-and-befriend literature. I will first give an overview of the tend-and-befriend literature before introducing the main hypothesis of the current experiment.

8.5.1. The Physiological Substrates of Affiliation

To recapitulate, polyvagal theory suggests that social engagement behaviours are an emergent property of the ventral vagal complex (VVC; i.e., activation of the PNS). In calm and self-soothing states we are able to explore our social environments and socially engage with others (Porges, 2001, 2003b). Without activation of the VVC, as in times of threat, there is a retraction of the neural regulation of the striated muscles of the face and head. Thus in times of stress we are supposedly less able to modulate our facial activity, which leads to the suppression of emotional facial expressions. Our ability to communicate and socially interact with others is impaired reducing the success of social interactions. Although polyvagal theory’s argument it logical, it is well documented that in times of threat people can often seek the presence of others, and that stress can
increase affiliation tendencies (Gump & Kulik, 1997; Schachter, 1959; Taylor et al., 2000). Thus polyvagal theory appears deficient.

The absence of VVC activation in the polyvagal framework does not mean that affiliation seeking behaviours cannot occur. Porges (2003a) does suggest that social behaviours can be sought to regulate physiological activation. Usually social engagement behaviours are considered an emergent property of the VVC, but it is possible that during stress socially-oriented behaviours become consciously mediated (this parallels the argument put forward in Chapter 6 regarding the voluntary control of facial expressions during stress; see section 6.2.3). In threat or challenge situations we may use our facial muscles to try to engage others and find safety cues to reactivate the VVC. The advantage of this strategy is that using facial muscles automatically activates the neural pathways of the social engagement system and provides afferent feedback to the VVC even in the absence of reinforcement, which may act as a further safety signal. Despite this, there is still the assumption made by polyvagal theory that threats activate a mobilising fight-flight response or an immobilising freeze response, neither of which promotes affiliative behaviours (Porges, 2007a).

Although attachment is possible within the polyvagal framework (Porges, 1998), this is usually confined to calm and self-soothing states. Attachment behaviours are generally not well-supported by the physiological or behavioural responses determined by the physiological systems that activated during times of challenge or threat (e.g., the SNS). In order for attachment and social engagement behaviours to occur the VVC must be up-regulated, meaning that in stressful environments if social engagement is to successfully occur threats need to be eliminated or reappraised as non-threatening. From a polyvagal perspective positive social interactions can occur and act as safety signals (Porges, 2003a), but social behaviours are not an emergent property of defensive behavioural states.

### 8.5.2. Tend-and-Befriend

In contrast to polyvagal theory, Taylor and colleagues (Taylor, 2006; Taylor et al., 2000; Taylor et al., 2008) advocate that stressful situations do not automatically result in the physiological or behavioural responses of fight or flight. Instead, Taylor et al. (2000) argue that in certain situations fight-flight behaviours are not always the most
adaptive responses to stress, for example if a female caring for her offspring is threatened, fight–flight behaviours may jeopardize their safety. Taylor et al. propose an alternative; that stress can prompt “tend-and-befriend” behaviours. These behaviours during periods of threat help to promote safety and reduce distress, as well as create and maintain social networks.

The ability to befriend others during times of stress has survival-related advantages. Schachter (1959) believed that increased affiliation during stress has two important purposes: it provides a chance for social comparison, as well as a source of mutual comfort and support. Schachter (1959) hypothesised that affiliation tendencies would be strongest during stress when being with others was likely to help eliminate threatening aspects of the stress, or increase one’s ability to cope with the stress. Taylor et al. (2000) propose that the biobehavioural mechanism underlying tend-and-befriend behaviours builds on attachment–care-giving processes. It is suggested that in situations of stress oxytocin and endogenous opioid mechanisms can be activated which down-regulate sympathetic and neuroendocrine stress responses. In experimental studies oxytocin has been found to enhance parasympathetic-vagal activity and decrease sympathetic activity, patterns of activation that are antithetical to the fight-or-flight response (Uvnäs-Moberg, 1998). Oxytocin is also known to help co-ordinate the causes and effects of positive social interactions (Uvnäs-Moberg, 1998). These findings help to explain why oxytocin can increase positive social behaviours, including social bonds, and how in turn these can reduce HPA activity (Carter, 1998; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003).

Tend-and-befriend behaviours are not incompatible with polyvagal theory; the reason they are neglected from the polyvagal framework is because they are mostly driven by the neuroendocrine system rather than the ANS (a caveat of polyvagal theory is that it concentrates on neurophysiological mechanisms, and tends to exclude the role of other homeostatic systems such as the neuroendocrine and immune systems). Theoretical isolation of the autonomic regulatory systems minimises the significant interrelationships between autonomic, neuroendocrine, and immune responses, and limits the range of physiological states and behaviours that can occur during periods of threat (Cacioppo et al., 1995). In polyvagal theory it is accepted that oxytocin and opioids inhibit SNS activation, which is why socialisation and attachment are safety behaviours that initiate and maintain activation of the VVC (Porges, 1998). However,
polyvagal theory assumes that threats automatically activate protective defensive behaviors driven by the ANS, rather than initiate the up-regulation of attachment and care-giving processes driven by the neuroendocrine system. Focusing on the flexibility of the ANS alone fails to appreciate the true range of physiological and behavioral repertoires that are available during times of threat. Instead we need to appreciate the dynamic interplay between the body’s systems that allows the promotion of fight–flight behaviors, immobilization behaviors, but also attachment and affiliation behaviors.

8.5.3. Current Aims and Hypotheses

The aim of Experiment 2 was to investigate the effects of defensive physiological arousal on individuals’ willingness to spend time with others. There is some evidence to suggest that stress may increase affiliation tendencies (Gump & Kulik, 1997; Taylor et al., 2000; Taylor, 2006), although as Park and Maner (2009) have pointed out, social support during threat can have its own risks (e.g., social rejection), which may prevent affiliative motivation from leading to strategic affiliation behaviours. A recent study carried out by von Dawans, Fischbacher, Kirschbaum, Fehr, and Heinrichs (2012) found that engaging participants in a laboratory stressor increased pro-social behaviours, namely trust and sharing, however these behaviours do not directly measure affiliation tendencies. In line with polyvagal theory, the following hypothesis was proposed:

Hypothesis 5. Increased activation of the SNS in response to a laboratory stressor will be associated with decreased affiliation tendencies.

To test this hypothesis the current experiment used a between subjects design. Participants completed the Rating Faces Task from Experiment 1, but similar to the previous studies, midway through the task participants were assigned to a stressor manipulation: half of the participants were instructed to prepare a speech (n = 35) and the other half were randomised to a reading task (n = 37). The stressor manipulation was carried out in between blocks of the Rating Faces Task (similar to Chapter 7, Experiment 2). It was hypothesised that after the stressor manipulation participants in the speech group would be less willing to spend with the regulators in the videos.
8.6. Method

8.6.1. Participants

Seventy-two undergraduate psychology students (15 males, 57 females) volunteered to participate in the study and were awarded course credits as part of their undergraduate course requirements. Exclusion criteria were assessed using self-report questionnaires and included current or past diagnoses of Axis I or II psychiatric disorders, and current psychological or pharmacological treatment. The participants ranged in age from 18–42 with a mean age of 20.11 years ($SD = 3.94$). 88.9% of these participants identified themselves as Caucasian, 6.9% as Mixed, and 4.2% as Asian. The participants were randomised into two groups during the experiment: a speech group ($n = 35$: 5 males, 32 females; mean age = 19.83, $SD = 2.84$) and a reading group ($n = 37$: 10 males, 25 females; mean age = 20.83, $SD = 4.78$).

8.6.2. Procedure

Experiment 2 followed a similar procedure to Experiment 1. The main differences between the two experiments were that trait measures of depression (BDI-II; Beck et al., 1996, see section 3.5.4) and anxiety GAD-7; Spitzer et al., see section 3.5.6) were added in Experiment 2, and the Rating Faces Task was altered. Two main changes were made to the Rating Faces Task. Firstly, the wording of the affiliation rating was changed so that participants were asked “how much time would you like to spend with this person right now” (1 not much time to 7 a lot of time). The “right now” wording was added to emphasise the state nature of this rating. Secondly, the e-Prime task was reprogrammed so that half of the video stimuli were randomly presented at baseline (T1: 18 stimuli in total; counterbalanced for instruction and valence). The remaining stimuli were subsequently presented in three blocks (T2: 6 stimuli per block); these blocks were counterbalanced with three one-minute blocks of the stressor manipulation (participants were assigned to either the speech task [see section 4.6.2] or the reading condition [see section 6.3.3]). During the manipulation blocks the e-Prime program instructed participants to complete the task assigned to them. Participants were signalled to resume the Rating Faces Task at the end of the one-minute blocks by a beep and an alert on the screen. A flowchart diagram of the rating faces task stressor procedure can be found in appendix 20.
8.7. Results

8.7.1. Statistical Analyses

In total, 2592 sets of ratings were completed: 24 (0.9%) of the regulators in the videos were marked as acquaintances of the observers and this data was subsequently removed from the analyses. The final data set consists of 2568 ratings (reading group T1 \( n = 659 \), speech group T1 \( n = 626 \); reading group T2 \( n = 660 \), speech group T2 \( n = 623 \)).

For the statistical analyses PSAW Statistics (version 18.0.2, SPSS Inc., Chicago IL) was used, with the alpha set to .05. The dependent variables were examined for normality of distribution using histograms and Kolmogorov–Smirnov tests. Mean HR, SCL, and HF-HRV values were calculated for the baseline period, each block of the Rating Faces Task, and each block of the stressor manipulation period. Mean HR and SCL were also calculated for each 10-second video. Similar analyses were conducted to Experiment 1 (see section 8.3.1). First, analyses were conducted to establish whether there were any differences between the stressor manipulation groups at baseline. Secondly, bivariate correlations were carried out to identify confounds that might affect the rating variables. Thirdly, the manipulation check was repeated to ascertain whether individuals were sensitive to the expressive regulation manipulation. Fourthly, analyses were carried out to investigate the effects of the repetitive stressor manipulations on physiology and mood state. Finally, analyses were conducted to evaluate the effects of the repetitive stressor manipulations on the affiliation ratings. As in Experiment 1, the Huynh-Feldt degrees of freedom correction was applied where necessary (i.e., when factors violated sphericity assumptions, as confirmed by Mauchly’s test). Significant main effects for all analyses were followed up with pairwise comparisons, and interactions were examined through analyses of simple effects. All pairwise contrasts were evaluated using Bonferroni critical values of .05.

8.7.2. Stressor Group Characteristics

The stressor groups were compared using their demographic data and baseline self-report measures (see table 8.1 for group characteristics). The groups did not significantly differ in terms of age, sex, smoking status, coffee consumption, self-
reported depression symptoms, current mood, or social safeness. The groups did
significantly differ in terms of trait anxiety, \( t(70) = -2.49, p = .015 \), with the speech
group reporting higher anxiety levels (\( M = 4.54, SD = 3.38 \)) than the reading group (\( M \\
= 2.70, SD = 2.89 \)). Independent \( t \)-tests did not reveal any significant differences
between the groups for HR, SCL, or HF-HRV at baseline or T1 (see table 8.2). Further
to this, mixed-factorial ANOVAs (repeated measure: Condition; between subjects
factor: Group) did not find any significant differences between the groups for
willingness to affiliate at T1. In the subsequent analyses comparing the stressor groups,
the analyses were repeated with GAD-7 scores included as a covariate to control for
group differences in trait anxiety. A caveat of carrying out the between subjects
analyses with GAD-7 scores as a covariate, is that controlling for trait anxiety may also
remove some of the effects of state anxiety that relate to the stressor manipulations (for
a discussion on the use of analyses of covariance, see Miller & Chapman, 2001).
Consequently, caution was used when interpreting the findings comparing the groups’
affiliation ratings.

Table 8.1.
Stressor group characteristics

<table>
<thead>
<tr>
<th></th>
<th>Reading Group (( n = 37 ))</th>
<th>Speech Group (( n = 35 ))</th>
<th>( t )</th>
<th>( df )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>5.76 (6.64)</td>
<td>7.83 (6.49)</td>
<td>-1.34</td>
<td>70</td>
<td>.185</td>
</tr>
<tr>
<td>GAD-7</td>
<td>2.70 (2.89)</td>
<td>4.54 (3.38)*</td>
<td>-2.49</td>
<td>70</td>
<td>.015</td>
</tr>
<tr>
<td>POMS-SF Total</td>
<td>3.84 (13.03)</td>
<td>3.77 (9.82)</td>
<td>0.02</td>
<td>70</td>
<td>.981</td>
</tr>
<tr>
<td>SSPS</td>
<td>33.27 (8.34)</td>
<td>30.23 (9.52)</td>
<td>1.45</td>
<td>70</td>
<td>.153</td>
</tr>
</tbody>
</table>

*Note. BDI-II = Beck Depression Inventory, GAD-7 = Generalised Anxiety Disorder Scale, POMS-SF = Profile of Mood States Questionnaire, SSPS = Social Safeness and Pleasure Scale.
Significant difference between the stressor groups * = \( p < .05 \)

8.7.3. Individual Differences in Observers’ Willingness to Affiliate.

Data collected from the first half of the Rating Faces Task (T1) were analysed to
identify individual differences in observers’ willingness to affiliate with others (as indexed by the mean amount of time individuals would be willing to spend with regulators across all conditions). Bivariate correlations revealed that the affiliation ratings were significantly correlated with self-reported social safeness (SSPS total; \( r = .24, p = .040 \)); the correlation between affiliation ratings and anxiety symptoms just
failed to research significance (GAD-7 total; $r = -0.23$, $p = 0.053$). The direction of the relationship between the affiliation ratings and the SSPS scores confirmed that willingness to affiliate was greater for individuals with higher social safeness. As a result, the SSPS scores were included as a covariate in the subsequent affiliation analyses.

