Anger Induction and Ambient Interventions: Effects on Cardiovascular Activity and Frontal EEG Asymmetry

by

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DEDICATIONS

This thesis is dedicated to my husband Dr Pascual Marqués

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ABSTRACT

Background and aims: The experience of anger could affect cardiovascular (CV) and electro-encephalographic (EEG) parameters but such parameters could vary within the motivational context. Although models of motivational contexts were proposed by CV literature as challenge/threat (Blascovich & Tomaka, 1996) and by frontal EEG asymmetry literature as approach/avoidance (Harmon-Jones, 2004a) little is known whether a negative emotion such as anger could be indexed by CV and EEG responses within motivational contexts. Anger in a threat context may be particularly detrimental for health due to low control compared to anger in a challenge context, where control is high. Hence, the aim of the research was twofold: 1. to investigate how a motivational context (challenge vs. threat) influences the cardiovascular system and frontal EEG asymmetry during anger induction protocols and 2. to analyse the efficacy of ambient interventions (music, light) to reduce the impact of anger on cardiovascular responses. Affective computing through the use of ambient intelligence technology could be used to promote positive emotion or to ameliorate negative moods.

Method: There were two anger induction protocols within the thesis. Firstly, anger was manipulated using an experimenter effect (i.e., rude vs. polite experimenter). Participants were exposed to a computer-based problem-solving task under conditions of control and no control which represented the motivational contexts of challenge/threat. Secondly, anger was induced by exposing participants to a time constrained driving schedule on a simulated route with financial penalties for any delays to arrive to the destination. Motivation was manipulated by exposing participants to traffic delays at an early (challenge) and later point (threat) on a simulated driving route. STAXI-2 (Spielberg, 1999) was used to measure anger states and motivation was measured by Confidence and Perceived Control Scale from Dundee Stress State Questionnaire (Matthews & Desmond, 1998). Psychophysiological variables included: blood pressure (BP), cardiovascular impedance (ICG), frontal EEG asymmetry, and facial electromyography (fEMG).

Results: The cardiovascular and EEG results of the present thesis pointed to a circumplex model of anger with quadruplet facets along cardiovascular responses to challenge/threat contexts in conjunction with approach/avoidance tendencies where a threat motivation with avoidance was indexed by increased blood pressure and cardiac output and by greater right frontal activation. The difference in the approach-threat responses was the activation of the left hemisphere. The challenge-avoidance state was defined by increased total peripheral resistance (TPR), systolic blood pressure (SBP), heart rate (HR), mean arterial pressure (MAP) and greater right frontal hemisphere activation. No frontal asymmetric activity was identified in the challenge-approach, but increased TPR, SBP, HR and MAP were observed. The ambient intervention results suggested that cardiovascular responses (e.g., SBP) could be reduced by low activation music or blue ambient light.

Discussion and conclusions: Anger in the context of challenge can be distinguished from anger in the context of threat via a specific pattern of CV (systolic BP) and EEG measures (frontal peripheral brain site). Ambient interventions (low activation music or blue light) could be factors in modulating physiological reactions while driving; discrepancies between self-report measures and physiological responses, low sensitivity of impendence data to manipulations and low impact of various colour ambient lights on cardiovascular responses were addressed within a theoretical and methodological.

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ABBREVIATIONS

Physiological measures

BPM	Breaths/min
CO	Cardiac Output
CV	Cardiovascular
DBP	Diastolic Blood Pressure
DC	Direct Current
ECG	Electrocardiography
EEG	Electroencephalography
EMG	Electromyography
fEMG	Facial electromyography
FFT	Fast Fourier transformer
FPA	Finger pulse amplitude
FPTT	Finger pulse transit time
FT	Finger temperature
HI	Heather index
HPA	Hypothalamic pituitary adrenal
HR	Heart rate
HRV	Heart rate variability
ICG	Impedance cardiography
LH	Left hemisphere
LVET	Left ventricular ejection time
MAP	Mean Arterial Pressure
MSD	Mean successive differences
PAC	Pituitary adrenocortical response
PEP	Pre-ejection period
RH	Right hemisphere
SAM	Sympathetic-adrenomedullary response
SBP	Systolic blood pressure
SV	Stroke volume
TPR	Total peripheral resistance
TWA	T-wave amplitude
VC	Ventricular contractility

Self-report measures

DSSQ	Dundee Stress State Questionnaire
STAXI 2	State-Trait Anger Expression Inventory 2

Conditions

BL1	Blue light condition 1 with priming
BL2	Blue light condition 2 without priming
GB	Green blue
HA	High arousal
LA	Low arousal
MI	Mood induction
NV	Negative valence
PV	Positive valence
RB	Red blue
RG	Red green
RGB	Red green blue
TJ1	Traffic jam 1
TJ2	Traffic jam 2

CHAPTER 1
INTRODUCTION

Anger and its behavioural consequences are ubiquitous in human beings. An exploration of the nature of anger with respect to its psychological and physiological correlates represents the core of the present thesis. This introduction defines the concept of anger with reference to theoretical models. Thereafter, health implications are discussed including an examination of the relation between anger, aggression and hostility, and the risk of coronary heart disease. The core analysis in this thesis concentrates on psychophysiological measures of anger with a focus on motivational disposition as a modulator of anger. The introductory chapter ends with a discussion on technological interventions from the domain of affective computing that serve as anger countermeasures, a debate on ambient interventions as manipulators and controllers of anger, finally an outline of the hypothesised physiological model of anger.

1. Anger and Motivation – Concepts and Psychophysiology

1.1. Anger – Theoretical Models

Within the literature, the term emotion is used interchangeably with the terms affect and feelings (e.g., Isen, 2000) or as a unique concept (Fredrickson, 2001). According to Frijda (2000), for most theorists emotions are in essence feelings. However, these feelings occur within the context of the events that elicit them (Ellsworth & Scherer, 2003, p. 575). For example, different kinds of events can elicit negatively valence feelings and the feelings that emerge would be differentiated by context (Carver & Harmon-Jones, 2009). This binding of event type to feeling expression can be understood in terms of appraisals (Scherer, Schorr, & Johnstone, 2001) or scripts (Izard, 2007; Russell, 2003). The binding between affect and type of event allows us to understand the meaning of valence. Anger may be construed as a feeling characterised by negative valence caused by the blockage of movement toward a desired goal (Berkowitz, 1993; Depue & Zald, 1993; Dollard, Doob, Miller, Mowrer, & Sears, 1939; Lewis, Alessandri, & Sullivan, 1990). It has been suggested that no additional appraisal is necessary for anger to occur

(Berkowitz & Harmon-Jones, 2004), nonetheless many theorists do incorporate other constituent elements into the appraisal of anger. For example, anger may occur when one person undergoes the experience of being intentionally hurt by another person (Frijda, 1986, p. 198). Anger has also been associated with displeasure and an urge to blame on someone else's actions (Ortony, Clore, & Collins, 1990, p. 147). In fact, anger has been portrayed as a reaction to a displeasing violation of what the person considers acceptable behaviour (Frijda, 1986, pp.198–199; Mascolo, Harkins, & Harakal, 2000, p. 137; Ortony et al., 1990, p. 152–153). Such violation of standards has been described by Ortony et al. (1990) as a thwarting of "interest" goals. Notwithstanding the different elements in the appraisal of anger, there is consensus in that anger is connected to an approach motivational orientation. Approach tendencies prompt a variety of different behavioral responses to anger. For instance, anger often motivates an effort to eradicate the violation of what "ought" to be, an attempt to modify the behavior of others (Fischer & Roseman, 2007), an effort to reestablish the path to the desired goal (Frijda, 1986). Anger can also promote a desire to inflict pain or harm on an offender; with the goals of attaining a particular desired condition, creating discomfort for someone else, or rectifying an injustice (Shaver, Schwartz, Kirson, & O'Connor, 1987). Thus, goal blockage and the concomitant disruption of ongoing movement toward a desired end point, violation of what "ought" to be, the associated failure to maintain an existing desired condition, and thwarting of an interest goal imply that anger emerges from disrupted approach (cf. Depue & Lacono, 1989; Fox, 1991; Fox & Davidson, 1987). Interestingly, the fact that anger often leads to restoring a desired psychological state could motivate the individual to perceive the situation as in reality being controllable (Lerner & Keltner, 2001; Mackie, Devos, & Smith, 2000; Roseman, 1991; Roseman, Antoniou, & Jose, 1996).

The current academic debate on emotional experience (e.g., Barrett, Niedenthal, & Winkielman, 2005; Davidson, Scherer, & Goldsmith, 2003: Frijda, 2007; Lane & Nadel, 2000; Lewis & Haviland-Jones, 2000; Rottenberg & Johnson, 2007) is characterised by two perspectives: on one

side emotions are defined as a set of distinct modular entities conferring them the status of basic emotions (e.g., Ekman, 1992; Izard & Ackerman, 2000), and on the other side there is the assumption that emotions should be identified by a set of underlying dimensions (e.g., Barrett, 2006a; Russell, 2003; energy vs. arousal and valence vs. arousal). Discrete (Ekman, 1992) and dimensional models of emotions (Eerola & Vuoskoski, 2010; Zentner & Eerola, 2009; Juslin & Sloboda, 2010) have offered different views of emotion over the years (Gendron & Barrett, 2009). The discrete emotion model (Ekman, 1992) implies that all emotions can be derived from a limited number of universal and innate basic emotions. Such basic emotions include, for example, fear, anger, disgust, sadness and happiness (Ekman, 1992, 1999). The basic emotion model assumes that an independent neural system sub-serves each of the discrete basic emotions (Ekman, 1999). However, neuroimaging and physiological studies have not attained reliable evidence to support this theory (for a review, see Barrett & Wager, 2006). At present, published meta-analyses have assessed the neuroimaging literature on emotion: three analyses assessed the locationist view (Fusar-Poli, Placentino, Carletti, Landi, Allen, Surguladze et al., 2009; Phan, Wager, Taylor, & Liberzon, 2002; Vytal and Hamann, 2010) which follows the line of discrete models and one analysis assessed the psychological construction view which supports the dimensional nature of emotions (Kober, Barrett, Joseph, Bliss-Moreau, Lindquist, & Wager, 2008). However, only two meta-analyses assessed both views (Murphy, Nimmo-Smith, & Lawrence, 2003; Wager, Phan, Liberzon, & Taylor, 2003). Regarding the *locationist* view, Phan et al. (2002) found that the amygdala showed increased activation during instances of *fear*, but also during instances of *happiness* and *sadness*. Murphy et al. (2003) found that the anterior mid-cingulate cortex had increased activation during instances of both *sadness* and *happiness*. Vytal and Hamann (2010) found that the left amygdala underwent consistent increases in activation during instances of *anger*, *fear*, and *disgust*. However, methodological differences between meta-analyses make it hard to ascertain the degree to which there is specificity in the localisation of discrete emotion.

Meta-analyses that have tested a psychological constructionist view prior to

2008 (Murphy et al., 2003; Wager et al., 2003) found very different results. Murphy et al. (2003) could not find any brain areas that consistently showed increased activity during positive or negative affect, or approach or withdrawal behavior. Wager et al. (2003), on the other hand, found brain areas with increased activation during instances of positive affect, negative affect, and approach and avoidance behavior. The most remarkable observation from the meta-analyses by Murphy et al. (2003) and Wager et al. (2003) is that many of the same regions associated with emotion also showed specialisation for the broader category of withdrawal-related affects. For example, anger-related stimuli may activate the left amygdala, which is part of a broader class of aversive stimuli that involve this region. The neuroimagining literature has found evidence that it is consistent with a psychological constructionist approach to the mind (Murphy et al., 2003; Wager et al., 2003). Hence, interacting brain regions typically involved in psychological operations of both an emotional and non-emotional nature are active during emotion episodes across a range of discrete emotion categories. Nonetheless, the limited evidence of discrete emotion category could be explained by methodological caveats (e.g., human research fails to evoke strong and distinctive basic emotional responses in the laboratory; measurement tools are erroneous, research designs lack precision). In contrast to the discrete theory, the dimensional model of emotion (Russell, 2003) refuted the existence of an independent neural system for every basic emotion (Posner, Russell & Peterson, 2005), and proposed that all affective states stem from two independent neurophysiological systems: one related to valence (a pleasure–displeasure continuum) and the other to arousal (activation–deactivation). Therefore, the literature suggests that all emotions consist of varying degrees of valence and arousal.

The dimensional theory contrasts with an earlier model proposed by Thayer (1989) that supports two underlying dimensions of affect with two separate arousal dimensions: energetic arousal (ranging from feeling sleepy to feeling awake) and tense arousal (ranging from feeling calm to feeling nervous); Matthews, Jones, and Chamberlain, 1990; Schimmack and Grob, 2000; Steyer, Schwenkmezger, Notz, and Eid, 1994; Thayer, 1989; Watson,

Wiese, Vaidya, and Tellegen, 1999. In Thayer's (1989) two dimensional model of activation, valence may be explained as varying combinations of energetic arousal and tense arousal (Fig. 1.1).

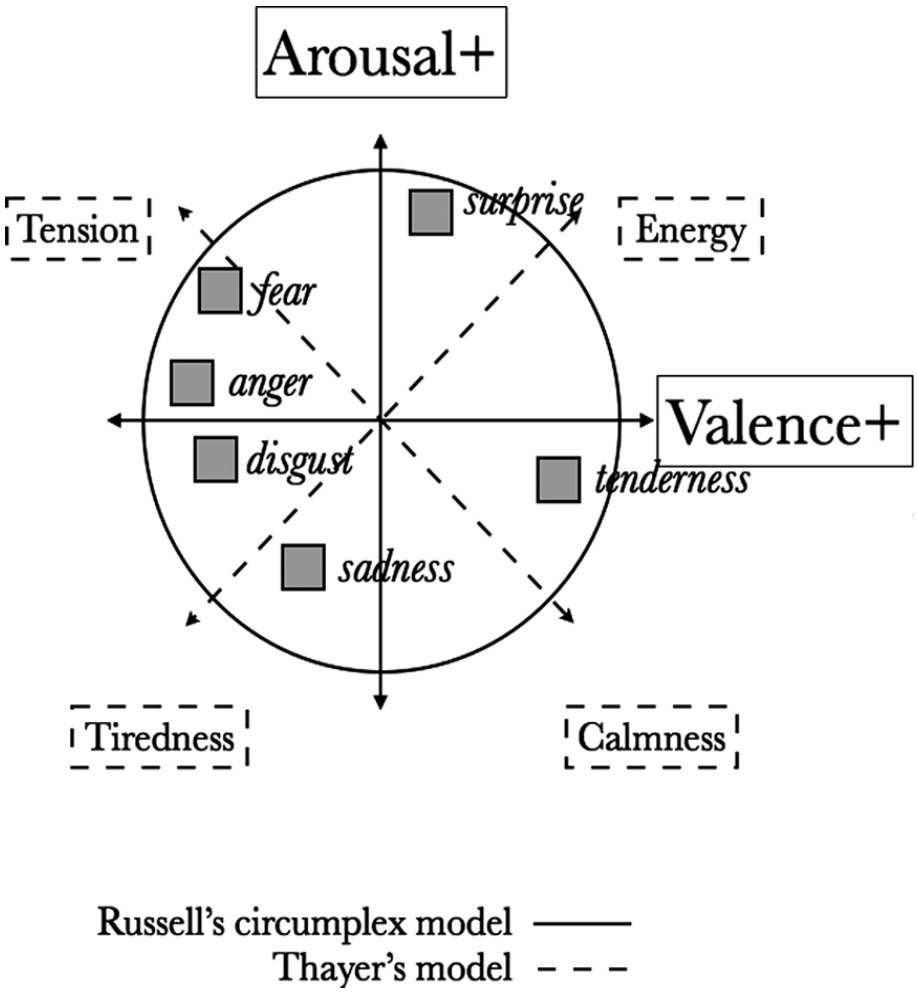

Fig. 1.1: The dimensional models of emotions. The common basic emotion categories are overlaid (reproduced from Eerola & Vuoskoski, 2010).

The two-dimensional conceptualisation of activation is supported by various studies. First, there is evidence that both dimensions of activation are related to different causal factors. For example, energetic arousal is induced by a circadian rhythm (Schimmack, 1999; Thayer, 1989; Watson et al., 1999) that elicits brain cells activity that regulate the organisms' sleep–wake cycle (Tucker & Williamson, 1984). Second, the two activation dimensions can shift in opposite directions. For example, Gold, MacLeod, Frier, and Deary (1995)

examined the influence of experimentally induced hypoglycemia on energetic arousal and tense arousal. Energetic arousal decreased in response to low blood sugar levels, whereas tense arousal increased, ostensibly as the result of an emergency response to mobilize the body and reacted to restore blood sugar levels. Third, the two types of activation have different consequences. For example, energetic arousal is a better predictor of performance on cognitive tasks than tense arousal, according to a body of research (Heller & Nitschke, 1997; Matthews & Davies, 2001; Matthews & Westerman, 1994). The two-dimensional conceptualisation of activation became the topic of renewed controversy (Russell & Barrett, 1999; Schimmack & Grob, 2000; Watson et al., 1999; Yik, Russell, & Barrett, 1999) in a quest to differentiate between energetic and tense dimensions of activation. However, both multi-dimensional models – two-dimensional model and circumplex model – could be criticised for their lack of differentiation when it comes to emotions that are close neighbours in the valence-activation space, such as anger and fear (see e.g., Tellegen, Watson, & Clark, 1999).

A link between anger and fear is also consistent with the well-known concept of “fight or flight” response (Cannon, 1929). Such response implies that the two actions involve activation of energy and can be considered to have a common pathway. A given adverse situation might lead to either action depending on several other factors (Lindsay & Anderson, 2000). In their evolutionary view, Lang, Bradley and Cuthbert (1998, p. 1249) suggest that both fear and anger arise from the aversive motivational system. It can also be viewed in terms of motivational processes underlying the affective state (Blascovish & Tomaka, 1996; Harmon-Jones, 2004a). These positions are by no means mutually exclusive viewpoints (Carver & Harmon-Jones, 2009). Motivational direction (approach vs. avoidance) might be an important basis for differentiating varieties of negative emotions. From this perspective, anger could be defined as positive or negative in terms of whether an individual accepts or rejects, likes or dislikes, the subjective experience of anger. In general, most people regard anger as a negative subjective experience, nonetheless some individuals find the experience of anger less aversive than others (Harmon-Jones, 2004b).

In sum, models of emotion described in discrete terms (Ekman, 1992) emphasise the importance of distinct emotions, whereas dimensional models (Barrett, 2006a; Christie & Friedman, 2004; Russell, 2003) focus upon the ways in which valence interacts with arousal and highlight the importance of both influences in order to describe the category of emotional experience.

1.2. Impact of Emotions on Health

Negative emotions, such as anger, are of particular interest to researchers because the experience of these emotions is associated with poor health in the long-term (Kassam & Mendes, 2013). It has been argued that the immune system is perturbed by prolonged negative emotions, like anger, that contribute to the production of proinflammatory cytokines (Kiecolt-Glaser, McGuire, Robles & Glaser, 2002a). Research has focused on inflammatory processes, given their key mediating roles in the development of cardiovascular disease (CVD) (Packard & Libby, 2008; Puterman, Epel, Donovan, Prather, Aschbacher, & Dhabhar, 2014). The pro-inflammatory cytokines, such as interleukin-6 (IL-6), are strong predictors of CVD morbidity and mortality in both healthy and unhealthy individuals (Honda, Qureshi, Heimbürger, Barany, Wang, Pecoits-Filho et al., 2006; Baune, Rothermundt, Ladwig, Meisinger, & Berger, 2011). The link between anger and cardiovascular health can be characterised as bidirectional in the sense that anger raises health risk and the presence of disease can amplify the physiological impact of anger (Suls, 2013). A concrete example of this bidirectional link is the development of atherosclerosis (caused by an increase of lipids in the blood that stick to the artery walls, increasing blood pressure and presenting greater chronic risk), which has been associated with acute anger responses (Ohira, Diez-Roux, Polak, Homma, Iso, & Wasserman, 2012).

1.3. Motivational-context of anger experience and health implication

Anger has implications for health and behaviour, however the way in which anger is manifested and measured can be variable due to context. The definition of autonomic manifestations of anger is complicated because context determines the observed pattern of psychophysiological activity.

Physiological responses may be linked to specific types of emotion, however this assumption has received minor support (Barrett, 2006b; Larsen, Berntson, Poehlmann, Ito, & Cacioppo, 2008; Levenson, 2003). It is known that the induction of anger causes increased heart rate and cardiac output (CO) (Mauss, Cook, Cheng & Gross, 2007; Mendes, Major, McCoy, & Blascovich, 2008) and research points to multiple physiological profiles of anger, rather than a definite physiological model. For example, there is supporting evidence that anger and anger with rumination elicits distinct physiological responses. Ray et al. (2008) asked participants to recall an unresolved anger experience and either ruminate or reappraise the experience. The physiological response of the participants was increased peripheral vasoconstriction during rumination compared to the reappraise condition. In an experiment in which anger was instilled in participants using a difficult task and an unreasonable experimenter, the participants responded with higher systolic blood pressure when asked to write about the provocation (Pedersen, Denson, Goss, Vasquez, Kelley, & Miller, 2011). Clinical and health psychologists distinguish between internalised and externalised anger (Spielberger & Sydeman, 1994). In fact, each construct yields unique predictions: internalised anger independently predicts depressive symptoms, and externalised anger is characterised by hostility (Bridewell & Chang, 1997; Clay, Anderson, & Dixon, 1993). Theoretical constructs on the health consequences of stress support this distinction of anger states (Gerin, Zawadzki, Brosschot, Thayer, Christenfeld, Campbell et al., 2012), whereby rumination allows stress to instigate damaging effects on the cardiovascular system. Evidence of a mechanism for this pathway has been provided by Kassam and Mendes (2013). On reporting on their emotional state, angry participants showed increased total peripheral resistance (TPR), which limits the flow of oxygenated blood to the peripheral circulation. Participants aware of their emotional state exhibited a response consistent with threat (Blascovich & Mendes, 2010), which has previously been associated with cardiovascular disease (Matthews, 2005). Differences in CO across anger conditions may also hold adverse health implications. In summary, the physiological differences observed in the anger conditions due to motivational context are of practical importance in promoting

cardiovascular health.

1.4. Physiological Signature of Anger

The autonomic manifestation of anger encompasses both *α*- and *β*-adrenergically mediated cardiovascular effects: increased HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), TPR, stoke volume (SV) and CO (Hamer, Tanaka, Okamura, Tsuda & Steptoe, 2007), decreased SV and increased CO (Prkachin, Mills, Zwaal & Husted, 2001; Stemmler, Heldmann, Pauls & Scherer, 2001), decreased SV and unchanged CO (Neumann and Waldstein, 2001), or decreased SV and CO ("anger out" - i.e., defined as anger directed outward away from the self) (Herrald & Tomaka, 2002). Other studies, that did not assess all indices, produce partial replications (Foster & Webster, 2001; Pauls & Stemmler, 2003). Various results were obtained across studies due to different levels of adrenaline released in rapport to the intensity of the stimuli. It is well known that Adrenaline reacts with both α- and β-adrenergic receptors and that this causes vasoconstriction and vasodilation. The α receptors are less sensitive to adrenaline, however they can override the vasodilation mediated by β-adrenoreceptors in conditions when they become activated during high stimulating tasks or are available in high doses. This is facilitated by the higher number of peripheral α1 receptors compared to β-adrenoreceptors. High levels of circulating epinephrine cause vasoconstriction as a result. It is also known that at lower levels of circulating epinephrine (physiologic epinephrine secretion), β-adrenoreceptor stimulation is dominant. This is due to the higher affinity for the β_2 adrenoreceptor in epinephrine than the α_1 adrenoreceptor. Physiologically, vasodilation is followed by a decrease of peripheral vascular resistance (Spielberger & Reheiser, 2010).

Apart from the influence of the intensity of the stimuli, conflicting cardiovascular responses to anger across studies could be also a product of both individual characteristics and stimulus duration. Blood pressure could be elevated to different degree in different individuals (Fahrion, 1991). High SBP follows a pattern of elevated CO, whereas high DBP coincides with high TPR. Alpha-adrenergic response involves moving the blood out of the areas

where is not needed for fight or flight, whereas beta-adrenergic moves this available blood into the area where is needed (muscle, heart) and the responses include increased CO because both HR and SV increase. However, in the first stages of a stress these patterns occur together. TPR is normal because of the increase of diameter of arteries in muscle tissues providing a significant arterial opening through which blood can easily flow. If the anger is sustained beta-adrenergic normalises but the alpha-adrenergic responses continues squeezing blood out of the peripheries and TPR becomes elevated and the whole arterial tree is constricted (Nooris, Fabrion and Oikawa, 2007). The pattern of cardiovascular response observed for the anger conditions across studies are dependent on the sympathetic arousal of the task and levels of habituation alongside individual responses (Dart, Du & Kingwell, 2002).

The anger response pattern is also characterized as *α*- and *β*-adrenergically mediated responses (Table 1.1) by measures that convey shortened pre-ejection period (PEP) (Herrald & Tomaka, 2002; Montoya, Campos & Schandry, 2005) and left ventricular ejection time (LVET) (Stemmler et al., 2001), lower T-wave amplitude (TWA) (Stemmler, Aue & Wacker, 2007), increased heather index (HI) (Montoya et al., 2005), and increased R–Z time (Stemmler et al., 2007). Cardiac parasympathetic inhibition is signposted by decreased heart rate variability (HRV) (calculated as the mean difference between successive RR intervals - MSD; Christie & Friedman, 2004) and spectral respiratory sinus arrhythmia (RSA) (Marci, Glick, Loh, & Dougherty, 2007); other research has found unchanged HRV (peak-valley and spectral RSA, root-mean-square of successive normal sinus RR interval differences (RMSSD), MSD, standard deviation of the normal-to-normal intervals (SDNN) (Rainville, Bechara, Naqvi & Damasio, 2006)). There are reports of increased electrodermal activity (increased skin conductance response (SCR) (Drummond, 1999); increased nonspecific skin conductance response rate (nSRR) (Pauls & Stemmler, 2003); increased skin conductance level (SCL) (Christie & Friedman, 2004), which additionally implicate cholinergically-mediated sympathetic effects at the eccrine sweat glands.

Table 1.1: Modal* cardiovascular responses to anger: Heart Rate (HR); Heart Rate Variability (HRV); Left Ventricular Ejection Time (LVET); Pre-ejection Period (PEP); Stroke Volume (SV); Cardiac Output (CO); Systolic Blood Pressure (SBP); Diastolic Blood Pressure (DBP); Total Peripheral Resistance (TPR)(adapted from as Kreibig, 2010)

Cardiovascular measure	Direction of responses
HR	⇧
HRV	⇩
LVET	⇩
PEP	⇩
SV	⇧⇩
CO	⇧⇩
SBP	⇧
DBP	⇧
TPR	⇧

Note. *Modal responses were defined as the response direction reported by the majority of studies, with at least three studies indicating the same response direction. Arrows indicate increased (↑), decreased (↓), or both increases and decreases between studies (↓↑). See the definition of these symbols in the Notation section.

1.5. Effects of Different Anger Induction Protocols on Model Response

Patterns of sympathetic activation are context-dependent (Kreibig, 2010) in that findings were different when manipulating anger using media (e.g., Jonsson and Sonnby-Borgstrom, 2003; Christie & Friedman, 2004) versus manipulating anger in real-life scenarios (e.g., Stemmler et al., 2001). Physiological responses to picture viewing of facial emotional expressions of anger cause heart rate (HR) to decelerate, SCL decreases instead of increase, and HRV (spectral RSA) increases instead of showing a decrease (Jonsson & Sonnby-Borgstrom, 2003; Dimberg & Thunberg, 2007).

The motivational context of anger induction, whether approach or avoidance orientated, will influence the precise autonomic manifestation of emotion. For example, anger manipulated by film viewing expresses in lower HR in the presence of decreased HRV or MSD of HR – Christie & Friedman, 2004),

pointing to sympathetic–parasympathetic cardiac deactivation that may possibly indicate passive sensory intake (Obrist, 1981; Schneiderman & McCabe, 1989). Another film based study of anger (Montoya et al., 2005) found that TPR decreased in association with increased HR, LVET, SV, CO, HI, SBP, DBP, and mean arterial pressure (MAP) and shortened PEP. Similarly, a response pattern labeled “anger in” (i.e., anger directed toward the self) is embodied by increased HR, SV, and CO, unchanged SBP and DBP, and decreased TPR (Adsett, Schottsteadt & Wolf, 1962). Increased HR, SBP, DBP, SV, CO, but decreased levels of TPR have also taken place under conditions of experimenter harassment in accompaniment of a friend (Lavoie, Miller, Conway & Fleet, 2001). Continuing this line of argument, Stemmler et al.’s findings (2007) showed that approach-oriented anger was indicated by an absence of HR change. In contrast, withdrawal-oriented anger showed decreased HR suggesting that motivational direction in anger influences the associated cardiovascular response. These findings provide evidence for the existence of various sub-forms of anger, differentiated by context of emotion manipulation that influences the heart rate and α-adrenergic response. In other words, the type of emotion induction protocol has an enormous influence over the precise autonomic manifestation of anger and other emotions.

However, the physiological signature of anger can be intermingled with other negative states like fear if the context of emotional induction (real life vs. imagination) is not carefully controlled. The somatic response pattern may be taken as suggestive of a fear response, rather than an anger response. This explains the relatively few consistent cardiovascular differences between fear and anger through several studies (Levenson, Ekman, & Friesen, 1990, Levenson, Ekman, Heider, & Friesen, 1992; Sinha, Lovallo & Parsons, 1992; Sinha & Parsons, 1996). In anger situations, increases in diastolic blood pressure and total peripheral resistance activity were larger than during fear. During fear, decreases in finger temperature and increases in CO were larger than during situations of anger. Stemmler et al. (2001) determined that the context of experimental manipulation (real-life vs imagination) affects the type of psychophysiological response in a significant way.

In contrast to cardiovascular responses, one expects minor influences of the context on self-reports of emotion. This is because feeling states are largely independent of situational properties. Stemmler et al. (2001) found that there were considerable cardiovascular profile pattern differences between fear and anger during real-life context. However, all of the cardiovascular variables were assessed by the fear and anger inductions into the same direction. A strong cardiovascular activation was the common denominator of fear and anger real-life emotion responses, which comprised of:

- tachycardia;
- left ventricular contractility increases leading to shortened pre-ejection period and left-ventricular ejection time;
- reduction of stroke volume;
- elevation of cardiac output, and systolic and diastolic blood pressure;
- heightened electrodermal activity;
- finger vasoconstriction.

Thus, there was a considerable overlap of fear and anger responses during real-life situations. An initial explanation for the overlap of cardiovascular fear and anger responses during real-life is found in the concept of emotion non-specificity. However, arguments for the hypothesis of emotion non-specificity are not compelling. In sum, both the fear and the anger real-life inductions provoked an alerting response characterised by a pattern that is very similar to the description of the “defense reflex.” During imagination, this alerting response was not present. Thus, Stemmler et al. (2001) found supporting evidence for considerable emotion–context confounds and the concept of context deviation specificity. The proposed Component Model of Cardiovascular Response Organisation (Stemmler et al., 2001) is an example of how physiological activity associated with emotion can encompass reactivity to several psychological variables. The model states that emotion–context confounds operate in two distinct ways: a component of response organisation is independent of the emotion, whereas another component is intertwined with the emotion. A third component of the model,

the emotion signatures of fear and anger, could be identified as specific, non-overlapping emotion responses and thus also be separated from the context-related components. Future research should disentangle the context effects of the intertwined component from emotion signatures experimentally.

1.6. Threat/Challenge Cardiovascular Model of Anger

It is known that anger induction causes changes in cardiovascular activity (Suls & Wan, 1993). The precise cardiovascular manifestation of anger may be affected by motivational disposition. However, there is limited research (Stemmler et al., 2007; Harmon-Jones, 2004a) on the manifestation of positive or negative affective states in the context of motivational direction (approach vs. avoidance). Different *patterning* of cardiac activation and vascular resistance has been associated with challenge and threat motivation (Blascovich & Tomaka, 1996). Challenge motivation can be triggered when the individual perceives a degree of control over the task, whereas threat may characterize a situation where the person perceives an absence of control. Blascovich and Tomaka (1996) claimed that the motivational states generated by an appraisal process are important when differentiating between positive and negative emotion. For example, a coping task could be appraised as a positive *challenge* when the perceived resources of the individual meet subjective demand or as a negative *threat* when perceived demand exceeds resources (Blascovich & Tomaka, 1996). Appraisal of stressful events as *challenges* (perceived personal resources that exceed situational demands) yields high cardiac activation coupled with lower vascular resistance. In comparison, appraising the same events as threats (perceived demands greater than resources) causes low to moderate cardiac activity in consonance with higher vascular resistance (Blascovich, Mendes, Hunter, Lickel & Kowai-Bell, 2001; Blascovich & Tomaka, 1996; Dienstbier, 1989).

In support of their theory, Blascovich and Tomaka (1996) argued that challenge is linked to a sympathetic-adrenomedullary (SAM) response, whilst threat reflects both SAM response and a pituitary-adrenocortical (PAC) response. For instance, SAM involves increased HR, a greater volume of

blood expelled by the heart (CO), amplified ventricular contractility (VC) and less resistance to blood flow in the systemic circulatory system (TPR). In the original 1996 model, both challenge and threat states increase SAM activation, and a threat state also augments PAC activation (Blascovich & Tomaka, 1996; Dienstbier, 1989). In a threat state increased PAC activation prevents vasodilation (Blascovich & Mendes, 2000). Using impedance cardiography (Sherwood, Allen, Fahrenberg, Kelsey, Lovallo, & van Doornen, 1990), a threat state can be detected from increases in TPR with no change or a slight increase in CO. A challenge state is observed by an increase in CO and a decrease in TPR (Blascovich & Mendes, 2000). In a situation in which appraisal of resources and demands results in challenge, SAM axis activation also leads to an increase in the release of epinephrine into the bloodstream, which in turn decreases the TPR (the measure of the resistance of the arteries) and increases cardiac output. In addition, threat triggers the hypothalamic pituitary adrenal (HPA) axis prompting the release of cortisol into the bloodstream, which counteracts the SAM effects on TPR and CO, and produces little change or even increases in TPR, and decreases or no changes in CO (Blascovich, 2008).

Challenge and threat states require an underlying motivated performance situation which implies that these states only occur when individuals are engaged with the task. Therefore, HR increases from baseline and PEP decreases indicating task engagement (Blascovich & Mendes, 2000). PEP is an index of isovolumic contraction time directly linked to cardiac contractile force and PEP is controlled exclusively by beta-adrenergic sympathetic responses. However, the empirical findings (Blascovich & Tomaka, 1996; Blascovich, Mendes, Hunter, Lickel, & Kowai-Bell, 2001) were presented in relation to the proposed threat/challenge model were not always accurate (Wright & Kirby, 2003). In particular, the heart rate parameter failed to distinguish between the states of challenge and threat (Blascovish & Tomaka, 1996). In more recent literature (Gendolla & Krüsken, 2002), the role of heart rate in distinguishing between emotional states as well as between motivational states was questioned. It has been explained that the validity of using heart rate as a cardiac parameter may be influenced by the

periodic fluctuations of the breathing pattern (Butler, Wilhelm & Gross, 2006). Nonetheless, Blascovich (2008) explained that VC - the strength of contractions of the left ventricle of the heart - could be a more reliable predictor of threat/challenge differences than HR (see Blascovich et al., 2002; Blascovich & Seery, 2006; Blascovich, 2008) most likely because VC is controlled predominantly sympathetically - also influenced by cardiac preload, cardiac afterload, and to some extent by parasympathetic activity. However, HR is only partially regulated sympathetically. Hence, in terms of cardiovascular (CV) activation, both challenge and threat induce increases in VC and HR from resting baseline in active coping situations; defined as situations that involve task engagement and instrumental cognitive responses (Obrist, 1981; Blascovich, 2008). The cognitive responses are the direct result of SAM axis activation and index task engagement. The cardiovascular patterns associated with challenge and threat proposed in the BPS model have been empirically validated (see Blascovich, Mendes, Vanman, & Dickerson, 2011; Seery, 2011 for reviews). However, the distinction between challenge and threat is minimal and reduced to a cognitive task engagement scenario with no consideration of how negative affect states such as anger could alter the challenge/threat balance.

In summary, according to BPS model of challenge/threat (Blascovich & Tomaka 1996), cardiovascular reactivity could be influenced by a motivational appraisal; in that when success is perceived as possible (demand meets resources) a challenge motivation response could be indexed by increased CO, but low TPR. On the contrary, an excessive demand in relation to resources will generate threat associated responses (lesser CO increase compared to challenge condition, but high TPR). An update of the model (Blascovich et al., 2001) included measures of SBP with lower values being linked to low TPR in the challenge context and HR increase rate in the threat context. Nonetheless, the model should be tested in regards to emotions such as anger that have been found to be associated with different sets of CV responses, either decreased SV and increased CO (Prkachin et al., 2001; Stemmler et al, 2001), decreased SV and unchanged CO (Neumann and Waldstein, 2001), or decreased SV and CO (Herrald &

Tomaka, 2002). Since studies produced partial replications (Foster & Webster, 2001; Pauls & Stemmler, 2003) it would be useful to identify whether motivational factors can exert an important influence on cardiovascular markers of anger.

1.7. Frontal Electroencephalographic (EEG) Asymmetry and Motivational Direction

From the cardiovascular literature we learn that anger should be measured during challenge/threat situations. In such cases, subjective appraisal triggers motivational predispositions for action to either *approach* (to respond to a challenge or remove the obstacle) or *avoid* the threatening situation. Challenge is regarded as an approach motivational state, whereas threat is not (e.g., Tomaka & Palacios-Esquivel, 1997). Such bidirectional motivational engagement (approach vs. avoidance) has been investigated extensively in the frontal brain asymmetry literature (Davidson, 1993; Hagemann, Naumann, Becker, Maier & Bartussek, 1998; Hewig, Hagemann, Seifert, Naumann & Bartussek, 2004; Harmon-Jones, 2004a). Davidson (1993) proposed a model of frontal brain asymmetry and emotion. An approach system is activated by the perception of achievable goals, which elicits positive emotion and it is thought to be associated with greater left prefrontal cortex (Hewig et al., 2004). On the other hand, the avoidance system is activated by aversive stimuli, which elicits negative emotions and leads to withdrawal. The neuroanatomical basis for the avoidance system is considered to be the right brain hemisphere (Hewig et al., 2004). In terms of negative emotion, fear is often conceptualized as involving withdrawal, whereas anger is seen in association with approach state. However, not all studies that induced a state of anger found evidence of neural left asymmetry (e.g., Waldstein, Kop, Schmidt, Haufler, Krantz, & Fox, 2000; Hewig et al., 2004).

The approach-withdrawal and valence models draw on the same theoretical foundation, whereby frontal asymmetries are the fundamental element. However, in the approach-withdrawal model rather than examining behaviours in terms of positive and negative valence, emotionality is

determined according to whether the emotion tends to provoke approach or withdrawal behaviours. This model proposes that emotions that tend to provoke withdrawal behaviours are associated with relative activation of the right hemisphere, in contrast emotions related to approach behaviours are associated with relative left hemisphere activation (Davidson, 1995). Several electroencephalograph studies of clinical populations have provided support for this model and produced evidence that among depressed patients, relative right activation or left deactivation is indicative of depressive symptom exhibition; in particular, withdrawal type behaviours (Heller, Nitschke & Miller, 1998; Henriques & Davidson 1991; Robinson & Downhill 1995). Numerous experiments with adults endorse the findings in depressed patients (Davidson 1992, 1993, 1995; Davidson, Abercrombie, Nitschke, & Putnam, 1999). Studies on anger at the Harmon Jones laboratory (Harmon-Jones & Allen 1998; Harmon-Jones, 2003, 2004a, 2004b) have provided substantial support for the approach-withdrawal model.

Anger is largely conceived as a negative emotion (Harmon-Jones, 2004b) because it is elicited by unpleasant or undesired events. Empirical findings by Harmon-Jones and Sigelman (2001) indicate that self-report anger and subsequent aggression correlate positively with left frontal brain activity in a group of participants that were insulted compared to a group of participants that were treated kindly. Subsequent studies have replicated these results (Jensen Campbell, Knack, Waldrip & Campbell, 2007) and also have indicated that state anger could be linked to increased left frontal activity and decreased right frontal activity (Harmon-Jones, Vaughn-Scott, Mohr, Sigelman & Harmon-Jones, 2004). At least one study has reported the opposite asymmetry (Zinner Brodish, Devine, & Harmon-Jones, 2008); however, the observation occurred in a context in which anger was mixed with anxiety. In such context, anger may have led to a desire to escape from the situation to prevent risking disapproval (Plant & Devine, 2003). At a close inspection, it would appear that the manipulation of anger was carried out in a way that aided a certain directional dimension; in particular, an approach, goal-orientated motivation (Carver & Harmon-Jones, 2009). Carver and Scheier (1998, 2002) comment on the existence of two kinds of motivational

systems that have contrary aims - one organized to approach incentives, the other organized to avoid threats. Each motivational system relates to both positive and negative affect. In other words, the approach dimension could present itself even in a negative emotional state such as anger.

Following this reasoning, Harmon-Jones (2003) varied the intensity of approach motivation independent of anger, by manipulating perceptions of control. The expectation of being able to act in order to resolve the event should yield greater approach motivational intensity than an expectation of being unable to act. Both conditions evoked increases in anger (compared to baseline). Consistent with their predictions, participants who expected to be able to act had more left frontal activity than those who expected to be unable to do so. Moreover, in the action-possible condition, those with greater left frontal activity after the anger-provoking event also reported more anger. This again is consistent with the idea that anger is an approach related emotional response. Indeed, those with greater left frontal activity in this condition were subsequently more likely to act to change the situation. In the condition in which action was not possible, greater left frontal activity did not relate to greater anger. In our view, this is because when action is not possible, motivational engagement is low, even if angry feelings are high.

The frontal asymmetry research (i.e., Harmon-Jones, 2004a; Carver & Harmon-Jones, 2009) suggests that motivational direction (approach vs. avoidance) is an important basis for differentiating between negative affective states. As Davidson (1995) explained, the determination of emotionality should be made based on whether the emotion tends to provoke approach behaviours or withdrawal behaviours; a view supported by findings that emotions that tend to provoke withdrawal behaviours are associated with relative activation of the right hemisphere while emotions related to approach behaviours are associated with relative left hemisphere activation (Carver & Harmon-Jones, 2009). In this respect, the involvement of approach versus avoidance motivational systems in the experience of anger could be echoed in left/ right anterior activation.

From all the studies presented above, a general criticism could be raised. In most previous work, the heart-brain interconnection has not been given due attention because it is hard to link the ideas from the Frontal EEG Asymmetry community with Blascovich and team's (1996, 2000, 2001) work on cardiovascular activity. Few studies have looked at the relation between cardiac reactivity and the responses of the central nervous system. Wittling (1990) found that the right hemisphere holistically is linked to the autonomic response. Right-hemispheric film presentation by means of a technique for lateralizing visual input that allows prolonged viewing while permitting free ocular scanning caused a significantly higher increase in systolic and diastolic pressure than left-hemispheric viewing of the same film.

Other studies (e.g., Waldstein et al., 2000) have found that a right frontal EEG response in an anger state was associated with increased blood pressure, suggesting that asymmetric frontal EEG responses to emotion produce different patterns of cardiovascular reactivity. Koslov, Mendes, Pajtas, & Pizzagalli (2011) conducted research on participants exposed to social rejection and found a significant association between left frontal asymmetry and cardiovascular stress responses. Under conditions of social pressure (challenges), left frontal asymmetry (average of alpha1 and alpha2 current density in Brodmann area 9) increased cardiac output and therefore cardiac efficiency, and decreased total peripheral pressure, which indicates dilation of the arterioles. Both responses correspond to a *challenge* state. The findings suggest that participants with higher resting activity in the left relative to right frontal cortex experience more adaptive, approach-oriented cardiovascular stress in response to social evaluative challenges. Therefore, it is essential to consider environmental and contextual factors in the study of the impact of brain-based traits on physiological and emotional responses. The context in the study of Koslov et al. (2011) was social evaluation triggered using a motivated performance situation in the presence of two interviewers. The interviewers provided positive feedback - a protective factor - or negative feedback, perceived as a situation of social evaluative challenges. The beneficial effects of left frontal asymmetry occurred only when participants were devoid of environmental protective factors; that is,

when they were most vulnerable to social stress.

Challenge states are often associated with positive affect, as well as anger (Mendes et al., 2008). Furthermore, left frontal asymmetry has been associated with anger; a negatively valenced, approach-related emotion (Harmon-Jones, 2003). Research has found evidence, therefore, for both positive and negative affective correlates of left frontal asymmetry and this necessitates careful interpretation of left frontal asymmetry relationships. Individuals with relatively higher left frontal activity underwent a blend of affective responses in the social threat condition - anger and challenge. From the work of Koslov et al. (2011) we conclude that left frontal asymmetry is associated with approach motivation. Future research should attempt to disambiguate the valence components of this response. In sum, Koslov et al.'s (2011) findings show that left resting frontal asymmetry may constitute a protective factor in individuals exposed to a threatening situational context. Right frontal asymmetry may be a crucial vulnerability factor in stress-diathesis models of disease etiology. However, the limited number of studies on heart-brain interaction have failed to provide an insight into the motivational facets of various emotions.

2. Affective Computing: Countermeasures

Negative emotion can be detrimental for health in the long-term (Kiecolt-Glaser et al., 2002a), but the experience of negative emotion is intensified depending on the appraisal of the situation (Stewart, Levin-Silton, Sass, Heller, & Miller, 2008). Therefore, is it possible to create technological systems designed to function as countermeasures for negative emotion in everyday life?

Affective Computing has proven useful in the field of Health-Care (Nasoz, Alvarez, Lisetti & Finkelstein, 2004) as it can provide communication between medical professionals and patients. For example, tele-home health care interventions are currently used to collect physiological data remotely (e.g., ECG, blood pressure, oxygen saturation, heart rates, and respiration), and assess aspects of mental or emotional status (Cearreta, López, López

de Ipiña, Hernandez, Garay, Graña et al., 2007). Monitoring is important in health related cases, and we can also identify its utility outside the clinical environment. For example, research on affective computing systems - a discipline that concerns itself with the development of computer interfaces for measuring and responding to users' emotions (Picard, 1997) - has often focused on detection of negative emotion in order to improve self-regulation (Picard & Klein, 2002; Picard, 2003) and has been applied to psychophysiology measures to delimit emotional states (Kapoor, Burleson, & Picard, 2007). The discipline of affective computing aims to give machines the ability to recognise and generate affective states. In part, affective computing addresses the shortcomings of traditional Human Computer Interaction (HCI) systems, which tend to neglect changes in affective states in users. Such neglect may explain why many users view interactions with computers as "cold, incompetent and socially inept." (Zeng, Pantic, Roisman & Huang, 2009; p.1). A solution is to warrant that user interfaces of the future are able to "detect subtleties of and changes in the user's behaviour, especially his/her affective behaviour, and to initiate interactions based on this information rather than simply responding to the user's commands" (Zeng et al., 2009; p. 1). These subtleties may be related to motivational states underlying the experience of interacting with technology (Mandryk, Inkpen & Calvert, 2006; Yannakakis, Hallam & Hautop-Lund, 2007). Consideration to motivational states promotes the concept of adaptive computer games, where software responds to the state of the player in order to challenge or help the individual according to the user's response (Dekker & Champion, 2007; Fairclough, 2007; Gilleade & Dix, 2004). Psychophysiology has the potential to quantify different psychological states (e.g., happiness vs. anger), to monitor changes in state along a psychological continuum (e.g., low vs. high anger) and to function as a proxy for input control (e.g., a brain-computer interface - BCI). Psychophysiological data may also be used to identify stable personality traits, including motivational tendencies (Coan & Allen, 2003) and predispositions related to health, such as stress levels (Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann et al., 1998).

Current perspectives in affective computing prioritise the development of systems that can diagnose states of frustration (Partala & Suraka, 2004). However, affective computing has moved from detection of specific categories of emotion to the detection of negative states related to performance on computer tasks (Kapoor et al., 2007). Although such systems are perceived as useful in the context of self-regulation, they lack psychological sophistication regarding the definition of *negative emotion within a motivational context*. Moreover, given that an interactive system can also be used as a health-monitoring tool (Gerasimov, Selker & Bender, 2002) careful consideration has to be paid to causes that transform a negative emotion into an *unhealthy* emotion. Therefore, it is important to develop valid affective systems in order to ensure a correct affective computing system that will allow self-regulation and assist in health monitoring during situations such as driving a car. The affective computing systems could incorporate an informative feedback function ('you are angry') and adaptive counter-interventions (i.e., relaxation music or calming light). Noticeably, the ambient interventions could only function as adaptive countermeasures once the user learns how the system works. However, the provision of the feedback should be functional (it should be ascertained whether the function 'you are angry' effectively counteracts a negative emotion), non-intrusive (it does not distract) and unprovocative (it does not cause even more anger).

Ambient environment (i.e., music, light) could be used as a way of providing feedback to the user and as a counter measure. There is an increasing interest in affective computing to enhance communication with the environment (Lisetti & Nasoz, 2004). In this way, some environmental variables can be adapted to user's current emotional state in order to improve the experience, for example by adjusting the levels of personalised environmental music or lighting (Cearreta et al., 2007). One particular technique of communication between the user and the environmental is called Ambient Intelligence (AmI) which is the opposite of virtual reality as it places the computer in the reality of the user rather than placing the user in a virtual reality. Aml strives to support the activity of the user through the use of information and communication technologies distributed pervasively in the

environment (from personal objects to vehicles) (Morganti & Riva, 2004). However, research in AmI areas is generally limited to passive inference, one–way communication and does not consider affective use (Li & Ji, 2005). Here, affective computing could provide the tools to assess the emotion experience in a changing ambient environment, and the necessity to develop high level of emotion-user-environmental models should be the focus of further research.

Functional psychophysiological correlates of emotion could be triggered within a driving context (Vanlaar, Simpson, Mayhew, & Robertson, 2008) and be modulated by environmental stimuli such as music (Wiesenthal, Hennessy, and Totten, 2000; Juslin & Sloboda, 2010) or ambient coloured light (Knez, 2001). Driving is a routine activity that raises negative emotions when road obstructions are present (McGarva, 2005). Links were found between high levels of systolic blood pressure and the appraisal of the impossibility of completing the journey schedule (Hennessy & Wiesenthal, 1997) due to dense traffic (Schaeffer, Street, Singer, & Baum, 1988; Stokols, 1978). Hence, the prolonged experience of a negative emotion in a driving context could impact on cardiovascular health. Ambient light as an environmental trigger of a wide range of emotions, from exhilaration to relaxation, happiness to anger, and even combinations of these, could be manipulated to regulate emotion in a driving scenario, similar to the effects achieved using music (Chanda & Levitin, 2013). Moreover, the acute effects of ambient light on emotional processing have implications for our understanding of the mechanisms by which changes in lighting environment could modulate emotions, not only in the case of mood disorders using light therapy, but also in day to day life, by paying more attention to our light environment in different contexts, including driving a car. An emerging avenue for research is the development of computerised affective systems that aid monitoring and manipulation of emotion (Picard, 2003).

An interactive system that identifies physiological expressions of motivation and emotion and offers biofeedback could raise self-awareness as a strategy for reducing the experience of a negative emotion (Mauss et al., 2007), with

beneficial consequences for health. The design of future affective computing systems should take into consideration the fact that the mechanism beyond self-regulation is a motivation trigger and that any emotion incorporates cognitive and motivational facets, as well as an affective dimension (Matthews, Campbell, Falconer, Joyner, Huggins, Gilliland et al., 2002). Therefore, the adverse impact of negative emotions on health may be attributed to motivation or to cognitions associated with a particular affective state. For instance, anger in combination with lack of coping resources creates a sense of helplessness (Stemmler et al., 2007), whereas anger coupled with sufficient resources increases motivation to approach a task constructively. Thus, negative emotions such as anger must be defined multi-dimensionally in relation to motivational contexts prior implementation of an emotional model in any cybernetic system.

3. Proposed Model and Predictions

Despite significant differences appearing both within and between the bodies of cardiovascular and encephalographic literature in relation to anger and its motivational context dependent facets (challenge/threat and approach/avoidance), there appears to be a great deal of overlap which may be conducive to the construction of a unified theoretical model. Such a theoretical model of anger is proposed where we interpret cardiovascular and electrocortical responses to anger induction within 2D psychological space (see Fig. 1.2). For example, a challenge/avoidance state will be indexed by right brain activation along SAM activation, whereas a threat/avoidance will activate SAM and PAC responses and the right frontal brain. Challenge/approach will activate left hemisphere and SAM responses, whereas threat/approach will be indexed by left frontal brain activation and PAC responses.

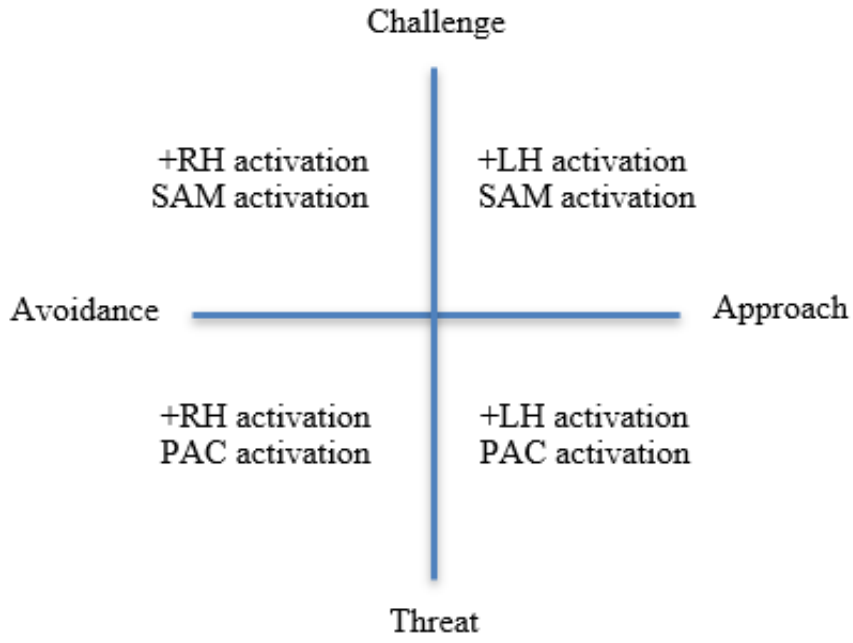

Fig. 1.2: The proposed cardiovascular and encephalographic dimensional model of anger (SAM = sympathetic-adrenomedullary response; PAC = pituitary-adrenocortical response; RH = right hemisphere; LH = left hemisphere)

Hence, the general aim of this thesis was to develop a multi-dimensional model of psychophysiological markers of anger within a motivational context of challenge/threat behaviours and test the malleability of this model in two modalities: auditory by means of personalised music and visual by manipulation of coloured ambient light.

For consistency, the thesis refers to right or left activation as a representation of the asymmetry of the logarithm score metric between left and right frontal hemispheric sites. Asymmetric measurements of cortical activity were computed as the difference of logarithm alpha power of contralateral homologous sites (Hagemann, 2004). Natural log transform scores are needed to limit skewness of the data due to individual differences. Because alpha power is inversely related to cortical activity, higher scores indicate greater left than right activity (Davidson, Jackson and Larson, 2000). By using logarithms, positive values in the index showed greater right alpha power with greater left

frontal activity. While negative values in this index indicated greater right frontal activity (Allen et al., 2004).

It was hypothesised that:

1. Challenge is characterised by effortful engagement and a degree of control over the situation.
2. Challenge with approach motivation can be measured by SAM responses: increased HR, SV, CO, LVET, and low TPR associated with low BP and by activation of the left area of the frontal cortex.
3. Challenge with avoidance motivation can be measured by SAM responses: increased HR, SV, CO, LVET, and low TPR associated with low BP and by activation of the right area of the frontal cortex.

4. Threat is characterized by low likelihood of success and low/no control over outcome.
5. Threat with avoidance motivation is characterized by increased avoidance measured by PAC responses - specifically an increase of HR, SV, CO and LVET at a lower rate compared to the increase in challenge approach - a high TPR, high SBP, and by activation of the right area of the frontal cortex.
6. Threat with approach motivation is characterized by increased avoidance measured by PAC responses, specifically a lower rate of increased HR, SV, CO and LVET compared to challenge; a high TPR, high SBP and by activation of the left area of the frontal cortex.
7. A successful ambient intervention reduces the magnitude of anger and the cardiovascular responses associated with threat/avoidance.

CHAPTER 2
METHODOLOGY

The core aim of the thesis was to find physiological correlates of anger within motivational context of challenge/threat and the induction procedures of anger and motivational context are the focus of this methodology chapter. The rationale for choosing an active anger-induction protocol over a passive one and the choice of motivation manipulation are described. The tools for manipulation checks are outlined and the physiological apparatus used across studies is presented. The protocols for ambient interventions on anger/motivation methodologies are not presented here but in their corresponding experimental chapters.

2.1. Review of Methods for Inducing Anger Under Experimental Conditions

This chapter will discuss methods for mood induction, describe the different options available (media, introspection, active method) and then talks about anger induction specifically. In previous studies, experimental scenarios incorporated either incentives or threat stimuli consequently the outcome measures included an approach motivation associated with positive feelings and avoidance motivation linked to negative feelings (for a review see Coan & Allen, 2004). However, previous studies focus on passive manipulation of motivational and affective states, such as participants being exposed to repellent film clips (Carver & Harmon-Jones, 2009) or pictures of delicious desserts (Gable & Harmon-Jones, 2008). Other manipulations of anger involved participants performing a difficult mental arithmetic task designed to elicit anger subliminally (Mauss et al., 2007; Mendes et al., 2008; Jamieson, Koslov, Nock & Mendes, 2013). This is considered an active manipulation of anger. However, previous research indicated that such protocols that are designed to manipulate anger at a non-self-conscious level could result in distinct cardiovascular and EEG profiles if brought to a self-conscious level which involves appraisal of the situation, in particular shame (Herrald & Tomaka, 2002; Mendes et al., 2008; Jamieson, Nock & Mendes, 2012). Active participation in the task that elicited anger was found to be more

effective in indexing anger via psychophysiological responses compared to passive participation (Stemmler et al., 2001). Therefore, the anger induction procedures within the present thesis used an active method as opposed to passive exposure to media (Gable & Harmon-Jones, 2008; Carver & Harmon-Jones, 2009).

2.2. Active Anger Induction

Two active anger induction scenarios were proposed to account for variability of the experience of anger in diverse environments. Participants in Study 1 were exposed to rude and aggressive messages from a fictional experimenter (Stemmler et al., 2007) while performing a task in a malfunctioning computer. Participants in Studies 2 - 5 were given a driving simulator task to complete on a scheduled time with financial penalties for failing to reach the destination in the designated time. Participants in Study 1 heard the 3 aggressive messages at equal intervals during their task and, to resemble this voice induction procedure, participants in Studies 2 - 5, heard 3 warnings messages with the remaining millage to finish the journey.

Across the experiments, anger levels were manipulated concomitantly with motivational dimensions of challenge/threat. In Study 1, participants were presented with a computer-based problem-solving task where the computer either worked correctly (control) or malfunctioned (no control). A controllable task was operationalised as being a challenge to solve the task, whereas a no control task predicted an avoidance state from the threat of not completing the task (Partala & Suraka, 2003). Subsequent studies used 2 simulated traffic jams to manipulate the challenge/threat motivational dichotomy; the first traffic jam (TJ1) allowed participants to complete the task on time without incurring financial penalties (challenge) while the second traffic jam (TJ2) annulated any chances for the participants to finish the task on time (threat).

2.3. Driving Simulator

A clock was visible next to the simulated scene and participants were instructed to complete the journey within 15 min in order to earn the £20

(approximately $32/22 €) payment for participation in the study 2. From studies 3 to 5, the instructions were to complete the journey in 8 min in order to earn £10 (approximately $16/11€). The deadline was presented to the participants within the context of a scenario; they were told that the purpose of the journey was to collect a child from school. The precedence of completing the journey on time was reinforced by providing feedback of journey progression via three instances of pre-recorded verbal messages, e.g., “five miles remaining,” “three miles remaining” and “one mile remaining.” If the driver crashed the vehicle more than twice, they were told they would lose their total participant payment. In addition, speeding warnings were in operation and participants were informed that they would be fined by £1 if they broke the speed limit or committed a driving violation such as overtaking where lane marking indicated that passing other vehicles was prohibited.

A simulated car journey was prepared using STI SIM Driving Simulator software (STI Inc.). This PC-based software allowed interaction via a steering wheel/pedals console and the driving scene was projected onto a large screen (approximately 3.66 m × 4.57 m), yielding a visual angle of approximately 80° (Figs. 2.1 and 2.2). The infrastructure of the simulated route included a number of bends, crossroad intersections with stop lines, and several sets of traffic lights. In the first design (study 2), the driver encountered a low level of traffic density in both lanes with two exceptions; after approximately 2 min the journey had elapsed participants encountered the first traffic jam where extremely slow moving traffic in the road lane was combined with high density traffic in the opposite lane, hence participants remained ‘trapped’ in the first traffic jam for 4 min. At a later point in the simulated journey (after approximately 12 min of driving), participants encountered a second traffic jam, identical to the first, that persisted for 5 min. The combined delay introduced by both traffic jams made it impossible for the participants to reach the destination within the required 15 min (Figs. 2.1 – 2.3).

Fig. 2.1: Driving simulator set up.

Fig. 2.2: Driving simulator test

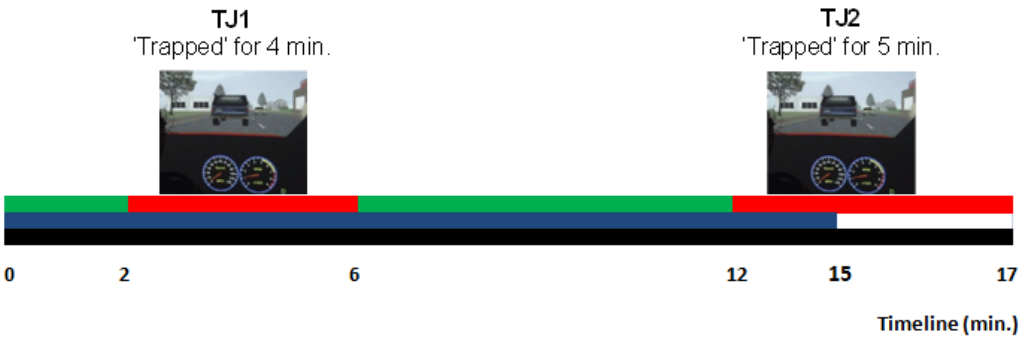

Fig. 2.3: Timeline: Simulated car journey 1 in the STI SIM Driving Simulator.

Note: TJ1 = traffic jam1; TJ2 = traffic jam 2; green = traffic-jam-free journey, red = traffic jam, blue = target journey time, black = actual journey time.

A shorter version of the simulated car journey was designed for study 3 and the subsequent two studies. The new version used the same road events and traffic jams but the times were reduced. In this short simulated journey, the driver encountered a first traffic delay after 3 mins into the drive and it lasted for 3 mins. The second traffic jam was set at the 7 min mark and lasted for 4 mins (Fig. 2.4).

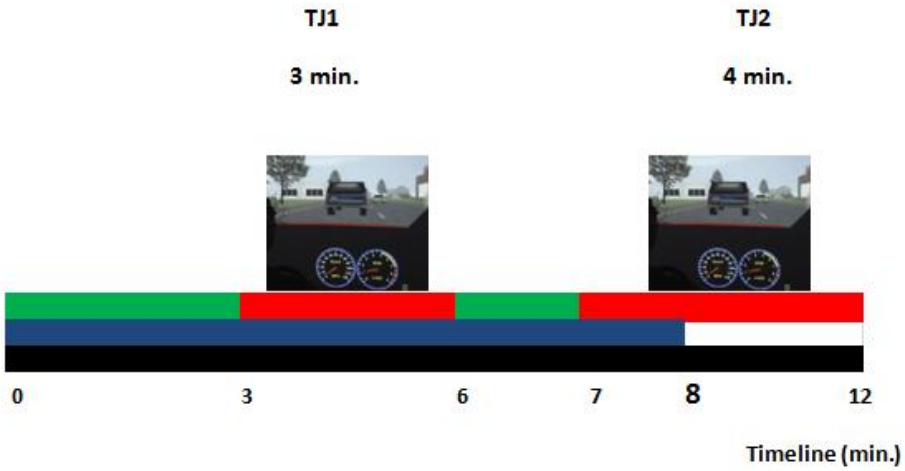

Fig. 2.4: Timeline: Simulated car journey 2 in the STI SIM Driving Simulator.

Note: TJ1 = traffic jam1; TJ2 = traffic jam 2; green = traffic-jam-free journey, red = traffic jam, blue = target journey time, black = actual journey time.

All the times given are estimates as each driver reached the traffic events at different times depending on their speed. Nonetheless, the set drive adjusted

for each driver and the time differences were in the range of 1 to 2 minutes, and the end of the drive was still no possible if the driver respected all the traffic rules.