### 8.7.4. Manipulation Check

Analyses of the expressivity ratings supported the manipulation of the expressive regulation strategies. A repeated-measures ANOVA revealed a significant effect of Condition (enhancement, suppression, maintain) on the ratings of expressivity, $F(1.52, 107.85) = 509.19$, $p < .001$. As expected, Bonferroni pairwise comparisons indicated that the expressivity ratings were significantly greater in the enhancement condition ($M = 4.67$, $SD = 0.53$) than the maintain condition ($M = 3.14$, $SD = 0.46$), and were significantly lower in the suppression condition ($M = 2.52$, $SD = 0.59$) than the maintain condition (all significant at $p < .001$).

### 8.7.5. Physiological Reactivity to the Stressor Manipulations

Mixed-factorial ANOVAS (repeated measure: Time; between subjects factor: Group) were carried out separately for the HR, SCL, and HF-HRV data; the raw means for each group are shown in table 8.2. For HR a significant main effect of Group was found, $F(1, 70) = 7.27$, $p = 0.009$. Bonferroni pairwise comparisons ($p < .05$) confirmed that HR in the speech group ($M = 84.09$, $SD = 13.08$) was consistently higher during the stressor manipulation blocks than HR in the reading group ($M = 77.02$, $SD = 8.91$). Both groups demonstrated an increase in SCL during the stressor manipulation blocks. A significant main effect of Time was revealed, $F(1.28, 89.49) = 19.84$, $p < .001$, as both groups demonstrate declines in SCL during the Rating Faces blocks at T2. Although the speech group tended to exhibit higher levels of SCL through the blocks, the main effect of Group did not reach significance, $F(1, 70) = 3.26$, $p = 0.075$. For HF-HRV there was a significant Time x Group interaction, $F(2, 140) = 9.83$, $p < .001$. During the manipulation blocks the reading group showed an initial increase in HF-HRV, whilst the speech group exhibited a significant decrease in HF-HRV during the first two stressor blocks ($p < .05$).
### Table 8.2.
Mean heart rate, skin conductance level, and high-frequency heart rate variability during the manipulation periods by stressor group

<table>
<thead>
<tr>
<th>Stressor Group</th>
<th>Baseline</th>
<th>Stressor Manipulation</th>
<th>F (time)</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Block 1</td>
<td>Block 2</td>
<td>Block 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>79.20 (11.20)</td>
<td>76.52 (8.72)**</td>
<td>77.06 (9.18)*</td>
<td>77.48 (9.25)*</td>
<td>6.32</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.51 (1.22)</td>
<td>4.20 (1.98)**</td>
<td>3.86 (2.00)**</td>
<td>3.86 (2.08)**</td>
<td>54.80</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.27 (0.81)</td>
<td>7.46 (0.67)</td>
<td>7.21 (0.75)</td>
<td>7.13 (0.63)</td>
<td>5.24</td>
</tr>
<tr>
<td>Speech Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>76.15 (10.88)</td>
<td>84.32 (13.71)**</td>
<td>83.69 (12.95)**</td>
<td>84.27 (13.68)**</td>
<td>22.32</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>2.34 (1.48)</td>
<td>5.04 (1.87)**</td>
<td>4.70 (1.88)**</td>
<td>4.65 (1.92)**</td>
<td>126.47</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.37 (1.17)</td>
<td>7.00 (0.95)*</td>
<td>7.06 (0.76)*</td>
<td>7.22 (0.79)</td>
<td>3.46</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from baseline * = p < .05 ** = p < .01 *** = p < .001

### Table 8.3.
Mean scores for the POMS-SF subscales before the first (T1) and second (T2) administrations of the Rating Faces Task.

<table>
<thead>
<tr>
<th></th>
<th>Reading Group (n = 37)</th>
<th>Speech Group (n = 35)</th>
<th>F (time x group)</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression–Dejection</td>
<td>1.22 (3.50)</td>
<td>0.92 (2.64)</td>
<td>0.57 (1.31)</td>
<td>1.46 (2.67)*</td>
<td>3.92</td>
</tr>
<tr>
<td>Vigour–Activity</td>
<td>6.11 (4.03)</td>
<td>4.46 (3.31)**</td>
<td>6.06 (3.76)</td>
<td>3.83 (3.83)**</td>
<td>1.27</td>
</tr>
<tr>
<td>Anger–Hostility</td>
<td>0.49 (1.10)</td>
<td>0.68 (1.08)</td>
<td>0.66 (2.11)</td>
<td>1.09 (3.02)</td>
<td>0.45</td>
</tr>
<tr>
<td>Tension–Anxiety</td>
<td>2.51 (3.00)</td>
<td>1.41 (1.66)**</td>
<td>3.11 (2.58)</td>
<td>7.09 (4.72)**</td>
<td>39.98</td>
</tr>
<tr>
<td>Confusion–Bewilderment</td>
<td>2.05 (2.19)</td>
<td>1.68 (2.10)</td>
<td>1.97 (1.92)</td>
<td>2.46 (2.20)</td>
<td>6.38</td>
</tr>
<tr>
<td>Fatigued–Inertia</td>
<td>3.68 (3.58)</td>
<td>3.46 (3.10)</td>
<td>3.51 (3.78)</td>
<td>2.83 (3.72)*</td>
<td>1.39</td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Significant difference from Rating Task T1 * = p < .05 ** = p < .01 *** = p < .001
When the analyses were rerun with the GAD-7 scores entered as a covariate, all of the effects remained significant with the exception of the main effect of Time for SCL; this was replaced with a significant Time x GAD-7 interaction, $F(1.31, 90.59) = 4.10, p = .035$. The SCL response of high and low GAD-7 scorers was evaluated by creating two groups using the sample median for the GAD-7: The groups demonstrated similar increases in SCL during the first stressor block, but individuals with higher GAD-7 scores demonstrated a quicker decline in SCL during the second and third stressor blocks ($p < .05$).

The POMS-SF subscales demonstrated that the speech group responded differently during the stressor manipulation period than the reading group (see table 8.3). A mixed-factorial ANOVA was carried out with Time and Scale as repeated factors and with Group as the between subjects factor. A significant main effect was found for Scale, $F(2.75, 192.49) = 31.97, p < .001$, as well as significant two-way interactions for Time x Group, $F(1, 70) = 13.81, p < .001$, Time x Scale, $F(2.59, 181.00) = 20.81, p < .001$, and Scale x Group, $F(2.75, 192.49) = 5.05, p = .003$. These were superseded by a significant three-way Time x Scale x Group interaction, $F(2.59, 181.00) = 18.45, p < .001$. Paired $t$-tests revealed that both groups reported being significantly less vigorous at T2, but whilst the reading group also reported being less tense, the speech group reported feeling significantly more tense and depressed but significantly less fatigued (significance is marked in table 8.3). These effects remained significant when the analysis was repeated the GAD-7 as a covariate; the main effect of the GAD-7 also reached significance: $F(1, 69) = 7.45, p = .008$, which is not surprising as both scales index anxiety symptoms.

To investigate whether changes in ANS function during the stressor manipulation period were related to changes in self-reported emotion, correlations were carried out between the mean HR, SCL, and HF-HRV reactivity scores and the mean POMS-SF reactivity scores. Significant correlations were found between HR reactivity and the POMS-SF confusion–bewilderment subscale ($r = .30, p = .011$), the POMS-SF tension–anxiety subscale ($r = .42, p < .001$), and the POMS-SF vigour–activity subscale ($r = .28, p = .018$). In addition to this, positive correlations were revealed between SCL reactivity and the POMS-SF tension–anxiety subscale ($r = .43, p < .001$), the POMS-SF confusion–bewilderment subscale ($r = .35, p = .003$), the POMS-SF anger–hostility subscale ($r = .25, p = .032$), and the POMS-SF depression–dejection subscale ($r =
Taken together these correlations suggest that larger increases in SNS activation were associated with larger increases in indices of negative affect.

Mixed-factorial ANOVAs were carried out on the blocks of the Rating Faces Task at T2 to establish if arousal was maintained during the secondary task (see table 8.4). HR was associated with a significant main effect of Time, $F(2.54, 178.00) = 2.95, p = .042$, as well as a Time x Group interaction, $F(2.54, 178.00) = 7.38, p < .001$. The speech group maintained increases in HR across all of the Rating Faces Task blocks at T2 ($p = .007$), whilst the reading group demonstrated a decrease in HR that was only significant during the first block. SCL also demonstrated a significant effect of Time, $F(1.67, 117.04) = 34.69, p < .001$, and a Time x Group interaction, $F(1.67, 117.04) = 8.10, p = .001$. Both stressor groups demonstrated significant increases in SCL at T2, but this increase was larger and maintained for longer in the speech group ($p < .05$). Finally, a significant main effect of Time was found for HF-HRV, $F(2.92, 204.07) = 5.08, p = .002$. HF-HRV demonstrated a steady decline during the Rating Faces blocks at T2 in both groups. Despite the speech group demonstrating a significant increase in HF-HRV from T1 during the first rating block at T2 ($p = .002$), none of the effects involving Group were significant. Again, when the analyses were repeated with GAD-7 scores entered as a covariate all the effects remained significant.
Table 8.4.
Mean heart rate, skin conductance level, and high-frequency heart rate variability by stressor group

<table>
<thead>
<tr>
<th></th>
<th>Rating Task (T1)</th>
<th>Rating Task (T2)</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Block 1</td>
<td>Block 2</td>
<td>Block 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>77.14 (9.79)</td>
<td>75.90 (9.54)*</td>
<td>77.01 (9.55)</td>
<td>76.47 (9.18)</td>
<td>2.46</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>3.58 (1.74)</td>
<td>3.93 (1.87)***</td>
<td>3.88 (1.94)**</td>
<td>3.88 (2.09)**</td>
<td>8.24</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.10 (0.72)</td>
<td>7.18 (0.68)</td>
<td>7.03 (0.67)</td>
<td>7.01 (0.72)</td>
<td>3.42</td>
</tr>
<tr>
<td>Speech Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>74.16 (10.63)</td>
<td>75.94 (10.82)**</td>
<td>76.14 (11.20)**</td>
<td>76.53 (11.07)**</td>
<td>7.57</td>
</tr>
<tr>
<td>SCL (µS)</td>
<td>3.59 (1.77)</td>
<td>4.53 (1.73)***</td>
<td>4.40 (1.75)***</td>
<td>4.51 (1.87)***</td>
<td>25.84</td>
</tr>
<tr>
<td>HF-HRV (ms²)</td>
<td>7.26 (0.93)</td>
<td>7.47 (0.87)**</td>
<td>7.33 (0.88)</td>
<td>7.32 (1.00)</td>
<td>2.84</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability. Significant difference from Rating Task T1 * = p < .05 ** = p < .01 *** = p < .001
8.7.6. The Effects on Expressive Regulation on Observers’ Physiology

To investigate whether the stressor manipulations influenced the effects of observing expressive regulation on HR and SCL, repeated-measures ANOVAs were carried out on the mean HR and SCL data for the 10-second video observations at T1 and T2. A repeated-measures ANOVA on the HR data revealed that observers’ physiological responses were again influenced by Condition, \( F(1, 70) = 30.61, p < .001 \); Bonferroni pairwise comparisons confirmed that HR was lowest when viewing enhanced expressions and highest when viewing suppressed expressions \( (p < .05) \). In addition to this there was also evidence of a significant Time x Group interaction, \( F(1,70) = 12.68, p = .001 \). As shown in figure 8.4, the reading group exhibited a slight overall decrease in HR at T2, whilst the speech group exhibited a significant overall increase in HR at T2 \( (p = .002) \). These effects remained significant when the GAD-7 was entered as a covariate.

![Figure 8.4](image_url)

*Figure 8.4. Mean heart rate by Condition as a function of Time and Group.*

A repeated measures ANOVA also revealed that observers’ physiological SCL responses were influenced by significant effects of Time, \( F(1, 70) = 48.87, p < .001 \), and a Time x Group interaction, \( F(1, 70) = 11.31, p = .001 \). As shown in figure 8.5, both groups exhibited significant increases in SCL at T2 \( (p < .001) \), but the magnitude of the increase was larger in the speech group. Again, these effects remained significant when the GAD-7 was entered as a covariate.
8.7.7. Effects of the Stressor Manipulations on Willingness to Affiliate

A mixed-factorial ANCOVA (repeated measures: Time, Condition, Valence; between subjects factor: Group; covariate: SPSS total) was carried out to assess the impact of defensive arousal on willingness to affiliate as measured by the Rating Faces Task (T1 compared to T2). Significant main effects were found for Condition, $F(1.59, 109.43) = 8.16, p = .001$, and Time, $F(1, 69) = 4.36, p = .041$. Significant interactions were also revealed for Condition x Valence, $F(2, 138) = 6.83, p = .001$, and Time x Condition x Valence, $F(2, 138) = 3.16, p = .045$. These were superseded by a four-way Time x Condition x Valence x Group interaction, $F(2, 138) = 3.37, p = .037$.