2.4. Ethics of Active Anger Inductions

It should be noted that the decision to deceive the participants was felt to be absolutely necessary in order to accomplish the goals of the experiment; i.e., to induce genuine experience of anger. All procedures for participant recruitment and data collection were approved by the Liverpool John Moores University's Ethics Committee prior to commencement of all the studies. Participants were fully debriefed as to why it was necessary to deceive them

2.5. Participants

Participants were recruited from a nonclinical population with no history of psychiatric illness or cardiovascular problems. Using participant self-reports, the inclusion criteria were: right handed, mentally and physically healthy, not being under a course of medication, absence of vision deficiencies (e.g., colour blindness), and not having high levels of anger (scored below the 80th population percentile on the Trait Anger Expression Inventory of the STAXI 2; Spielberger, 1999) to reduce the likelihood of the researcher being exposed to aggressive behaviour during the anger induction protocols used in the studies. Blood pressure and resting heart rate were measured prior to the experiment to ascertain that the participants were not hypertensive or experienced elevated heart rate.

2.6. Dependent Variables

2.6.1. Self-report measures

A number of self-report questionnaires were used as manipulation checks in all experimental studies: The State Anger Expression Inventory 2 (Spielberger, 1999) was used to measure the anger level differences pre- and post-test across all 4 studies , UWIST Mood Adjective Check List (Matthews, Jones and Chamberlain, 1990) was utilized to indicate the valence changes after the test in Studies 1, 3, 4 and 5, and the Confidence and Perceived Control Scale from Dundee Stress State Questionnaire

(Matthews & Desmond, 1998) was employed to index confidence and control while performing the task in the first 2 studies.

The State-Trait Anger Expression Inventory (STAXI-2) is a 57-item questionnaire developed to measure *Trait Anger, State Anger*, and Expression and Control of Anger (Spielberger, 1999). The State Anger Scale assesses the intensity of anger as an emotional state at a particular time. Fifteen items measured on a scale from 1 to 4 (1 = not at all, 5 = very much so) form three 5-item State Anger subscales. The three sub-scales are: a) feelings of anger (*S-Ang/F*), (b) feel like expressing anger verbally (*S-Ang/V*), and (c) feeling like expressing anger physically (*S-Ang/P*). The Trait Anger Scale was used to measure how often angry feelings are experienced over time. The Trait Anger measure was based on 10 Trait Anger items validated by Spielberger (1999) with 2 subscales: 4-item anger temperament scale and 4-item angry reaction scale. The internal consistency for all scales and subscales was satisfactory with Cronbach alpha values ranging from .76 for the 4-item T-Anger/R subscale to greater than .84 (Spielberger, 1999; Spielberger and Reheiser, 2004); Table 2.1.

Table 2.1: State Anger Scale and Trait Anger Scale

Construct	Subscales	Number of items
State Anger Scale (intensity of anger)	Feelings of anger (*S-Ang/F*)	5
	Feel like expressing anger verbally (*S-Ang/V*)	5
	Feel like expressing anger physically (*S-Ang/P*)	5
		Total 15
Trait Anger Scale (frequency of anger)	Anger temperament (*T-Anger/T*)	4
	Angry reaction (*T-Anger/R*)	4
		Total 10

On the subject of construct/factor analytic validity, several exploratory and confirmatory factor analytic studies have reported empirical support for the STAXI-2 structure (Lindqvist, Waterman & Hellström, 2003; Borteyrou, Bruchon-Schweitzer & Spielberger, 2008; Maxwell, Sukhodolsky & Sit, 2009;

de la Rubia, González & Landero, 2010). Evidence of predictive validity of the STAXI-2 in the measurement of anger has been provided in several research (Spielberger & Reheiser, 2010; Deschênes, Dugas, Fracalanza, & Koerner, 2012; Antypa, Giegling, Calati, Schneider, Hartmann, Friedl et al*.,* 2013).

The UWIST Mood Adjective Check List (Matthews et al., 1990) was also employed as a validated measure (Matthews et al., 1990) with high internal consistency across 4 dimensions: Energetic arousal - Cronbach α = 0.88, Tense arousal - Cronbach α = 0.86, Hedonic tone - Cronbach α = 0.88, and General arousal - Cronbach α =0. 75.

Apart from the affective nature, psychological states also have motivational and cognitive elements (Matthews et al., 2002). Hence, a measure of control state was employed using Confidence and Perceived Control Scale from Dundee Stress State Questionnaire (Matthews & Desmond, 1998). Confidence-Control scale had 6 items - 'I feel confident about my abilities' - showed Cronbach α = .80 (pre) and .84 (post). Test-retest reliability was: across-task r = .49, 3-week = .54, 6-month =. 32. Matthews et al, 2002). The resulting scales met psychometric criteria for state measures. For instance, high internal consistency but lower test–retest reliability than traits.

2.6.2. Physiological measures

Cardiac activity

Cardiac activity was recorded using NICO100C Noninvasive Cardiac Output Module (BIOPAC Systems Inc.) at an operational frequency of 50 kHz and a magnitude range of 5 Ohms/volt in conjunction with the MP150 data recording system (BIOPAC Systems Inc.); see Fig. 2.5.

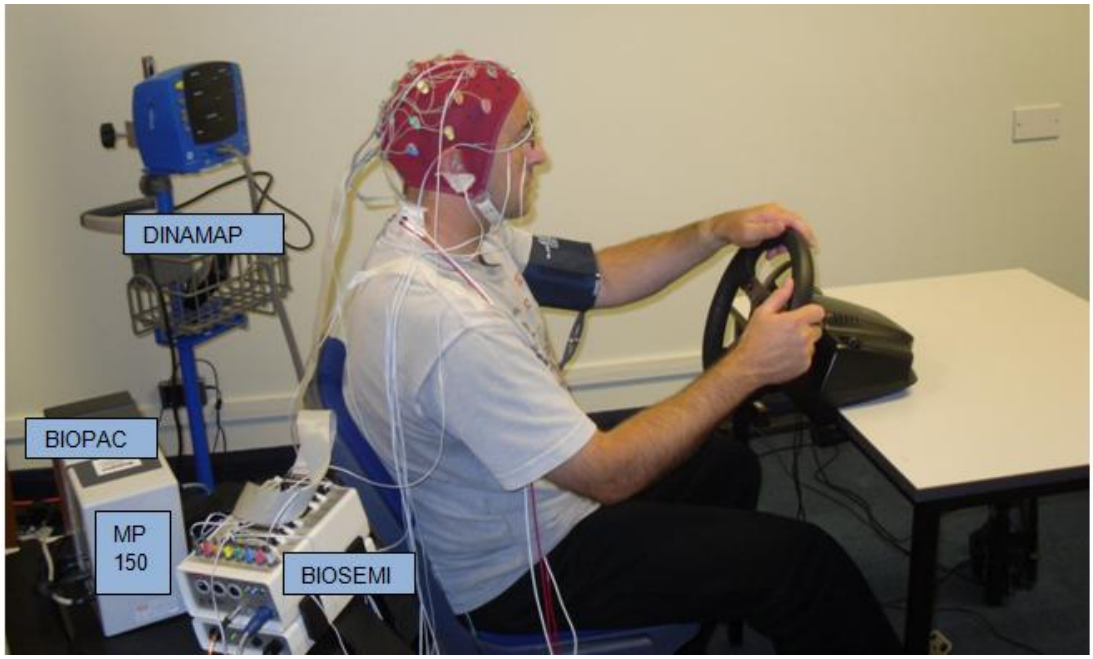

Fig. 2.5: Hardware apparatus: DINAMAP module for BP and MAP; BIOPAC MP150 module with NICO100C(for ICG), TEL100C (for HR and respiration), EMG100C(for fEMG) and BIOSEMI amplifier for 32-channel EEG recording.

Within Study 1, cardiac activity was recorded using a 4-dual electrode configuration that resembled band electrodes placed on each side of the neck and thorax (adapted from Stemmler et al., 2007); Fig. 2.6.

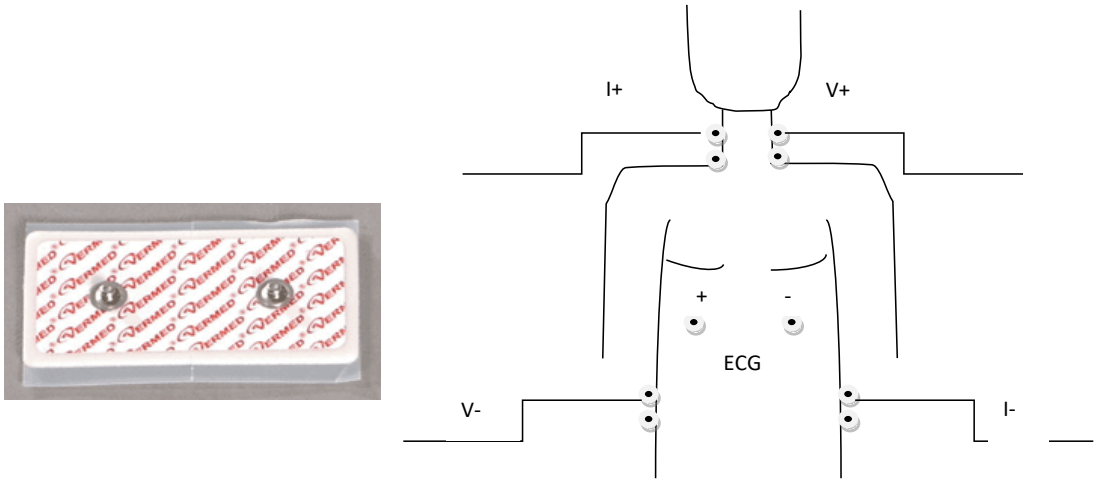

Fig. 2.6: Fixed spacing spot electrode pairs used for recording of impedance cardiography (ICG) and their placement on anatomical sites. Note: ECG was recorded concomitantly with the ICG using single spot electrodes. (Source left image: BIOPAC Systems, Inc.).

Two fixed spacing spot electrodes pairs (size: 41 mm wide x 82 mm long x 1.5 mm thick) were used instead of just one spot electrode in order to closely resemble the band electrodes. Using the configuration described by Penney, Patwardhan and Wheeler (1985), two sets of electrodes were placed parallel to the base of the neck and centred about the prominence of the seventh cervical vertebra. The other two sets of electrodes were placed on the left anteriolateral chest surface; one at the end of the ninth intercostal space near the mid-clavicular line, and the other in the tenth intercostals space near the mid-axillary line. The electrical current electrodes were on the right of the neck and on the left of the thorax, with the remaining two electrodes used as voltage electrodes. Studies 2 to 5 used a 4 - band electrode configuration placed on the back of the neck and the thorax (Sherwood et al., 1990); Fig. 2.7.

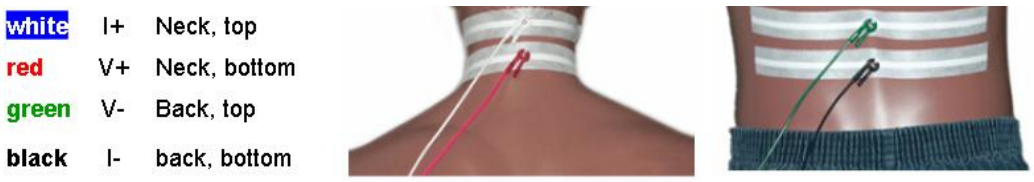

Fig. 2.7: ICG 4-band electrode configuration (BIOPAC Systems, Inc.)

The signal from the electrodes was filtered through the BIOPAC module that delivered impedance magnitude (Zo) and derivatives (dZ/dt) at 1000 Hz. The impedance signals were analysed using BIOPAC software in the first study and an algorithm developed in our own laboratory in order to detect the following measures for each cardiac cycle: Cardiac Output (CO), Stroke Volume (SV), Left Ventricular Ejection Time (LVET), Pre-Ejection Period (PEP), and Total Peripheral Resistance (TPR).

Electrical bioimpedance is the characteristic resistivity of a volume of tissue and fluid. In the case of cardiac output (CO), the relevant tissues are located around the heart and thorax (Sherwood et al., 1990). CO was calculated by multiplying the *stroke volume* by the *heart rate*. *Stroke volume* is the pulsatile volume of blood ejected from the heart and was calculated in Study 1 using

the Sramek-Bernstain approach which views the thorax as “a volume conductor that is geometrically similar to a truncated cone” (Sherwood et al., 1990; p. 14). The equation includes a measure of body height and weight as an accurate estimate of the volume of participating electrical tissues for each individual (Quail, Traugott, Porges & White, 1981). However, the BIOPAC algorithm was found to be error-prone. Consequently, an algorithm was developed to detect significant points in the ICG wave (e.g., Q, B, X) and ECG based on existing research (e.g., Sherwood et al., 1990; Lozano Norman, Knox, Wood, Miller, Emery et al., 2007). The algorithm provided a calculation for impedance measures using the Kubicek formula (Kubicek, Karnegis, Patterson, Witsoe, & Mattson, 1966). The C point was defined as the maximum point in the dz/dt signal in a time window 60–200 ms from the R peak (Gratze, Fortin, Holler, Grasenick, Pfurtscheller, Wach et al., 1998), the X point was defined as the minimum point over the course of the cycle after the C point whereas B was set as the maximum derivative of the dz/dt signal in a time window 150–100 ms before the C point. The algorithm was validated based on visual inspection and manual scoring from a trained observer. Baseline data (10 minute) from the study from 20 participants were scored manually by the trained experimenter and compared to the results from the algorithm. With respect to LVET times, the mean deviation between the manual and computerised scores was 82 ms (s.d. = 30 ms; range = 23–110 ms). For PEP, the mean deviation was 25 ms (s.d. = 23 ms; range = 5–79 ms). A correlation was conducted across the whole data set to assess PEP between manual scoring from a trained observer vs. computerised analysis, it was found that scores were highly correlated (r = 0.89) indicated a high reliability of the algorithm.

Heart rate measures

The Inter-Beat Interval (IBI) from the heart was calculated from an electrocardiographic (ECG) signal filtered between 0.5 and 0.35 Hz and sampled at 1000 Hz. This signal was collected via a two-lead electrode sensor placed on the participants’ left and right rib cage (Fig. 2.8) connected to the TEL100C data capture signal (BIOPAC Systems Inc.) that was attached to the MP150 system (i.e., the ground electrode for the ECG was

not required as there was a ground electrode already incorporated in the NICO100C apparatus; Fig. 2.8). This stable configuration provides better noise performance and stability. The TEL100C system includes a portable amplifier/transmitter, which converts up to four channels of data into a modulated data stream. This data stream travels over a single lightweight coaxial cable to the receiver module. The receiver module demodulates the data and sends it to the MP for recording and analysis.

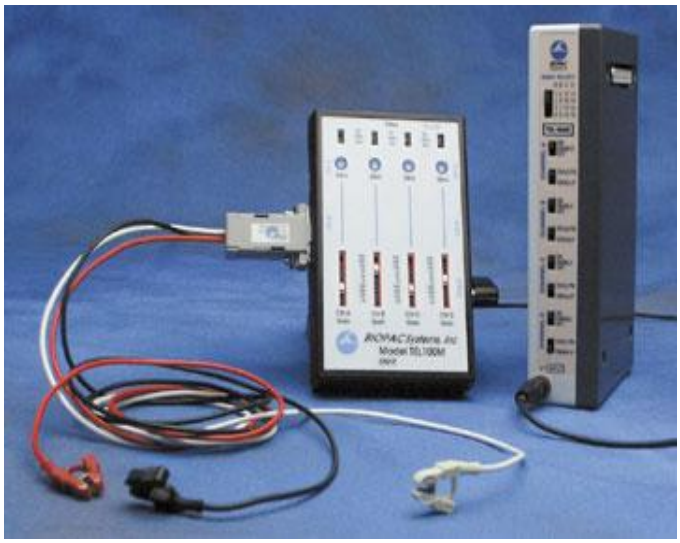

Fig. 2.8: TEL100C (Lead, Tel100M and ECG 100C) used to measure ECG (BIOPAC Systems, Inc.)

Blood pressure was measured using a CARESCAPE Vital Signs Monitor (V100) (DINAMAP Inc.) which involved placement of an inflatable cuff on the upper left arm. Readings of systolic blood pressure, diastolic blood pressure, heart rate and Mean Arterial Pressure (MAP) were all obtained using the oscillometric method (note: for the purpose of analysis, heart rate was measured from the ECG trace obtained via the MP150, not the CARESCAPE monitor).

Other cardiovascular measures included: Ventricular Contractility and pre-ejection period (with smaller values of pre-ejection period indicating greater VC) derived from the ECG and the ICG waves. Pre-ejection period was identified as the time elapsed between the Q point on the ECG wave (the left

ventricle contracting) and the B inflection on the ICG wave (the opening of the aortic valve) (Stemmler et al., 2007). Total Peripheral Resistance (TPR) was calculated by combining information from the CARESCAPE and the NICO100C, i.e. TPR = MAP x 80 / CO).

Respiration

Respiration was measured by using two elasticised TSD201 BIOPAC bands; one band placed on the chest and a second on stomach (Fig. 2.9) and then integrated the two signals to accommodate people who breathe from chest or stomach (Fairclough & Venables, 2006). Respiration rate was amplified using BIOPAC TEL100C apparatus with the filter setting at .05 - 35 Hz. The time difference between the maximum of each successive inhalation was taken as the duration of the respiration cycle (Grossman & Taylor, 2007). The amount of respiration cycles per minute was taken as respiration frequency. The respiration was recorded as a validation of the impedance data, where no disturbances in the measurements occurred due to the respiration cycle.

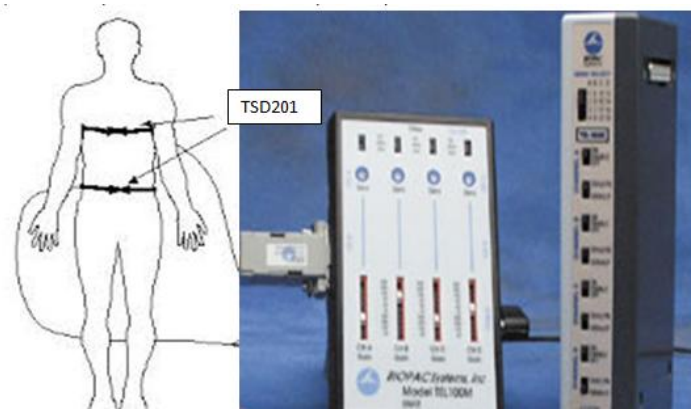

Fig. 2.9: TEL100C system for respiration rate (BIOPAC Systems, Inc.)

Facial Electromyogram (fEMG)

fEMG were obtained from the Zygomaticus major and the Corrugator supercilii muscles as indicators of positive and negative emotion, respectively. fEMG data were sampled at 1000 Hz and filtered between 30 and 500 Hz. The EMG100C Electromyogram Amplifier was added to the MP150 system for recording of the zygomaticus facial muscle electrical

activity. The Zygomaticus activity was recorded through two electrodes connected to the EMG100C module attached to the BIOPAC MP150 system. Corrugator muscle activity was indexed through two external electrodes from the BIOSEMI apparatus.

During post-processing, the sample rate was reduced to 512 Hz and artifacts due to eye blinks were removed (vertical EOG was recorded separately and this signal was subtracted from the corrugator trace). The resulting fEMG data was normalised using a root-mean-square (RMS) transformation. The signal was then rectified, time integrated, and divided by the duration of the segment to obtain the mean rectified voltage (Fridlund & Cacioppo, 1986). Corrugator activity was analysed in all the studies and Zygomaticus activity was measured in Studies 1 and 3.

Frontal brain asymmetry - 32 channels configuration

Measures of frontal EEG asymmetry were used in studies 1 and 2. Studies of the thesis were designed to compile an emotional model of anger with challenge/threat dimensions transposed in cardiovascular changes correlated to frontal brain asymmetry measures. However, the subsequent studies looked at applicability of such model in a changing environment via music or ambient light. EEG was recorded monopolarly from 32 Ag-AgCl pin-type active electrodes mounted in a BioSemi stretch-lycra head cap connected to a BioSemi amplifier (BioSemi Inc.) at a sampling of 512 Hz and a continuous digitization using windows of 16.4 sec (16,384 points); Fairclough and Roberts (2011).

Electrodes were positioned using the 10–20 system (Fig. 2.10) and the EEG activity recorded from the following sites: frontal pole (FP1, FP2), anterior frontal (AF3, AF4), pre-frontal (F7, F8), frontal (F3, Fz, F4), frontocentral (FC5, FC1, FC2, FC6), central (C3, Cz, C4), temporal (T7, T8), parietocentral (CP5, CP1, CP2, CP6), parietal (P7, P3, Pz, P4, P8), occipitoparietal (PO3, PO4) and occipital (O1, Oz, O2). Electrodes were also placed at earlobe sites (A1, A2) and around eye allowing offline re-referencing with a linked ears montage and correction of the artifacts.

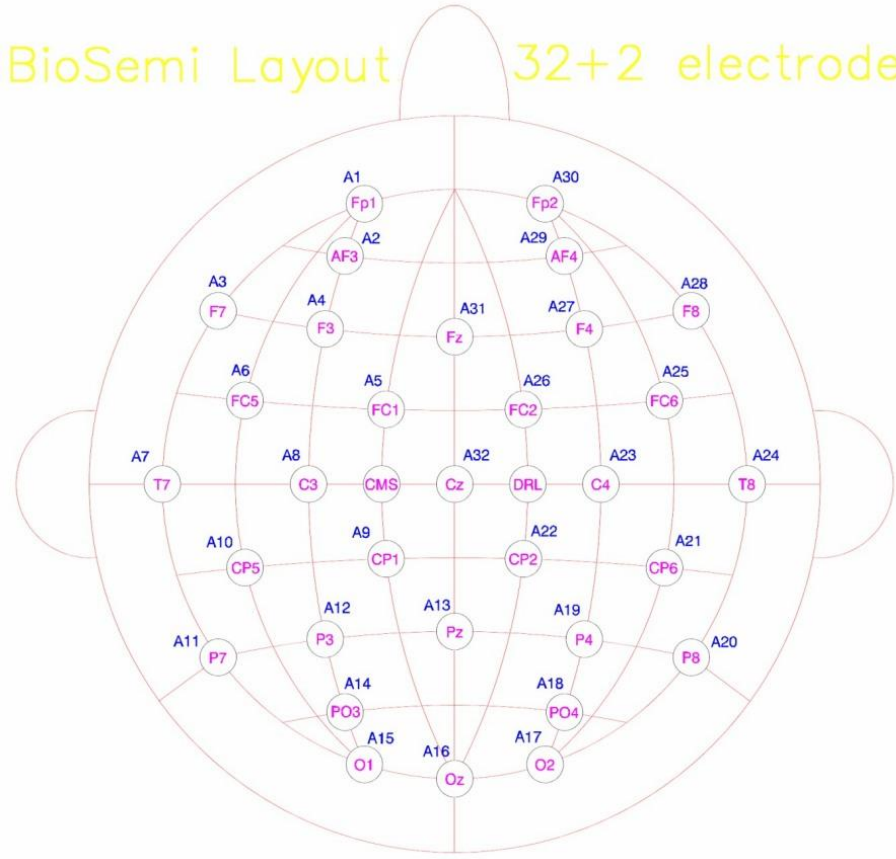

Fig. 2.10: The 32-electrode EEG configuration plus 2 ground electrodes (www.biosemi.com)

EEG was recorded continuously throughout baseline prior to the task (with eyes open and eyes closed) and continuously throughout the task. Analysis was performed using BESA software (MEGIS software GmbH, Gräfelfing, Germany). Offline filtering was performed using high and low pass filters of 0.05 Hz and 60 Hz respectively and a notch filter of 50 Hz.

In order to identify the spectral power at the frontal sites without reflecting the activity at a reference lead, an inactive linked ears reference montage was chosen (Hagemann, Naumann & Thayer, 2001). The common vertex (Cz) reference, one of the most frequently used reference schemes, has the disadvantage of a high signal to noise ratio. The vertex is an electrically active site and it could be presumed that the frontal sites have a lower alpha activity than vertex. Hence, the derivations of frontal sites (F3, F4) against vertex reflect less pre-frontal structure and more vertex activity (Hagemann,

2004). The solution to this problem with the use of linked ear-lobes/mastoids (A1 + A2) could be a better solution because of lower electrical activity compared to vertex (Hagemann, 2004). The topographic validity of this reference scheme is based on the assumption that the actual reference site (A1 + A2) is substantially less active than the cephalic target site. Although there is no empirical data to support this assumption, a study by Hagemann et al (2001) indicated that bipolar derivation of the ears (A1 + A2) show smaller activity than the activity of a central derivation indicating that (A1 + A2) reference is more appropriate for the measurement of frontal asymmetry than the Cz reference (Dien, 1998).

Another factor that may be influencing asymmetry measures is the instruction to keep eyes open or closed while the resting EEG is recorded. However, studies indicated good internal consistencies of EEG baselines that were recorded with eyes open and closed irrespective of the EEG reference used (Sutton and Davidson, 1997). After the recording of the EEG, the analysis involves the rejection or correction of artifacts. The ocular activity could have generated substantial power in the alpha range which may distort EEG alpha symmetry. However, ocular artifacts do not affect measures of individual differences of alpha asymmetry. The control of ocular artifacts is necessary for the present thesis because the analyses involved mean comparisons (Hagemann and Naumann, 2001). The signal of ocular origin could appear in the 8-13 Hz alpha range used in EEG asymmetry research (Allen et al., 2004). The concern that the ocular signal may contain alpha band neural activity prompted the need for employing correction procedures post recording. The recording involved measures of alpha activity with the eyes open and eyes closed. By subtracting alpha activity when the eyes were closed from alpha activity when the eyes were open we eliminate the ocular neural activity in the alpha band. In the initial study of the thesis both resting conditions (eyes open and eyes closed) were reported because of their significant differences compared to test. However, in the second study only the eyes open condition was reported. Automatic correction of blink artifacts and horizontal and vertical saccades was performed using detection through predefined topographies. Muscle activity over 100 μV was

also excluded (Fairclough & Roberts, 2011). An average of 5.8% of analysed data was rejected for each participant due to artifacts.

Fast Fourier transforms (FFT) were computed over overlapped windows of 2 s (1024 points). Fast Fourier is a method used to extract power spectra from assumed periodic raw signals (Allen et al., 2004). Although, the EEG signals are not strictly periodic, in that they do not repeat at short intervals, by selecting short epochs allowed identifying similar features at different points in the EEG waveform and by overlapping, the similarities received maximum weighting. Overlapping also prevented the data near the end of the epoch to be discharged as data minimally weighted at the end of an epoch *x* was weighted more heavily in the subsequent epoch (Allen et al, 2004).

From the two spectra results of the FFT (a power spectrum and a phase spectrum), the analyses focused on the power spectrum which reflected the power in the signal at each frequency from DC to Nyquist's (1928) frequency on the principle that accurate highest frequencies represent one-half of the sampling rate. At the next step, average power spectra were computed for each experimental condition and summarised within the alpha frequency band (8-13 Hz). Although, other studies used some divisions of alpha band for specific tasks and applications (Koslov et al, 2011) the global 8-13 Hz definition of alpha band has been found to give a fairly uniform baseline in adult participants (Allen et al., 2004) and has been used extensively in previous research (e.g., Peterson, Shackman & Harmon-Jones, 2008). Alpha power in μV2 as an index of the inverse of cortical activity (Davidson, 1988) was natural log transformed to avoid skewness of the data (Allen et al., 2004). Because frontal asymmetry was ultimately of interest, a difference score metric between left hemisphere sites (AF3, F7, F3, FC1, FC5) and right hemisphere sites(AF4, F8, F4, FC2, FC6) was employed using the formula Ln [right total alpha power]−Ln [left total alpha power] to generate an index of asymmetry for each homologous pair of electrodes in alpha frequency band. Positive values on the index indicated greater relative right alpha power and greater relative left frontal activity, while greater relative

right frontal activity was indicated by negative values (Allen et al., 2004). An advantage of using this difference of natural log transformed scores is that it provided some degree of correction for overall alpha power, as the magnitude of the asymmetry could be confounded by individual differences.

CHAPTER 3
STUDY 1

Psychophysiological markers of anger in combination with levels of control during a computer-based problem-solving task.

Abstract

Psychophysiological responses during states of anger can be confounded with reactions associated with the perception of control. The aim of the present study was to identify psychophysiological markers of anger in combination with levels of control during a laboratory task. Forty participants were split into four experimental groups: anger/no control, anger/control, neutral/no control, and neutral/control. Anger (anger state vs. neutral state) was manipulated via an experimenter effect (i.e., rude vs. polite experimenter). Participants were exposed to a computer-based problem-solving task where the computer either worked correctly (control) or malfunctioned (no control). A number of psychophysiological variables (BP, cardiovascular impedance and frontal EEG asymmetry) were collected in addition to subjective data. Self-report measures indicated that the manipulation of anger and control was successful. Relative to baseline, systolic BP increased significantly only in the anger/no control condition, whereas diastolic BP increased in the anger/no control, anger/control, and neutral/no control conditions (compared to neutral/control). Cardiovascular impedance measures were insensitive to the experimental manipulations. With respect to measures of frontal EEG asymmetry, frontal peripheral sites (FP2-FP1) showed the expected increase of right-brain activation in the anger/no control condition. Conclusively, systolic BP and frontal peripheral EEG asymmetry were indicators of anger in combination with the perception of a state of no control, suggesting that emotion and motivation interact in order to intensify psychophysiological measures of anger.

3.1. Introduction

From the pioneering work of Picard (1997) on affective computing, psychophysiology was used to quantify emotional states, in particular frustration (Kapoor et al., 2007), or motivational states underlying the experience of entertainment technology (Mandryk et al., 2006; Yannakakis et al., 2007). Within the affective computing literature the term frustration is used loosely to illustrate a negative state that easily indicates anger. However, constant development of 'smart' technology to support human health and well-being could be directed towards systems that allow increased autonomy and adaptive capabilities, and enhanced symbiotic human-machine interaction (Norman, 2007).

Research on affective computing systems has employed a one-dimensional model of emotional valence (Picard & Klein, 2002; Picard, 2003) or a two-dimensional space of activation and valence (Kulic & Croft, 2005, 2006). Nevertheless, reliance on a one-dimensional representation of the user's psychological state may restrict the range of adaptive responses that can be expressed within that framework (Norman, 2007). Affective computing has moved from detection of specific categories of emotion (Partala & Suraka, 2004) to the detection of negative states related to performance on computer tasks (Kapoor et al., 2007). Although such systems are perceived as useful in the context of self-regulation (Scerbo, Freman, Mikulka, Parasuraman, Di, Nocero et al., 2001) they lack psychological sophistication regarding the definition of *negative emotion*. Moreover, given that an interactive system can also be used as a health-monitoring tool (Gerasimov, Selker & Bender, 2002) careful consideration should be paid as to what constitutes an *unhealthy* emotion. An interactive system that identifies physiological expressions of motivation and emotion could extend self-awareness as a strategy for reducing the experience of a negative emotion (Mauss et al., 2007) with beneficial consequences for health.

It has been argued that the immune system is perturbed by prolonged negative emotions, which contribute to the production of proinflammatory proteins (Kiecolt-Glaser, McGuire, Robles & Glaser, 2002b). A 9-year

longitudinal study found that chronic anger doubled the risk of cardiovascular mortality (Everson, Kauhanen, Kaplan, Goldberg, Julkunen, Tuomilehto et al., 1997). A meta-analytic review (Kreibig, 2010) indicated that anger-in, which can be interpreted as anger in combination with a coping strategy of passive avoidance (Stewart et al., 2008), could be associated with higher blood pressure and could increase the severity of cardiovascular disease. However, a lack of methodological control over the full spectrum of health behaviors generated a full list of contradictory results (Schulz, Beach, Ives, Martire, Ariyo & Kop, 2000). The discrepancies could be understood to arise from a bidirectional interpretation of the findings. In fact, anger may raise the risk for cardiovascular disease and the presence of disease can also trigger higher levels of anger. It has been suggested that absence of an individual's control over a disease can be an external source for anger (Kreibig, 2010). Therefore, it is important to develop valid affective computing systems that allow self-regulation and assist in health monitoring (Norman, 2007).

The design of future affective computing systems should take into consideration the fact that the mechanism beyond self-regulation is a motivation trigger and that any emotion incorporates cognitive and motivational facets, as well as an affective dimension (Matthews et al., 2002). Therefore, the adverse impact of negative emotions on health may be attributed to motivation or to cognitions associated with a particular affective state. For instance, anger in combination with lack of coping resources creates a sense of helplessness (Stemmler et al*.,* 2007), whereas anger coupled with sufficient resources increases motivation to approach a task constructively. Thus, negative emotions that are unhealthy often represent a combination of affect within a motivational context and the same distinction should be attempted by an affective computing system.

The role of motivational systems in the experience of emotion has received considerable attention in the frontal asymmetry literature (Berntson, Norman & Cacioppo, 2011) and cardiovascular research (Stemmler et al., 2007). The theory that generated this interest claimed that a behavioural activations system incorporates positive emotions and motivates approach behaviour

(Carver & Harmon-Jones, 2009). Also, a behavioural inhibition system responds to negative emotion and leads to avoidance of actions (Gray, 1987; Gray & McNaughton, 1996). The model of emotion-motivation was questioned in the work of Harmon-Jones (2004a), which distinguishes approach from avoidance responses in an anger-provoking situation based on *asymmetrical frontal brain activity*. In this case, a single negative emotional category is split by considering two different motivational dispositions as context. Specifically, left hemisphere activation is associated with anger/approach whilst right hemisphere activation characterizes anger/avoidance. Nonetheless, there is a large body of literature (Davidson, 1993; Hagemann et al., 1998; Hewig et al., 2004; Harmon-Jones, 2004a) on the role of the brain in the understanding of emotion and motivation. Davidson (1993) proposed a model of frontal brain asymmetry and emotion. An approach system is activated by the perception of achievable goals, which elicits positive emotion and it is thought to be associated with greater left prefrontal cortex (Hewig et al., 2004). On the other hand, the avoidance system is activated by aversive stimuli, which elicits negative emotions and leads to withdrawal. The neuroanatomical basis for the avoidance system is considered to be the right brain hemisphere (Hewig et al., 2004). In terms of negative emotion, fear is often conceptualized as involving withdrawal, whereas anger is seen in association with approach state. However, not all studies that induced a state of anger found evidence of neural left asymmetry (e.g., Waldstein et al., 2000; Hewig et al., 2004). Hence, there is limited research (Stemmler et al., 2007; Harmon-Jones, 2004a) that analyses each type of emotion, either positive or negative, in relation to each motivator trigger (approach or avoidance). Stemmler et al. (2007) attempted to detach motivational disposition from affect in order to describe different affective-motivational states, such as fear and anger. Such work is based on a division between fear-withdrawal from goals (noradrenergic response) and anger-approach towards goals (adrenergic response) (Stemmler, 2004). The merit of the paper by Stemmler et al. (2007) consisted in opening the debate regarding whether each affective state (anger and fear, in this instance) could be coupled with each motivational state (approach or withdrawal). As expressed by Stemmler et al. (2007), approach motivation serves to stay

actively engaged in order to narrow the distance to a challenging goal. On the other hand, withdrawal motivation serves to increase the distance to a threatening stimulus. However, the results obtained were not always accurate as they were based on a mathematical estimation of some missing raw values. Also, the self-reports did not reflect a successful manipulation of the motivational states.

Within the cardiovascular field, the challenge/threat model finds some support in the work of Lovallo, Pincomb and Wilson (1986), which emphasizes the effects of reward/punishment incentives on cortisol levels. Findings consistent with those of Lovallo et al. (1986) have been reported by Frankenhauser (1986). Loss of control increased cortisol levels leading to an "effort with distress" state, as opposed to the "effort with stress" effect observed in the control condition. Hence, challenge motivation can be triggered when the individual perceives a degree of control over the task, whereas threat may characterize a situation where the person perceives an absence of control. Nonetheless, distinguishing only between motivational states failing to consider the emotion is limited in scientific quality. Blascovich and Tomaka (1996) claimed that the motivational states generated by an appraisal process are important when differentiating between positive and negative emotion. For example, a coping task could be appraised as a positive *challenge* when the perceived resources of the individual meet subjective demand or as a negative *threat* when perceived demand exceeds resources (Blascovich & Tomaka, 1996). This type of appraisal triggers motivational predispositions for action to either *approach* (to respond to a challenge or remove the obstacle) or *avoid* the threatening situation (Stemmler et al., 2007). In support of their theory, Blascovich and Tomaka (1996) argued that challenge is linked to a sympathetic-adrenomedullary (SAM) response, whilst threat reflects a pituitary-adrenocortical response (PAC). For instance, SAM involves increased heart rate (HR), a greater volume of blood expelled by the heart (cardiac output, CO), amplified contraction of the left ventricle (left ventricular ejection time, LVET) and less resistance to blood flow in the systemic circulatory system (total peripheral resistance, TPR). In contrast, PAC activity entails the same pattern of

response but not to the same extent. An update of the model (Blascovich & Mendes, 2000; Blascovich et al., 2001) included blood pressure as a parameter that distinguish between threat and challenge, with an increased level during threat appraisal. In particular, the anger response is characterized by α- and β-adrenergically mediated cardiovascular effects: increased HR, increased SBP and DBP, and increased TPR, accompanied either by increased SV and CO (Hamer et al., 2007). This response pattern is further characterized by measures indicating shortened PEP (Montoya et al., 2005) and LVET (Montoya et al., 2005), lower TWA (Stemmler et al., 2007), increased HI (Montoya et al., 2005; Stemmler et al., 2007), increased R–Z time and decreased FPA (Stemmler et al., 2007) or unchanged FPA, and FT (Rochman & Diamond, 2008). However, the empirical findings (Blascovich & Tomaka, 1996; Blascovich et al., 2001) were presented in relation to the proposed threat/ challenge model were not always accurate (Wright & Kirby, 2003). In particular, the heart rate parameter failed to distinguish between the states of challenge and threat (Blascovish & Tomaka, 1996). In more recent literature (Gendolla & Krüsken, 2002), the role of heart rate in distinguishing between emotional states as well as between motivational states was questioned. It has been explained that identifying heart rate as a valid cardiac parameter is difficult because of the periodic fluctuations of the breathing pattern (Butler et al., 2006).

A number of previous studies (Boiten, Frijda & Wientjes, 1994; Ritz, 2004; Homma & Masaoka, 2008) relate respiration to emotional arousal, whereas the actual contribution of respiration during motivational states remains poorly understood. Respiration sinus arrhythmia (RSA) is associated with the vagal influences of the heart and can be considered a measure of the parasympathetic nervous system (Grossman & Taylor, 2007). RSA seems an important measure in emotion research; higher values are related to negative emotions (Nyklíček, Thayer & Van Doornen, 1997; Butler et al., 2006). However, hyperventilation (HV) can be considered a respiration parameter that distinguishes between states of challenge and threat. van Diest, Winters, Devriese, Vercamst, Han, van de Woestijne et al. (2001) explained that HV is a defense response indicating the preparation to

escape, thus to avoid the threatening stimulus. In their study, van Diest et al., (2001) compared the respiration pattern between a threat state and a pleasant affective state. Although problematic, their study brought into light the possibility of separating motivation and emotion. If respiration rate is found to distinguish between challenge and threat motivation, then heart rate should follow a similar trend. Nonetheless, the respiration literature remains weak in supporting the challenge/threat model proposed by Blascovich and Tomaka (1996).

A general criticism could be raised of most previous work. The heart-brain interconnection has not been given due attention and few studies have looked at the relation between cardiac reactivity and electrocortical activity. A right frontal electroencephalographic (EEG) response in an anger state was associated with increased blood pressure, suggesting that asymmetric frontal EEG responses to emotion produce different patterns of cardiovascular reactivity (Waldstein et al., 2000). However, the limited number of studies on heart-brain interaction have failed to provide an insight into the motivational facets of various emotions. The development of a taxonomy of anger and its physiological implementations, whether neural (Harmon-Jones, 2004a) or cardiovascular (Stemmler et al., 2007), needs to consider both motivational directions (approach vs. avoidance). Thus, the current study will focus on measuring cardiovascular activity and frontal EEG asymmetry) in order to differentiate between a *high anger state* with a passive motivational orientation (anger/no control) from a *low anger state* with an active response (anger/control). The following hypotheses were formulated.

1. Anger in combination with no-control (avoidance) motivation will be accompanied by a PAC response characterized by higher total peripheral resistance and an increased magnitude of cardiac output, left ventricular contractility and pre-ejection period (threat).
2. Left frontal activation increases in an anger/control condition *while* right frontal activation increases in an anger/no-control condition.

3.2. Method

3.2.1. Participants

Forty one right-handed participants, 17 males (age = 27.18 ± 8.91 years) and 24 females (24.79 ± 7.15 years) were allocated to four different experimental groups: (anger/no control, anger/control, neutral/no control and neutral/control; see Table 3.1). To eliminate variance due to trait anger only participants with a score below 80 percentile on the Trait Anger Expression Inventory (Spielberger, 1999) were selected for the experiment. Relevant personality traits (Ten-Item Personality Inventory validated by Gosling, Rentfrow & Swann (2003)) were controlled. That is, neuroticism and extraversion were equivalent across all four groups; $F(3,37) = 0.35$, $p > .05$ and $F(3,37) = 0.47$, $p > .05$, respectively. The participants were also selected on the basis of their mental fitness (no history of psychiatric illness; not taking anti-depressant medication) and health (normal blood pressure; no history of a heart conditions; not on any prescribed medication; healthy body weight). All procedures for recruitment and running the study were cleared by Liverpool John Moores University Ethics Committee.

Table 3.1: Gender ratio and mean ± SD age of the participants

	Number of participants		
	Male	**Female**	**Age (years)**
Anger / Control	4	6	28.8 ± 9.51
Anger / No control	4	6	24.3 ± 6.18
Neutral / Control	5	6	26.6 ± 8.51
Neutral / No control	4	6	23.4 ± 7.03

3.2.2. Study Design

A between participants design was employed with the combination of the two factors (Emotion (anger/neutral) and Motivation (control/no control) resulting in 4 experimental conditions *(*control/anger; control/no anger; no

control/anger; no control/no anger). The dependent variables were the subjective self-report scales (self-reported anger and control) and psychophysiological responses (frontal EEG asymmetry, cardiovascular activity, facial muscle activity, respiration) at two levels (baseline and test).

3.2.3 Apparatus and material

Self-report measures

A number of self-report electronic questionnaires were used as manipulation checks: The State Anger Expression Inventory 2 (Spielberger, 1999) was used to measure subjective anger both at pre and post-test stages. The Confidence and Perceived Control Scale from Dundee Stress State Questionnaire (Matthews and Desmond, 1998) was employed to index subjective levels of confidence and control while performing the task.

Psychophysiological measures

Cardiovascular measures

Cardiac activity was recorded using NICO100C Impedance cardiogram (ICG) at an operational frequency of 50 kHz and a magnitude range of 5 Ohms/volt. Electrical bioimpedance is the characteristic resistivity of a volume of tissue and fluid. In the case of cardiac output (CO), the relevant tissues are located around the heart and thorax (Sherwood et al., 1990). CO was calculated by multiplying the *stroke volume* by the *heart rate*. *Stroke volume* is the pulsatile volume of blood ejected from the heart and was calculated using the Sramek-Bernstain approach which views the thorax as "a volume conductor that is geometrically similar to a truncated cone" (Sherwood et al., 1990; p. 14). The equation, unlike the widely used Kubicek equation which assumes that blood resistivity is equal for all individuals, includes a measure of body height and weight as a more accurate estimate of the volume of participating electrical tissues for each individual (Quail et al., 1981). The *heart rate* was calculated from R–R intervals in the electrocardiogram (ECG). The ECG was also used to capture the mid-frequency component of heart rate variability (0.09-0.13Hz known as the 0.1 Hz component). The 0.1 Hz component provides an index of mental effort (Nickel & Nachreiner, 2000), which can indicate how anger and no control

influence effort. The upper band (0.14-0.40Hz) was used to represent vagal tone which is an index of parasympathetic activation.

ECG was recorded through two electrodes placed on the participants' left and right rib cage. The electronic signal was amplified by BIOPAC TEL 100C with the filters set at 0.5 Hz and 35 Hz, respectively. The ground electrode was incorporated in the impedance cardiogram (ICG) configuration. Two spot electrodes (for each of the four leads) were used instead of just one spot electrode in order to closely resemble the more accurate band electrodes. Using Penney et al.'s (1985) configuration, two sets of electrodes were placed parallel to the base of the neck and centred about the prominence of the seventh cervical vertebra. The other two sets of electrodes were placed on the left anteriolateral chest surface; one at the end of the ninth intercostal space near the mid-clavicular line, and the other in the tenth intercostals space near the mid-axillary line. The electrical current electrodes were on the right of the neck and on the left of the thorax, with the remaining two electrodes used as voltage electrodes.

Other cardiovascular measures included: Mean Arterial Pressure (MAP) and systolic/diastolic blood pressure (BP) which were obtained using the Dinamap apparatus. Total peripheral resistance (TPR) was calculated as (MAP × 80) / CO. Ventricular Contractility and pre-ejection period (with smaller values of pre-ejection period indicating greater VC) derived from the ECG and the ICG waves. Pre-ejection period is identified as the time elapsed between the Q point on the ECG wave (the left ventricle contracting) and the B inflection on the ICG wave (the opening of the aortic valve) (Stemmler et al., 2007). The PEP is an index of peripheral sympathetic activation. AcqKnowledge Software derived by Biopac (version 3.8.3) was used to calculate the formula; however, the identifications of points in the ICG wave (e.g., Q, B, X) and ECG based on existing research (e.g., Sherwood et al., 1990; Lozano et al., 2007) was done manually by a trainer observer.

Respiration

Respiration was measured by using two elasticised bands placed around the

chest and diaphragm (Fairclough & Venables, 2006). Respiration rate was amplified using BIOPAC TEL100C apparatus with the filter setting at .05 - 35 Hz. The time difference between the maximum of each successive inhalation was taken as the duration of the respiration cycle (Grossman & Taylor, 2007). The amount of respiration cycles per minute was taken as respiration frequency.

Facial EMG activity

Facial Electromyograms were obtained from the Zygomaticus major and the Corrugator supercilii muscles as indicators of positive and negative emotion, respectively (Lang, Greenwald, Bradley, & Hamm, 1993). Corrugator muscle activity was indexed through two external electrodes from the BIOSEMI apparatus and the Zygomaticus activity through two electrodes attached to the BIOPAC TEL100C system. The signal was then rectified, time integrated, and divided by the duration of the segment to obtain the mean rectified voltage (Fridlund & Cacioppo, 1986).

Frontal brain asymmetry

Thirty two channels of Electroencephalography (EEG) were recorded in order to capture frontal EEG asymmetry. Frontal asymmetry was expressed as alpha suppression from left and right frontal sites (Fp1 and Fp2; F3 and F4; F7 and F8; Af3 and Af4) following the procedure used by Harmon-Jones and Allen (1998). The EEG signals were amplified using BIOSEMI apparatus. The high and low bandpass filters were set at 0.1 Hz and 35 Hz, respectively (Harmon-Jones & Sigelman, 2001). The raw EEG data was corrected for ocular artifacts and physical movement using BESA software. The EEG data were then converted to a linked-ears reference montage and analyzed via Fast Fourier Transformer in steps of 2 seconds. Mean -power- amplitude values were obtained for the alpha band (8-13 Hz).

3.2.4. Procedure

Pre-test

The participants who passed the screening procedure were invited to the laboratory. After signing the informed consent form, participants completed a

set of demographic questions and electronic self-report questionnaires of their state anger and mood state. Also, participants were measured for height and weight, which were needed for the calculation of the ICG measure of the stroke volume. Using a blind protocol, participants were led to believe that their task requires “participation in a cognitive task”. It was necessary to initially deceive the participants in order to elicit the desired emotional reactions in ways that closely resemble a real-life situation. However, all participants were fully debriefed afterwards as to the true nature of the experiment and were reminded of their right to withdraw their data so should they wish.

Following the results on pre-screening personality trait questionnaires, participants were quasi randomly allocated to conditions. A series of measures were put into place in order to establish the illusion of the presence of an unseen researcher that sent neutral and angry messages over the telecom. First, the actual experimenter did the set-up acting as an assistant and then passed the participant into the hands of a fictitious unseen experimenter. The fictitious experimenter communicated with the participants once they entered the experimental room. In order for this illusion to work, a sense of another person being present in the other room was created. The unseen female experimenter addressed the participant via an audio link after all the physiological apparatus was put into place (ECG, ICG, BP cuff, respiration belts, EEG, fEMG) instructing them in a calm tone to begin the computer task.

Test

The computer task comprised of a Number Stroop task (Fig. 3.1) where the participants had to press the number key that accounted for the number of digits presented on the computer screen.

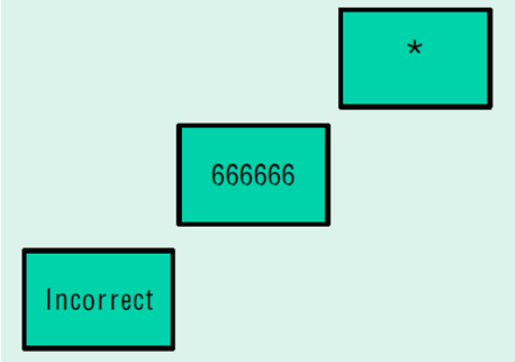

Fig. 3.1: Screenshots of the sequences of the Number Stroop task used in the study

The task was created using E-prime software and comprised of 40 sets of number strings of combined 3 to 6 digits of which half were similar (i.e., 333 = 3) and 26 dissimilar (i.e., 3333 = 4). The string of digits were presented on a 15" PC monitor for 100 msec in black font on white background. Physiological measures were recorded by the researcher prior the task as a baseline, and then during the task.

Baseline measures were taken in two epochs of 5 minutes each, while the participant sat down on a chair in a relaxed position with the eyes open for the first 5 minutes and with the eyes closed for the second baseline. The second baseline was only monitored for the EEG measures as a standard procedure to remove ocular artifacts (Harmon-Jones, 2004a). Baseline measures were taken after the experiment.

Emotional reactions and motivational directions were manipulated during the performance of the cognitive task. The anger manipulation was adapted from Stemmler (1997). Participants in the anger condition were warned three times by a recorded voice through telecom link that:

(1) *You are moving too often. You are making the physiological measures useless* (at beginning of the task).
(2) *Stop moving so often* (in the middle of the task).
(3) *You have to come back to do the testing again. Your data cannot be used at this point* (at the end of the task).

The control vs. no-control manipulation was adapted from Partala and Suraka (2004). Participants in the no-control condition experienced problems with a malfunctioning computer system (e.g., key number 4 did not respond to the assigned use) and participants received erroneous feedback. In total, there were 8 acceptable correct answers randomly alternated with 16 answers requiring the use of the malfunctioning key number 4 and 16 incorrectly verified answers. The feedback received on the PC screen included information about the length of the response time and the average percent of correct answers.

Post-test

After completing the cognitive task, participants were asked to complete again the set of questionnaires presented in Section 1.2 plus the electronic version of the Confidence and Perceived Control Scale from Dundee Stress State Questionnaire (Matthews & Desmond, 1998). In the event that the participant complained about the task (especially those in the no-control condition) or about the virtual experimenter (especially those in the anger condition), the procedure involved standardised responses. For example, in the case of complaints about the experimenter “We’ll take a look at the data and let you know if we need you again in a moment, just complete the questionnaire for now”. In terms of handling complaints about the apparatus, the experimenter feigned surprise: “Really? It wasn’t working? OK, if you could fill in this questionnaire for now and I’ll take a look at it when you’re done.”

After completion of the questionnaires, the physiological apparatus was removed from the participants and a full debriefing was provided as to the true nature of the experiment. Participants received the debriefing sheet corresponding to the experimental group in which they were placed and asked the extent to which they were aware of the true nature of the “cognitive performance task” (anger provocation). Also, the participants were informed that they could listen to some positive music (Mozart’s "Eine Kleine Nachtmusik" was validated as positive mood induction music by Moore & Oaksford, 2002) if they felt that their mood had been altered to an

uncomfortable level during the experiment.

3.3. Results

The psychophysiological data were corrected for skewness using natural logarithm transformation (Allen, Coan and Nazarian, 2004). The experimental data were subsequently analyzed using SPSS *version*-16 and any outliers (± 3 SD) removed. A Mixed 4 x 2 ANOVA for each individual physiological variable was carried out to investigate differences between the four experimental conditions at 2 levels (baseline and test). Post-hoc tests were performed using a *Bonferroni* procedure.

Self-report measures

According to the STAXI data (the extremes are from a minimum score of 15 to a maximum score of 60), the anger manipulation was successful ($F(1,37) = 10.88$, $p < .001$). Bonferroni pairwise comparisons revealed that anger state increased significantly (ps < .02) in the anger/no control and anger/control conditions compared to the neutral/control condition. Descriptive statistics for subjective anger are presented in Table 3.2.

Table 3.2: Descriptive statistics (mean ± SD) for subjective anger (STAXI), for all four experimental groups (N = 41).

	Pre-test Anger	Post-test Anger
Anger / Control	15.4 ± 1.0	29.0 ± 5.9
Anger / No control	15.6 ± 1.1	26.3 ± 8.8
Neutral / Control	16.0 ± 3.0	16.2 ± 3.9
Neutral / No control	15.6 ± 1.0	19.9 ± 5.7

The effect of the control manipulation on subjective control and confidence was significant ($F(3,37) = 36.59$, $p < .001$). No differences in control were apparent at baseline. Bonferroni-adjusted pairwise comparisons showed that participants in the anger/no control group reported significantly less control (ps < .001) than participants in the two control groups (anger/control and

neutral/control); Table 3.3.

Table 3.3: Descriptive statistics (mean ± SD) for subjective Control scores, for all four experimental groups (N = 41)

Condition	Test
anger/ no control	13.05 ± 4.63
anger/ control	24.80 ± 5.98
neutral/ no control	15.20 ± 4.89
neutral/control	27.27 ± 4.54

Cardiovascular responses

Mean levels of systolic and diastolic blood pressure (BP) were subjected to separate 4 (groups) x 2 (baseline, test) ANOVA procedures. The results showed significant differences in systolic BP between baseline and test ($F(1,37) = 21.25$, $p < .01$). There was also a significant group x test interaction ($F(3,37) = 3.35$, $p < .05$) with anger/no control group having the highest blood pressure increase during test. The same pattern of results was obtained for diastolic BP ($Fs > 1$, $ps < .03$) (Figs. 3.2 and 3.3). Bonferroni-corrected post-hoc t-tests ($p < .02$) showed that both systolic and diastolic BP increased significantly in the *anger/no control* conditions ($ps < .01$) compared to baseline. In the *anger/control* condition, diastolic BP during the task increased significantly relative to the baseline ($t(9) = 7.24$, $p < .001$), while systolic BP almost reached significance ($p = .032$) in the same condition. Only diastolic BP was found to be significant in the *neutral/no control* condition ($t(9) = 3.40$, $p < .01$). In the *neutral/control* condition no significant differences were found.

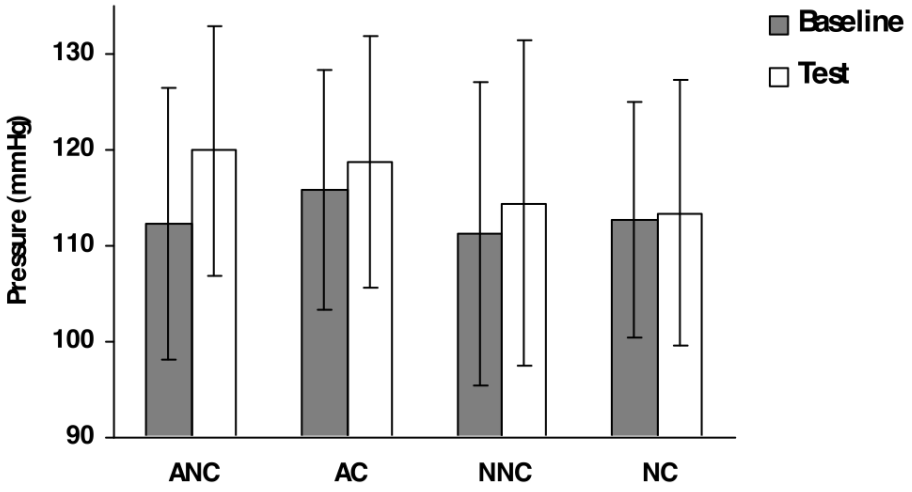

Fig. 3.2: Mean ± SD systolic blood pressure (mmg/Hg) for each experimental group (NB: ANC = Anger/No Control, AC = Anger/Control, NNC = Neutral/No Control, NC = Neutral/Control). N = 41.

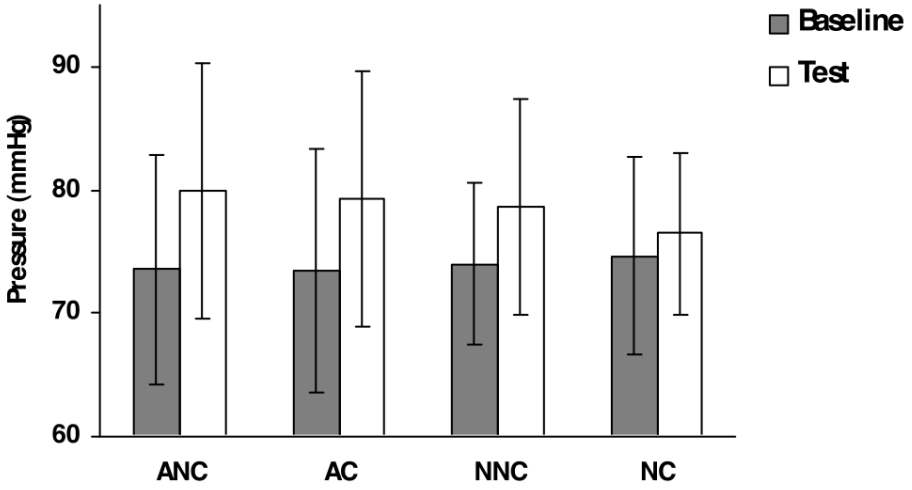

Fig. 3.3: Mean ± SD diastolic blood pressure (mmg/Hg) for each experimental group (NB: ANC = Anger/No Control, AC = Anger/Control, NNC = Neutral/No Control, NC = Neutral/Control). N = 41.

There was significant decrease of PEP during test compared to baseline across conditions ($F(1,38) = 5.79$, $p < .05$); Fig. 3.4. However, there were no significant changes between conditions ($p > .05$).

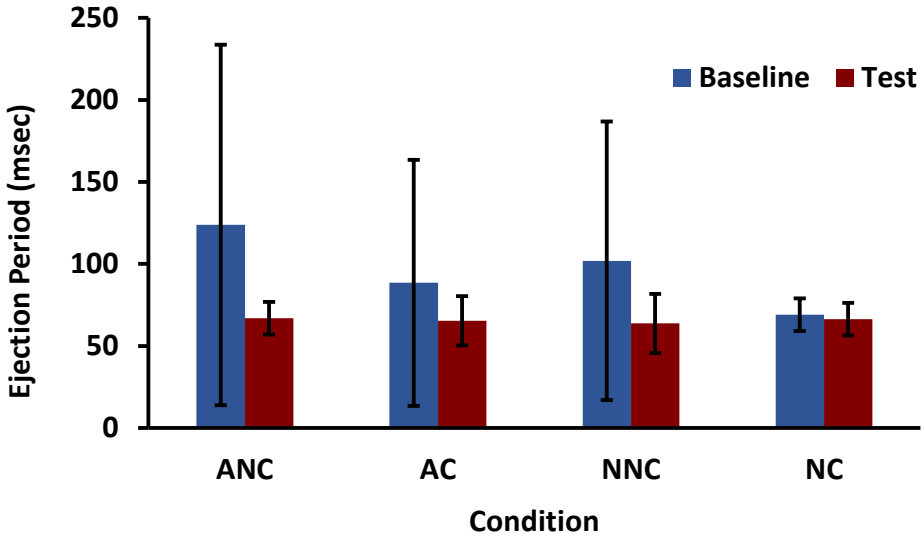

Fig. 3.4: Mean ± SD pre-ejection periods (msec) for each experimental group (NB: ANC = Anger/No Control, AC = Anger/Control, NNC = Neutral/No Control, NC = Neutral/Control). N = 41.

The analysis of cardiovascular impedance yielded three dependent variables: cardiac output (CO), left ventricular ejection time (LVET) and total peripheral resistance (TPR). Descriptive statistics (Table 3.4) indicated CO increase, higher LVET and a decline in TPR in the control conditions. Suppressed cardiovascular activity was observed in the no-control (avoidance) conditions. However, differences between baseline and test across all groups failed to reach statistical significance (ps > .05) for all three measures.

Table 3.4: Mean (± SD) of cardiovascular measures (N = 41).

	Cardiac output (ml/min)		Left ventricular ejection time (min)		Total peripheral resistance (mmHg/ml/min)	
Condition	Baseline	Test	Baseline	Test	Baseline	Test
anger/no control	6.2 ± 1.4	6.1 ± 1.3	0.31 ± 0.03	0.31 ± 0.03	7.02 ± 0.29	7.09 ± 0.25
anger/control	5.2 ± 1.4	5.5 ± 0.9	0.31 ± 0.03	0.32 ± 0.06	7.19 ± 0.29	7.10 ± 0.18
neutral/no control	5.8 ± 1.1	5.7 ± 0.8	0.31 ± 0.04	0.32 ± 0.04	7.05 ± 0.23	7.10 ± 0.23
neutral/control	5.1 ± 1.1	5.3 ± 2.0	0.30 ± 0.03	0.30 ± 0.03	7.19 ± 0.29	7.23 ± 0.45

The mid frequency (0.09 - 0.13Hz) component of the heart rate variability

was significantly different between conditions F(3,32) = 3.85; p < .02); with a suppressed activity in during anger/no control condition and an increased activity during neutral control condition; this effect is illustrated in Fig. 3.5.

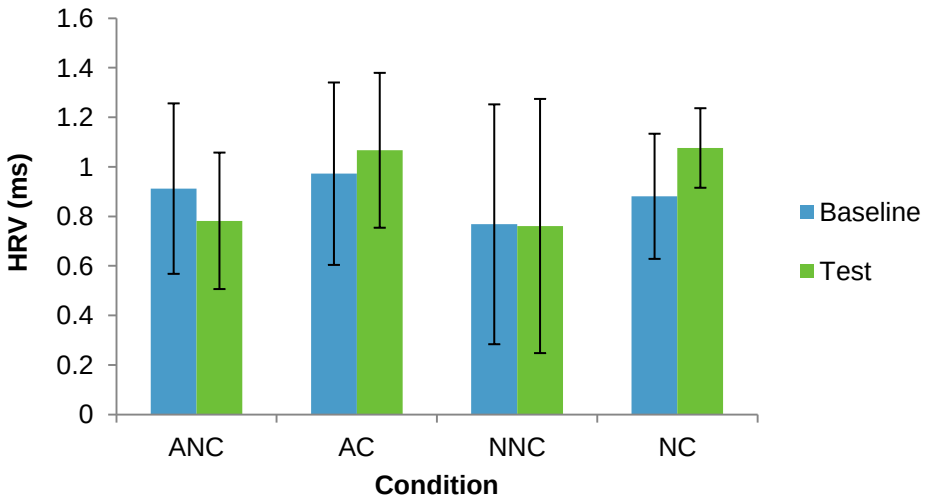

Fig. 3.5: Mean ± SD heart rate variability; 0.1 Hz component for each experimental group (NB: ANC = Anger/No Control, AC = Anger/Control, NNC = Neutral/No Control, NC = Neutral/Control). N = 41.

However, post–hoc tests did not reveal any significant results; the closest to significance was the difference between anger/no control and neutral control conditions (p < .08). The measure of vagal tone (bandwidth 0.14-0.40 Hz) did not differ significantly between the four groups (F(3,34) = 0.39, p >.05).

Respiration

The cardiac measures were not influenced by the respiration rate, since the respiration rate did not differ significantly between conditions (F(3,37) = 1.4; p > .05). However, the descriptive analysis (Table 3.5) indicated that the rate of respiration in breaths/min (BPM) increased only in the neutral/ no control condition.

Table 3.5: Mean ± SD of respiration measures in BPM (N = 41).

Condition	Baseline	Test
anger/ no control	26.46 ± 6.43	24.85 ± 4.83
anger/ control	33.22 ± 8.74	29.28 ± 7.54
neutral/ no control	27.10 ± 6.67	28.87 ± 8.70
neutral/control	27.25 ± 8.13	25.45 ± 5.83

Facial EMG activity

After carrying out ANOVAs between baseline and test across conditions for normalized corrugator and zygomaticus activity no significant differences were found (Fs < .1). However, trends in the data (Tables 3.6 and 3.7) suggest that the corrugator muscle was more active during test compared to the baseline in the anger/no control condition, and less contracted during test compared with baseline in the neutral condition. In contrast, the zygomaticus activity decreased during test compared to baseline in the anger/no control condition, and increased during test compared to baseline (M = 3.90; SD = .69) in the neutral/no control condition. However, no conclusion can be drawn due to non-significant results.

Table 3.6: Mean ± SD of corrugator muscle activity (N = 41).

Condition	Baseline	Test
anger/ no control	2.91 ± 0.38	2.98 ± 0.44
anger/ control	2.92 ± 0.23	2.99 ± 0.20
neutral/ no control	3.02 ± 0.34	2.92 ± 0.15
neutral/control	2.94 ± 0.30	2.89 ± 0.17

Table 3.7: Mean ± SD of zygomaticus muscle activity (N = 41).