The main effect of Condition and the Condition x Valence interaction effect parallel the findings from Experiment 1: Observers were more willing to spend time with regulators expressing more emotion, particularly if they expressed positive emotion. The main affiliation finding of Experiment 2, was the four-way interaction between Time x Condition x Valence x Group, which suggests that the stressor manipulation did influence observers’ willingness to affiliate (see figure 8.6). Generally speaking, at T2 observers in the reading group exhibited increases in the amount of time they would spend with regulators. In particular, the reading group demonstrated a notable increase in their willingness to spend time with regulators suppressing negative emotion ($p < .001$). The speech group on the other hand did not exhibit increases in the amount of time they would spend with the regulators. Repeating the ANCOVA with GAD-7 scores to control for group differences in trait anxiety meant that the four-way interaction was no longer significant, $F(2, 136) = 2.97, p = .055$. As mentioned
previously, entering the GAD-7 scores as a covariate is an imperfect way of removing the group differences in trait anxiety. In this instance, controlling for GAD-7 scores did not alter the behaviour of the speech group only the control group. The current experiment therefore suggests that a stressor manipulation did not have a significant impact on observers’ willingness to affiliate with others.

Figure 8.6. Willingness to spend time with regulators as a function of Condition, Valence, Time (T1 vs. T2), and Group. Covariates were entered into the model at the following values: SPSS total = 31.79.

8.8. Discussion

Experiment 2 extended the findings of Experiment 1. First of all, the use of a different sample was able to confirm that social safeness was predictive of observers’ willingness to affiliate at T1. The positive relationship between the SSPS and the affiliation ratings from Rating Faces Task suggests that the safer we feel the more time we are willing to spend with others. A. C. Kelly, Zuroff, Leybman, and Gilbert (2012)
have reported an association between low social safeness and social avoidance, the
direction of this relationship is yet to be established. It is possible that social withdrawal
reduces opportunities for individuals to yield feelings of safeness, however our results
lend support to the argument that individuals low in social safeness are less willing to
seek social interactions. Further research to confirm the direction of this relationship is
warranted. It should be noted that the SSPS is a trait measure of social safeness, and
how safe we feel can vary according to the immediate environment (A. C. Kelly et al.,
2012). A measure of state changes in social safeness would have been useful in this
experiment to investigate the effect of the stressor manipulation on this construct.

8.8.1. Defensive Arousal and Observers’ Willingness to Affiliate

Experiment 2 confirmed that the repetitive stressor manipulation is effective at
maintaining defensive physiological arousal in the speech group; the speech group
exhibited larger increases in HR and SCL that were maintained during the blocks of the
Rating Faces Task at T2. As well as the speech groups exhibiting differences in arousal,
they also demonstrated differences in their willingness to affiliate during the secondary
task. Observers in the reading group demonstrated increases in their willingness to
affiliate with regulators, particularly for regulators who displayed suppressed negative
emotion. Observers in the speech group however showed little change in their
willingness to affiliate over the two tasks. Controlling for trait anxiety removed the
significant change in affiliation in the reading group, but did not affect the performance
of the speech group. The hypothesis that a laboratory stressor would decrease affiliation
tendencies was therefore not supported. It is proposed that the observers in the reading
group may have been more willing to spend time with regulators at T2 because they
were better able to perceive safety in the facial displays of the regulators over time. The
observers in the speech group failed to show this pattern of responding, suggesting that
their perception of the regulators may have been impaired by their physiological state.
This hypothesis would be in line with Porges’ (2004b, 2009a) theory of neuroception
(i.e., the ability for the nervous system to detect danger in the environment). However,
as there were significant group differences in trait anxiety these findings are tenuous.

Porges (2007a) has suggested that withdrawal of the VVC can affect “social
awareness” as well as limit spontaneous social engagement behaviours. It is not clear
which faculties are impaired when “social awareness” is affected, although Porges
(2007a) does suggest that the reading of social cues is one capacity that can be impaired during defensive physiological states. This supposition relates nicely to the theory that perceived social safeness is dependent on the immediate context (A. C. Kelly et al., 2012). Future research should endeavour to investigate the links between defensive physiological arousal, state social safeness, and affiliation tendencies. Relevant findings in this area may help to shed light on both polyvagal theory and the tend–and–befriend hypothesis.

8.8.2. Limitations

As with the previous studies there are several limitations to consider. Firstly, the Rating Faces Task in a new measure of individuals’ willingness to affiliate. Although the task has face validity it has not been validated as to whether or not affiliation ratings actually index how much time observers would spend with the regulators in real-life settings. It is also unknown whether or not the task is sensitive to social desirability effects. Although observers indicated how much time they would spend with regulators, it would have been interesting to relate this to other affiliation measures (i.e., quantity and quality of usual social interactions, and other established measures of affiliation). In addition to this, the current sample was homogenous in terms of age, sex, and ethnicity, which means that the results may not be generalisable to the wider population. This is in spite of the group differences in self-reported anxiety, which may be a significant confound of the current findings. An interesting finding of previous research is that females tend to be more affiliative than males (Luxen, 2005), which was not demonstrated in the current research, possibly because the current sample was not diverse enough in terms of sex. Repeating the experiment using more diverse samples in terms of age, sex, and ethnicity would help to establish the veracity of the current findings.

Before concluding, it is worth considering some of the strengths of the current experiments. The videos used in current study have good ecological validity. Participants were contemporary students of the current sample, and were filmed modulating their facial expressions in the belief that they were being observed. The videos are therefore good representations of voluntary expressive regulation. A second strength is that the current experiments are the first to report the simple effects of expressive regulation on observers’ physiology: participants were exposed to regulators
enhancing and suppressing both positive and negative emotion whilst HR and SCL were recorded. Previous studies have tended to only focus on one dimension of expressive regulation (i.e., enhancement or suppression), and more often than not the focus has been on the regulator rather than their intended target. Taken together with the findings from Chapter 6, the current research emphasises the need to evaluate both the sending and receiving aspects of emotional communication.

8.9. General Discussion

The current study provided some interesting findings relating to the effects of expressive regulation on observers, as well as how an observer’s physiological state affects their willingness to spend time with regulators. Experiment 1 demonstrated that observers are more willing to spend time with regulators who express more emotion, particularly if they express positive emotion. Experiment 2 confirmed this pattern of responding, but also suggested that defensive arousal affects one’s willingness to spend time with others: whilst participants in the reading group were likely to increase their willingness to spend time with regulators over the task, participants in the speech group were not. These findings are not consistent with the claims of the tend–and–befriend literature, however they do provide some support for polyvagal theory (Porges, 1995, 2001, 2003a). It is hypothesised that the speech group were less willing to spend time with regulators during the second administration of the Rating Faces Task because they were less receptive to safety cues in the facial displays presented. Future research would benefit from measuring state changes in social safeness during defensive physiological arousal, as this may be a mechanism underlying the observed effects.
Chapter 9: General Discussion

Porges’ (1995, 2001, 2003a) polyvagal theory purports that successful social engagement is contingent on activation of the ventral vagal complex (VVC; i.e., calm and self-soothing states). When individuals are challenged or threatened, activation of the sympathetic nervous system (SNS) or dorsal vagal complex (DVC) is hypothesised to be accompanied by withdrawal of the VVC and reduced accessibility of the social engagement system. Although polyvagal theory is consistent with other models of physiological responding such as the neurovisceral integration model (Thayer & Lane, 2000) and Schauer and Elbert's (2010) fear response cascade, only polyvagal theory makes claims about the effects of defensive arousal on social functions such as facial expressivity and social awareness. To date there is very little empirical work with adult populations that supports Porges’ claims. The current research was designed to address this gap in the literature and investigate the social consequences of defensive physiological states.

Due to methodological limitations it is currently impossible to directly challenge Porges’ claims by investigation of the VVC and DVC pathways. Although this has led to some criticism (see Berntson, Cacioppo, & Grossman, 2007; Grossman & Taylor, 2007; Ritz, 2009), this does not mean that tenets arising from polyvagal cannot be empirically tested. In Chapter 2 five main research hypotheses were identified: (1) laboratory stressors will be associated with decreased parasympathetic nervous system (PNS) activation, increased SNS activation, and increased negative affect (2) emotion regulation strategies will be associated with increased activation of the PNS, and accelerate the down-regulation of physiological and psychological arousal after a stressor, (3) increased activation of the SNS in response to a laboratory stressor will be associated with decreased facial expressivity, (4) increased activation of the SNS in response to a laboratory stressor will be associated with decreased emotional sensitivity, and (5) increased activation of the SNS in response to a laboratory stressor will be associated with decreased affiliation tendencies.

Before addressing the hypotheses stemming from polyvagal theory the thesis first considered which methodological techniques were suitable for the current research (Chapter 3). The following chapters then established a suitable way of activating and down-regulating defensive physiological states as a test of hypotheses 1 and 2: Chapter
4 confirmed that speech tasks are a suitable stressor manipulation whilst Chapter 5 compared the effects of several emotion regulation strategies on physiological return to baseline and self-reported affect. The thesis then focused on the investigation of Porges’ claims regarding socio-emotional responding (testing hypotheses 3-5); experiments were carried out to examine the effects of defensive physiological arousal on facial expressivity (Chapter 6), emotional sensitivity (Chapter 7), and affiliation tendencies (Chapter 8). The findings suggest that the social consequences of defensive physiological arousal in healthy adult populations are minimal, with social evaluative threat having little or no influence on the social functions explored. As a result several of the hypotheses in this thesis were not supported.

9.1. Summary of the Main Findings

As discussed in chapters 2 and 3, polyvagal theory’s (Porges, 1995, 2001, 2003a) claims regarding the effect of defensive physiological arousal on social functioning are largely theoretical in nature, and there is a dearth of experimental research testing Porges’ hypotheses in adult populations. The following sections will provide an overview of the main findings from the current thesis.

9.1.1. Social Evaluative Threat and Defensive Physiological Arousal

Although social evaluative threat is often invoked as a stressor manipulation, previous studies have failed to report on how well defensive physiological states are maintained during secondary tasks (e.g., Mansell, Clark, Ehlers, & Chen, 1999; Mansell, Ehlers, Clark, & Chen, 2002). Chapter 4 demonstrated some of the difficulties of using virtual reality as a stressor induction, however the findings confirmed that speech tasks result in reliable increases in heart rate (HR) and skin conductance (SCL), and reliable decreases in high-frequency heart rate variability (HF-HRV) (Feldman, Cohen, Hamrick, & Lepore, 2004; Kirschbaum, Pirke, & Hellhammer, 1993). This pattern of autonomic responding was interpreted as being consistent with a defensive physiological response; with increases in systems corresponding to SNS activation and decreases in systems corresponding to PNS activation (Lang, Davis, & Öhman, 2000; Tuvblad et al., 2010). In conjunction with Feldman et al. (2004) and Gregg, James, Matyas, and Thorsteinsson (1999), the current experiments suggested that utilising the manipulation period of the speech task was more effective at maintaining arousal than
the presentation period. From this it was hypothesised that the residual arousal may be sufficient to impact on the behavioural tasks employed in chapters 5 to 8.

Chapter 5 utilised the speech task and demonstrated that regulatory strategies can facilitate the down-regulation of residual arousal (for full discussion of the effects see section 9.1.2). However when the speech task was employed along with secondary tasks measuring social behaviours (particularly in chapters 6 and 7), it was discovered that arousal in the speech group did not remain significantly higher than arousal in the control group during the secondary tasks. At first it was conjectured that the expressive regulation task in Chapter 6 may have been initially too arousing, which may have limited observable group effects in response to the stressor manipulation, however similar group responses were found in Chapter 7 when using an emotion recognition task that was less arousing. As a result it was concluded that the original stressor was ineffective at maintaining arousal, and in the later experiments the speech task was adapted by splitting the preparation period into blocks that were counterbalanced with blocks of the secondary tasks. This adaptation to the speech task was considered to be a more suitable design than simply lengthening the stressor period, because individuals tend to habituate to stressors over time (Kelsey, Ornduff, & Alpert, 2007; Kelsey, Soderlund, & Arthur, 2004). The later findings confirmed the utility of splitting the preparation period into blocks because continued arousal in the stressor group allowed the groups to be differentiated on the basis of their psychophysiological profiles. Consequently, future research using stressor manipulations may wish to consider how physiological responses to stressor tasks can change over time, particularly when using healthy control samples. Where prolonged arousal is required researchers may want to consider repeating short blocks of a stressor task to reinforce the effects of the stressor manipulation.

A caveat of using stressor manipulations in laboratory settings is the ability to initiate measurable changes in physiology that reflect physiological responses to real-life stressors. Laboratory-based experiments allow for high levels of control, but laboratory stressors often elicit milder physiological responses than natural stressors (Dimsdale, 1984). It has long been debated how well responses to laboratory stressors predict responses to real-life stressors outside of the laboratory (Kamarck & Lovallo, 2003; van Egeren & Sparrow, 1989). This poses a difficulty for researchers as experiments can lack ecological validity. A further challenge of designs using
laboratory-based stressors is that the observed physiological responses to stress are likely to be short-lived. Stressors result in measurable changes in physiology, as the body shifts out of a state of homeostasis (Fabes & Eisenberg, 1997; Jänig, 2006). As soon as the body moves into a defensive physiological state, processes will be at work to return the body to its natural baseline: Increased SNS activation is likely to be rapidly followed by increased PNS activation to counter increases in arousal (Mezzacappa, Kelsey, Katkin, & Sloan, 2001). Returning to physiological baseline after a stressor is a sign of physiological health, with chronic elevations in arousal being indicative of poor autonomic functioning (Rozanski & Kubzansky, 2005; Sapolsky, 1997; Tuvblad et al., 2010). Physiological health means that control populations are likely to respond to laboratory stressors by showing elevations in arousal that are mild in magnitude and short in duration.