Condition	Baseline	Test
anger/ no control	4.24 ± 0.65	4.07 ± 0.55
anger/ control	4.75 ± 0.96	4.16 ± 0.56
neutral/ no control	3.90 ± 0.69	3.99 ± 0.70
neutral/control	4.15 ± 0.75	3.97 ± 0.77

Frontal EEG Asymmetry

A difference in scores between Ln (right) – Ln (left) alpha power was calculated for the frontal brain sites (FP2-FP1, F4-F3, AF4-AF3, FC2-FC1) with higher scores demonstrating relatively greater left frontal activity, thus indicative of approach motivation (Harmon-Jones, 2004a). For each site mixed 2 x 4 (baseline1/baseline 2 x condition) ANOVAs were carried out. The asymmetry score provided a degree of correction for overall alpha power, which could be founded with the magnitude of the asymmetry (Allen, Coan and Nazarin, 2004). The asymmetry score of alpha power at the frontal peripheral sites (Fp) decreased significantly during the task (relative to baselines) regardless of condition ($F(2,64) = 10.02$, $p < .001$); Fig. 3.6.

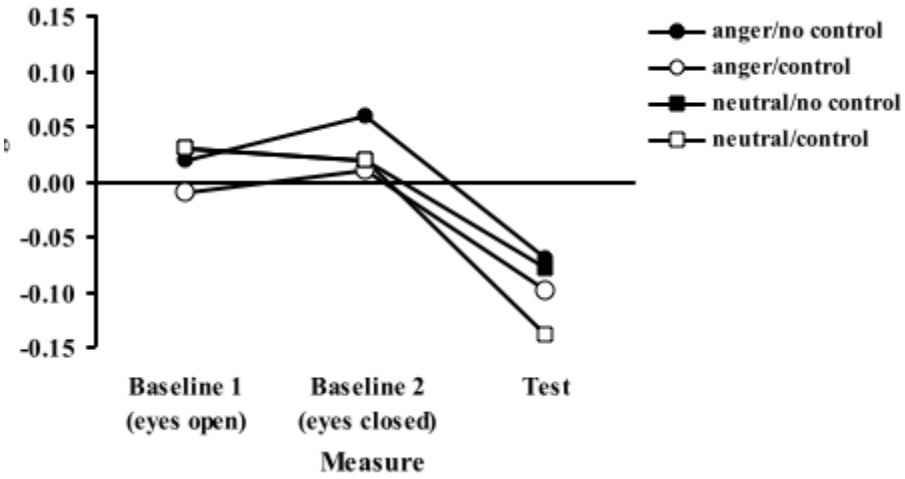

Fig. 3.6: Alpha power expressed as frontal asymmetry score at the Fp1-Fp2

The significant difference found was between baseline 2 (eyes-closed) and

test in the anger/no control condition (t(9) = 2.82, p < .03), indicating a possible right site activation. However, the pattern of results for the FP site was neither replicated at the mid frontal site (F3-F4) nor at the anterior frontal site (AF3-AF4). In fact, the pattern was reversed (Fig. 3.7) with increased left hemisphere activation in all experimental groups compared to baseline 1 (eyes open); Fig. 3.7.

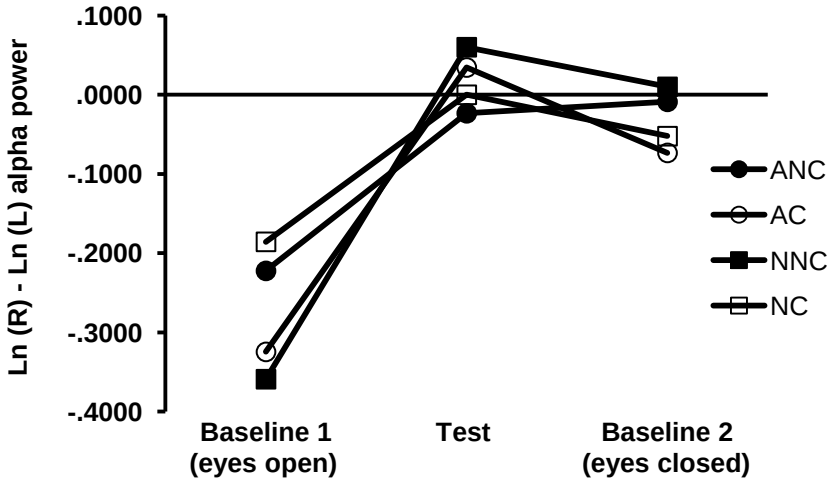

Fig. 3.7: Alpha power expressed as frontal asymmetry score at the AF3 – AF4 site

An increased left hemisphere activation during the test (M = .068; SE = .05) compared to baseline 1 (eyes open) (M = .044; SE = .031) was observed in the anger/no control condition, but the increase was not significant at the F3-F4 site. At the AF3-AF4 site, the increased left hemisphere activation was statistically significant regardless of condition (F(1,63; 50.5) = 32.56, p < .001), however post hoc tests did not reveal any significant changes in any of the conditions. At the FC5-FC6 site no significant statistical differences were obtained.

3.5. Discussion

The present study investigated whether different anger states (anger/approach vs anger/avoidance) are associated with distinct patterns of cardiovascular responses and frontal EEG asymmetry. It was predicted that anger/avoidance is indexed by a PAC response, higher blood pressure,

increased heart rate, augmented respiration rate and the occurrence of right frontal activation. In contrast, it was hypothesized that anger/approach is defined through a SAM response, lesser increases in blood pressure, heart rate and respiration rate and the presence of left brain activation.

Cardiovascular findings

The findings regarding blood pressure were generally in agreement with the dual model proposed by Blascovich et al. (2001) in that higher BP should occur in a threatening avoidance state. There were higher increases in systolic and diastolic BP during the anger/avoidance states than during the avoidance state. Therefore, the results suggest that loss of control failed to increase BP. However, in the same manner as motivation can provide a context for the experience of an emotion, an emotion (especially a negative one) could increase or reduce motivation. As Kreibig, Gendolla and Scherer (2008) pointed out, the use of mood induction to affect motivation could explain why increased negative affect reduced motivation. Health studies (Everson et al., 1997; Stewart et al., 2008) found that anger is a risk factor that produces high BP (Kiecolt-Glaser et al., 2002a). However, the present work identified a specific anger state that causes increased BP; that is, anger within an avoidance context. Such findings could be applied in an affective system where the user's psychological state is identified multi-dimensionally to increase the array of adaptive responses (Kulic & Croft, 2005) state. This could be a first step in raising awareness and preventing prolonged negative emotion/motivational states that lead to cardiac problems.

It was expected that the cardiac parameters (CO, LVET, TPR, PEP) would discriminate between groups. However, the impedance data failed to find significant differences between groups. The results indicate a SAM trend for the anger/approach (increased CO, a drop in TPR), whereas anger/avoidance was defined by a lower increase in CO and a higher TPR. Although, the data trends replicate the model of Blascovich and Tomaka (1996), the absence of significant findings brings into question the accuracy of the theory as well as the effectiveness of the experimental manipulations. Considering that previous studies (Mauss et al., 2007; Stemmler et al., 2007)

also found it difficult to predict emotion/motivational states from purely cardiovascular variables, the present results cast some doubt over the motivational model put forward by Blascovich and Tomaka (1996) and emphasize the need to better characterize the multidimensionality of emotion and motivation.

Facial EMG activity

Corrugator muscle activity, an indicator of negative affect as claimed by previous research (Cacioppo Bush & Tassinary, 1990), did not distinguish between the anger and neutral conditions. The null results of the corrugator muscle activity in the anger condition could be explained by the fact that the present work tested for differences between negative and neutral emotional states; whereas in the literature differences have been reported between negative and positive affect.

Frontal EEG asymmetry

Results from the analysis of frontal EEG asymmetry revealed a possible distinction between the states of anger/avoidance and anger/approach. At the FP1/FP2 frontal peripheral site, there was an increase in right brain activation in the anger/no control condition, indicating an avoidance motivation. However, the trend at the F3/F4 and AF3/AF4 sites showed the contrary pattern, therefore linking avoidance motivation with the left-hemisphere. Such contradictory findings do not support Harmon-Jones's (2004a) theory which postulates that asymmetric left activation is observed only in an approach state. Other studies have also reported inconsistent findings. For example, Wacker, Heldmann, & Stemmler (2003) found that anger with a withdrawal state presented asymmetric left frontal EEG activation. Waldstein et al. (2000) found comparable left and right activation during states of anger, however only right activation correlated with other physiological changes. Asymmetric right frontal EEG activation was found to concide with BP reactivity, without a significant change in heart rate (Wittling, 1990; Waldstein et al., 2000). Considering that systolic BP was also sensitive to the anger/avoidance state in the present study, it can be suggested that the right activation at the FP site increases vascular resistance.

Limitations and future directions

The lack of any real-life consequences for performance (Lovallo et al., 1986) and the dull nature of the Stroop task probably reduced the cardiovascular responses observed in the current study (Cacioppo, Klein, Berntson & Hatfield, 1993). Future studies should consider a more engaging task that prevents boredom from becoming a confounded variable. Also, financial incentives may be introduced as manipulators of motivation. Another possible limitation was the use of a large array of apparatus used. Employing different apparatus to measure BP and the cardiovascular indicators of SAM and PAC may have led to a lack of consistency of significant findings. As pointed out by Wright and Kirby (2003), different methodologies yield different results. Nonetheless, the analysis of the cardiac impedance measures was performed manually and with a high degree of accuracy. Hence, it can be concluded that the ICG results do not support Blascovich's theory (Blascovich & Tomaka, 1996).

In the light of the present findings, the design of future affective computing systems should take into consideration that motivation offers a context for the experience of emotion and that avoidance motivation in combination with anger could lead to unhealthy reactions (Kielcot-Glaser et al., 2002), disengagement from the task (Kapoor et al., 2007) or rushed actions (Beeli, Koeneke, Gasser & Janke, 2008). An interactive system that identifies physiological expressions of motivation and emotion and offers biofeedback could raise self-awareness as a strategy for reducing the negative health impact of the anger/avoidance state. Although it is desirable to use systolic BP as a physiological monitoring tool because of its high sensitivity to changes in emotional states, systolic BP is a measure susceptible to movement artifacts. Non-invasive measures such as impedance measures, which are correlated with BP (Richer & Gendolla, 2009), could be employed as an alternative. Although the present research did not find significant differences in ICG parameters, the trend of the data indicated an optimistic outcome for future studies that employ a more ecologically valid methodology and recruit only a single gender sample. In this context, simpler

sensors of cardiovascular reactivity could detect the psychophysiological state of the participant and provide feedback by means of a visual message whenever a negative emotional state reaches undesirably high levels.

CHAPTER 4
STUDY 2

*Cardiovascular and electroencephalography-based markers of anger and threat/challenge motivation during a simulated driving task**

Abstract

Given that Study 1 found systolic blood pressure and EEG asymmetry differences in expressing anger within motivational contexts but limited delimitating ICG parameters due to mixed gender sample and reduced ecological validity, the current study was designed to investigate how motivational context (challenge vs. threat) influenced the cardiovascular system and frontal EEG asymmetry during anger provocation in an ecologically valid context using a male sample. A group of 29 male participants completed a simulated driving journey with a fixed time schedule in a driving simulator. Anger was induced by exposing participants to traffic delays at an early (challenge) and later point (threat) on the simulated route. A number of dependent variables were recorded, including 32 channels of EEG, measures of cardiovascular impedance, BP and fEMG activity from the Corrugator Supercilii. The results indicated that traffic delays significantly increased BP, HR, TPR and Corrugator activity whilst reducing the relative level of left frontal activation in the EEG. However, there was little evidence for any consistent distinction between motivational context (challenge vs. threat) for any of the dependent variables. The consequences of these findings for capturing the cardiovascular and electroencephalography based responses to anger induction are discussed.

***** Study published in IJPP: Fairclough, S.H., & Spiridon, E. (2012). Cardiovascular and electrocortical markers of anger and motivation during a simulated driving task. *International Journal of Psychophysiology*, *84*, 188–93.

4.1. Introduction

The research literature suggests that there is a link between anger and certain cognitions (e.g., perceived injustice), increased cardiovascular activation, and a greater behavioural disposition towards aggression (Al'Absi & Bongard, 2006). It has been suggested that repeated episodes of anger have detrimental consequences for the health of the individual, as these episodes can lead to the development of coronary heart disease (CHD) (Everson-Rose & Lewis, 2005) and hypertension (Everson, Goldberg, Kaplan, Julkunen & Salonen, 1998); however, this relationship is mediated by trait variables such as hostility (Davis, Matthews, & McGrath, 2000) and expressive style like anger-in which represent how often angry feelings are experienced but not expressed (Vella & Friedman, 2009).

The autonomic manifestation of anger is characterised by sympathetic activation in combination with an elevated rate of respiration (Kreibig, 2010). This autonomic pattern has been validated using protocols designed to induce anger in the laboratory. Autobiographical recall of anger-provoking events (Sinha et al., 1992; Prkachin et al., 2001; Hamer et al., 2007) elicited increased blood pressure, accelerated heart rate (HR), greater cardiac output (CO) and higher total peripheral resistance (TPR); these changes were accompanied by a decline in both left ventricular ejection time (LVET) and pre-ejection period (PEP). This pattern is typified by alpha- and beta-adrenergically mediated changes in the cardiovascular system (Stemmler et al., 2007; Kreibig, 2010).

Variability pertaining to the autonomic correlates of anger may derive from the influence of non-emotional factors submersed within the evaluative context of anger provocation. Stemmler et al. (2007) have argued that the psychophysiological measurement of emotion exemplifies an aggregation of three components: 1) a non-psychological contribution to the physiological response (e.g., motor activity, temperature), 2) an emotion-related component associated with somatovisceral adaptation, and 3) cognitive and motivational aspects associated with the emotion context. With regard to the

latter, Stemmler et al. (2007) examined the influence of motivational disposition (i.e., approach vs. avoidance) on psychophysiological quotas of anger. Anger/approach was associated with higher HR, and shorter PEP and LVET, compared to anger/avoidance scenario.

Provocation of anger in the laboratory represents a number of methodological and ethical challenges; nevertheless, laboratory work attains greater ecological validity if active forms of induction are used, rather than passive manipulation of anger. For example, Stemmler et al. (2001) exposed participants to a demanding task consisting of a provocation from the experimenter and a high rate of failure. The experimental conditions elicited significant shortening of PEP (i.e., defined as the time interval between the onset of ventricular depolarisation and the opening of aortic valve) and LVET (i.e., described as the time from the opening to the closing of the aortic valve) combined with increased diastolic blood pressure (DBP). In contrast, Dimberg and Thunberg (2007) used the passive manipulation of anger by picture viewing and found only differences in HR.

The study of Herrald and Tomarka (2002) consisted of participants enduring an interview with an experimenter who made various demeaning remarks and gestures designed to provoke anger. Such conditions generated increased heart rate and contractility in combination with relatively low vascular resistance. Thus, the study corroborated that anger may be expressed via divergent patterns of cardiovascular activity using different protocols for anger induction. The influence of motivational disposition on the experience of anger has been researched with respect to frontal EEG asymmetry (Harmon-Jones, Gable, & Peterson, 2010). Previous EEG research has found greater activation of the left frontal hemisphere associated with induction of angry mood (Wacker et al., 2003), exposure to insult (Harmon-Jones & Sigelman, 2001) and social rejection (Harmon-Jones, Peterson & Harris, 2009). However, evidence suggests that this relationship is strongly moderated by the motivational context. Harmon-Jones (2003) reported a connection between left frontal activation and anger where recourse to action was readily available; however, Zinner et al. (2008)

reported greater right frontal activation in a social context where anger expression was not socially accepted. Thus, the development of a taxonomy of anger and its neural implementations (Harmon-Jones, 2004a) needs to consider motivational directions (approach vs. avoidance).

The conceptual partition between approach/avoidance resembles the biopsychosocial model of challenge and threat proposed by Blascovich, Berry-Mendes, Hunter and Salomon (1999) and Blascovich and Mendes (2000). According to Blascovich et al. (1999), a state of challenge stimulates sympathetic-adrenomedullary activity. Myocardial contractility is elevated causing higher cardiac output in combination with vasodilation, thus blood pressure remains unchanged. An experience of threat can be associated with pituitary-adrenocortical activity, which inhibits the release of adrenaline and produces a pattern characterized by increased blood pressure and reduced peripheral resistance. This model has been the subject of some debate (Blascovich, Mendes, Tomaka, Salomon, & Seery, 2003; Wright & Kirkby, 2003). However, recent work on the psychophysiology of social support/rejection conducted by Koslov et al. (2011) supports the model based on measurements of cardiovascular impedance and frontal EEG asymmetry. Koslov et al. (2011) reported that baseline levels of frontal EEG asymmetry are good indicators of cardiovascular reactivity in response to anger during the exposure to social rejection. Participants that manifested greater left hemispheric activation at baseline tended to respond to social rejection as a challenge (i.e., increased cardiac output but no change in blood pressure). In contrast, participants who exhibited greater right hemispheric activation at baseline interpreted social rejection as a threat (i.e., cardiac output was stable but blood pressure increased).

Koslov et al. (2011) found significant associations between left frontal asymmetry and cardiovascular stress in participants exposed to social threat conditions; whereby, left frontal asymmetry increased cardiac output and decreased total peripheral pressure (dilation in the arterioles) indicating challenge or approach stress states. Conversely, lower left frontal asymmetry was associated with a maladaptive cardiovascular response (threat). This

supports Blascovich et al.'s (2003) model. Consequently, participants with higher resting activity in the left prefrontal cortex exhibited more adaptive, approach-oriented cardiovascular stress responses when exposed to social evaluative threat. The findings of Koslov et al. (2011) reveal the importance of environmental and contextual factors on brain-based traits on physiological and emotional outcomes. In the study of Koslov et al. (2011) based on a motivated performance situation, two interviewers gave either positive feedback (a protective factor) or negative feedback (creating a situation of social evaluative threat). The beneficial effects of left prefrontal asymmetry occurred only when participants were disposed of environmental protective factors and were, therefore, most vulnerable to social stress.

Challenge is usually associated with positive affect. However, these states have also been associated with anger (Mendes et al., 2008). Left prefrontal asymmetry has been linked to anger, a negatively valenced approach-related emotion (Harmon-Jones, 2003; Harmon-Jones & Allen, 1998). Therefore, research demonstrates both positive and negative affective correlates of left frontal asymmetry. However, individuals with relatively higher left frontal activity possibly experienced intermingling of affective responses in the social threat condition - anger and challenge. Past research points therefore suggests that left frontal asymmetry can be linked to approach motivation, and future research should attempt to disambiguate the valence components of this postulation. An individual's trait frontal asymmetry and the types of social stressors s/he encounters in life may determine their physical and psychological health. Individuals with right prefrontal asymmetry demonstrated malignant acute reactivity to a social threat and such pattern may accumulate over time leading to vulnerabilities in the form of coronary disease or hypertension. Further, increased sensitivity to and vigilance for social threat may facilitate the development of social anxiety or depression. To substantiate this argument, both depression (Pizzagalli et al., 2002) and social anxiety disorders (Davidson et al, 2002) have been linked to increased right frontal asymmetry. It can be concluded that left resting prefrontal asymmetry can serve as a protective factor in a threatening situational context. Right prefrontal asymmetry, on the contrary, suggests vulnerability

in stress-diathesis models of disease etiology.

The current study was conducted to investigate the relationship between motivational context and the psychophysiological manifestation of anger within a simulated driving task. Participants were frustrated by exposure to traffic delays strategically placed in the simulated journey to induce state of anger/approach/challenge and anger/avoidance/threat. The primary hypothesis was to investigate how measures of cardiovascular reactivity and frontal EEG asymmetry responded to both challenge and threat. Earlier research (e.g., Study 1 - Spiridon & Fairclough, 2009) was used to generate specific predictions. Thus, challenge would lead to greater left frontal activation in conjunction with increased cardiac output and no change in blood pressure, whereas threat would be associated greater right frontal activation in combination with increased blood pressure. In addition, it was investigared whether motivational markers of approach/avoidance were correlated across cardiovascular and electrocortical measures as indicated by Koslov et al. (2011).

4.2. Method

4.2.1. Participants

Twenty-nine male participants (age: M = 25.5 yrs., SD = 7.5 yrs.) were recruited via advertisements posted on the campus. Participants had no history of psychiatric illness or cardiovascular problems, were not currently taking any medication, and scored below the 80th population percentile on the Trait Anger Expression Inventory of the STAXI2 (Spielberger, 1999). The latter restriction was included to reduce the likelihood of the researcher being exposed to aggressive or abusive behaviour during the study. All procedures for participant recruitment and data collection were approved by the University Ethical Committee prior to commencement of the study.

4.2.2. Simulated driving task

A simulated car journey was prepared using a STI SIM Driving Simulator software (STI Inc.). This PC-based software allowed interaction via a steering wheel/pedals console and the driving scene was projected onto a large screen (approximately 3.66 m × 4.57 m), yielding a visual angle of

approximately 80°. The simulated journey consisted of a two-lane roadway passing through countryside and urban settings.

The route took approximately 12 min to complete if participants travelled at the maximum speed that was permitted. A clock was visible next to the simulated scene and participants were instructed to complete the journey within 15 min in order to earn the £20 (approximately $32/22 €) payment for participation in the study. A deadline was presented to the participants within the context of a scenario, they were told that the purpose of the journey was to collect a child from school. The emphasis on completing the journey on time was reinforced by providing feedback of journey progression via three instances of pre-recorded verbal messages, e.g. “five miles remaining,” “three miles remaining” and “one mile remaining.” If the driver crashed the vehicle more than twice, they were told they would lose their total participant payment. In addition, auditory speeding warnings were in operation and participants were informed that they would be fined by £1 if they broke the speed limit or committed a driving violation such as overtaking where lane marking indicated that passing other vehicles was prohibited.

The infrastructure of the simulated route included a number of bends, crossroad intersections with stop lines and several sets of traffic lights. The driver encountered a low level of traffic density in both lanes with two exceptions; after approximately 2 min of the journey had elapsed, participants encountered the first traffic jam where extremely slow moving traffic in the lane was combined with high density traffic in the opposite lane, hence participants remained ‘trapped’ in the first traffic jam for 4 min. At a later point in the simulated journey (after approximately 12 min of driving), participants encountered a second traffic jam, identical to the first, which persisted for a period of 5 min. The combined delay introduced by both traffic jams made it impossible for the participants to reach the destination within the required 15 min. There was a short two-minute section at the end of the drive when the traffic jam had cleared and participants reached the school. It should be noted that the threats to withhold participant payment or enforce speeding fines were a deception and all participants were fully debriefed

when the experiment had been completed.

4.2.3. Experimental design

The experiment was designed to compare psychophysiological activity during both early and the late traffic jam (TJ) sections. The early traffic jam (TJ1) occurred after only 2 min of the simulated journey and introduced a four minute delay; we assumed that most participants would feel that it was still possible to complete the journey on time at this point, hence the first traffic jam represented an instance of anger provocation in context of challenge, whereas the late traffic jam (TJ2) elicited anger in the context of threat. The timing of TJ2 in combination with the previous delay meant that participants effectively had no opportunity to complete the journey within the required schedule.

4.2.4. Experimental measures

Subjective questionnaires

The state scale of the State-Trait Anger Expression Inventory 2 (STAXI 2) (Spielberger, 1999) was administered to participants before and after the simulated journey. This scale is designed to capture the subjective experience of anger. The Confidence and Perceived Control scale from the Dundee Stress State Questionnaire (DSSQ) (Matthews, Joyner, Gilliland, Campbell, Falconer, & Huggins, 1997) was also completed by participants before and following the simulated journey. This sub-scale from the DSSQ measures the level of confidence (in one's own ability to perform a task adequately) and the level of perceived control over performance.

Cardiovascular Impedance

Cardiovascular activity was recorded using band electrodes placed on the back of the neck and the thorax (Sherwood et al., 1990). These signals were processed via the NICO100C Noninvasive Cardiac Output Module (BIOPAC Systems Inc.) in conjunction with the MP150 data recording system (BIOPAC Systems Inc.). This module delivered impedance magnitude (Zo) and derivatives (dZ/dt) at 1000 Hz. The impedance signals were analysed using an algorithm developed in our own laboratory in order to detect the following measures for each cardiac cycle: Pre-Ejection Period (PEP), Cardiac Output

(CO), Stroke Volume (SV), Left Ventricular Ejection Time (LVET). (An algorithm was developed to detect significant points (e.g. Q, B, X) in the impedance signals and ECG based on existing research (e.g., Sherwood et al., 1990; Lozano et al., 2007). This algorithm provided a calculation for impedance measures based on the Kubicek formula (Kubicek et al., 1966). For a description of the validation of the algorithm see the General Methodology section.

Heart rate and blood pressure

The Inter-Beat Interval (IBI) from the heart was calculated from an ECG signal filtered between 0.5 and 0.35 Hz and sampled at 1000 Hz. This signal was collected via a two-lead electrode sensor connected to the TEL100C data capture signal (BIOPAC Systems Inc.) that also worked with the MP150 system (i.e. the ground signal for the ECG was obtained from the cardiovascular impedance apparatus). Blood pressure was measured using a CARESCAPE Vital Signs Monitor (V100) (DINAMAP Inc.) which involved placement of an inflatable cuff on the upper left arm. Readings of systolic blood pressure, diastolic blood pressure, heart rate and Mean Arterial Pressure (MAP) were all obtained using the oscillometric method (note: for the purpose of analysis, heart rate was measured from the ECG trace obtained via the MP150, not the CARESCAPE monitor). Recordings from the CARESCAPE were taken during every 2 min of the journey and coincided with the middle point of each traffic jam. Total Peripheral Resistance (TPR) at baseline and during both traffic jams was calculated by combining information from the CARESCAPE and the NICO100C, i.e. TPR=MAP/CO*80.

Facial electromyography (fEMG)

fEMG activity was recorded from the corrugator supercilii (located just above the eyebrow) using two external electrodes (Cacioppo et al., 1990). fEMG data were sampled at 1000 Hz and filtered between 30 and 500 Hz. During post-processing, the sample rate was reduced to 512 Hz and artifacts due to eye blinks were removed (vertical EOG was recorded separately and this signal was subtracted from the corrugator trace). The resulting fEMG data

was normalised using a root-mean-square (RMS) transformation.

Frontal EEG asymmetry

EEG was recorded monopolarly from 32 Ag-AgCl pin-type active electrodes mounted in a BioSemi stretch-lycra head cap. Electrodes were positioned using the 10–20 system and recorded activity from the following sites: frontal pole (FP1, FP2), anterior frontal (AF3, AF4), pre-frontal (F7, F8), frontal (F3, Fz, F4), frontocentral (FC5, FC1, FC2, FC6), central (C3, Cz, C4), temporal (T7, T8), parietocentral (CP5, CP1, CP2, CP6), parietal (P7, P3, Pz, P4, P8), occipitoparietal (PO3, PO4) and occipital (O1, Oz, O2). Electrodes were also placed at earlobe sites (A1, A2) allowing of line re-referencing with a linked ears montage. Offline filtering was performed using high and low pass filters of 0.05 Hz and 60 Hz respectively and a notch filter of 50 Hz. EEG was recorded continuously throughout baseline prior to the task (with eyes open) and continuously throughout the task. Analysis was performed using BESA software (MEGIS software GmbH, Gräfelfing, Germany).

A linked ears montage was applied offline for asymmetry analysis and an average reference was used for analysis of alpha modulation. Automatic correction of blink artifacts and horizontal and vertical saccades was performed using detection through predefined topographies. Muscle activity over 100 μV was also excluded. An average of 5.8% of analysed data was rejected for each participant due to artifacts. Fast Fourier transforms were computed over 50% overlapped windows of 2 s (1024 points). Average power spectra were then computed for each experimental condition by averaging mean FFT results of both blocks for each level. Power spectra in μV2 were log transformed (using the natural log) to normalise distribution. Alpha power was measured as the average power within the 8-13 Hz band. Frontal asymmetry values were obtained using a linked-ears reference scheme from the following sites: AF4, F8, F4, FC2, FC6 (right hemisphere sites) AF3, F7, F3, FC1, FC5 (left hemisphere sites). Power estimates for the alpha (7.5–13 Hz) band were then used in the following formula: Ln [right total alpha power]−Ln [left total alpha power] to generate an index of asymmetry for each homologous pair of electrodes in each band. Positive

values on the index indicated greater relative right alpha power and greater relative left frontal activity, while greater relative right frontal activity was indicated by negative values (Allen et al., 2004).

4.2.5. Procedure

Participants who responded to flyers and emails were screened via a health questionnaire and the Trait Anger Expression Inventory prior to recruitment; see exclusion criteria in the Participants section. Participants who passed this screening procedure were invited to the laboratory where they received information about the test and signed a consent form. They were subsequently provided with a short training session on the driving simulator of approximately 5 min duration. On completion of the training session, participants were fitted with cardiovascular electrodes. This equipment was tested before the EEG and fEMG electrodes were fitted to the participant. This process took approximately 40 min and the participant remained seated throughout.

Baseline levels of psychophysiology were collected before and after the experiment; participants were asked to sit and relax for 10 min; blood pressure values were collected at two minute intervals and participants' eyes were closed for five minutes at the beginning and open for the last five minutes of the baseline period. Data from the last 5 min of this period were averaged to yield baseline values with the exception of the blood pressure data, which was averaged from three readings taken at 2 min, 5 min and 10 min. Participants completed two pre-test questionnaires: the State Anger Expression Inventory and the Control/Confidence scale from the DSSQ. They were subsequently presented with a set of standardised instructions, which are paraphrased as follows:

"You are expected to collect a child from school in 15 minutes and therefore, you must complete the journey in this time. Please check the clock to ensure that you complete the journey on schedule. If you fail to do so, you will not be reimbursed for your time. You will receive a £1 penalty if you exceed the speed limit or overtake where the lane markings indicate that overtaking is

illegal. If you crash the car more than twice, you will not be reimbursed for your time. Remember, you can withdraw from the task at any time without the need for explanation." After reading these instructions, participants performed the simulated journey as described earlier. When the participants had completed the simulated drive, they were asked to complete post-test versions of both questionnaires. Participants were then fully debriefed as to the true nature of the experimental task.

4.2.6. Ethical considerations

The decision to deceive the participants was felt to be absolutely necessary in order to accomplish the goals of the experiment, i.e. to induce genuine experience of anger. By inviting participants to the laboratory to give their time, we were obliged to compensate them financially but also needed to introduce real-world consequences associated with failure to complete the journey on schedule. Therefore, a decision was taken to give participants an impression that financial compensation was dependent on performance.

4.3. Results

All data were subjected to a repeated measures ANOVA analysis. Initial tests were performed on the subjective data as a manipulation check. A comparison of pre- and post-test values of the state scale from the STAXI 2 (Spielberger, 1999) revealed a significant increase of subjective anger during the simulated journey [$F(1,28) = 34.52$, $p < .01$, $\eta^2 = .55$]. The opposite trend was observed for the subjective level of confidence/control experienced by our participants [$F(1,28) = 148.64$, $p < .01$, $\eta^2 = .84$]; i.e., participants experienced a significant fall in control during the simulated journey. Descriptive statistics for both subjective data are presented in Table 4.1.

Table 4.1: Mean and standard deviations for subjective levels of anger and control (N = 29).

Subjective measure	Pre-test	Post-test
Anger	15.38 (3.75)	22.55 (6.91)
Control	24.79 (3.97)	14.72 (5.11)

A repeated measures ANOVA was conducted on both systolic and diastolic blood pressure for three periods: baseline, the first traffic jam (TJ1) and during the second traffic jam (TJ2). The analysis of systolic blood pressure (SBP) revealed a significant main effect [$F(2,27) = 16.71$, $p < .01$, $\eta^2 = .56$] and descriptive statistics are illustrated in Table 4.2. Post-hoc Tukey HSD tests revealed that SBP was significantly higher during both TJ1 and TJ2 compared to baseline ($p < .01$). The same analysis was applied to diastolic blood pressure (DBP) and also revealed a significant main effect [$F(2,27) = 15.29$, $p < .01$, $\eta^2 = .54$]. Post-hoc Tukey tests indicated that DBP was significantly elevated during both traffic jams ($p < .01$).

The ANOVA model was applied to a number of cardiovascular variables. The analysis of heart rate revealed a significant main effect [$F(2,26) = 8.14$, $p < .01$, $\eta^2 = .39$], i.e. heart rate was significantly elevated during both traffic jams compared to baseline.

The data record was incomplete for six participants for cardiovascular impedance analysis due to equipment failure. Analysis of PEP revealed a null effect and the analysis of LVET failed to reach significance. A main effect was found for TPR [$F(2,21) = 3.14$, $p = .05$, $\eta^2 = .24$]; post-hoc Tukey tests revealed that TPR was significantly higher during TJ1 compared to baseline ($p = .05$) whereas the increase of TPR at TJ2 did not significantly differ from baseline. Both analyses of CO and SV failed to reach significance. All descriptive statistics for cardiovascular measures are presented in Table 4.2.

Muscle activity from the corrugator supercilii was captured and normalised (via RMS transformation) prior to analysis. The ANOVA revealed a main effect [$F(2,27) = 9.93$, $p < .01$, $\eta^2 = .43$] and subsequent Tukey tests indicated that: (1) corrugator activity was higher during both traffic jams compared to baseline ($p < .02$), and (2) corrugator activity increased during TJ2 compared to TJ1 ($p < .01$).