The stressor manipulation employed in the preceding chapters increased SNS activation and this was assumed to represent increases in defensive physiological arousal, however there is no guarantee that the observed changes in physiology were not attributable to co-activation of the SNS and VVC, as opposed to pure defensive arousal (i.e., SNS activation coupled with withdrawal of the VVC). This pattern of responding could be why the increases in arousal in the speech groups were not maintained during the secondary tasks and why there were no noticeable changes in social behaviour in the speech groups after the stressor manipulations. Further discussion of the limitations of using a healthy control sample is given in section 9.2.

9.1.2. Regulatory Strategies and their Effects on Defensive Physiological Arousal

The subjective experience of emotion is an important concomitant of physiological responding: How we feel is likely to impact on how we think and behave, and our ability to regulate our emotions has been shown to affect our social competence (Eisenberg, Fabes, Guthrie, & Reiser, 2000). Negative affect is often coupled with increases in SNS activation (Feldman et al., 1999; Sloan et al., 1994), which in turn is hypothesised to affect social engagement behaviours (Porges, 2001, 2003a; but see Taylor, 2006). Chapter 5 compared the psychophysiological effects of several regulatory strategies on down-regulating defensive physiological arousal. The focus was on emotion regulation strategies that are hypothesised to target activation of the PNS and increase positive affect. In general the findings suggest that engaging in active
emotion regulation may be able to facilitate the down-regulation of psychophysiological arousal (when compared to resting quietly). The mindful breathing and neutral listening strategies had the most pronounced effects on HR and SCL, which may be a function of the human voice activating the VVC via the muscles of the middle ear (Porges, 2001, 2003a; Porges & Lewis, 2010). Importantly, the role of distraction was also considered as a potential mechanism underlying the down-regulation seen in arousal.

In the short term the loving-kindness meditation was able to significantly increase positive affect (see Fredrickson, Cohn, Coffey, Pek, & Finkel, 2008), whilst the mindful breathing meditation demonstrated the most notable decrease in physiological arousal (see Jerath, Edry, Barnes, & Jerath, 2006). It is argued that these strategies work by mechanisms other than distraction; distraction is helpful in the short term, but in the long term it becomes maladaptive (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Increased practice of mindfulness and loving-kindness has been shown to enhance the observed effects of meditation practices (Carson et al., 2005; Pace et al., 2009), suggesting that mechanisms other than distraction are at work. The effects of loving-kindness and mindful breathing meditations make them promising strategies for reducing subjective distress and defensive physiological arousal. Further research is needed however, to identify the mechanisms that make these strategies so successful in order to maximise their efficacy.

Consistent with previous research, none of the regulation strategies examined demonstrated signs of buffering against future stress in the short term (Fredrickson, Mancuso, Branigan, & Tugade, 2000). Interestingly, the groups that carried out the emotion regulation strategies rated their preparation and performance of the speech task as being significantly poorer than the group that simply rested quietly. Consequently, although emotion regulation strategies can be beneficial, the timing of their deployment is an important consideration. Individuals relying on emotion regulation strategies will need to be taught which techniques are appropriate, as well as when to recognise that the behaviours are needed (Sheppes & Gross, 2011). Timing is important in two respects: Firstly, one must consider the timing of the strategy in relation to the emotion generation process (i.e., early vs. late processing), and secondly it is important to recognise whether or not the chosen strategy is feasible and/or appropriate in the current context (Sheppes & Gross, 2011). The present research has confirmed the potential role of emotion regulation strategies in down-regulating defensive physiological arousal,
however further research is needed to explore their effectiveness in clinical populations, as well as their acceptability and accessibility.

A strength of the current research is that it highlights the role of doing nothing in times of stress. Often researchers compare different emotion regulation strategies in the belief that they will facilitate coping and self-regulation; in healthy populations however sometimes the most effective way forward may be to take a step back from the situation. When an individual is stressed, that is not always the most appropriate time to be learning a new emotion regulation skill. If an individual does not feel overly distressed by the presence of physiological arousal, then doing nothing may be a surprising way of helping individuals to cope with an impending stressor. For example, in the current experiment participants who did nothing reported higher levels of satisfaction with their speech performance than those who carried out an emotion regulation strategy.

9.1.3. Defensive Physiological Arousal and Facial Expressivity

A key issue raised in the initial literature review (Chapter 2) was the premise that defensive physiological arousal should reduce facial expressivity (see Porges, 2001, 2003a). To date there have been few empirical studies that have approached this question, and most of the research that has been carried out has been with infants rather than adult populations (for example Stifter & Fox, 1990; Stifter, Fox, & Porges, 1989). A comprehensive review relevant to this topic was carried out by Cacioppo et al. (1992). The model formulated by Cacioppo and colleagues acknowledged that different relationships can occur between SNS function and facial expressivity: Individuals can exhibit increases in SNS activation coupled with low facial expressivity (internalisers) or high facial expressivity (externalisers). The mechanisms underlying this relationship are unclear (individual differences in expressivity may result from natural tendencies and/or conscious control), however Cacioppo et al.’s (1992) model suggests that there is not a transparent link between autonomic functioning and facial expressivity.

Despite research indicating that the relationship between autonomic responding and facial expressivity tends to vary within and across individuals, polyvagal theory posits that increased activation of the SNS should result in withdrawal of the social engagement system and subsequently reduce facial expressivity (Porges, 2001, 2003a, 2007b). To examine this proposition, two experiments were carried out in the present
thesis using healthy adult populations: In Chapter 6 participants completed an expression regulation task whilst facial expressivity was captured using eMotion (an emotion recognition software package; Gevers, 2008), and in Chapter 7 participants completed an emotion recognition task whilst facial mimicry was measured using electromyography. In both experiments facial expressivity was measured before and after a stressor manipulation to investigate changes in facial expressivity that may have been associated with increases in defensive physiological arousal. The findings of each chapter will be discussed in turn.

The ability to modulate one’s facial expression in accordance to situational demands is often considered to be a trait ability (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004), however it has also been shown that the immediate context can influence facial expressivity (Westphal, Seivert, & Bonanno, 2010). In Chapter 6 participants carried out an expressive regulation task that required the participants to enhance, maintain, or suppress their facial expressions in response to positive and negative visual stimuli (Bonanno et al., 2004). The participants were divided into a stressor group and a control group so that the role of defensive physiological arousal in expressive enhancement ability and expressive suppression ability could be explored. Following Porges (1995, 2001, 2003a), it was hypothesised that increased SNS activation would be associated with reduced facial expressivity in the stressor group after the stressor manipulation but not the control group. Although participants in the stressor group did demonstrate reduced enhancement ability and increased suppression ability these changes were not significant within or across the groups. Consequently the hypothesis was not supported. It was concluded that the findings may have been obscured because both groups demonstrated increased physiological arousal during the expressive regulation task. However, a second line of reasoning suggested that defensive arousal may reduce spontaneous facial expressivity but not consciously mediated facial expressivity. This potential confound was addressed in the following chapter.

In Chapter 7 two experiments used electromyography to measure spontaneous facial expressivity in response to facial displays of emotion. It is well-established that viewing facial expressions results in facial mimicry in observers (Dimberg, 1982; Hatfield, Cacioppo, & Rapson, 1993). In the current experiments, participants completed an emotion recognition task (the Multimorph Facial Affect Task; Blair,
Colledge, Murray, & Mitchell, 2001) and were again divided into a stressor group and a control group. The general hypothesis was that participants in the stressor groups would show reduced spontaneous facial mimicry to the facial displays presented after the stressor manipulation, and that this in turn would increase the amount of time it would take to correctly identify the emotions (this second measure was termed *emotional sensitivity* and is discussed in section 9.1.4). The hypothesis regarding spontaneous facial expressivity was not supported by the results: The findings in Experiment 1 indicated that the stressor group did not vary in the amount of emotion expressed across the two administrations of the emotion recognition task. Although concerns were raised regarding the effectiveness of the stressor manipulation, these were addressed in Experiment 2 by splitting the stressor manipulation into blocks that were counterbalanced with blocks of the emotion recognition task. Despite increasing the physiological arousal of the stressor group during the second administration of the emotion recognition task in Experiment 2, this still failed to affect the spontaneous facial mimicry of the stressor group.

Taken together, the findings of chapters 6 and 7 indicate that there is not a direct relationship between defensive physiological arousal and facial expressivity. The current research did not support a central tenet of polyvagal theory, which suggests that increased activation of the SNS inhibits the social engagement system (Porges, 2001, 2003a). This has important implications for some of the proposals made in the literature review, where by reduced facial affect was identified as a characteristic of several psychiatric disorders (see sections 2.4.5.1-2.4.5.5). Although polyvagal theory (Porges, 1995, 2001, 2003a, 2007b) is a compelling model for explaining reduced facial expressivity in clinical populations, the current evidence provides some doubt for Porges’ model as a model of universal responding.

**9.1.4. Defensive Physiological Arousal and Emotional Sensitivity**

The ability to recognise emotions in others was identified as an area of interest because it has been suggested that emotional sensitivity is contingent on facial feedback (Oberman, Winkielman, & Ramachandran, 2007). If defensive physiological arousal results in decreased facial expressivity, as suggested by polyvagal theory, one’s ability to recognise emotional facial expressions may also be impaired during defensive physiological arousal due to reduced afferent feedback from the facial muscles. Chapter
7 was designed to concurrently investigate the effects of defensive physiological arousal on facial expressivity and emotion recognition. Surprisingly, there has been little research on the effects of stress responding on emotion recognition. Hänggi (2004) carried out an Internet-based experiment where participants carried out emotion recognition tasks during an online procedure designed to induce stress (stressors included negative feedback, form malfunction, and increased time pressure). Hänggi’s results suggested that stress impairs decoding ability, with individuals in the stress condition demonstrating poorer recognition of emotional facial displays.

In Chapter 7 participants completed the Multimorph Facial Affect Task (Blair et al., 2001). The task requires participants to classify 6 basic emotional facial expressions as quickly as they can whilst each stimulus morphs through 39 stages into a prototypical emotional expression. On the basis of Porges (2001, 2003a) and Hänggi (2004) it was hypothesised that participants would take longer to correctly classify the emotional expressions after a stressor manipulation (i.e., they would be less sensitive to the emotional facial expressions). This hypothesis was not supported in Experiment 1, as the stressor group did not show a significant change in emotional sensitivity after the stressor manipulation. After reviewing the physiological data from Experiment 1 it was conjectured that the stressor manipulation may have been ineffective at maintaining arousal during the secondary emotion recognition task. The stressor manipulation was revised in Experiment 2 and counterbalanced with blocks of the emotion recognition task, which maintained arousal in the speech group for longer. Although the change in design resulted in greater differences between the groups in terms of physiology, these differences were not associated with measurable behavioural differences between the groups in terms of emotional sensitivity (i.e., both groups took similar amounts of time to correctly identify the emotions presented). Consequently, neither of the experiments supported the hypothesis that defensive physiological arousal dampens facial expressivity which in turn reduces emotional sensitivity.

The lack of evidence to support the link between defensive physiological arousal and emotional sensitivity undermines Porges’ claims that activation of SNS impacts on “social awareness” (Porges, 2003a, 2009a). The findings of this thesis suggest that physiological arousal does not necessarily impair one’s ability to perceive and decode others’ emotional states. Previous research has suggested that although emotion recognition and emotion regulation are complementary proficiencies, they are not
analagous of each other (Papousek, Freudenthaler, & Schulter, 2008). Thus the emotion recognition deficits seen in specific clinical disorders may not result from the deficits in autonomic function that also tend to be observed in these populations.

A finding from Chapter 7 that does provide some limited support for polyvagal theory, was the relationship found between the activation of the levator labii and the speed at which participants recognised disgust. This finding is in line with the work of Stel and colleagues (Stel & van Knippenberg, 2008; Stel & van den Bos, 2010), and suggests that emotional sensitivity for disgust may be contingent on afferent feedback from the face. If reduced activation of the levator labii decreases emotional sensitivity for disgust, this would support the notion that decreased facial expressivity in response to a threat may reduce "social awareness" (Porges, 2009b). It is possible that different forms of threat result in differential reductions in facial expressivity and/or emotional sensitivity that are emotion-specific. This has implications for researchers who only focus on one or two emotions as representative facial expressions of positive and negative emotion (e.g., happiness and anger are often used as prototypical positive and negative emotions respectively; Dimberg & Lundquist, 1990; Dimberg & Petterson, 2000; Jönsson & Sonnby-Borgström, 2003). This was not however a limitation of the current experiment.