Table 4.2: Mean and standard deviations for cardiovascular and fEMG measures during baseline and both traffic jams. Note: HR (heart rate), SBP (Systolic Blood Pressure), DBP (Diastolic Blood Pressure), fEMG_corr (activity of the corrugator supercilii muscle), PEP (Pre-Ejection Period), LVET (Left Ventricular Ejection Time), TPR (Total Peripheral Resistance), CO (Cardiac Output), and SV (Stroke Volume) and frontal EEG asymmetry at F3/F4. (N = 23). Note: data in italics is significantly different to baseline levels.

Measure	Units	Baseline	TJ1	TJ2	Sig
HR	bpm	66.37 (12.16)	*73.75 (13.45)*	*75.20 (15.95)*	<.01
SBP	mmg/Hg	118.69 (8.53)	*126.86 (11.65)*	*129.39 (13.05)*	<.01
DBP	mmg/Hg	77.70 (7.45)	*84.75 (7.86)*	*82.89 (8.02)*	<.01
fEMG_corr	mV(RMS)	12.37 (0.79)	*16.34 (1.73)*	*18.35 (1.72)*	<.01
PEP	ms	114.5 (43.39)	112.5 (28.39)	129.6 (41.94)	n.s.
LVET	ms	276.3 (65.67)	291.6 (55.98)	295.8 (38.24)	n.s.
TPR	dyne-s*cm^{-5}	996.56 (370.10)	*1239.30 (383.72)*	1131.12 (385.38)	<.05
CO	L/min	6.06 (3.98)	7.22 (3.81)	7.32 (2.82)	n.s.
SV	ml	93.36 (49.11)	104.40 (38.11)	112.52 (33.61)	n.s.
F3/F4	Difference score	0.14 (0.22)	*−0.02 (0.37)*	0.03 (0.36)	<.05

Frontal EEG asymmetry was captured using a measure of relative difference between left and right side at five pairs of frontal sites: AF3/4, F3/4, F7/8, FC1/2 and FC5/6. These data were subjected to individual ANOVA analyses. A significant main effect was found at F3/4 [$F(2,27) = 3.03$, $p = .05$, $\eta^2 = .16$]. A post-hoc Tukey test revealed a decrease of left hemisphere activation during TJ1 compared to baseline condition ($p = .05$). Descriptive statistics for both fEMG and EEG frontal asymmetry are presented in Table 4.2. Correlations approaching significance between EEG and cardiovascular measures are shown in Table 4.3.

Table 4.3: Correlation coefficients above .40 for frontal EEG asymmetry and cardiovascular measures for baselines and both traffic jams. Note: frontal EEG asymmetry at F4-F3, FC2-FC1 and FC6-FC5 sites, and SBP (Systolic Blood Pressure), DBP (Diastolic Blood Pressure), and CO (Cardiac Output). (N = 23).

			Cardiovascular		
			SBP Baseline	DBP Baseline	CO Tj1
EEG	Tj1	F4-F3	-0.49		
		FC2-FC1			-0.57
		FC6-FC5		-0.44	
	Tj2	F4-F3	-0.54		
		FC2-FC1			-0.56
		FC6-FC5	-0.48	-0.42	

ns at Bonferroni-corrected $p < 0.0004$

4.4. Discussion

It was predicted that challenge would lead to greater left frontal activation together with increased cardiac output and no change in blood pressure. On the contrary, threat would be associated with greater right frontal activation in combination with increased blood pressure. Motivational markers of approach/avoidance were expected to correlate across cardiovascular and electrocortical measures (Koslov et al., 2011).

Subjective measures of anger and control (Table 4.1) supported the success of the manipulation in which the simulated driving task induced feelings of anger and loss of control/confidence; however, these data were only collected on pre- and post-task basis only. EMG activity from the corrugator supercilii indicted that negative affect peaked during the second traffic jam (Table 4.2). Corrugator activity could be an ambiguous measure of anger expression via facial musculature; and increased activity could be associated with other concepts, such as increased mental information processing (Topolinski, Likowski, Weyers & Strack, 2009). However, this seems unlikely

as the mental demands of the driving task were minimal during the traffic jam. The presence of a second traffic jam could have been a surprise, and the facial coding for the element of surprise could have had a negative valence (Topolinski & Strack, 2015). This explanation is more in line with studies that have indicated that a range of variables, such as inconsistencies, disruption, and lack of structure, are experienced as unpleasant (Gawronski & Strack, 2012; Noordewier & Breugelmans, 2013).

The pattern of cardiovascular reaction observed during the first traffic jam suggested a state of threat as blood pressure and TPR were both elevated whilst CO remained stable (Table 4.2). It has been argued that this pattern is associated with a reduction of adrenaline and lower levels of vasodilation (Blascovich & Mendes, 2000). The absence of any significant change in PEP indicated that observed changes in systolic blood pressure were moderated by variation in vascular resistance as opposed to a beta-adrenergic influence on the force of myocardial contraction. The cardiovascular pattern observed in TJ1 did not fit either state of challenge or threat (as defined by Blascovich et al., 1999; Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996) as CO remained relatively unaffected.

The analysis of frontal EEG asymmetry revealed a reduction of left hemisphere activation at F3/F4 during both traffic jams (Table 4.3). It was anticipated that the first traffic jam would provoke anger in the motivational context of challenge/approach. By contrast, the anger response to the second traffic jam would occur in the context of threat/avoidance, because participants had no chance of completing the journey on schedule due to the timing of this delay. We found little evidence to support either hypothesis. Changes in frontal EEG asymmetry during the first traffic jam indicated a significant reduction of left frontal activation/approach motivation when an increase of left activation had been anticipated (Harmon-Jones, 2003). On one side, such results might point to the fundamental bivariate structure of positive and negative evaluative substrates. This is consistent with data indicating that the left hemisphere is associated with parasympathetic activity related to nourishment, positive affect; whereas the right hemisphere is more

linked to sympathetic regulation and negative affect, aversive reactions and defensive behaviours (Craig, 2005). On another side, it could be a case of adaptive responses, where higher level neural systems permitted "avoidance" responses as a goal strategy (Rutherford & Lindell, 2011). In this situation it could be expected to find an association between lower left frontal asymmetry and a maladaptive cardiovascular response (threat) in support of Koslov et al.'s study (2011). However, we found no direct support as none of the cardiovascular variables showed any significant association with frontal EEG asymmetry and all the correlations where negative indicting that the more left activation, the less CO increase (Table 4.2). In hindsight, the present findings could caution against a fundamental hemispheric coding of approach/avoidance behaviors in a simple manner. Future work could be directed towards clearer insights into the approach-avoidance motivational substrates of specific emotions (e.g., anger) with thorough analysis in diverse contexts and with various environmental stimuli.

The current study presented some methodological limitations. A drawback of the current work was the lack of counterbalancing between challenge/approach and threat/avoidance scenarios. This is an obvious and binding limitation given the role of elapsed time and journey schedule in our methodology. It is also possible that the initial traffic jam came as an unexpected surprise, resulting in higher reactivity than anticipated, whilst experience of the second traffic jam was less novel and psychophysiological responses were subdued as a direct result. It is possible that the use of financial penalties to prevent participants from subverting the time schedule manipulation could have reduced approach-related anger. Alternatively, the task scenario (to collect a child from school) may have produced an empathetic response that mitigated approach-related anger. In addition, we focused on the subjective measurement of anger and control, when in hindsight, a broader approach would have been useful to capture related emotional categories (e.g., fear) that may have played a role. The study was also limited with respect to the decision to recruit male participants that was taken to preserve the homogeneity of the psychophysiological response to the anger induction; it remains to be seen whether these findings may be

generalised to a female population. From a methodological perspective, the simulated driving task proved to be a reliable method for anger induction compared to the previous study (Spiridon & Fairclough, 2009) where a combination of a computer malfunction and harassment by experimenter were used to provoke anger.

The anger-inducing properties of the current methodology were derived from the presence of real-world consequences (payment) associated with failure and a resonance between the simulated traffic jam and participants' actual experience as drivers. The absence of a control condition meant that it was impossible to differentiate between the effects of anger provocation (traffic jams) and task context (journey schedule/financial penalties) on psychophysiological markers. However, the protocol does run the risk of producing domain-specific instances of anger, i.e. the expression of anger in a driving scenario may not generalise to the measurement of anger other domains of life such as home or the workplace; i.e., Bongard and Al'Absi (2005).

In sum, the results of the present study demonstrated increased cardiovascular reactivity and reduced left brain activation in response to anger in the context of a simulated traffic jam. There may be important physical and psychological health applications of the present findings since the context of traffic jams generated an increased cardiovascular reactivity and a decreased left frontal asymmetry in response to anger. High cardiovascular reactivity may accumulate over time to vulnerabilities such as coronary disease or hypertension (Stewart et al., 2008) and less left prefrontal asymmetry could be a sign of depression (Allen, Urry, Hitt & Coan, 2004) or social anxiety disorders (Davidson, Marshall, Tomarken, & Henriques, 2000). Nonetheless, pervasive environmental stimuli such as music could modulate psychophysiological reactions (Juslin and Sloboda, 2010; Knez, 2001); hence, the next studies in this thesis investigated anger in a driving context being moderated by environmental stimuli. Within this framework, affective systems could be proposed for self- regulation of anger state in a threatening situation.

CHAPTER 5
STUDY 3

The impact of music on psychophysiological reactions during anger inducing drives

Abstract

Study 2 pointed to an increased cardiovascular reactivity in response to anger in a context of a simulated traffic jam; however, other factors within a car driving environment could have a counter effect on anger. Music is known to possess mood inducing properties, hence music may be proposed as a potential medium in the prevention of anger during driving. In this study, the influence of music on anger, systolic BP was investigated during anger inducing scenarios using a driving simulator. A group of 100 participants were split into five groups: four groups listened to different types of music (high/low activation music in combination with both positive/negative valence) and one group served as a no-music control group. Anger inducement was highest during high energy negative music compared to positive music, irrespective of music activation level. Systolic BP was higher during high activation negative music and no music compared to low activation music. Music was effective to mediate the state of anger; thus, music may promote road safety and positive health in the long run.

***** Study published in Physiology and Behaviour.

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7.1. Introduction

The experience of anger elicits a pattern of cardiovascular reactivity that can be a precursor of coronary heart disease (Samuel, 2007) and hypertension (Everson et al., 1998), most prevalent in people with high trait anger or hostility (Williams, Paton, Siegler, Eigenbrodt, Nieto, & Tyroler, 2000). Cardiac conditions such as myocardial ischemia and ventricular arrhythmia may be provoked by elevation of cardiovascular stress caused by episodes of extreme anger (Kop, Verdion, Gottdiener, O'Leary, Merz, & Krantz, 2001; Lane, Cowie & Chow, 2005; Strike & Steptoe, 2005). It has also been suggested that frequent and repeated experiences of anger may prompt susceptibility to cardiovascular disease through allostatic mechanisms characterised by neural wear and tear (Ganzel, Morris & Wethington, 2010). The connection between anger and cardiovascular health is attributed to several factors, such as: trait hostility (Everson-Rose & Lewis, 2005; Vella & Friedman, 2009), situations that elicit anger response (Bongard and Al'Absi, 2005) and expressive style (Davidson & Mostofsky, 2010). These anger moderating variables may interact to return specific cardiovascular responses. For example, both the exaggerated expression and suppression of anger have been related to increased cardiovascular reactivity (Bongard & Al'Absi, 2003; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003). Cardiovascular response has been related closely to the context of the anger experience (Al'Absi & Bongard, 2006); therefore, anger measurement must be understood within particular domains of everyday life, such as work vs. home (Bongard & Al'Absi, 2005).

Driving is a ubiquitous activity in everyday life where the experience and expression of anger associated with driving scenarios has implications for the health and safety of the individual (Vanlaar et al., 2008). The experience of anger manifests itself in the form of unwelcome behaviours on the road, including physical/verbal abuse, speeding, and tailgating (Deffenbacher, Lynch, Oetting, & Swaim, 2002; Nesbit, Conger, & Conger, 2007). The manifestation of anger may originate from unexpected or erratic action from other road users. Frequently, anger is instigated by impedance to the journey due to the characteristics of the traffic; notably stop lights, traffic congestion

(McGarva, 2005). Fieldwork by means of ambulatory monitoring of drivers responses points to an association between high traffic congestion and augmented systolic blood pressure caused by loss of control of the journey schedule (Stokols, 1978; Schaeffer et al., 1988).

The psychophysiological manifestation of anger is characterised by increased sympathetic activation and intensified respiration in the form of greater amplitude and rate of breathing (Kreibig, 2010). Both alpha- and beta-adrenergic activation of the sympathetic nervous system stimulate the heart to beat faster and to augment the volume of blood being pumped through the circulatory system. The effect is increased myocardial contraction and peripheral resistance (Stemmler et al., 2007; Kreibig, 2010). This psychophysiological response has been observed in several laboratory studies where participants recalled and relived previous episodes of anger-provoking experiences (Prkachin et al., 2001; Hamer et al., 2007). Researchers have utilised different method to incite anger under laboratory conditions: threat of punishment via electric shock (Schachter, 1957) accompanied by demanding cognitive activity and harassment from the experimenter (Drummond, 1999; Lavoie et al., 2001), high rate of task failure in addition to provocation from the experimenter (Stemmler et al*.,* 2001), interviews conducted by a demeaning experimenter (Herrald & Tomaka, 2002), and failure of technical equipment (Partala, 2003). It has been reported that the induction of anger increases heart rate and blood pressure whilst considerably shortening the pre-ejection period (PEP) and left ventricular ejection time (LVET); Stemmler et al., 2001. However, total peripheral resistance (TPR) remained unchanged. By contrast, the study of Herrald and Tomaka (2002) reported an increase of both cardiac output (CO) and TPR when participants were demeaned by the experimenter.

Previous research into the influence of music on cardiovascular activity have returned inconclusive results. Several studies reported that arousing music tended to increase heart rate, whereas sad music yielded the opposite effect (Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006; Hodges, 2010). Other research sustains that the presence of music per se causes increased heart

rate (Iwanaga, Ikeda, & Iwaki, 1996; Rickard, 2004). Conflicting findings may originate from methodological variations across different studies; including the context of music listening (music in the background or as primary activity), the duration of the musical piece, and the interaction between musical stimuli and personal taste for music. Emotional states may be described as a series of distinct categories (happiness, anger, and other; Russell, 1980) or within the context of dimensional models; the reader is hereby referred to the General Introduction to further scrutinize dimensional models. It can be suggested, therefore, that mood music has an effect on the cardiovascular response but the actual impact is determined by the methodological context.

The current study investigated the impact of music on the cardiovascular manifestation of negative affect during a simulated driving task. Participants were exposed to an unavoidable delay during a simulated driving journey with a fixed time schedule to complete the road trip in order to induce negative affect. This protocol was validated in a previous study conducted by Fairclough and Spiridon (2012). The underlying protocol was supplemented by exposing participants to four different categories of music plus a no-music control group. The four categories were: high arousal (HA)/positive valence (PV), HA)/negative valence (NV), low arousal (LA)/PV, and LA/NV. Our main aim was to investigate how the dimensions of the musical pieces (activation and valence) influenced cardiovascular correlates of negative affect within the context of an unavoidable delay during the simulated car journey. We expected HA/NV music to augment cardiovascular reactivity, whereas LA/PV music was presumed to have the opposite effect. Earlier research (Fairclough & Spiridon, 2012) was used to generate specific predictions. Thus, challenge (Traffic jam 1, TJ1) would lead to increased cardiac output and no change in blood pressure, whereas threat (Traffic jam 2, TJ2) would be associated with increased blood pressure. In addition, we wish to investigate whether changes in activation or valence induced by the music were sufficient to enhance or mitigate cardiovascular reactivity to anger during both challenge (TJ1) and threat (TJ2) to the experience of negative affect in comparison to the control condition.

7.2. Method

5.2.1 Participants

The study used a between-participant design using five groups, whereby each group included 20 volunteers (10 males, 10 females) amounting to 100 participants in total. The mean age of the participants was 21.2 years (SD = 4.7 years, see Table 5.1). Each participant received a £10 voucher for taking part. The trait anger measures capture two aspects of trait anger: Trait Anger-Reaction (T-Ang/R) and Trait Anger-Temperament (T-Ang/T) (Spielberger, 1999). The former refers to the frequency with which anger is experienced during frustration or negative evaluation, whereas the latter represents the disposition of the individual to experience anger in the absence of provocation.

Table 5.1: Trait variables for each participant group including means and standard errors (N = 100).

	Age (M,SE)	Gender (M/F)	T-Ang/R (M,SE)	T-Ang/T (M,SE)
HA/PV	20.79 (1.07)	9/11	2.05 (.13)	1.53 (.61)
LA/PV	20.65 (1.04)	10/10	2.08 (.13)	1.53 (.77)
HA/NV	20.95 (1.04)	10/10	2.01 (.13)	1.61 (.72)
LA/NV	20.15 (1.04)	10/10	1.96 (.13)	1.46 (.37)
No music	23.45 (1.04)	10/10	2.42 (.13)	1.61 (.64)

5.2.2 Simulated Driving Task

A simulated car journey was devised using STI SIM Driving Simulator software (STI Inc.). This PC-based software allowed interaction via a steering wheel/pedals console and the driving scene which was projected onto a large screen (approx. 12 ft. x 15 ft.), yielding a visual angle of approx. 80 degrees. The simulated journey consisted of a two-lane roadway passing through countryside and urban settings. The route was planned to take approximately 8 minutes to complete assuming that the participants travelled at the maximum allowed speeds.

A clock was visible next to the simulated scene and participants were

instructed to complete the journey within 8 minutes in order to earn an additional £10 monetary incentive for participation in the study. The deadline was presented to the participants within the context of a scenario; they were told that the purpose of the journey was to collect a child from school. The precedence of completing the journey on time was reinforced by providing feedback of journey progression via three instances of pre-recorded verbal messages; e.g., “five miles remaining,” “three miles remaining”, and “one mile remaining.” If the driver crashed the vehicle more than twice they were told that they would lose 70% of their total participant payment. In addition, speeding warnings were in operation and participants were informed that they would be fined by £2 if they broke the speed limit or committed a driving violation.

To further induce anger the following manipulations were adapted from (van der Hulst, Meijman, & Rothengatter, 2001). During the drive, participants were exposed to a number of discrete obstacles, such as traffic lights that always turned red on their approach; drivers also encountered a number of vehicles that accelerated and decelerated in a sinusoidal pattern at a point with traffic coming in the opposite direction, preventing any attempt to overtake.

The driver encountered a low level of traffic density in both lanes with two exceptions; after approximately 3 minutes of the journey had elapsed, participants encountered the first traffic jam where extremely slow moving traffic in the lane was combined with high density traffic in the opposite lane, hence participants remained ‘trapped’ in the first traffic jam for 3 minutes. At a later point in the simulated journey (after approx. 7 minutes of driving), participants encountered a second traffic jam, identical to the first, that persisted for 4 minutes. The combined delay introduced by both traffic jams made it impossible for the participants to reach the destination within the required 8 minutes. It should be noted that the threats to withhold participant payment or enforce speeding fines were a deception and all participants were fully debriefed when the experiment had been completed.

5.2.3 Experimental Design

The experiment consisted of two sessions. During the first session, all participants were required to rate music via the internet in order to personalise the music choice of those individuals in the four music groups: HA/PV, LA/PV, HA/NV, LA/NV. The second session took approximately 60 minutes in a laboratory and involved a baseline measurement, a mood induction session (when participants were exposed to music only) and a simulated driving task. The study used a mixed design where music group functioned as a between-participants' factor; whilst baseline, induction and exposure to two traffic jams were manipulated on a within-participants' basis. The experimental protocol and all associated procedures were approved by the University Ethics Research Committee prior to data collection.

5.2.4 Experimental Measures

Subjective Questionnaires

The State Anger Expression Inventory 2 (Spielberger, 1999) was administered to participants before and after the simulated journey. This scale is designed to capture the subjective experience of anger. The UMACL from the Dundee Stress State Questionnaire (DSSQ) (Matthews et al., 1997) was also completed by participants before and following the simulated journey.

Cardiovascular Impedance

Cardiovascular activity was recorded using band electrodes placed on the back of the neck and the thorax (Sherwood et al., 1990). These signals were processed via the NICO100C Noninvasive Cardiac Output Module (BIOPAC Systems Inc.) in conjunction with the MP150 data recording system (BIOPAC Systems Inc.). This module delivered impedance magnitude (Zo) and derivatives (dZ/dt) at 1000 Hz. The impedance signals were analysed using an algorithm developed in our own laboratory in order to detect the following measures for each cardiac cycle: Pre-Ejection Period (PEP), CO, Stroke Volume (SV), LVET.

Heart Rate and Blood Pressure

The Inter-Beat Interval (IBI) from the heart was calculated from an ECG signal filtered between 0.5 and 35 Hz and sampled at 1000 Hz. This signal was collected via a two-lead electrode sensor connected to the TEL100C data capture signal (BIOPAC Systems Inc.) that also worked with the MP150 system (i.e., the earth signal for the ECG was obtained from the cardiovascular impedance apparatus). Blood pressure was measured using a CARESCAPE Vital Signs Monitor (V100) (DINAMAP Inc.) which involved placement of an inflatable cuff on the upper left arm. Readings of systolic blood pressure, diastolic blood pressure, heart rate and mean arterial pressure were all obtained using the oscillometric method. Recordings from the CARESCAPE were taken during every two minutes of the journey and coincided with the middle point of each traffic jam. TPR at baseline and during both traffic jams was calculated by combining information from the CARESCAPE and the NICO100C; i.e., TPR = MAP/CO * 80.

5.2.5 Procedure

Once the participants provided written consent, they were asked to take part in two sessions; the first was conducted remotely to personalise the choice of music for each individual and the second session represented the simulated drive.

Music selection

The music selection was achieved via an online survey exercise. Participants were asked to rate 80 songs, which were preselected to vary with respect to activation and valence. The classification of the music with these labels was carried out using an automatic music classification algorithm that uses audio signals to classify music into mood classes (Skowronek, McKinney, & van de Par, 2006, 2007). Participants were asked to listen to each song at different places within the song to get a good impression of the song and to rate the level of activation (energetic - no energy) and valence (unpleasant to pleasant) using 7-point Likert scales. For each participant the 6 songs (i.e., 2 songs for the induction and 4 songs for the drive) with the highest rating in their mood state were selected; i.e., for a participant in the positive high

energetic, HA/PV condition songs with the highest scores for valence and energy were selected. The average valence (V) and activation or energy (E) ratings of the selected song stimuli per music state were as follows: LA/PV V = 5:1, E = 3:5, HA/PV V = 6:7, E = 6:6, LA/NV V = 1:8, E = 1:3, HA/ NV V = 1:8, E = 5:1; Fig. 5.1.

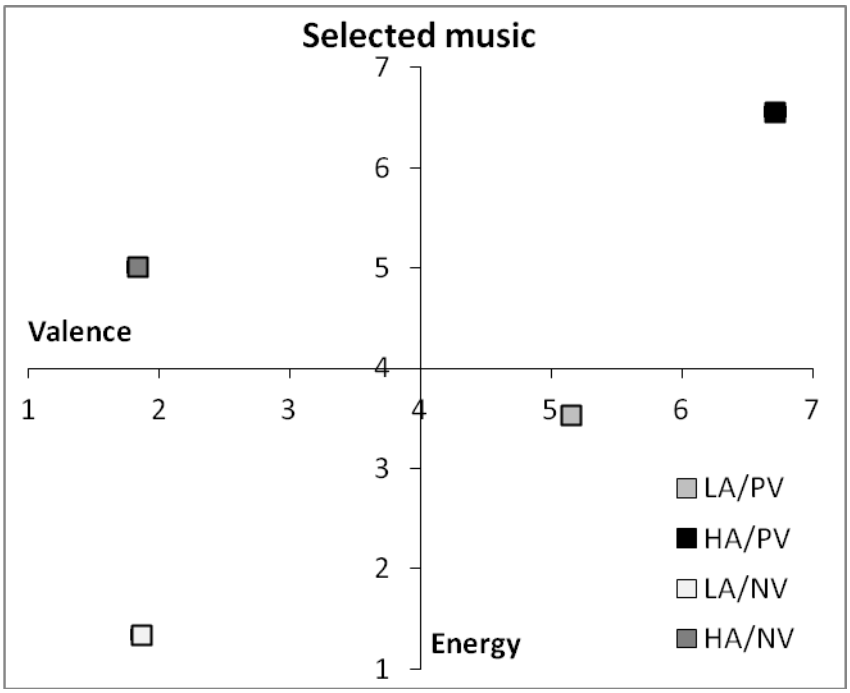


Fig. 5.1: The average valence and activation (energy) values for the selected songs. Error bars represent ± 1 SE. Note: The SEs are so small that they appear as black lines around the means.

Simulator Trial

In the lab, participants were randomly assigned to one of the five experimental groups. They were seated in the simulator and psychophysiological apparatus was attached. Once the signals had been checked, a baseline period commenced where participants were asked to relax and watch a neutral video for eight minutes (Piferi, Kline, Younger, & Lawler, 2000); Fig. 5.2. The data acquired during this time provided the baseline measurements. The participants were then asked to complete the UMACL and STAXI scale and listen to the music presented for six minutes; this was the music induction session. To ensure that the participants paid attention to the music they were told that questions would be asked about

the music at the end of the experiment. After the music induction, participants completed the STAXI and UMACL questionnaires. Participants were presented with written instructions in which they were told that they had to take some children to school in an eight minute drive. It was emphasised that it was important to arrive on time as the children had an exam and they would not be allowed to start the exam if they arrived too late. The monetary penalties for road offences were made clear. If the participants had no further questions the drive began. During the 12-minute ride the chosen music was played continuously. After twelve minutes the drive was stopped and the STAXI and UMACL were presented for completion. Finally, there was a recovery period in which the participants were given the debriefing form and the money voucher.

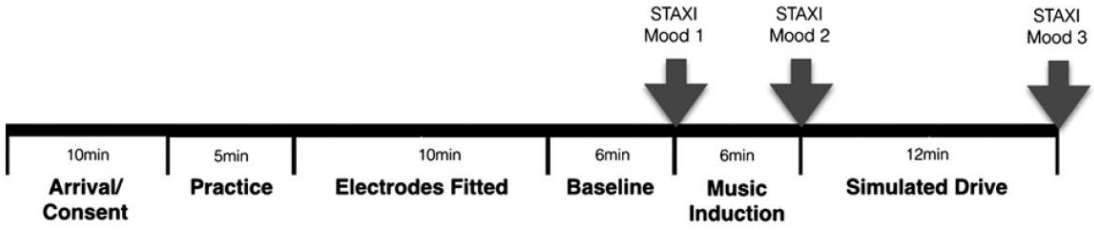


Fig. 5.2: Timeline of events during the simulator trial.

7.3. Results

Data was derived from self-reported questionnaires and measures of cardiovascular reactivity. The analysis of the former was analysed principally using MANOVA analyses to test the effects of music and the simulated drive on subjective anger and mood. The analysis of cardiovascular reactivity is divided into two sections, an initial test of between-participant differences at baseline and a series of 5 × 3 ANOVA models, including Music (HA/PV, HA/NV, LA/PV, LA/NV, no music) and Test phase (TJ1, TJ2, and mood induction MI) as the independent variables.

5.3.1. Analysis of self-reported questionnaire data

State anger was captured using the STAXI with reference to three sub-scales: (a) feelings of anger, (b) feel like expressing anger verbally, and (c) feel like expressing anger physically. All three STAXI variables collected after the baseline (STAXI 1 in Fig. 5.2) were subjected to a 5 × 3 MANOVA to test

for differences between the participant groups at baseline; however, no significant differences were found. Change scores were derived by subtracting post-baseline STAXI scores (STAXI 2 in Fig. 5.2) from post-induction score (STAXI 1) values to capture the effect of music only; i.e., a positive number is associated with increased anger during the music mood induction. There were significant main effects for music ($F(4,95) = 5.02$, $p < .01$, $\eta^2 = .18$); post-hoc tests indicated that total anger (a combination of feelings, verbal anger and physical anger) was significantly higher in the HA/NV condition (M = 0.19) compared to: HA/PV (M = −.03), LA/NV (M = .01), LA/PV (M = .02) and no music (M = .02).

Post-drive STAXI scores (STAXI 2 in Fig. 5.2) were subtracted from post-induction scores (STAXI 2) to obtain a series of change scores. All three scales were subjected to a 5 × 3 MANOVA (Group × State Anger). Significant effects were found for Group with respect to feelings of anger ($F(4,94) = 4.15$, $p < .01$, $\eta^2 = .15$) and feel like expressing anger verbally ($F(4,94) = 3.36$, $p < .05$, $\eta^2 = .13$). There was no significant effect for the scale related to expression of physical violence. Descriptive statistics are presented in Table 5.2. Post-hoc comparisons showed that feeling angry scores were significantly higher for the HA/NV group compared to all other groups ($p < .05$). A post-hoc analysis of the verbal anger scale indicated higher scores for the HA/NV group relative to all groups except for the Control (no music) group.

Table 5.2: Means and standard errors for three sub-scales of the STAXI-2 based on baselined scores (post-drive minus pre-drive) (N = 100).

Group	Feeling angry	Verbal anger	Physical anger
HA/PV	0.62 [.11]	0.21 [.11]	0.07 [.06]
LA/PV	0.39 [.12]	0.05 [.11]	0.01 [.06]
LA/NV	0.60 [.12]	0.25 [.11]	0.08 [.06]
HA/NV	1.05 [.12]	0.57 [.11]	0.18 [.06]
Control	0.67 [.12]	0.32 [.11]	0.09 [.06]

Two unidimensional scales from the UMACL, activation (energetical arousal: active vs. tired) and valence (hedonic tone: happy vs. sad) were converted to

change scores; i.e., post-test minus pre-test during both the music induction (Mood 2 − Mood 1 in Fig. 5.2) and driving (Mood 3 − Mood 1) parts of the test session. These data were analysed as a manipulation check that four categories of music had the predicted effects on subjective mood. A 5 × 2 × 2 MANOVA was conducted on these data (Group × induction/driving × activation/valence), which revealed significant interaction effects for Group × activation/valence ($\Lambda(4,95) = .81$, $p < .01$, $\eta^2 = .19$), induction/driving × activation/valence ($\Lambda(1,95) = .44$, $p < .01$, $\eta^2 = .57$) and a three-way interaction ($\Lambda(4,95) = .83$, $p < .01$, $\eta^2 = .17$). Post-hoc Bonferroni tests revealed that activation was significantly higher for the HA/PV and HA/NV groups compared to all other groups during the music induction session ($p < .01$). The post-hoc analysis of valence during music induction revealed that both negative valence categories of music (HA/NV and LA/NV) resulted in significantly lower valence scores compared to all other groups ($p < .01$); i.e. lower valence scores are indicative of negative affect. It was also found that activation significantly increased and valence decreased during the simulated drive compared to the music induction session ($p < .01$). By contrast, the analysis of subjective mood data during the driving task failed to reveal any significant differences between the five groups of participants. All descriptive statistics for the mood data during both music induction and the simulated drive are illustrated in Fig. 5.3.

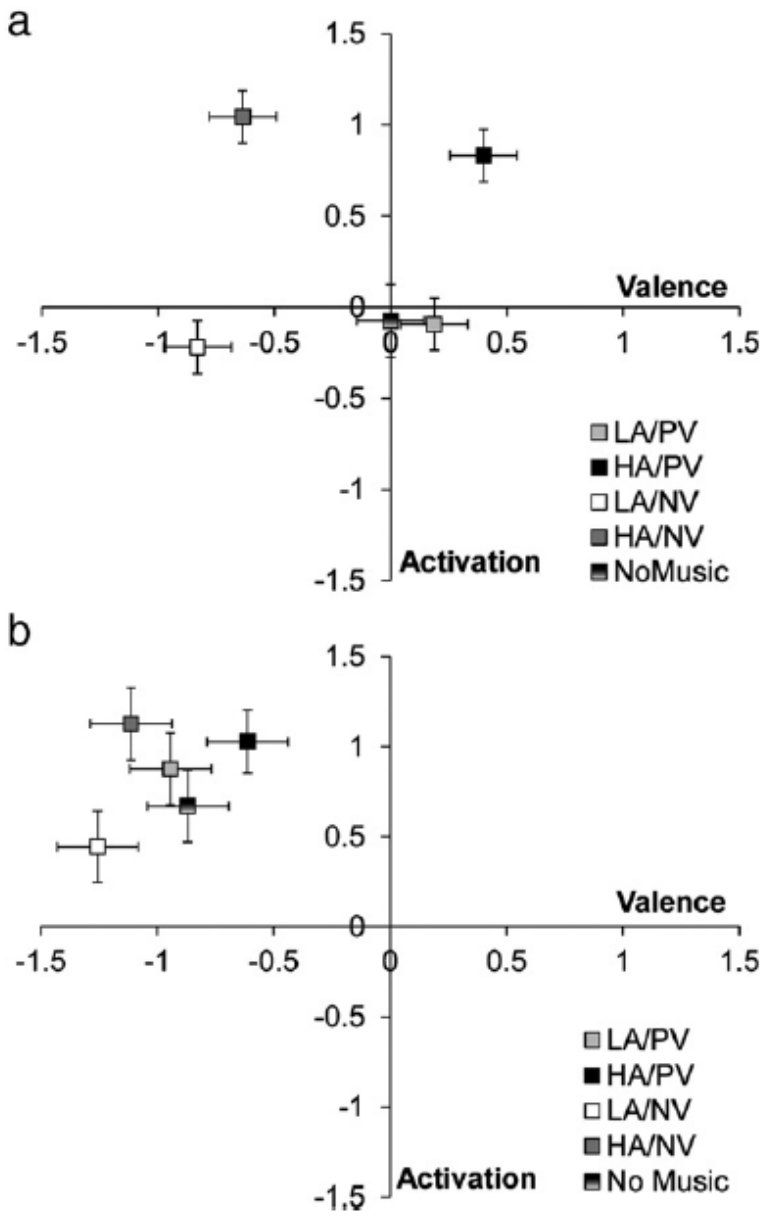


Fig. 5.3: Self-reported mood data (activation and valence) during music induction (a) and simulated drive (b) for all conditions (N = 100).