9.1.5. Defensive Physiological Arousal and Affiliation

The final area of interest in this thesis was Porges’ claims regarding social engagement and affiliation (see Porges, 2001, 2003a, 2007b). Porges proposed the existence of functional neuroanatomical networks that link physiological state and affiliation tendencies, but this relationship has not been subjected to empirical testing. From a theoretical point of view polyvagal theory is inconsistent with the tend–and–befriend model: Porges (1995, 2001, 2003a) believes that social engagement only occurs in calming and self-soothing states, whereas Taylor and colleagues (Taylor et al., 2000; Taylor, 2006; Taylor et al., 2008) claim that stressful situations can elicit affiliation tendencies. In Chapter 8 two experiments were carried out to shed further light on this research question: Experiment 1 investigated the assertion that facial displays influence social interactions, whilst Experiment 2 investigated how the relationship between facial expressivity and willingness to affiliate is affected by stress.
The findings from Experiment 1 indicate that facial expressivity does play an important role in interpersonal interactions. Participants watched videos of individuals voluntarily regulating their facial expressions and rated their willingness to spend time with them (the Rating Faces Task was created using the video stimuli collected from the experiment in Chapter 6). Greater expression of emotion was associated with increases in the amount of time observers would be willing to spend with regulators. This relationship was accompanied by significant changes in heart rate; regulators who suppressed their emotional expressions elicited increases in heart rate in observers. The rationale given for this pattern of results was the premise that facial displays are important signals of safety and danger that help to regulate social interactions (McHugo & Smith, 1996; Orr & Lanzetta, 1980). Whilst greater expression of emotion signals safety, the absence of facial expressions may signal danger or deception, resulting in decreased likability and a reduced willingness to interact with the person signalling (see Riggio & Friedman, 1986). Experiment 1 therefore provided partial support for polyvagal theory by confirming a link between facial expressivity and others’ willingness to affiliate.

A second line of evidence in favour of polyvagal theory found in Experiment 1 relates to perceived safety. Bivariate correlations established that one’s willingness to affiliate with others is positively related to feeling safe, as indexed by the Social Safeness and Pleasure Scale (Gilbert et al., 2009) and HF-HRV, which has been linked to the ability to perceive safety (Thayer & Lane, 2000). The current findings are consistent with research suggesting that individuals with low trait social safeness are less willing to seek social interactions with others (A. C. Kelly, Zuroff, Leybman, & Gilbert, 2012). The findings also corroborate the notion that perceived safety is associated with affiliation tendencies (Porges, 2001, 2003a). Consequently Porges’ (2009a) claims that feelings of safety promote positive social interactions are supported by the current research.

Experiment 2 sought to further elaborate the relationship between facial expressivity and affiliation by investigating whether this relationship is affected by defensive physiological arousal. Two contrasting theories have arisen regarding affiliation during stress: polyvagal theory would predict decreased affiliation during stress whilst the tend–and–befriend hypothesis would predict increased affiliation during stress. A between subjects design was used to investigate the effects of a stressor
manipulation on the Ratings Faces Task developed in Experiment 1. Despite
confirmation that social safeness was related to observers’ willingness to spend time
with others, the stressor group did not exhibit changes in the affiliation ratings after the
stressor manipulation. Consequently the results were not in favour of polyvagal theory
or the tend–and–befriend hypothesis.

9.2. Limitations of the Research

There are several limitations in the current thesis that need to be addressed when
interpreting the usefulness of the findings. First of all the use of healthy control
populations to challenge polyvagal theory may have obscured the relationship between
autonomic functioning and social engagement behaviours. If polyvagal theory is a valid
model of socio-emotional responding, all populations should experience reductions in
social functioning during defensive physiological arousal because of a functional
research with healthy adult populations has tended to investigate links between
autonomic function and socio-emotional behaviours during passive tasks such as
viewing films, as opposed to during threat. A strength of the current experiments is that
socio-emotional responding was investigated during laboratory challenge, making the
findings more applicable to the predictions made by polyvagal theory. Despite this,
several of the hypotheses were not supported, and this may be a result of using healthy
control populations to test the tenets of polyvagal theory. It should be noted that the
findings may suggest that polyvagal theory does adequately model socio-emotion
responding in healthy populations, although this does not mean that polyvagal theory is
not a valid model of socio-emotional responding in clinical populations.

It is possible that healthy populations may not show physiological responses to
laboratory stressors that are large enough to impact on the socio-emotional behaviours
measured in the current studies. In healthy populations, proficiencies in emotion
regulation, emotion perception, and emotion production skills, may buffer against the
emergence of deficits in social functioning during a social stressor. Arguably stronger
stressor manipulations could be used to induce larger shifts in physiological state to
encourage full withdrawal of the VVC, however this would be difficult to achieve in
laboratory settings using stressor manipulations that abide by ethical considerations. As
a result, a major limitation of the current research is that it was difficult to suitably stress participants, and then keep them in a stressed state.

One way forward could be to try and find a more effective way of stressing healthy control participants. It is proposed that perceived safety should be a key target for laboratory stressors; this is for two reasons. Firstly, in the current research perceived social safeness was significantly correlated with both self-reported emotion (Chapter 6) and affiliation tendencies (Chapter 8). Taken together, these findings suggest that perceived safety is an important moderator of socio-emotional responding. Secondly, it is claimed that feelings of perceived safety vary according to the immediate environment, and that in threatening situations individuals will feel less safe (A. C. Kelly et al., 2012). It is therefore argued that stressors which challenge participants’ perceived safety may be the most effective means of eliciting stress responses in laboratory settings. Tasks that reduce social safeness may not only influence the autonomic nervous system, but may also be the mechanism by which stressors induce changes in socio-emotional behaviours. Research is therefore needed to further investigate the effects of state feelings of perceived safety on socio-emotional responding.

An alternative way forward for the current research could be to replicate the experiments from this thesis using clinical populations. Although this is a viable avenue for further research, as mentioned in section 3.2, using clinical populations to test polyvagal theory limits the generalisability of the findings. It would be impossible to demonstrate that any deficits in social function in clinical populations did not result from disorder specific abnormalities, such as anatomical flaws. A second alternative could be to repeat the experiments with healthy populations responding to life stressors, for example Bonanno and colleague have investigated the role of facial expressivity in coping with bereaved populations, survivors of sexual abuse, and New York City college students adjusting to college life after the 9/11 terrorist attacks (Bonanno et al., 2002, 2004; Gupta & Bonanno, 2011). This latter approach may provide a test of polyvagal theory that is more ecologically valid. At the same time it is also possible that populations responding to life stressors may still show signs of healthy social functioning, and not respond to social stressors with deficits in socio-emotional behaviour.
One must consider the possibility that environmental stressors may only reduce social functioning in individuals with other notable predisposing factors, such as genetic and/or socio-demographic influences: A combination of individual differences in neural processing and environmental stressors may be required for the development of psychiatric symptomatology and associated deficits in social function (Leppänen, 2006). This would suggest that polyvagal theory may be appropriate as a model of socio-emotional responding in clinical populations, but does not adequately represent normal socio-emotional functioning. To test this theory, researchers need to capture a range of indices, including autonomic functions, genetic polymorphisms, socio-demographic factors, self-reports from participants and peers, as well as observable behaviours, in order to establish how and when defensive physiological arousal can influence socio-emotional responding (Porges, 2006).

A further limitation of the current thesis is that even though the samples used were healthy control participants, the generalisability of the findings is still limited by the characteristics of the populations sampled. All of the samples comprised of undergraduate students with no history of psychiatric treatment or symptomatology, and the samples were fairly narrow in age, sex, and ethnicity. This means that the ability to detect significant effects of socio-demographic factors was restricted. For example, gender differences were identified in Chapter 7 in emotion recognition as expected (with female participants demonstrating higher emotional sensitivity than male participants), but gender effects were not found in chapters 6 (facial expressivity) and 8 (affiliation) which conflicts with previous research on gender differences (see for example Hess & Bourgeois, 2010; and Luxen, 2005). Although the findings cannot be extended beyond this demographic group, the internal validity of the studies is enhanced by having such a narrow sample demographic.

Another consideration that is important to address is methodological issues regarding the psychophysiological measures. In line with previous research, skin conductance level (SCL) was used as an index of SNS activation and high-frequency heart rate variability (HF-HRV) was used as a measure of PNS activation (Berntson et al., 1997; Dawson, Schell, & Filion, 2000; Jänig, 2006). These measures have been shown to map onto two separate latent factors of autonomic activity (Tuvblad et al., 2010), however their ability to accurately reflect the overall physiological state of an individual is questionable. SCL indexes widespread arousal but does not indicate the
effects of the SNS on specific effector organs (Boucsein, 1992), whilst HF-HRV measures *cardiac* vagal control, but not widespread PNS function (Ritz, 2009). Although some researchers choose to measure the influences of the SNS and PNS on the same effector organ (e.g., HRV and pre-ejection period respectively index PNS and SNS innervation of the heart; Cacioppo, Uchino, & Berntson, 1994), this still does not overcome the problem of inferring how the autonomic nervous system is responding as a whole (both the SNS and PNS are heterogeneous in their effects on effector organs). Arguably it would be better to use measures of autonomic functioning that reflect the central outflow of vagal and sympathetic activity, however current imaging techniques are not refined enough to isolate the origins of neural signals from the brainstem (Grossman & Taylor, 2007; Ritz, 2009).

As a final limitation, some attention should also be given to the behavioural measures and their utility in revealing the effects of defensive physiological arousal on social engagement behaviours. It is debatable how accurately the behavioural measures reflect competencies in emotional expression, emotion recognition, and affiliation. The measures did not reflect the outcomes of real-time interactions, and as tasks that are analogues of real world behaviours they may not translate directly to how individuals would behave in face-to-face interactions in real-life (Kamarck & Lovallo, 2003). Thus prospective research is needed to address how well the chosen behavioural measures predict social behaviour outside the laboratory.

### 9.3. Final Conclusions

The experiments presented in this thesis are some of the first to evaluate polyvagal theory as a model of socio-emotional functioning in healthy adult populations. The findings only provide partial support for the polyvagal theory: afferent feedback from the face was associated with emotional sensitivity (even if only for disgust), emotional expressivity was shown to influence observers’ willingness to affiliate with regulators, and self-reported social safeness was also associated with affiliation tendencies. It is interesting to note, that the strongest evidence in the current thesis is not related to the biological tenets of polyvagal theory, but comes from its hypotheses regarding psychological and behavioural systems. Despite these encouraging findings, several of the assumptions of polyvagal theory were not
supported, including the main hypothesis that defensive physiological arousal would be associated with changes in facial expressivity and emotional sensitivity.

Although some of the conclusions of this thesis did not support the main tenets of polyvagal theory, several of the findings are in line with previous research. The current findings confirmed that social stress results in increased activation of the SNS (Kirschbaum et al., 1993; Schubert et al., 2009). It was also confirmed that emotion regulation strategies can be deployed to down-regulate increases in physiological arousal, however their ability to buffer against impending stress is limited (Fredrickson & Levenson, 1998; Fredrickson et al., 2000). Despite the chosen stressors resulting in increased defensive physiological arousal, no direct links between physiological arousal and facial expressivity, emotional sensitivity, or affiliation tendencies were identified. The lack of a positive association between increased SNS activation and the behaviours of interest may be a function of using healthy control populations, as their ability to successfully regulate their emotional responses to the stressor task may mask notable associations between autonomic functioning and social engagement behaviours. Future research should continue to assess the utility of polyvagal theory as a valid model of socio-emotional responding in both healthy and clinical populations.

Although this thesis did not find conclusive evidence to support the existence of the social engagement system per se, polyvagal theory’s (Porges, 1995, 2001, 2003a) assertion that heart rate variability is a marker of autonomic health is being widely recognised elsewhere. Low heart rate variability has been identified as a marker for poor autonomic functioning (Pumpula, Howorka, Groves, Chester, & Nolan, 2002), and it has been proposed that increased vagal activity is associated with greater psychological (Thayer, Hansen, Saus-Rose, & Johnsen, 2009) and physiological health (Thayer & Lane, 2007). Increased psychological and physiological health may help to bolster social functioning in a way that is difficult to capture in laboratory experiments.

A promising future avenue for researchers is the link between physiological state and perceived feelings of safety. Infant studies have demonstrated that heart rate variability is a suitable index of self-soothing capabilities (Fox, 1989), whilst Gilbert has demonstrated that self-reported social safeness is related to heart rate variability (P. Gilbert, personal communication, October 2, 2010). Taken together, this evidence suggests that the VVC is indeed a system that indexes self-soothing and calm states, and
promotes feelings of safety (in line with Porges, 1998, 2001, 2003a). If increasing activation of the PNS can be used to increase feelings of safety, this may be a potential mechanism for increasing affiliation tendencies. As demonstrated in Chapter 8, higher self-reported social safeness was associated with greater willingness to affiliate with others. Increasing one’s willingness to affiliate with others may function to increase social contact and support (A. C. Kelly et al., 2012), and social contact can act as an added source of safety signals to reinforce the activation of the VVC (Porges, 2003a). Consequently, polyvagal theory is still in need of verification, and this thesis has highlighted several potential areas for future research.
APPENDIX 1: Demographic Screening Questionnaire

1. Gender: Male ____  Female ____

2. Age: _____ years  Date of Birth  _____/_____/_____
   day  month  year

3. Current marital status: (check all that apply)
   ____ married with spouse
   ____ living with partner
   ____ separated
   ____ divorced
   ____ widowed
   ____ in an intimate relationship but not living together
   ____ never married

4. Highest level of education reached: (please tick any that apply)
   ____ Left school before 16
   ____ Finished school at 16
   ____ Finished school at 18
   ____ Attended university or equivalent
   ____ Completed university or equivalent
   ____ Completed postgraduate qualification

4b. Total number of years of Higher Education (e.g. university) completed _____ years
   If the above options do not fit exactly (e.g. you left education at 16 and then returned as
   a mature student), please specify here:
   ……………………………………………………………………………………………
   ……………………………………………………………………………………………

5. Ethnicity:
   What is your ethnic group? (please tick as many boxes as you feel apply to you)
   1  ____ White
      11  ____ British (white)
          111  ____ English
          112  ____ Scottish
          113  ____ Welsh
          114 Other British (white) - please specify…………………..
   12  ____ Irish
   13  ____ Any other White background - please specify………………..
   2  ____ Mixed
      21  ____ White & Black Caribbean
      22  ____ White & Black African
      23  ____ White & Asian
      24  ____ Any other Mixed background - please specify………………..

Continued overleaf……………..
### APPENDIX 1: DEMOGRAPHIC SCREENING

#### 3 Asian, Asian British, Asian English, Asian Scottish or Asian Welsh
- [ ] Indian
- [ ] Pakistani
- [ ] Bangladeshi
- [ ] Any other Asian background - please specify……………………

#### 4 Black, Black British, Black English, Black Scottish or Black Welsh
- [ ] Caribbean
- [ ] African
- [ ] Any other Black background - please specify……………………

#### 5 Other ethnic background
- [ ] Chinese
- [ ] Middle Eastern/North African
- [ ] Any other background - please specify…………………………

#### 6. Have you ever been prescribed medications for emotional or psychiatric problems?
- [ ] Yes  [ ] No

If yes, please complete the following chart:

<table>
<thead>
<tr>
<th>Medication name</th>
<th>Dates used (e.g., January, 2000-December, 2001)</th>
<th>Was the medication helpful for you?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
</tbody>
</table>

#### 7. Have you ever been in therapy?  [ ] Yes  [ ] No:

If yes, are you currently in therapy?  [ ] Yes  [ ] No

Please complete the following chart

<table>
<thead>
<tr>
<th>What was the treatment for? Include diagnosis, if known.</th>
<th>Dates of treatment</th>
<th>Was the therapy helpful for you?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yes / No</td>
</tr>
</tbody>
</table>

---
APPENDIX 2: Profile of Mood States - Short Form (Shacham, 1983)

Please rate the following statements according to how you feel right now by circling the corresponding number.

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>A little</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Tense</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2.</td>
<td>Angry</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3.</td>
<td>Worn out</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4.</td>
<td>Unhappy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5.</td>
<td>Lively</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6.</td>
<td>Confused</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7.</td>
<td>Peeved</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8.</td>
<td>Sad</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9.</td>
<td>Active</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10.</td>
<td>On edge</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11.</td>
<td>Grouchy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12.</td>
<td>Blue</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13.</td>
<td>Energetic</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14.</td>
<td>Hopeless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15.</td>
<td>Uneasy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16.</td>
<td>Restless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17.</td>
<td>Unable to concentrate</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18.</td>
<td>Fatigued</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19.</td>
<td>Annoyed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20.</td>
<td>Discouraged</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>21.</td>
<td>Resentful</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>22.</td>
<td>Nervous</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>23.</td>
<td>Miserable</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>24.</td>
<td>Cheerful</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>25.</td>
<td>Bitter</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>26.</td>
<td>Exhausted</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>27.</td>
<td>Anxious</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>28.</td>
<td>Helpless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>29.</td>
<td>Weary</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>30.</td>
<td>Bewildered</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>31.</td>
<td>Furious</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>32.</td>
<td>Full of pep</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>33.</td>
<td>Worthless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>34.</td>
<td>Forgetful</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>35.</td>
<td>Vigorous</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>36.</td>
<td>Uncertain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>37.</td>
<td>Bushed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
APPENDIX 3: Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983)

[This material has been removed by the author of this thesis for copyright reasons]
[This material has been removed by the author of this thesis for copyright reasons]
APPENDIX 4: Beck Depression Inventory II (Beck, Steer, & Brown, 1996)

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[This material has been removed by the author of this thesis for copyright reasons]
APPENDIX 5: Generalised Anxiety Disorder-7 Scale
(Spitzer, Kroenke, Williams, & Löwe, 2006)

Over the last two weeks, how often have you been bothered by the following problems?

<table>
<thead>
<tr>
<th>Problem</th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Feeling nervous, anxious or on edge</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Not being able to stop or control worrying</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Worrying too much about different things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Having trouble relaxing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Being so restless that it is hard to sit still</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Becoming easily annoyed or irritable</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Feeling afraid that something awful might happen</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
## APPENDIX 6: Berkeley Expressivity Questionnaire (Gross & John, 1995)

For each statement below, please indicate your agreement or disagreement. Do so by filling in the blank in front of each item with the appropriate number from the following rating scale:

1 --------------- 2 --------------- 3 --------------- 4 --------------- 5 --------------- 6 --------------- 7

<table>
<thead>
<tr>
<th>strongly disagree</th>
<th>neutral</th>
<th>strongly agree</th>
</tr>
</thead>
</table>

(1) Whenever I feel positive emotions, people can easily see exactly what I am feeling.

(2) I sometimes cry during sad movies.

(3) People often do not know what I am feeling.

(4) I laugh out loud when someone tells me a joke that I think is funny.

(5) It is difficult for me to hide my fear.

(6) When I’m happy, my feelings show.

(7) My body reacts very strongly to emotional situations.

(8) I’ve learned it is better to suppress my anger than to show it.

(9) No matter how nervous or upset I am, I tend to keep a calm exterior.

(10) I am an emotionally expressive person.

(11) I have strong emotions.

(12) I am sometimes unable to hide my feelings, even though I would like to.

(13) Whenever I feel negative emotions, people can easily see exactly what I am feeling.

(14) There have been times when I have not been able to stop crying even though I tried to stop.

(15) I experience my emotions very strongly.

(16) What I’m feeling is written all over my face.
APPENDIX 7: Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004)

Please indicate how often the following statements apply to you by writing the appropriate number from the scale below on the line beside each item:

1------------------2----------------------3---------------------4-------------------5
almost never  sometimes  about half the time  most of the time  almost always
(0-10%) (11-35%) (36-65%) (66-90%) (91-100%)

(1) I am clear about my feelings.               _____
(2) I pay attention to how I feel.               _____
(3) I experience my emotions as overwhelming and out of control.  _____
(4) I have no idea how I am feeling.            _____
(5) I have difficulty making sense out of my feelings.  _____
(6) I am attentive to my feelings.               _____
(7) I know exactly how I am feeling.            _____
(8) I care about what I am feeling.             _____
(9) I am confused about how I feel.             _____
(10) When I’m upset, I acknowledge my emotions. _____
(11) When I’m upset, I become angry with myself for feeling that way. _____
(12) When I’m upset, I become embarrassed for feeling that way. _____
(13) When I’m upset, I have difficulty getting work done. _____
(14) When I’m upset, I become out of control.   _____
(15) When I’m upset, I believe that I will remain that way for a long time. _____
(16) When I’m upset, I believe that I’ll end up feeling very depressed. _____
(17) When I’m upset, I believe that my feelings are valid and important. _____
(18) When I’m upset, I have difficulty focusing on other things. _____
(19) When I’m upset, I feel out of control.     _____
(20) When I’m upset, I can still get things done. _____
(21) When I’m upset, I feel ashamed with myself for feeling that way. _____
(22) When I’m upset, I know that I can find a way to eventually feel better. _____
(23) When I’m upset, I feel like I am weak.     _____
(24) When I’m upset, I feel like I can remain in control of my behaviours. _____
<table>
<thead>
<tr>
<th></th>
<th>almost never</th>
<th>sometimes</th>
<th>about half the time</th>
<th>most of the time</th>
<th>almost always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>(0-10%)</td>
<td>(11-35%)</td>
<td>(36-65%)</td>
<td>(66-90%)</td>
<td>(91-100%)</td>
</tr>
</tbody>
</table>

(25) When I’m upset, I feel guilty for feeling that way.

(26) When I’m upset, I have difficulty concentrating.

(27) When I’m upset, I have difficulty controlling my behaviours.

(28) When I’m upset, I believe that there is nothing I can do to make myself feel better.

(29) When I’m upset, I become irritated with myself for feeling that way.

(30) When I’m upset, I start to feel very bad about myself.

(31) When I’m upset, I believe that wallowing in it is all I can do.

(32) When I’m upset, I lose control over my behaviours.

(33) When I’m upset, I have difficulty thinking about anything else.

(34) When I’m upset, I take time to figure out what I’m really feeling.

(35) When I’m upset, it takes me a long time to feel better.

(36) When I’m upset, my emotions feel overwhelming.
APPENDIX 8: Acceptance and Action Questionnaire–II (Bond et al., 2011)

Below you will find a list of statements. Please rate how true each statement is for you by circling a number next to it. Use the scale below to make your choice.

<table>
<thead>
<tr>
<th></th>
<th>never true</th>
<th>very seldom true</th>
<th>seldom true</th>
<th>sometimes true</th>
<th>frequently true</th>
<th>almost always true</th>
<th>always true</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. My painful experiences and memories make it difficult for me to live a life that I would value.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>2. I’m afraid of my feelings.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>3. I worry about not being able to control my worries and feelings.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>4. My painful memories prevent me from having a fulfilling life.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>5. Emotions cause problems in my life.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>6. It seems like most people are handling their lives better than I am.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>7. Worries get in the way of my success</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
</tbody>
</table>
APPENDIX 9: Social Safeness and Pleasure Scale (Gilbert et al., 2009)

We are interested in how people experience pleasure, positive feelings and emotions in social situations. Below are a series of statements about how you may feel in various situations. Please read each statement carefully and circle the number that best describes how you feel.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Almost never</th>
<th>Almost all the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>I feel content within my relationships</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>2.</td>
<td>I feel easily soothed by those around me</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>3.</td>
<td>I feel connected to others</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>4.</td>
<td>I feel part of something greater than myself</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>5.</td>
<td>I have a sense of being cared about in the world</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>6.</td>
<td>I feel secure and wanted</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>7.</td>
<td>I feel a sense of belonging</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>8.</td>
<td>I feel accepted by people</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>9.</td>
<td>I feel understood by people</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>10.</td>
<td>I feel a sense of warmth in my relationships with people</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>11.</td>
<td>I find it easy to feel calmed by people close to me</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
**APPENDIX 10: Flowchart of the Laboratory Stressors Procedure**

*Figure 10.1.* Flowchart of the laboratory stressors procedure (from Chapter 4, Experiment 1). Mean cell values for each arm are shown in Table 10.1 and Table 10.2 (as the experiment was a within subjects design the HADs scores [cell 1] were the same across the two arms; the qualitative results of the post-task questionnaire [cell 8a and 8b] are described in section 4.3.3.1 and 4.3.3.2).
Table 10.1
Mean values for each cell from the flowchart diagram for the virtual reality maze arm (figure 10.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Pre-task baseline</th>
<th>POMS-SF</th>
<th>Virtual reality maze</th>
<th>Recovery</th>
<th>POMS-SF</th>
</tr>
</thead>
<tbody>
<tr>
<td>3a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Total Score</td>
<td>5.07 (8.11)</td>
<td></td>
<td></td>
<td></td>
<td>7.73 (9.40)</td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>76.11 (10.20)</td>
<td></td>
<td>75.00 (8.99)</td>
<td>76.08 (9.03)</td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.71 (3.06)</td>
<td></td>
<td>3.18 (2.16)</td>
<td>2.97 (2.13)</td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.24 (1.18)</td>
<td></td>
<td>6.91 (0.74)</td>
<td>7.18 (0.84)</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 7.87 (SD = 3.93); HADS-D mean = 3.87 (SD = 3.56). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.

Table 10.2
Mean values for each cell from the flowchart diagram for the speech task arm (figure 10.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Pre-task baseline</th>
<th>POMS-SF</th>
<th>Speech Task Preparation</th>
<th>Speech Task Presentation</th>
<th>Recovery</th>
<th>POMS-SF</th>
</tr>
</thead>
<tbody>
<tr>
<td>3b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5c</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Total Score</td>
<td>6.20 (10.70)</td>
<td></td>
<td>86.99 (11.57)</td>
<td>89.92 (9.65)</td>
<td>76.60 (10.23)</td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>77.05 (9.71)</td>
<td></td>
<td>86.99 (11.57)</td>
<td>89.92 (9.65)</td>
<td>76.60 (10.23)</td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>1.78 (1.26)</td>
<td></td>
<td>3.77 (2.11)</td>
<td>4.50 (2.93)</td>
<td>3.56 (2.49)</td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>6.98 (1.02)</td>
<td></td>
<td>6.71 (1.02)</td>
<td>--</td>
<td>7.44 (0.93)</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 7.87 (SD = 3.93); HADS-D mean = 3.87 (SD = 3.56). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
APPENDIX 11: Flowchart of the Speech Task Anticipation Procedure

Figure 11.1. Flowchart of the speech task anticipation procedure (from Chapter 4, Experiment 2). Mean cell values are shown in Table 11.1.
Table 11.1
Mean values for each cell from the flowchart diagram for the speech task anticipation procedure (figure 11.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Pre-task baseline</th>
<th>POMS-SF</th>
<th>Speech Task Preparation</th>
<th>Anticipation</th>
<th>POMS-SF</th>
<th>Speech Task Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>8.90 (12.07)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>18.05 (20.11)</td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>78.74 (9.85)</td>
<td>90.44 (12.79)</td>
<td>84.91 (11.19)</td>
<td>91.75 (10.35)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>1.43 (1.04)</td>
<td>3.32 (1.65)</td>
<td>3.73 (1.77)</td>
<td>4.42 (2.26)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.27 (1.07)</td>
<td>6.52 (1.08)</td>
<td>7.19 (1.00)</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 8.25 (SD = 3.40); HADS-D mean = 3.75 (SD = 2.94). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability
APPENDIX 12: Mindful Breathing Script
(abridged from Kabat-Zinn, 2005)

Over the next 5 minutes you will be guided through a mindful breathing practice. Before we begin gently rest your spare hand over the green key on the keyboard in front of you. Now, as you sit in a comfortable, upright position close your eyes. Bring your awareness to your breath and body as a whole. Rest here for a moment in time, and allow your attention to alight gently on the breath, as it moves in and out of the body.