5.3.2. Analysis of cardiovascular reactivity data

Baseline cardiovascular data was analysed via MANOVA to test for a priori differences between the five participant groups. Data from 6 to 9 participants were lost due to erroneous data resulting from excessive movement during the baseline phase. There were no significant differences between the five groups at baseline for any of the cardiovascular variables and all descriptive statistics are presented in Table 5.3. Cardiovascular data was extracted from three periods: baseline and music induction (Fig. 5.2) and the 2 min period of

the simulated drive when participants encountered the first traffic jam (TJ1). A set of baselined data were created for music induction (MI) and traffic jam (TJ1) where cardiovascular measures at baseline were subtracted from measures obtained during MI and TJ1; i.e., positive numbers are equated with an increase of each measure during MI and TJ1. The two phases of the experiment, MI and TJ1, were collectively labelled as the test phase and all cardiovascular data were subjected to 5 × 3 ANOVA (Group × Test phase) testing.

Table 5.3: Mean and standard errors for all cardiovascular variables during the baseline session: SBP (systolic blood pressure), DBP (diastolic blood pressure), HR (heart rate), PEP (pre-ejection period), LVET (left ventricular ejection time), SV (stroke volume), CO (cardiac output), TPR (total peripheral resistance).

Group *(units)*	**SBP** *mmg/Hg*	**DBP** *mmg/Hg*	**HR** *bpm*	**PEP** *ms*	**LVET** *ms*	**SV** *ml*	**CO** *L/min*	**TPR** *dyne-s*cm^{-5}*
HA/PV	118.50 [2.49]	75.07 [1.56]	74.98 [2.71]	108 [.03]	298 [.01]	45.93 [9.06]	3.45 [.68]	1603.56 [292.31]
LA/PV	120.18 [2.70]	78.24 [1.69]	75.59 [2.94]	105 [.03]	309 [.01]	65.22 [9.06]	4.85 [.68]	1123.44 [292.31]
HA/NV	121.97 [2.56]	78.54 [1.60]	71.67 [2.78]	116 [.03]	316 [.01]	48.11 [9.06]	3.16 [.68]	1540.35 [317.06]
LA/NV	122.17 [2.63]	79.56 [1.64]	75.28 [2.86]	149 [.03]	302 [.01]	54.17 [9.06]	3.85 [.68]	1177.06 [299.91]
Control	120.70 [2.49]	77.53 [1.56]	68.75 [2.71]	141 [.03]	322 [.01]	43.49 [8.82]	3.77 [.66]	1655.50 [292.31]
N	94	94	94	91	91	91	91	91

The presence of movement artefacts in the data resulted in the removal of six participants from the blood pressure data. The analysis of systolic reactivity (SBP) yielded main effects for both Group ($F(4,89) = 4.08$, $p < .01$,

η^2 = .34) and Test phase (F(1,88) = 19.54, p < .01, η^2 = .17). Post-hoc Bonferroni tests indicated that systolic reactivity was significantly reduced for the LA/PV and LA/NV groups compared to both HA/NV and Control groups (p < .01). This effect is illustrated in Fig. 5.4. The main effect for Test phase indicated that systolic reactivity was highest during TJ1 (M = 6.17, SE = 0.95) compared to MI (M = 1.81, SE = 0.49) (p < .01). The same ANOVA model was applied to diastolic reactivity (DBP). This analysis yielded a significant effect for Test phase (F(1,89) = 14.91, p < .01, η^2 = .13); as with SBP, DBP was significantly higher during TJ1 (M = 4.08, SE = 0.92) compared to MI (M = 0.65, SE = 0.38, p < .01).

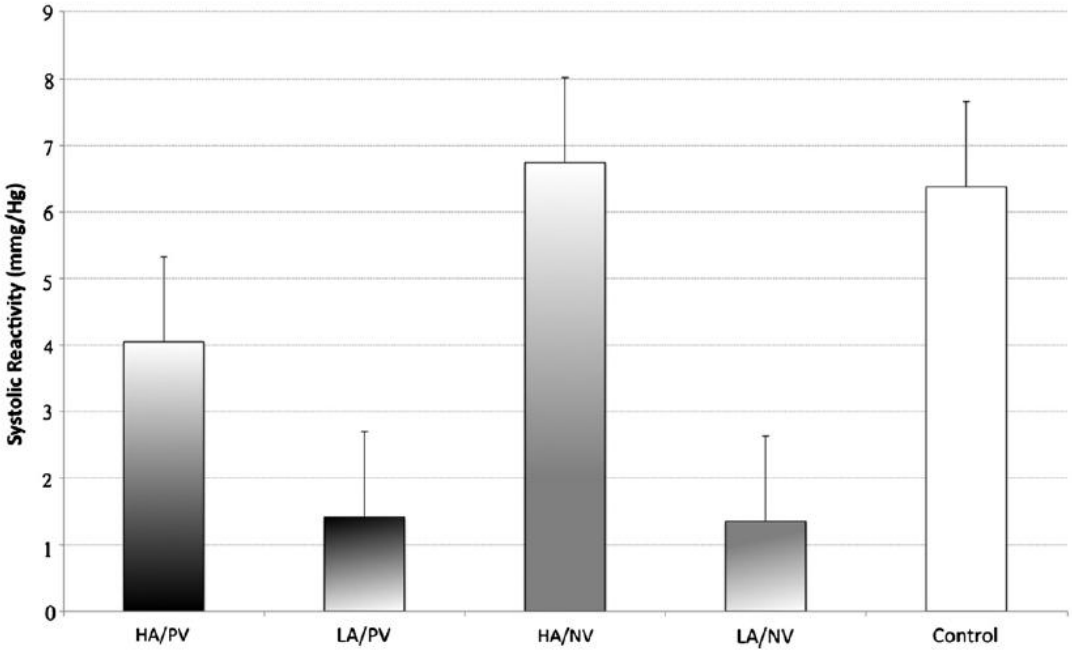


Fig. 5.4: Mean + SE systolic reactivity (SBP) for all five groups averaged across both test phases (MI, TJs); N = 98.

A number of participant data records from the ECG and impedance data were corrupted due to movement artefacts in the driving simulator environment; this loss of data resulted in only 81 participants being eligible for subsequent data analysis of heart rate and all markers of cardiovascular impedance (PEP, CO, LVET, SV, TPR); i.e., 16 participants remained in all groups with the exception of the HA/PV group where data for 17 participants were available.

The analysis of baselined heart rate (extracted from ECG trace) revealed a significant effect for Test phase [$F[2,75) = 18.32$, $p < .01$, $\eta^2 = .17$]. Post-hoc tests indicated that heart rate was significantly higher during TJ1 compared to MI. The same ANOVA model was applied to the baselined values of PEP, which also revealed a significant main effect for Test phase [$F[2,75) = 11.09$, $p < .01$, $\eta^2 = .24$]; i.e., baselined PEP was significantly reduced at TJ1 compared to MI.

The ANOVA analyses of LVET failed to reveal any significance influence from Group or Test phase. The analysis of CO showed a significant interaction between both main effects [$F[8,152) = 2.97$, $p < .05$, $\eta^2 = .10$]. Post-hoc Bonferroni tests indicated that CO was significantly lower for the LA/PV group compared to the HA/NV group, but only during TJ1 ($p < .05$). This effect is illustrated in Fig. 5.5.

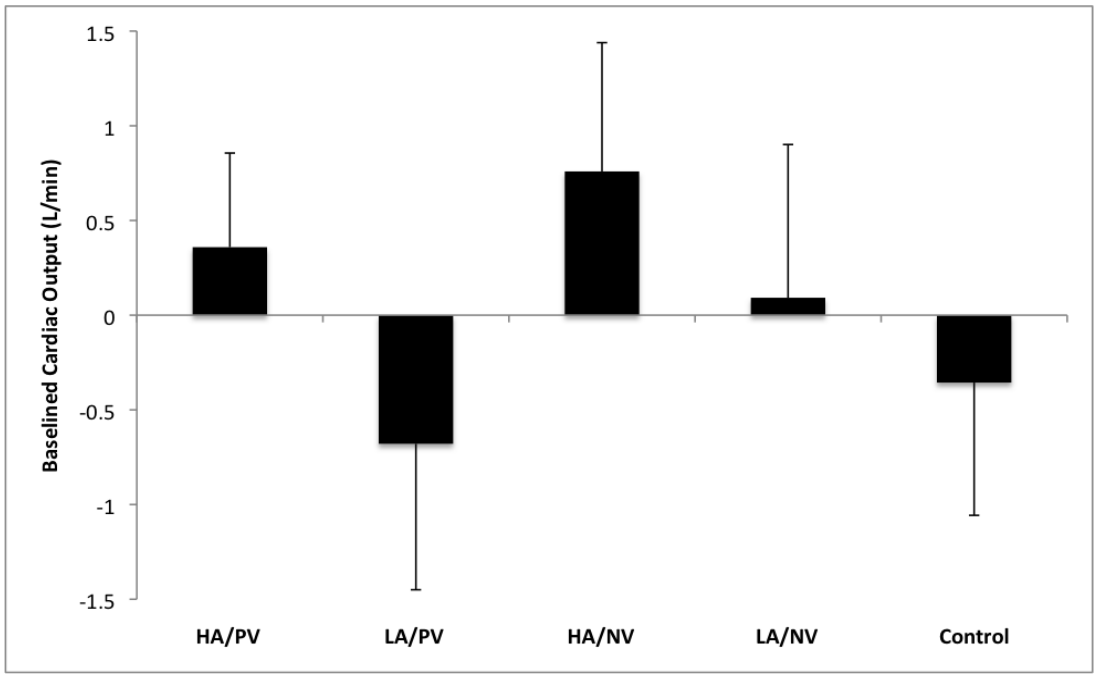


Fig. 5.5: Mean + SE group means for baselined CO during TJ1 (N = 81).

There was a significant interaction between Group and Test phase [$F[8,152) = 1.95$, $p = .05$, $\eta^2 = .11$] when TPR data was subjected to the ANOVA model. Post-hoc tests revealed that TPR was significantly reduced for the HA/NV group compared to the HA/PV group, however this effect was confined to TJ1 ($p < .05$). The results are illustrated in Fig. 5.6.

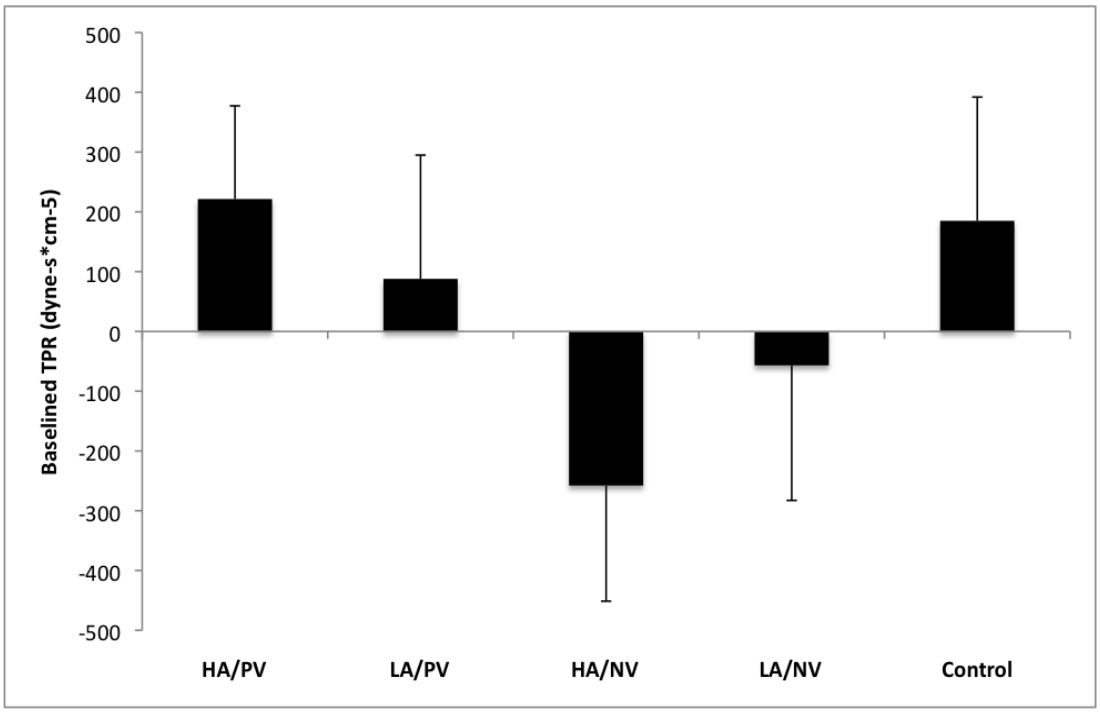
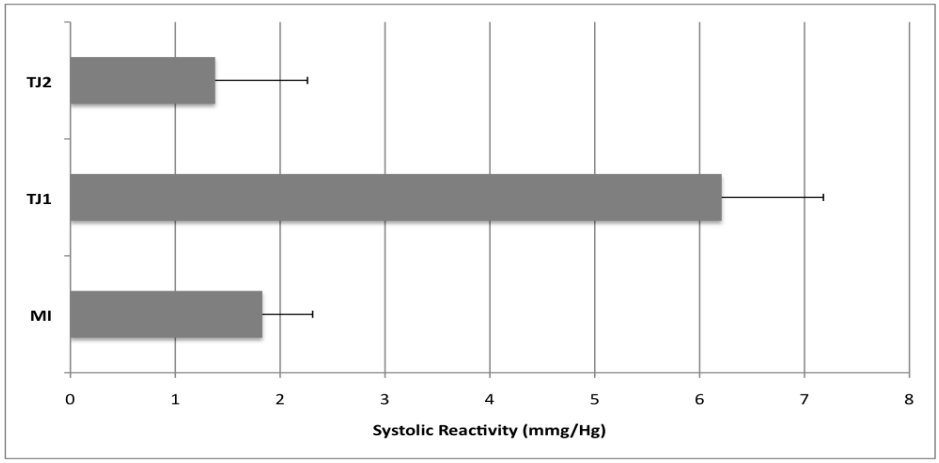


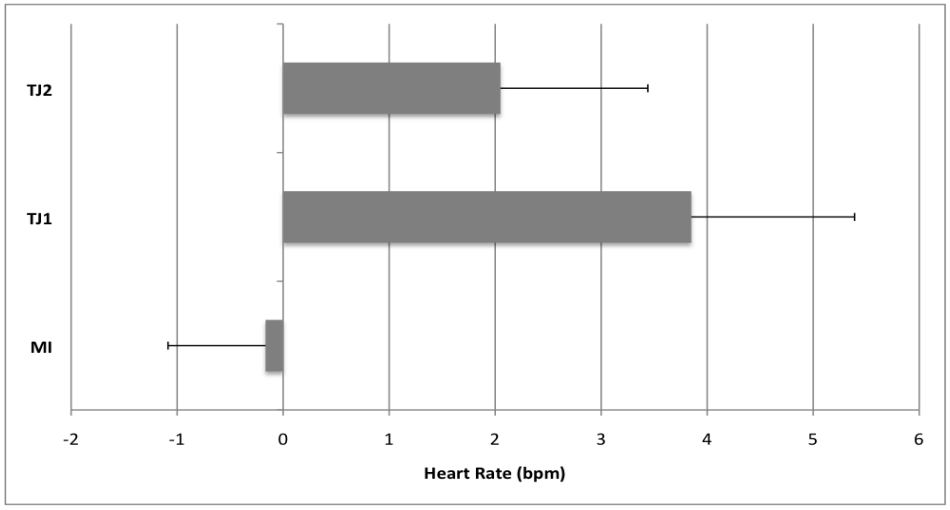
Fig. 5.6: Mean + SE group means for baselined TPR during TJ1 (N = 81).

When the second traffic jam (TJ2) was added into analysis, calculating MI vs. TJ1 and TJ2 between music groups, there were no significant differences between music groups; but specific cardiovascular differences were noted (Appendix 7). Systolic blood pressure ((F(2,186) = 18.32, $p < .01$, $\eta^2 = .17$) and diastolic blood pressure (F(2,186) = 9.29, $p < .01$, $\eta^2 = .09$) had significant effects on the TJ1 and TJ2 with decreased values in the TJ2 compared to TJ1 (ps < .01). On the contrary to SBP, PEP had lesser decreased values at TJ2 compared to TJ1 ($p < .05$) relative to MI (F(2,62) = 32.04, $p < .01$, $\eta^2 = .51$); Fig. 5.7.

(a)



(b)



(c)

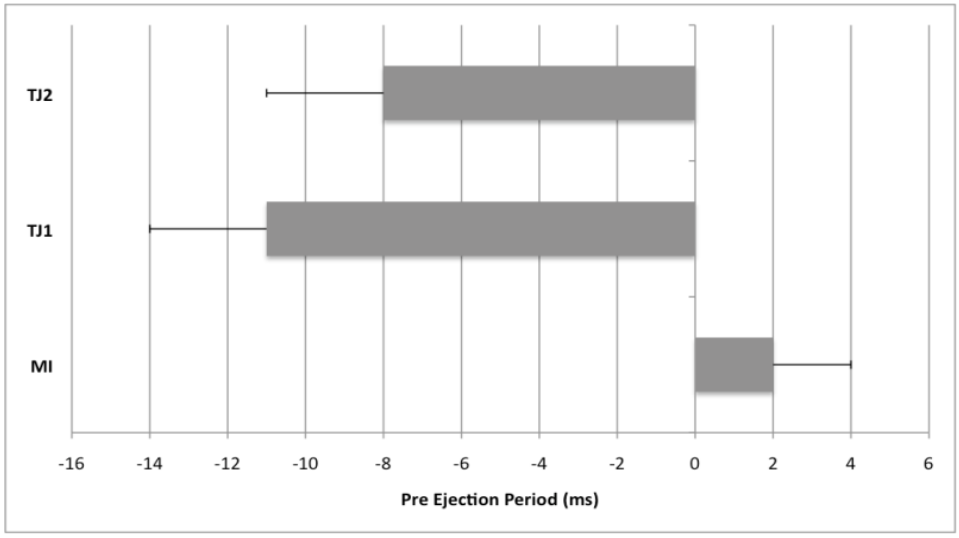


Fig. 5.7: Mean + SE effects of test phase (MI, TJ1, TJ2) on baselined cardiovascular variables: (a) systolic reactivity, (b) heart rate, and (c) pre-ejection period.

7.4. Discussion

The experimental design entailed inducing negative affect in participants by hindering their journey schedule during a simulated drive scenario. The protocol successfully elicited a mood state in the participants that coupled high activation with negative valence (Fig. 5.5b). TJ1 caused cardiovascular changes, manifested as increased blood pressure, augmented heart rate, and reduced PEP. Such physiological changes suggest an increase in sympathetic activation, which can be considered an indication of anger (Stemmler et al., 2007). Nonetheless, the profile of subjective mood and cardiovascular psychophysiology observed during the driving protocol can be related to the manifestation of anxiety in response to a perception of failure (inability to complete the journey on time) and the impact of real-world consequences (the threat of not being remunerated for participation in the study). Such adverse factors were considered equally capable of inducing states of anxiety and fear, and also states closely related to anger including frustration and annoyance. The experiment did not attempt to discriminate between different categories of negative emotion, like anger and fear (Stemmler et al., 2001, 2007), and this limited the specificity of our experimental manipulation.

Our hypothesis stated that HA/NV music would enhance cardiovascular reactivity to anger provocation, in contrast LA/PV music was expected to elicit the opposite effect. The first hypothesis was substantiated since exposure to HA/NV music was associated with high systolic reactivity and CO, alongside a reduction of TPR. This pattern of cardiovascular activity is consistent with previous work on the psychophysiology of anger (Kreibig, 2010). Regarding the latter hypothesis, some evidence was found of reduced systolic reactivity for both positive and negative types of low activation music in relation to the no-music control condition. It was expected that subjective self-report measures would follow a similar pattern. HA/NV music actually increased feelings of anger and the tendency to express anger verbally, however there was no evidence of changes in subjective mood during the simulated journey. (1) HA/NV produced high systolic reactivity but it was not higher than control condition, (2) LA music reduced systolic reactivity but

only relative to HA/NV and control.

It was apparent that potential for the music to induce a state of low activation was the most significant influence of systolic reactivity, rather than positive or negative valence. In addition, low activation music prompted systolic reactivity relative to the no-music, control condition, demonstrating how mood manipulation using music can downgrade the impact of anger during driving (Wiesenthal et al., 2000). Systolic reactivity is influenced by the force of myocardial contraction and changes in TPR. The primary mechanism responsible for the force of contraction is beta-adrenergic activity in the autonomic system (Ganong, 2005). However, diastolic reactivity is strongly influenced by changes in TPR, whereby the impact of beta-adrenergic activity may be masked (Richter, 2010). Neither diastolic reactivity or TPR exhibited the same pattern as systolic reactivity, therefore it may be proposed that low activation music exerted an influence on beta-adrenergic activity. However, this result may be interpreted with caution since PEP, widely regarded as robust marker of beta-adrenergic activation (Sherwood et al*.,* 1990; Richter and Gendolla, 2009), did not manifest any significant pattern of reactivity to the music manipulation.

The combination of anger provocation and HA/NV music significantly increased CO and reduced TPR. This outcome is supported by previous research that used film clips to provoke anger (Montoya et al., 2005). In contrast to the effects of music on systolic reactivity, both effects on CO and TPR were closely related to the participants' initial exposure to anger provocation (i.e., TJ1). The significant increase of CO for HA/NV was only relative to the LA/PV music condition. The reduction of TPR during HA/NV music was significantly different from only the HA/PV condition. Reduced TPR can be attributed to decreased vasoconstriction and active vasodilation in the muscle and arteries (Herrald & Tomaka, 2002). The former is associated with decreased sympathetic arousal and habituation. In contrast, active vasodilation is linked to the release of adrenaline from the sympathetic nervous system. The pattern of cardiovascular activity for the HA/NV music condition is indicative of active vasodilation. The reduced TPR would cause

increased blood flow back to the heart, hence increasing CO in such circumstances.

The participants in the current study experienced traffic delays on two occasions, TJ1 occurred early in the drive when successful completion of the journey schedule was possible, whilst TJ2 was timed to effectively eliminate any possibility of completing the drive on schedule. This manipulation was applied to examine anger provocation under conditions of challenge (TJ1) and threat (TJ2) (Blascovich et al., 1999). Cardiovascular reactivity to such experimental manipulation yielded elevated heart rate, blood pressure and PEP during TJ1 compared to TJ2. Therefore, cardiovascular reactivity was enhanced when success was deemed to be possible, a pattern that corresponds with the postulates of motivational intensity theory (Brehm & Self, 1989) and not with the challenge/threat model proposed by Blascovich et al. (1999) and Blascovich (2000). Brehm and Self (1989) theorised that *potential* motivation (the upper limit of individual's willingness to complete a task) could be overwritten by the variations of *motivation intensity* in a non-linear manner, first rising - then declining steeply, which resembles the reactions of participants at TJ1 and TJ2 in the present study.

Exposure to music with different psychological attributes had a significant effect on cardiovascular correlates of anger. Previous research reported that music can modulate activity in the several regions of the amygdala (Blood & Zatorre, 2001; Baumgartner, Lutz, Schmidt, & Jancke, 2006). Some of these regions can be activated and others deactivated by pleasant music. How music exactly enhances or inhibits anger responses via amygdale activity is a topic for further research. Music may further influence the experience of anger via connections between the frontal lobe and amygdala (Koelsch, Siebel, & Fritz, 2010) and therefore produce a conscious modulation of mood. In addition, exposure to music may exert an influence at the hypothalamus (Chanda & Levitin, 2013); in such case, the effect is not likely to be perceived consciously by the individual (Lovallo, 2005; Brosschot, 2010). There is evidence that background music elicits physiological responses, however music does not necessarily cause significant differences

in the perception of subjective mood (Van der Zwaag, Dijksterhuis, de Waard, Mulder, Westerink, & Brookhuis, 2012). This is in line with theories that support that mood should first pass a certain intensity and saliency before it can be consciously noticed (Gendolla, 2000). Further, music may be used unconsciously to diminish body stress. The exploration of conscious and unconscious routes to anger reduction may be suggested as an avenue for further research.

The present study was, however, marred by a limitation. The findings remain inconclusive because the time schedule manipulation employed to enhance motivation during the simulated journey did not allow counterbalancing of TJ1 and TJ2. It is consequently thought that the pattern of increased cardiovascular reactivity at TJ1 may have simply reflected a novelty effect due to initial exposure to the traffic delay. One strength of the current study was that the journey schedule manipulation and associated financial penalty provided a valuable motivational context effective in provoking a genuine anger response (Fairclough & Spiridon, 2012). However, the protocol was also limited in some respects.

In the present study, the protocol used to personalise the choice of music to produce a desired effect on mood (Skowronek et al., 2006, 2007) was validated. Specifically, the mood music was calibrated to the preferences of the individual in advance (Table 5.1). The advantage of such approach is that individual differences are catered for; the disadvantage is that experimental control is diminished (because people hear different kinds of music). Anger was provoked using a combination of short-term events (traffic jams) within the context of a long-term challenge to complete the journey on schedule and avoid a financial penalty. Anger was brought about in the participants utilising short-term boredom/displeasure as a means of impedance to the journey and implication of the delay to successfully meet the goal of the task. Further research is warranted required to investigate several aspects that were not explored in the current study, for example: 1) ascertain the relative contribution of each factor to the cardiovascular manifestation of anger, 2) explain how anger influences psychophysiology in

the presence of a reward rather than punishment, and 3) to confirm whether our findings with the respect to the impact of music on cardiovascular measures of anger generalise to other domains of life (Bongard & Al'Absi, 2005). It should also be noted that the present study took place in a simulated environment that represented a simplification of the real driving environment. Consequently, the relationship between the findings observed in the current study and actual cardiovascular reactivity during real driving remains unknown. In particular, systolic reactivity during the traffic jams was high for the control group and we question whether the relative paucity of sensory stimulation in the experiment may have: a) exaggerated the impact of music on cardiovascular reactivity, and b) amplified the aversive experience of the traffic jams for participants in the no-music control condition. In our study, threat of punishment was employed as a means of inducing negative mood. Future work could evaluate whether the availability of an additional reward yielded the same effect in this scenario.

To summarise, the study supports the notion that low activation music, regardless of valence, reduces systolic reactivity associated with a state of high-activation and negative valence although in a simulated driving environment. Further research is required to explore whether our observations can be replicated in the field and whether other environmental factors could modulate cardiovascular responses to anger.

CHAPTER 6
STUDY 4

The impact of ambient lights on anger states within the motivational context of challenge/threat in a simulated driving scenario

Abstract

Apart from music which has been found in Study 3 to be a factor in reducing cardiovascular reactivity to anger, other environmental factors such as light should also be tested to understand how ambient lighting influences psychophysiological reactivity in healthy individuals. It was hypothesised that different colours of ambient light would modulate anger state within the motivational contexts of challenge/threat. Forty healthy volunteers of mixed gender took part in the experiment. Participants were exposed to 4 ambient light conditions (red, blue, white, or no light) prior to and during a 12-minute anger induction procedure in a driving simulator. Anger reactivity was measured using questionnaires and physiological measures: BP, cardiac output, HR, and facial muscle activity. Motivation was manipulated by exposing participants to two traffic jams in a simulated driving scenario; the first traffic jam (TJ1) allowed participants to complete the task on time (challenge) while the second traffic jam (TJ2) eliminated any possibility to complete the driving schedule (threat). Subjective responses after light induction phase revealed that participants in the red light condition experienced higher levels of anger compared to the blue light condition. Physiological responses (SBP, HR, MAP) were more accentuated in TJ1 with the exception of CO that increased significantly in TJ2 compared to the light induction stage prior the task. The interactions between light effect and anger effect counterbalancing physiological responses were discussed.

6.1. Introduction

Colour is a fundamental aspect of human perception that has an effect on cognition and human behaviour (Mehta & Zhu, 2009). Research findings (Kwallek & Lewis, 1990; Stone, 2003) suggested that red colour is superior in

enhancing cognitive task performance compared with blue or green colour; however, colour theorists explained that colour influences cognition and behaviour by means of learned associations (Elliot, Maier, Moller, Friedman, & Meinhardt, 2007). Red is commonly associated with danger, mistakes and warning (Elliot et al., 2007), as well as the highest level of hazard and of compliance (Braun & Silver, 1995; Williams and Noyes, 2007). Blue tends to be associated with openness, peace, and tranquillity (Kaya & Epps, 2004). It can be proposed that the different associations pertaining red and blue colour induce alternative motivations. Red is associated with dangers and is likely to activate an avoidance motivation (Friedman & Forster, 2010; Koch, Holland, & van Knippenberg, 2008). Blue, in contrast, is likely to activate an approach motivation that allows people to behave in a more explorative and risky manner and engage in creative tasks. In support of this argument, Mehta and Zhu (2009) found evidence that red (versus blue) colour induces an avoidance (versus approach) motivation and enhances performance on a detail-oriented task performance. On the contrary, blue colour enhances performance in a creative task (Mehta & Zhu, 2009). It could be understood that the enactment of approach and avoidance behaviours was possible by incidental exposure to colours signaling safety vs. danger. Elliot et al. (2007) found that red could act as a cue to signal danger which evokes a motivation to avoid threats. In the same line of reasoning, blue could function as a cue for a benign situation evoking a challenging motivation.

However, the threat /challenge model proposed by colour theorists (Mehta & Zhu, 2009; Elliot et al., 2007) was based on overt behavioural reactions without thorough analysis of the physiological processes enabling such behaviour. Blascovich and Tomaka (1996) claimed that the threat/challenges motivational states generated by an appraisal processes preceding behaviour could be indexed by cardiovascular markers. For example, cardiovascular processes associated with the appraisal of a situation as threat (perceived demands greater than resources) was claimed to cause low to moderate cardiac activity in consonance with higher vascular resistance (Blascovich et al., 2001; Blascovich & Tomaka, 1996; Dienstbier, 1989), whereas appraisal of tasks as *challenges* (perceived personal

resources that exceed situational demands) was claimed to yield high cardiac activation coupled with lower vascular resistance. Aligning Blascovich and Tomaka's (1996) model of threat/challenge to the colour red/blue one would expect to find an association between colour and cardiovascular responses which is a clear avenue for future research.

6.1.1. Neuroanatomical Pathways of Light Sensitive Receptors

The neuroanatomical pathways of light-sensitive retinal ganglion cells (RGC) provide a mechanism by which changes in irradiance can impinge upon brain functions such as circadian entrainment, pupillary constriction, arousal, attention, emotion regulation, and vision (Lockley Evans, Scheer, Brainard, Czeisler, & Aeschbach, 2006; Dacey, Liao, Peterson, Robinson, Smith, Pokorny et al., 2005; Wirz-Justice, Terman, Oren, Goodwin, Kripke, Whybrow et al*.,* 2004). Research has shown that both acute and longer-term human nonvisual responses are particularly sensitive to monochromatic light of wavelengths between 460 and 480 nm (Lockley et al., 2006). This sensitivity is less than the average maximum sensitivity of the photopic system (555 nm). Light responses of the melanopsin-expressing RGC are detected seconds after light onset. However, firing is maintained for several minutes after the end of the light exposure which suggests that these cells are capable of producing long term effects on the nonvisual system (Berson, Dunn, & Takao, 2002; Dacey et al., 2005). In addition, melanopsin-expressing RGC receive extrinsic inputs from rods and cones which enable melanopsin-expressing RGC to immediately respond to light exposure (Dacey et al., 2005). A shortcoming of previous research is that nonvisual responses to different wavelengths in humans have only been characterised employing long duration exposures (tens of minutes). This leaves a gap in research, whereby the relative contributions of colour lights to nonvisual responses to light in humans needs further investigation.