Feel the breath sensation at the tip of your nose where the passage of air enters your body. And as you feel your breath pass from your nose into your lungs, press the green key on the keyboard each time.

(30-second pause)

Continue to press the green key as the in-breath begins. Keep your awareness on your breath so that you are fully present for the full duration of the in-breath and the full duration of the out-breath. If you notice that you mind wanders from the breath, without giving yourself a hard time, gently guide your attention back to it. Without pulling the breath in or pushing the breath out. Without any forcing whatsoever. Just allowing the breath to be as it is, moment-by-moment, and breath, by breath, by breath.

(90-second pause)

Continuing to press the key, allow your attention to include the full embracing of each and every breath. Gently, lightly with mindfulness, so the breath is known, felt, experienced in the moment of its arising and full unfolding of the in-breath and falling away in the out-breath. Remember, if you feel your mind wandering gently guide it back to your breath.

(60-second pause)

So just this very moment with the breath moving in, the feeling, sensing, knowing of the breath, as it is, moment by moment, breath by breath, sitting here, resting in awareness itself. Featuring in this moment. This breath.

(90-second pause)

Continue to sit here peacefully concentrating on your breathing until the researcher tells you otherwise.
To begin with, sit in a comfortable, upright position. Open the folder that contains the picture of the loving individual you have experience of feeling loving-kindness for. As you look at this picture, bring into your heart the feelings of deep affection, appreciation or positive connection you have.

Rest in the warmth and radiance of this feeling of loving-kindness as you gaze at the picture. Feel the loving-kindness from the centre of your chest. Give yourself over to these feelings and qualities of kindness and love. Take in the whole aura or field of the love you have experienced. Right here, right now. Breathe in these feelings, bathe in them. Rest in the warm and radiance of their heartfelt embrace of you. Just as you are.

(45-second pause)

Feeling the loving-kindness from the centre of your chest, extend warm wishes of loving-kindness to this loved one. Resting here in this field of loving-kindness, silently say to yourself: May this person be at ease... May they be content with their life... May they be joyful... May they be safe and secure.

(15-second pause)

Gently, at your own pace, over and over. Inwardly whispering, inwardly hearing, feeling, sensing, affirming: May this person be at ease... May they be content with their life... May they be joyful... May they be safe and secure.

(60-second pause)

May this person be at ease... May they be content with their life... May they be joyful... May they be safe and secure.

(30-second pause)

Allow yourself to bask in the feelings of loving-kindness as best as you can whilst you gaze at the picture. Continue to sit there peacefully feeling the loving-kindness until the researcher tells you otherwise.
APPENDIX 14: Neutral Listening Script
(replicated from Arch & Craske, 2006)

You will now be guided through a brief 5-minute exercise. Sit in a comfortable upright position with your eyes closed. Simply think about whatever comes to mind. Let your mind wander freely without trying to focus on anything in particular.

(45-second pause)

You may find that your mind becomes very active, with thoughts, memory or plans. Allow this to happen and give attention to whatever comes to mind.

(45-second pause)

As you sit here indulging in your mental activity. Fully embrace your daydream. Simply think about whatever comes to mind.

(30-second pause)

Let your mind wander, let your mind go wherever it wants to go, without restricting it, or challenging it, or pushing it in any direction. Simply get involved with whatever your mind wants to do.

(30-second pause)

As you sit here continuously follow these thoughts, fantasies, ideas and concerns. Mindlessly follow these patterns of mental activity.

(30-second pause)

Continue to let your mind wander freely without focusing on anything in particular.

(10-second pause)

Continue to let your thoughts wander until the researcher tells you to do the next task.
APPENDIX 15: Flowchart of the Emotion Regulation Strategies Procedure

Figure 15.1. Flowchart of the emotion regulation strategies procedure (from Chapter 5). Mean cell values are shown in Tables 15.1-15.5 (the qualitative results of the post-task questionnaire [cell 9] are described in section 5.4.3.2).
Table 15.1
Mean values for each cell from the flowchart diagram for the smiling arm of the emotion regulation strategies procedure (figure 15.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>POMS-SF 3</th>
<th>Speech Task Preparation 4</th>
<th>Smiling 6a</th>
<th>POMS-SF 7a</th>
<th>Speech Task Presentation 8a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>5.50 (11.93)</td>
<td>12.60 (16.28)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>82.48 (12.99)</td>
<td>91.63 (12.97)</td>
<td>83.72 (11.35)</td>
<td>91.75 (10.35)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.22 (1.35)</td>
<td>4.45 (1.97)</td>
<td>4.56 (1.94)</td>
<td>5.93 (2.52)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.14 (1.13)</td>
<td>6.88 (1.12)</td>
<td>7.40 (1.04)</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 6.65 (SD = 4.61); HADS-D mean = 1.90 (SD = 1.59). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.

Table 15.2
Mean values for each cell from the flowchart diagram for the mindful breathing arm of the emotion regulation strategies procedure (figure 15.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>POMS-SF 3</th>
<th>Speech Task Preparation 4</th>
<th>Mindful breathing 6b</th>
<th>POMS-SF 7b</th>
<th>Speech Task Presentation 8b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>8.00 (10.13)</td>
<td>11.40 (15.62)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>80.95 (11.81)</td>
<td>90.25 (13.21)</td>
<td>80.80 (12.52)</td>
<td>92.76 (15.35)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.27 (1.33)</td>
<td>4.77 (4.16)</td>
<td>4.45 (3.43)</td>
<td>6.05 (4.28)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.18 (0.75)</td>
<td>6.80 (0.71)</td>
<td>7.60 (0.93)</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 6.45 (SD = 3.32); HADS-D mean = 2.70 (SD = 2.16). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
### Table 15.3
Mean values for each cell from the flowchart diagram for the loving-kindness arm of the emotion regulation strategies procedure (figure 15.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline</th>
<th>POMS-SF</th>
<th>Speech Task Preparation</th>
<th>Loving-kindness</th>
<th>POMS-SF</th>
<th>Speech Task Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>2</td>
<td>4.70 (8.33)</td>
<td>6c</td>
<td>12.30 (11.56)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>84.25 (11.31)</td>
<td>92.02 (11.56)</td>
<td>82.80 (11.25)</td>
<td>94.93 (12.86)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.02 (1.52)</td>
<td>3.84 (2.16)</td>
<td>3.78 (2.37)</td>
<td>5.31 (2.55)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.15 (0.87)</td>
<td>6.85 (0.81)</td>
<td>7.34 (0.72)</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 6.20 (SD = 3.68); HADS-D mean = 2.15 (SD = 1.73). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.

### Table 15.4
Mean values for each cell from the flowchart diagram for the neutral listening arm of the emotion regulation strategies procedure (figure 15.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline</th>
<th>POMS-SF</th>
<th>Speech Task Preparation</th>
<th>Neutral listening</th>
<th>POMS-SF</th>
<th>Speech Task Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>2</td>
<td>3.90 (10.55)</td>
<td>6d</td>
<td>14.00 (15.58)</td>
<td>7d</td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>79.90 (13.94)</td>
<td>89.17 (12.00)</td>
<td>79.29 (12.93)</td>
<td>95.91 (12.84)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.10 (1.45)</td>
<td>3.97 (2.33)</td>
<td>3.84 (2.46)</td>
<td>4.99 (3.10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>6.99 (1.13)</td>
<td>6.67 (1.19)</td>
<td>7.30 (1.08)</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 7.10 (SD = 3.40); HADS-D mean = 2.45 (SD = 2.14). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
Table 15.5
Mean values for each cell from the flowchart diagram for the resting quietly arm of the emotion regulation strategies procedure (figure 15.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline</th>
<th>POMS-SF</th>
<th>Speech Task Preparation</th>
<th>Resting quietly</th>
<th>POMS-SF</th>
<th>Speech Task Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>7.72 (10.65)</td>
<td>19.57 (15.91)</td>
<td>94.97 (13.78)</td>
<td>6e</td>
<td>8e</td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>79.27 (10.08)</td>
<td>90.44 (12.79)</td>
<td>84.92 (11.19)</td>
<td>7e</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>1.53 (1.34)</td>
<td>3.32 (1.65)</td>
<td>3.73 (1.77)</td>
<td>8e</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.23 (0.92)</td>
<td>6.47 (1.04)</td>
<td>7.16 (1.00)</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: HADS-A mean = 7.45 ($SD = 3.68$); HADS-D mean = 2.72 ($SD = 2.59$). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
Figure 16.1. Flowchart of the expressive regulation procedure (from Chapter 6). Mean cell values are shown in Table 16.1 and Table 16.2.
### Table 16.1
Mean values for each cell from the flowchart diagram for the speech task arm of the expressive regulation procedure (figure 16.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>ER Task T1 3</th>
<th>POMS-SF 5a</th>
<th>Speech Task Preparation 6a</th>
<th>POMS-SF 7a</th>
<th>ER Task 2 8a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>3.89 (10.17)</td>
<td>14.11 (14.24)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>78.55 (9.90)</td>
<td>76.92 (8.43)</td>
<td>83.34 (12.75)</td>
<td>75.58 (9.14)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.93 (2.03)</td>
<td>5.10 (2.75)</td>
<td>5.75 (2.74)</td>
<td>4.99 (2.48)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.28 (1.04)</td>
<td>7.44 (0.88)</td>
<td>7.11 (0.97)</td>
<td>7.41 (0.91)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Expressive Ability</td>
<td>0.15 (0.20)</td>
<td>0.12 (0.18)</td>
<td>0.12 (0.18)</td>
<td>0.16 (0.20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Suppressive Ability</td>
<td>0.12 (0.18)</td>
<td>0.12 (0.18)</td>
<td>0.12 (0.18)</td>
<td>0.16 (0.20)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note.** Standard deviations are reported in parentheses. Cell 1: AAQ-II = 37.50 (SD = 6.86); BDI-II = 8.29 (SD = 9.05); BEQ Mean = 4.48 (SD = 0.81); GAD-7 = 4.92 (SD = 4.03); SSPS = 31.89 (SD = 8.67). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.

### Table 16.2
Mean values for each cell from the flowchart diagram for the reading task arm of the expressive regulation procedure (figure 16.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>ER Task T1 3</th>
<th>POMS-SF 5b</th>
<th>Reading Task 6b</th>
<th>POMS-SF 7b</th>
<th>ER Task 2 8b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>2.62 (9.52)</td>
<td>9.78 (12.96)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>74.45 (11.07)</td>
<td>74.06 (10.79)</td>
<td>72.42 (9.73)</td>
<td>72.19 (9.48)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.13 (1.42)</td>
<td>3.98 (1.91)</td>
<td>4.15 (2.17)</td>
<td>4.07 (2.26)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.64 (0.82)</td>
<td>7.62 (0.68)</td>
<td>7.64 (0.85)</td>
<td>7.62 (0.78)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Expressive Ability</td>
<td>0.12 (0.17)</td>
<td>0.12 (0.17)</td>
<td>0.12 (0.17)</td>
<td>0.14 (0.20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Suppressive Ability</td>
<td>0.09 (0.03)</td>
<td>0.09 (0.03)</td>
<td>0.09 (0.03)</td>
<td>0.11 (0.19)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note.** Standard deviations are reported in parentheses. Cell 1: AAQ-II = 39.38 (SD = 7.47); BDI-II = 5.59 (SD = 6.27); BEQ Mean = 4.53 (SD = 0.87); GAD-7 = 4.68 (SD = 4.79); SSPS = 34.51 (SD = 8.41). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
APPENDIX 17: Flowchart of the Emotion Recognition Procedure

Figure 17.1. Flowchart of the emotion recognition procedure (from Chapter 7, Experiment 1). Mean cell values are shown in Table 17.1 and Table 17.2.
Table 17.1
Mean values for each cell from the flowchart diagram for the speech task arm of the emotion recognition procedure (figure 17.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline</th>
<th>Multimorph Task T1</th>
<th>POMS-SF</th>
<th>Speech Task Preparation</th>
<th>POMS-SF</th>
<th>Multimorph Task T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>2</td>
<td>3</td>
<td>5a</td>
<td>6a</td>
<td>7a</td>
<td>8a</td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>71.65 (7.87)</td>
<td>71.25 (8.28)</td>
<td>83.43 (12.40)</td>
<td>9.60 (11.52)</td>
<td>71.99 (8.54)</td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>1.55 (1.29)</td>
<td>1.95 (1.54)</td>
<td>2.91 (1.81)</td>
<td>7.13 (0.93)</td>
<td>2.45 (1.73)</td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.48 (0.93)</td>
<td>7.35 (0.98)</td>
<td>7.38 (1.02)</td>
<td>22.32 (3.38)</td>
<td>7.41 (0.92)</td>
<td></td>
</tr>
<tr>
<td>Mean Emotional Sensitivity</td>
<td>23.87 (3.53)</td>
<td>22.62 (3.06)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Emotional Accuracy</td>
<td>89.67 (8.03)</td>
<td>91.53 (6.29)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Cell 1: BDI-II = 6.23 (SD = 5.61); BEQ Mean = 4.60 (SD = 0.89); DERS = 83.13 (SD = 18.59); GAD-7 = 3.80 (SD = 3.01); SSPS = 33.13 (SD = 6.43). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.

Table 17.2
Mean values for each cell from the flowchart diagram for the reading task arm of the emotion recognition procedure (figure 17.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline</th>
<th>Multimorph Task T1</th>
<th>POMS-SF</th>
<th>Reading Task</th>
<th>POMS-SF</th>
<th>Multimorph Task T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>2</td>
<td>3</td>
<td>5b</td>
<td>6b</td>
<td>7b</td>
<td>8b</td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>74.19 (9.28)</td>
<td>74.20 (9.56)</td>
<td>75.12 (9.47)</td>
<td>3.75 (8.52)</td>
<td>74.29 (9.82)</td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>1.69 (1.18)</td>
<td>2.06 (1.40)</td>
<td>2.53 (1.77)</td>
<td>2.32 (1.65)</td>
<td>7.38 (1.02)</td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.58 (1.13)</td>
<td>7.34 (1.01)</td>
<td>7.57 (1.03)</td>
<td>22.62 (3.06)</td>
<td>90.39 (5.75)</td>
<td></td>
</tr>
<tr>
<td>Mean Emotional Sensitivity</td>
<td>22.35 (3.54)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Emotional Accuracy</td>
<td>87.31 (7.65)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Cell 1: BDI-II = 6.80 (SD = 6.06); BEQ Mean = 4.60 (SD = 0.73); DERS = 79.97 (SD = 20.28); GAD-7 = 4.23 (SD = 3.96); SSPS = 32.95 (SD = 7.17). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
Figure 18.1. Flowchart of the adapted emotion recognition procedure (from Chapter 7, Experiment 2). Mean cell values are shown in Table 18.1 and Table 18.2.
## Table 18.1

Mean values for each cell from the flowchart diagram for the speech task arm of the adapted emotion recognition procedure (figure 18.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>Multimorph Task T1 3</th>
<th>POMS-SF 6a</th>
<th>Speech Task Preparation 7a</th>
<th>Multimorph Task T2 9a</th>
<th>Speech Task Preparation 10a</th>
<th>Multimorph Task T2 11a</th>
<th>POMS-SF 12a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>5.72 (11.90)</td>
<td>71.54 (8.55)</td>
<td>71.29 (8.57)</td>
<td>77.37 (10.51)</td>
<td>71.61 (8.70)</td>
<td>77.16 (10.06)</td>
<td>72.20 (8.34)</td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>71.88 (8.92)</td>
<td>71.54 (8.55)</td>
<td>78.78 (11.27)</td>
<td>71.29 (8.57)</td>
<td>77.37 (10.51)</td>
<td>71.61 (8.70)</td>
<td>77.16 (10.06)</td>
<td>72.20 (8.34)</td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.86 (1.55)</td>
<td>3.06 (1.73)</td>
<td>4.09 (1.62)</td>
<td>4.20 (1.87)</td>
<td>4.01 (1.71)</td>
<td>4.27 (2.10)</td>
<td>4.11 (1.66)</td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.54 (1.00)</td>
<td>7.35 (0.92)</td>
<td>7.66 (0.83)</td>
<td>7.35 (0.96)</td>
<td>7.60 (0.83)</td>
<td>7.34 (0.89)</td>
<td>7.68 (0.73)</td>
<td></td>
</tr>
<tr>
<td>Emotional Sensitivity</td>
<td>23.65 (3.87)</td>
<td>21.51 (5.83)</td>
<td>22.51 (8.69)</td>
<td>23.28 (5.80)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional Accuracy</td>
<td>90.24 (7.45)</td>
<td>92.71 (10.32)</td>
<td>86.98 (15.11)</td>
<td>92.19 (11.18)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: BDI-II = 8.81 (SD = 7.20); DERS = 78.91 (SD = 19.72); GAD-7 = 4.09 (SD = 3.08); SSPS = 33.97 (SD = 7.84). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.

## Table 18.2

Mean values for each cell from the flowchart diagram for the reading task arm of the adapted emotion recognition procedure (figure 18.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>Multimorph Task T1 3</th>
<th>POMS-SF 5b</th>
<th>Reading Task 6b</th>
<th>Multimorph Task T2 7b</th>
<th>Reading Task 8b</th>
<th>Multimorph Task T2 9b</th>
<th>Reading Task 10b</th>
<th>Multimorph Task T2 11b</th>
<th>POMS-SF 12b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>6.84 (14.71)</td>
<td>72.13 (8.88)</td>
<td>72.34 (8.32)</td>
<td>72.99 (9.40)</td>
<td>72.63 (8.93)</td>
<td>73.06 (8.63)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>74.00 (4.49)</td>
<td>72.13 (9.30)</td>
<td>72.34 (8.32)</td>
<td>72.99 (9.40)</td>
<td>72.63 (8.93)</td>
<td>73.06 (8.63)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.79 (1.75)</td>
<td>3.72 (2.18)</td>
<td>3.28 (2.16)</td>
<td>3.30 (2.25)</td>
<td>3.36 (2.26)</td>
<td>3.36 (2.32)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.44 (0.83)</td>
<td>7.76 (0.82)</td>
<td>7.36 (0.73)</td>
<td>7.34 (0.79)</td>
<td>7.47 (0.81)</td>
<td>7.45 (0.81)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional Sensitivity</td>
<td>23.99 (4.67)</td>
<td>23.01 (5.23)</td>
<td>22.53 (5.13)</td>
<td>20.89 (4.90)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional Accuracy</td>
<td>89.72 (8.22)</td>
<td>90.10 (11.87)</td>
<td>92.71 (12.66)</td>
<td>93.23 (10.25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. Cell 1: BDI-II = 10.22 (SD = 9.27); DERS = 81.41 (SD = 26.16); GAD-7 = 4.88 (SD = 4.69); SSPS = 31.81 (SD = 8.81). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
APPENDIX 19: Flowchart of the Rating Faces Task Procedure

![Flowchart of the Rating Faces Procedure](image)

*Figure 19.1.* Flowchart of the rating faces task procedure (from Chapter 8, Experiment 1). Mean cell values are shown in Table 19.1.

Table 19.1

Mean values for each cell from the flowchart diagram for the speech task arm of the rating faces task procedure (figure 19.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Questionnaires</th>
<th>Baseline</th>
<th>Rating Faces Task</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean POMS-SF Total</td>
<td>3.06 (13.18)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SSPS Total</td>
<td>35.59 (6.52)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>71.25 (12.66)</td>
<td>70.77 (11.35)</td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.06 (1.73)</td>
<td>3.11 (2.48)</td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.36 (1.04)</td>
<td>7.37 (0.88)</td>
<td></td>
</tr>
<tr>
<td>Mean Attractiveness Rating</td>
<td></td>
<td></td>
<td>3.54 (1.44)</td>
</tr>
<tr>
<td>Mean Familiarity Rating</td>
<td></td>
<td></td>
<td>2.38 (1.57)</td>
</tr>
<tr>
<td>Mean Distinctiveness Rating</td>
<td></td>
<td></td>
<td>3.45 (1.50)</td>
</tr>
<tr>
<td>Mean Affiliation Rating</td>
<td></td>
<td></td>
<td>3.46 (1.45)</td>
</tr>
<tr>
<td>Mean Emotion Rating</td>
<td></td>
<td></td>
<td>3.53 (1.39)</td>
</tr>
</tbody>
</table>

*Note.* Standard deviations are reported in parentheses. HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
APPENDIX 20: Flowchart of the Rating Faces Task Stressor Procedure

**Figure 20.1.** Flowchart of the rating faces task stressor procedure (from Chapter 8, Experiment 2). Mean cell values are shown in Table 20.1 and Table 20.2.
### Table 20.1

Mean values for each cell from the flowchart diagram for the speech task arm of the rating faces task procedure (figure 20.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>Rating Faces Task T1 3</th>
<th>POMS-SF 5a</th>
<th>Speech Task Preparation 6a</th>
<th>Rating Faces Task T2 7a</th>
<th>Speech Task Preparation 8a</th>
<th>Rating Faces Task T2 9a</th>
<th>Speech Task Preparation 10a</th>
<th>Rating Faces Task T2 11a</th>
<th>POMS-SF 12a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>76.15 (10.88)</td>
<td>74.16 (10.63)</td>
<td>84.32 (13.71)</td>
<td>75.94 (10.82)</td>
<td>83.69 (12.95)</td>
<td>76.14 (11.20)</td>
<td>84.27 (13.68)</td>
<td>76.53 (11.07)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.34 (1.48)</td>
<td>3.59 (1.77)</td>
<td>5.04 (1.87)</td>
<td>4.53 (1.73)</td>
<td>4.70 (1.88)</td>
<td>4.40 (1.75)</td>
<td>4.65 (1.92)</td>
<td>4.51 (1.87)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.37 (1.17)</td>
<td>7.26 (0.93)</td>
<td>7.00 (0.95)</td>
<td>7.47 (0.87)</td>
<td>7.06 (0.76)</td>
<td>7.33 (0.88)</td>
<td>7.22 (0.79)</td>
<td>7.32 (1.00)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attractiveness Rating</td>
<td>3.21 (1.39)</td>
<td>3.38 (1.41)</td>
<td>3.19 (1.36)</td>
<td>3.19 (1.36)</td>
<td>3.23 (1.46)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Familiarity Rating</td>
<td>2.19 (1.27)</td>
<td>2.22 (1.31)</td>
<td>2.34 (1.43)</td>
<td>2.22 (1.43)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distinctiveness Rating</td>
<td>3.19 (1.44)</td>
<td>3.39 (1.46)</td>
<td>3.27 (1.49)</td>
<td>3.46 (1.39)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affiliation Rating</td>
<td>3.10 (1.45)</td>
<td>3.22 (1.45)</td>
<td>3.09 (1.37)</td>
<td>3.08 (1.42)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion Rating</td>
<td>3.48 (1.44)</td>
<td>3.45 (1.35)</td>
<td>3.47 (1.34)</td>
<td>3.45 (1.40)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note.** Standard deviations are reported in parentheses. Cell 1: BDI-II = 7.83 (SD = 6.49); GAD-7 = 4.54 (SD = 3.38); SSPS = 30.23 (SD = 9.52). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.
Table 20.2
Mean values for each cell from the flowchart diagram for the reading task arm of the rating faces task procedure (figure 20.1)

<table>
<thead>
<tr>
<th>Flowchart Cell</th>
<th>Baseline 2</th>
<th>Rating Faces Task T1 3</th>
<th>POMS-SF 5b</th>
<th>Reading Task 6b</th>
<th>Rating Faces Task T2 7b</th>
<th>Reading Task 8b</th>
<th>Rating Faces Task T2 9b</th>
<th>Reading Task 10b</th>
<th>Rating Faces Task T2 11b</th>
<th>POMS-SF 12b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Total Score</td>
<td>3.84 (13.03)</td>
<td>3.68 (8.64)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HR (bpm)</td>
<td>79.20 (11.20)</td>
<td>77.14 (9.79)</td>
<td>76.52 (8.72)</td>
<td>75.90 (9.54)</td>
<td>77.06 (9.18)</td>
<td>77.01 (9.55)</td>
<td>77.48 (9.25)</td>
<td>76.47 (9.18)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean SCL (µS)</td>
<td>2.51 (1.22)</td>
<td>3.58 (1.74)</td>
<td>4.20 (1.98)</td>
<td>3.93 (1.87)</td>
<td>3.86 (2.00)</td>
<td>3.88 (1.94)</td>
<td>3.86 (2.08)</td>
<td>3.88 (2.09)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean HF-HRV (ms²)</td>
<td>7.27 (0.81)</td>
<td>7.10 (0.72)</td>
<td>7.46 (0.67)</td>
<td>7.18 (0.68)</td>
<td>7.21 (0.75)</td>
<td>7.03 (0.67)</td>
<td>7.13 (0.63)</td>
<td>7.01 (0.72)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attractiveness Rating</td>
<td>3.46 (1.43)</td>
<td>3.50 (1.50)</td>
<td>3.49 (1.37)</td>
<td>3.60 (1.41)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Familiarity Rating</td>
<td>2.37 (1.31)</td>
<td>2.18 (1.25)</td>
<td>2.29 (1.29)</td>
<td>2.44 (1.45)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distinctiveness Rating</td>
<td>3.37 (1.41)</td>
<td>3.47 (1.45)</td>
<td>3.51 (1.45)</td>
<td>3.67 (1.50)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Affiliation Rating</td>
<td>3.16 (1.37)</td>
<td>3.30 (1.26)</td>
<td>3.26 (1.35)</td>
<td>3.42 (1.37)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotion Rating</td>
<td>3.47 (1.35)</td>
<td>2.40 (1.22)</td>
<td>3.36 (1.38)</td>
<td>3.41 (1.39)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Standard deviations are reported in parentheses. Cell 1: BDI-II = 5.76 (SD = 6.64); GAD-7 = 2.70 (SD = 2.89); SSPS = 33.27 (SD = 8.34). HR = heart rate; SCL = skin conductance level; HF-HRV = high-frequency heart rate variability.


REFERENCES


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