6.1.2. Effects of Coloured Light on Mood

The finding that melanopsin is a photopigment highly sensitive to blue wavelengths (Gamlin, McDougal, Pokorny, Smith, Yau, & Dacey, 2007) has triggered substantial research on the effects of blue light (Bailes & Lucas,

2010). Aris et al., 2010 claimed that the functional organisation of the brain could be affected by blue light. They assessed brain activity of healthy participants while they listened to "angry voices" and "neutral voices". The participants were also exposed to blue or green light. Blue light enhanced emotional stimuli in the "voice area" of the brain and in the hippocampus. Blue light also promoted interaction between the voice area, the amygdala - a key area in emotion regulation - and the hypothalamus - essential for biological rhythms regulation by light. These findings inform our understanding of the mechanisms by which changes in lighting environment could improve mood.

Also, the use of blue light therapy has increased considerably supported by the realisation that patients with seasonal affective disorder have shown deficient melanopsin genes (Roecklein, Rohan, Duncan, Rollag, Rosenthal, Lipsky, 2009). It has been proposed that the impact of light therapy on mood is mediated through a long-term circadian adaptation (Vandewalle, Schwartz, Grandjean, Wuillaume, Balteau, Degueldre et al., 2010). Increased illuminance (< 3 h) has led to increases in subjective alertness and physiological measures of arousal (Cajochen, 2007; Vandewalle, Schmidt, Albouy, Sterpenich, Darsaud, Rauchs et al., 2007). In the study of Vandewalle et al. (2010) the two dimensions of illuminance and colour of lighting were considered. The research addressed mental stress and issues of wellbeing following a short period of demanding tasks in a simulated office environment. Light-emitting diode (LED) lighting was used which competes with traditional lighting such as incandescent light bulbs. The findings of research are, however, inconsistent possibly due to discrepancies in the timing of light exposure and the laboratory procedures. The evidence that lighting colour affects mood, emotions, and psychological wellbeing remains inconsistent. Most evidence originates from chronobiological studies that have investigated the effects of light colour usually during nighttime and demonstrate that blue light possesses an arousing quality in contrast with other light conditions (Cajochen, Münch, Kobialka, Kräuchi, Steiner, Oelhafen et al., 2004; Gordijn, Beersma, Rüger, & Daan, 2005). However, other studies (e.g., Varkevisser, Raymann, & Keyson, 2011) did not find that

blue light increase arousal. These conflicting findings could be attributed to experimental set-ups (night vs day light) and the different luminance level.

6.1.3. Physiological Effects of Colour Lighting

The study by Varkevisser et al. (2011) explored mood changes indexed by cardiovascular mechanisms in different colour lighting combinations and levels of illuminance using an RGB model of light. RGB is an additive colour model in which red, blue and green light are combined in various ways to produce a broad array of colours (i.e., adding red to blue yields magenta; adding red to green yields yellow; adding green to blue yields cyan; adding all three primary colors together yields white; Fig. 6.1).

Fig. 6.1: RGB Colour Model (R = red; G = green; B = blue)

Varkevisser et al. (2011) investigated how ambient LED colour light and illuminance level influenced momentary wellbeing as measured subjectively in relation to tension, fatigue, motivation, and annoyance items on a Likert type scale from 1 to 5, and in relation to self-reported arousal and valence and physiologically by cardiac reactivity following short, repeated episodes of mental stress. Perceived arousal and valence were measured using the self-assessment manikin (SAM) (Bradley & Lang, 1994). The colour combinations with a *red* component at opposing illuminance levels (RB with high illuminance (195 Lux) and RG with low illuminance (45 Lux) were found to increase fatigue, annoyance and indexed heart rate increases relative to white light condition. In these conditions (RB, RG) participants also expressed negative valence but no direct conclusions could be drawn in

rapport to arousal. In fact, arousing properties of the blue light combinations were expected to be present as reported in previous research (Berson et al., 2002; Brainard, Hanifin, Greeson, Byrne, Glickman, Gerner et al., 2001; Mills, Tomkins, & Schlangen, 2007). Their results showed a cardiac effect for the RB condition, but not for the GB condition (also with a blue component). This may be explained by the increases in annoyance and negative valence which could influence cardiac reactivity (Al'Absi, Bongard, Buchanan, Pincomb, Licinio, & Lovallo, 1997) but there were no significant effects on heart rate variability to support such claim. The absence of heart rate variability in conditions of different illuminance levels was also reported in other studies (Leproult, Colecchia, L'Hermite-Balériaux, & van Cauter, 2001; Rüger et al*.,* 2005). Perhaps, the subjective appraisal of colour was more important than the illuminance or the illuminance levels were relatively too low (45-195Lux) to elicit significant cardiovascular effects. Stronger effects are expected for brighter lighting (500 Lux) compared to standard lighting (300 Lux) (Goven, Laike, Raynham & Sansal, 2011); or the experimental set up evoked a specific vagal withdrawal, possibly related to the mental stressors and not to the different lighting conditions. The subjective measures were not validated and the measures of annoyance could have been confounded with self-reports of mood. In sum, light with a red wavelength component showed more cardiac responses and a negative affect. However, no clear alignment was made between self-report measures and a cardiovascular model. Further analysis should look into the individual influence of red/ blue colour light on clearly defined cardiovascular parameters (i.e., threat/challenge model proposed by Blascovich & Tomaka, 1996).

6.1.4. Concept of Coloured Light as Ambient Intervention

Awareness of emotion is an important focus in the design of consumer devices. Emotion recognition using physiological signals has been proposed as an optimum design solution (Kim, Kim, Lee, Whang, & Cho, 2011; Kim, Ahn, Park, & Whang, 2013). Different types of interactive lighting systems can increase immersion and trigger specific emotions (Shi et al*.,* 2010). Affective changes in a user's emotional state can be sensed using variations

in colour temperature, colour, and brightness from lighting systems. In particular, heart rate can be manipulated using changes in colour and lighting conditions (Varkevisser et al., 2011; Shi et al*.,* 2010). It would be a step forward to implement such physiological interfaces in different situational environment (i.e., driving).

Driving has been described as a cognitively demanding task (Löcken, Unni, Müller, Rieger, Heuten, & Boll, 2013). When designing the car information display, it is important to account for the driver's cognitive load. It may be desirable to keep the driver's workload low by adapting the information display to the driver's cognitive abilities. It can be argued that the driver's load can be reduced by presenting information via a less demanded resource – according to Wickens (2008) multiple resources theory. It is known that foveal vision is used during driving task and is under high demand whereas peripheral vision has the potential to provide spare capacity. Löcken et al*.* (2013) also advocate that peripheral vision is a less involved resource driving a car and therefore ambient light may be proposed as an alternative modality to enhance information presentation. It may be desirable to manipulate the driver's mood and anger using ambient light in the vehicle. Recently, there has been a great interest in developing interior lighting systems with more aesthetic and psychological appeal (Wördenweber, Wallaschek, Boyce, & Hoffman, 2007; Boyce, 2009). The use of interior coloured light in a car is feasible at night. The question remains whether implementing an ambient light system that would modulate mood in a car is effective in daylight because of the interaction with natural external light. One way around this problem could be to use shaded car windscreens and window glasses that partially block the outside light and enhance the intensity of interior lights. However, our knowledge of the effects of automotive interior lighting on the drivers' visual performance and emotional responses is limited.

Previous research has investigated the underlying influence of colour and ambient light on emotions and physiological markers. Red colour induced avoidance, threat (Friedman & Forster, 2010; Koch et al., 2008) whereas

blue colour was associated with approach, openness, risk taking (Metha & Zhu, 2009). Red colour increased arousal in a detailed task, whereas blue light increased relaxation in a creative task. These colour studies could balance the debate in the light studies where the influence of blue light on arousal has been contradictory. On one side, the blue light has been linked to arousal (Cajochen et al., 2004; Gordijn et al., 2005) and on the other side blue light did not elicit arousal (e.g., Varkevisser et al., 2011). It could be suggested that the motivational context could be an important factor when investigating the effect of light colour on physiological markers of emotions, in particular negative ones. Nonetheless, the literature on the effect of light on negative emotions remains inconclusive. Red light in combination with blue was found to elicit negative effect (Varkevisser et al., 2011), and people in a negative affective state were found to be deficient in melanopsin (Roecklein et al., 2009), a photopigment sensitive to blue light. Blue light increase responses to emotional stimuli (Aris et al., 2010), but no direct link with the red light has been found.

Hence, the current study will explore the impact of ambient coloured light on cardiovascular activity in the context of anger-provoking scenarios. It was hypothesised that different ambient colour lights modulate anger state within the motivational contexts of challenge/threat.

1. A red colour ambient anger intervention will increase the magnitude of anger, whereas a blue light will reduce the magnitude of anger.

2. SAM responses associated with challenge (increased HR, SV, CO, LVET, and low TPR) are expected to be stronger in blue light during TJ1 compared to red/white because it should potentiate approach motivation.

3. Red light will potentiate avoidance motivation during TJ2 compared to blue/white light as measured by pituitary-adrenocortical (PAC) responses: a lower rate of increased HR, SV, CO and LVET compared to challenge.

6.2 Method

6.2.1. Participants

Forty healthy volunteers of mixed gender and between 18-45 years of age were randomly allocated to one of the four ambient light conditions (red, blue, white, and no light). There were 10 participants (5 males and 5 females) in each light condition (Table 6.1).

Table 6.1: Age and driving experience of participants on each light condition.

	Age (yrs)		Driving (yrs)	
Light Colour	**Mean**	**SD**	**Mean**	**SD**
Red	28.8	7.1	5.5	7.3
Blue	23.1	3.2	2.0	3.5
White	26.9	8.7	5.9	7.5
No light	23.0	2.7	2.4	2.3

6.2.2. Experimental design

A mixed design was employed. Exposure to light was a between-participants variable as participants were exposed to red/blue/white/no light. The second variable was a within-participants variable and represented exposure to traffic jams (TJ1 and TJ2). Subjective measures and cardiovascular reactivity to both traffic jams were compared were compared to a baseline condition where data were collected in the presence of the different light conditions.

Photic stimulation

The light stimuli were presented using a 43.2 cm (17 in) flat-panel LCD monitor to avoid interference with the light generated by the projector. The lighting system consisted of two fluorescent tubes with led lights (Philips Master TL-D 18W/ 452 ActiViva) placed behind the LCD monitor in the upper part to provide a diffuse ambient light (Fig. 6.2). Light levels were measured using a Lux meter (Model CEM DT-1301 Light Meter). The average horizontal luminance measured at eye level while sitting was 500 Lux in all 3

coloured light conditions; the LED tubes were red, blue or white. The rationale for choosing 500 Lux was based on the findings that stronger effects are expected for brighter lighting (500 Lux) compared to standard lighting (300 Lux) (Goven, Laike, Raynham, & Sansal, 2011). Windows in the room were covered with opaque blinds during the study to block outdoor light interference. Following previous experimental protocols on light exposure investigations (Iyilikci, Aydin & Cambeyli, 2009; Dauchy, Wren, Dauchy, Hoffman, Hanifin, Warfield et al., 2015) a control group was formed and included in the study. Participants in the control group did not receive photic stimulation.

The early traffic jam, TJ1, occurred after 3 min of the simulated journey and introduced a 3 minute delay; we assumed that most participants would feel that it was still possible to complete the journey on time at this point, hence the first traffic jam presented a *challenge* to the goal of completing the journey on schedule. However, the timing of the late traffic jam (at the 7 minute mark), TJ2, meant that participants effectively had no opportunity to complete the journey within the required 8 minute schedule; therefore, the TJ2 represented a source of *threat*. See general method for a detailed description of the simulator protocol.

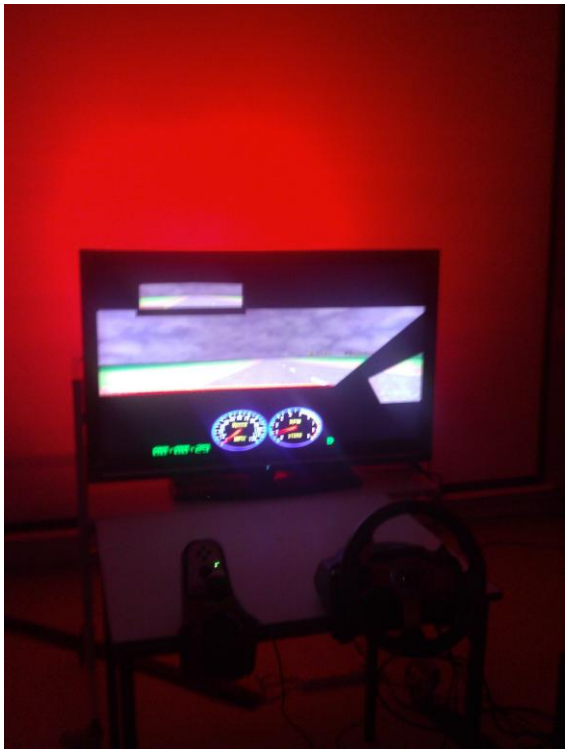


Fig. 6.2: Light induction procedure

6.2.3. Self-report measures

The State Anger Expression Inventory 2 (Spielberger, 1999) was used to measure the differences in anger levels between light conditions at baseline, post-light induction and post-driving. See general method.

6.2.4. Psychophysiological measures

Physiological measures included: systolic/diastolic blood pressure, pre-ejection period, cardiac output, heart rate, Heathers Index ventricular contractility, and Total Peripheral Resistance– see the General Methodology section for a detailed methodology. The methodology for fEMG and RMS normalization of the Corrugator supercilii and the procedures for anger induction through a simulated driving task are also described in the General Methods section of the thesis.

6.2.5. Simulator trial

Procedural sequences of events and collection of self-reported questionnaire data were replicated from study 3 and consisted of the following: practice trial (5 minutes); attachment of psychophysiological apparatus (10 minutes),

baseline period while watching a neutral video (6 min; Piferi et al., 2000); *ambient light induction* (6 minutes); *simulated drive* (12 minutes). The corresponding ambient light for each experimental group was switched on throughout the drive. Participants were asked to complete the STAXI after baseline, after the light induction, and after the drive. Finally, there was a recovery period in which the participants were given a debriefing form and a money voucher.

6.2.6. Statistical analysis

All statistical analyses were conducted using SPSS using multivariate statistical testing (ANOVA). The alpha level for significance was $p < .05$ and violations of sphericity were examined using the Mauchly's Test; if the Mauchly's Test was significant, the degrees of freedom for the corresponding ANOVA test were adapted via the Greenhouse–Geisser adjustment. Effect sizes were also included calculated as eta-squared, which provides a representation of the amount of variance in the data explained by the effect, i.e. 0.2 = 20% of variance. The Bonferroni test was used for post hoc analyses of significant main effects. All data were examined for the presence of outliers (defined as any value ± 3 SD from the mean) and outliers were omitted from statistical testing (Tabachmick & Fidell, 2001). The number of participants for each conditions after removing the outliers is presented in Tables 6.4 and 6.5.

6.3 Results

Subjective measures

State anger was captured using the STAXI in three sub-scales: (a) feelings of anger, (b) feel like expressing anger verbally, and (c) feel like expressing anger physically.

Change scores were calculated by subtracting post-baseline STAXI scores from post-induction scores to capture the effect of colour of light only, i.e., a positive number is associated with increased anger during light induction procedure. All the three subscales were subjected to 4 x 3 MANOVA (Group x State Anger). The analysis showed a significant effect for Group with

respect to feelings of anger F(3,36) = 3.64, p < .05, η^2 = .23). Post-hoc tests indicated stronger feelings of anger in the red condition (M = 1.2; SE = .41) compared to the blue condition (M = -1.2; SD = .41), p < .05; Table 6.2.

Table 6.2: Means and standard deviations for three sub-scales of the STAXI-2 based on difference scores (post-light induction minus baseline) (N = 40).

Light Condition	Feeling Angry	Verbal Anger	Physical Anger
Red	1.20 [.47]	0.80 [.60]	0.10 [.10]
Blue	0.0 [.0]	0.0 [0.0]	0.0 [0.0]
White	0.40 [.16]	0.10 [.10]	0.0 [0.0]
No light	1.00 [.30]	0.20 [.13]	0.20 [.13]

Post-drive STAXI scores were subtracted from Post-induction scores to obtain a series of change scores (Table 6.3). A 4 x 3 MANOVA (Group x State Anger) showed that the changes were not significant.

Table 6.3: Means and standard deviations for three sub-scales of the STAXI-2 based on difference scores (post-drive minus pre-drive) (N = 40).

Light Condition	Feeling Angry	Verbal Anger	Physical Anger
Red	4.3 [3.7]	2.4 [2.8]	0.7 [1.8]
Blue	2.5 [1.6]	1.7 [2.2]	0.0 [0.0]
White	3.5 [3.0]	1.0 [2.8]	0.8 [2.2]
No light	2.5 [1.9]	1.4 [2.1]	-0.1 [0.6]

6.3.1. Physiological measures

Muscle activity from the corrugator supercilii was captured and normalised (via RMS transformation) prior to analysis. EMG muscle activity at baseline was significantly different between light conditions; $F(3, 36) = 9.09$, $p < .001$, $\eta 2 = .43$. It was therefore necessary to baseline the data for corrugator supercilii EMG (Tabachmick & Fidell, 2001). The baseline value was subtracted from data obtained during the light induction and TJ and a positive number indicated an increase from baseline.

In the statistical analysis, a 4 x 3 ANOVA (4 light groups - Red, Blue, White, No light) x 3 experimental stages (Light induction, TJ1, TJ2) was carried out. The ANOVA test revealed a main effect for light conditions [$F(2, 66) = 19.41$, $p < .001$, $\eta^2 = .37$] and an interaction effect [$F(6, 66) = 12.75$, $p < .001$, $\eta^2 = .54$]. Subsequent Bonferroni tests indicated that: (1) corrugator activity was higher during both traffic jams across light conditions compared to light induction ($p < .001$), and (2) corrugator activity was equivalent across TJ1 and TJ2. Further analyses of the differences at each experimental stage, indicated that the induction stage had an effect [$F(3,36) =4.47$, $p < .01$, $\eta^2=.27$] with participants exposed to red light displaying less corrugator activity compared to the control group ($p < .01$). Fig. 6.3.

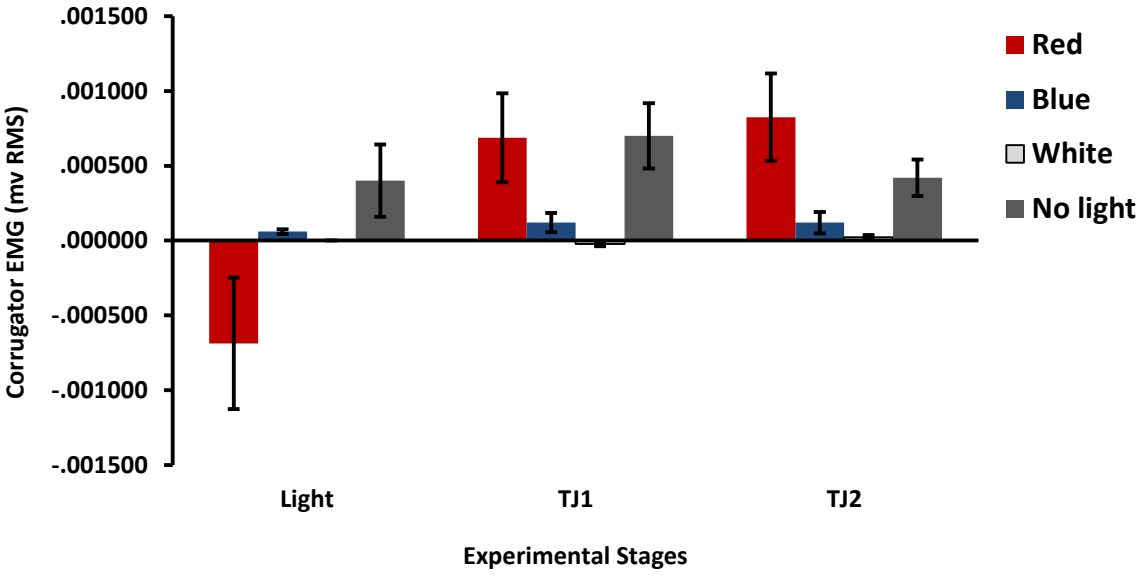


Fig. 6.3: Means and SE for baselined Corrugator Muscle within experimental stages.

6.3.2. Cardiovascular measures

Cardiovascular responses were tested at baseline between light conditions. There was a significant difference between light conditions in relation to systolic BP [$F(3, 36) = 2.93$, $p < .05$, $\eta^2 = .43$]. Hence, it was necessary to baseline the data for systolic BP (Tabachmick & Fidell, 2001). For reasons of consistency all other cardiovascular variables were also baselined (i.e, Diastolic BP, CO, MAP, PEP). In the statistical analysis, a 4 (light groups) x 3 (light induction, TJ1, TJ2) ANOVA model was applied to all cardiovascular data.

Systolic BP was significantly higher during TJ1 compared to induction had a significant effect across experimental stages: induction, TJ1 and TJ2 ($F(2,62) = 3.91$, $p < .05$, $\eta^2 = .24$). Post hoc tests showed that there was a significant increase in systolic BP during TJ1 (M = 5.48, SE = .98) compared to the induction period (M = 2.101, SE = .80); $p < .05$; Table 6.4.

There was a significant diastolic BP effect across experimental stages: induction, TJ1 and TJ2 ($F(2,68) = 3.37$, $p < .05$, $\eta^2 = .09$). Multiple comparisons corrected using Bonferroni adjustment did not indicate significant post-hoc results for diastolic BP (ps > .05).

The ANOVA results for CO indicated significant difference between experimental conditions ($F(2, 62) = 5.87$, $p < .05$, $\eta^2 = .16$). Post-hocs indicated a higher CO at TJ2 (M = .52, SE = .20) compared to light induction stage (M = - .09, SE = .13). Although red light and no light conditions showed a prominent increase in CO in the TJ1 and TJ2 stages (Table 6.5) the interaction between light condition and stages was not significant.

Table 6.4: Mean and standard error for baselined cardiovascular measures during light induction and both traffic jams. Note: Heart rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP).

		Light Induction	TJ1	TJ2
SBP (mmHg)	Red (9)	2.22 [1.58]	6.00 [1.93]	5.33 [2.54]
	Blue (9)	.56 [1.58]	3.22 [1.93]	1.44 [2.54]
	White (8)	2.63 [1.68]	5.38 [2.05]	5.50 [2.69]
	Control (9)	3.00 [1.58]	7.33 [1.93]	5.56 [2.54]
DBP (mmHg)	Red (10)	0.50 [1.17]	2.40 [2.96]	2.60 [2.88]
	Blue (10)	-1.2 [.83]	-0.40 [1.82]	1.20 [2.60]
	White (8)	0.88 [1.56]	3.123 [.83]	0.88 [.93]
	Control (10)	-0.60 [1.28]	5.00 [1.73]	2.60 [1.59]
HR (bpm)	Red (8)	4.0 [1.5]	11.0 [2.3]	3.6 [.9]
	Blue (9)	-0.4 [-.9]	3.3 [1.9]	3.4 [1.9]
	White (9)	2.4 [1.4]	4.8 [2.3]	1.3 [4.3]
	Control (9)	1.0 [1.3]	6.6 [2.2]	3.6 [1.7]
MAP (mmHg)	Red (9)	2.00 [1.70]	6.00 [1.90]	3.67 [2.23]
	Blue (8)	0.25 [.68]	1.00 [1.43]	0.25 [2.22]
	White (9)	0.22 [.57]	1.89 [1.11]	-.33 [.90]
	Control (10)	0.20 [1.25]	5.00 [2.14]	2.80 [1.88]

Number of participants in round brackets.

MAP indicated a significant difference between experimental stages: induction, TJ1, TJ2 ($F(2, 64) = 4.86$, $p < .05$, $\eta^2 = .13$) with significantly higher values at TJ1 ($M = 3.47$, $SE = .87$) compared to light induction ($M = .69$, $SE = .59$); $p < .02$, and TJ2 ($M = 1.6$, $SE = .94$), $p < .05$.

HR followed the same trend as MAP with significant differences between experimental stages ($F(2, 62) = 9.86$, $p < .001$, $\eta^2 = .24$). TJ1 had the largest effect on HR ($M = 6.42$, $SE = 1.09$) when compared to induction period ($M = 1.75$, $SE = .64$); $p < .001$, and TJ2 ($M = 2.99$, $SE = 1.31$), $p < .05$.

Table 6.5: Mean and standard errors for baselined impedance results during light induction and both traffic jams. Note: Pre-Ejection Period (PEP), Left Ventricular Ejection Time (LVET), Stroke Volume (SV) and Cardiac Output (CO).

		Light Induction	TJ1	TJ2
PEP (ms)	Red (10)	-.004 [.002]	-.011 [.010]	-.010 [.007]
	Blue (9)	-.004 [.002]	-.005 [.003]	-.003 [.005]
	White (10)	-.000 [.004]	-.005 [.006]	-.004 [.006]
	Control (9)	-.006 [.005]	-.014 [.005]	-.011 [.006]
LVET (ms)	Red (8)	-.010 [.005]	-.011 [.011]	-.018 [.009]
	Blue (9)	-.001 [.004]	.002 [.010]	-.003 [.011]
	White (9)	-.006 [.006]	-.003 [.008]	-.004 [.010]
	Control (9)	-.008 [.009]	.014 [.014]	.005 [.010]
SV (ml)	Red (10)	-3.26 [5.05]	10.60 [13.77]	7.94 [10.26]
	Blue (9)	-.95 [1.73]	3.90 [2.39	.15 [3.77]
	White (9)	-.23 [2.31]	-2.82 [4.21]	-3.15 [4.32]
	Control (9)	2.4 [8.83]	16.14 [4.17]	15.43 [4.25]
CO (L/min)	Red (8)	-0.22 [-.15]	0.76 [.66]	0.78 [.68]
	Blue (9)	-0.01 [.14]	0.37 [.19]	0.20 [.27]
	White (9)	0.10 [.29]	0.05 [.44]	0.25 [.31]
	Control (9)	-0.25 [-.37]	0.84 [.42]	0.85 [.24]

Number of participants in round brackets.

6.4. Discussion

The study was designed to identify the effect of ambient colour light (red, blue, white, no light) on subjective anger and cardiovascular responses during exposure to anger inducing simulated driving scenario within two motivational contexts of challenge/threat (TJ1/TJ2). The ambient light was presented to participants during the induction phase for 6 mins and during the duration of the drive for 12 mins. However, very little evidence was found to support the hypotheses that red light stimulated threat or that blue light promoted challenge. The effects of light colour were constrained to subjective measures - with participants in the red condition expressing higher

feelings of anger than participants in the blue condition, which may indicate social desirability or effect of past association rather than a genuine effect. Nonetheless, the threat and challenge parts of the experiment were incorporated within the task and we have no indication of subjective interpretation at TJ1 and TJ2.

The significant differences in subjective feelings of anger between red and blue light groups could be linked to a social learned effect of colour (Elliot et al., 2007); in this situation red was associated with negative affect. Moreover, exposure to colour with different psychological attributes had a significant effect on subjective anger. Previous research reported that blue light can enhance the connectivity between the amygdala and the hypothalamus (Aris et al., 2010), hence, the effect is not likely to be perceived consciously by the individual (Lovallo, 2005; Brosschot, 2010) which can explain the null differences between the baseline and light induction experimental stages for the participants in the blue condition. The absence of any effect for blue light may indicate that it does not provide a social cue in the same way that red light does; i.e., it is not salient from a social perspective. This is in line with theories that support that mood should first pass a certain intensity and saliency before it can be consciously noticed (Gendolla, 2000), but it does not explain the differences between the blue light group and the red light group in the present study. The exploration of the influences of red light on unconscious routes to anger could be suggested as an avenue for further research. Even if the effect is at an unconscious level it still produces changes in the nonvisual systems. Previous studies found that blue has an arousal quality (Cajochen et al., 2004; Gordijn et al., 2005). However, other studies (e.g., Varkevisser et al., 2011) did not find that blue light increases arousal. Such studies did not investigate the valence component and anger as an emotion defined by high arousal with negative valence that could clarify the debate. In the present study, the Corrugator muscle as a direct indicator of valence underwent the highest electromyographical reactivity in the TJ1, TJ2, motivational challenge/threat triggers within an anger inducting test. In this situation, negative valence was enforced by demands of the task and not by colour. Unexpectedly, red colour group showed less negative

valance as expressed by corrugator reactions when exposed to light only. It could be that red colour acted as a danger cue at subjective level (Elliot et al., 2007), but did not produce negative feelings. On the other side, blue light did not enhance reactivity to angry stimuli as found by Arie et al. (2010). It would be suggested to further investigate whether the conscious appraisal of colour light properties can actually enhance the physiological responses.

It was hypothesised that the type of colour light will impinge differently upon cardiovascular reactivity (BP, HR, MAP, PEP, CO) to an anger inducing task. Anger provocation was examined under conditions of challenge (delay on the route with a possibility to finish the drive; TJ1) and threat (impossibility to complete the drive; TJ2) (Blascovich et al., 1999). The results were partially in resonance with the cardiovascular model (Blascovich et al., 2001; Blascovich & Tomaka, 1996; Dienstbier, 1989). Cardiac reactivity to such experimental manipulation yielded elevated HR and MAP during TJ1 compared to TJ2. Therefore, cardiovascular reactivity was enhanced when success was deemed to be possible, a pattern that corresponds with the threat/challenge model (Blascovich, 2000). However, SBP and CO did not discriminate between threat/challenge conditions with one being higher at TJ1 and the other higher at TJ2. SBP increased in TJ1 compared to induction stage, and CO increased in TJ2 compared to induction experimental stage. The fact that the CO increased more in TJ2 compared to the induction experimental phase might indicate that TJ2 was not perceived as a threat after all but more like a challenge. The conflicting trends between CO responses and SBP pointed to a counter reactivity to light. Nonetheless, the juxtaposition between red/threat and blue/challenge taken from colour theorists (Elliot et al., 2007; Williams & Noyes, 2007; Friedman & Forster, 2010; Koch et al., 2008) was not translated in cardiovascular markers but advocated by self-reports indicating that the colour/motivation link could only work because of social cues (Mehta & Zhu, 2009).

On methodological grounds, the use of ambient light in the driving simulator could have been impaired by the luminosity of the display screen (Löcken et al., 2013); different coloured lights might have generated variations of tones

and nuances which in turn may have generated different cardiovascular responses. Ambient light could only exert an effect on peripheral vision and the darkness of the room was necessary for the simulation to work. It is known that foveal vision is used during driving tasks and that it is under high demand, whereas peripheral vision is a less involved resource driving a car (Löcken et al., 2013) and therefore ambient light had less effect on the task and on the physiological reactions. Although the ecological validity of a simulator study might be questioned, it represents a ramp to further studies in the applied field of the influence of ambient light in automotive interiors.

To summarise, the study supports the notion that red light elicits subjective anger, and found no support for the impact of various colour lights in cardiovascular reactivity in a simulated driving environment. Further research was required to explore conscious appraisal of coloured light properties known to be perceived as countermeasures of anger and associated with relaxation such as blue light.

CHAPTER 7
STUDY 5

Adaptation of cardiovascular parameters using blue light illumination

Abstract

Lack of cardiovascular differentiation between coloured conditions in Study 4 and the self-reported anger levels in the red light condition questioned wheather blue light from the monitor had a biological effect on its own and whether overt subjective interpretation of colour would allow for self-regulation of cardiovascular responses to anger scenarios. Blue light which has been claimed to influence the processing of emotional stimuli (Arie *et al*., 2010); however, previous studies (Elliot, Maier, Binser, Friedman, & Perkun, 2009) have examined the effect of ambient light as an implicit external cue. The goal of the present study was to assess the cardiovascular effects of blue light as an explicit ambient cue for relaxation. Thirty healthy participants were invited to carry out a 12-minute anger-inducing task in a driving simulator. Three equal experimental groups were formed: the first group was primed to the relaxation effects of blue light (BL1), the second group was exposed to blue light without priming (BL2) and the third group was not exposed to any ambient blue light (control group). Light was presented at three stages: prior to the task for 5 minutes and during anger induction due to simulated traffic jams. Psychological state of anger was measured using questionnaires and psychophysiological measures including: BP, CO, HR, and fEMG. Motivation was manipulated by exposing participants to the two traffic jams within the driving scenario; TJ1 allowed participants to complete the task on time without incurring financial penalties (challenge) while TJ2 annulated any chances for the participants to finish the task on time (threat). There was a decrease in subjective feelings of anger in BL1 condition relative to the control condition. Systolic BP was also significantly reduced in the BL1 condition compared to the control condition. In addition, corrugator muscle activity and SV were lower in BL2 compared to control condition. Limited differentiations between BL1 and BL2 conditions were discussed in terms of the time-on task demands and methodological challenges.

7.1. Introduction

7.1.1. Blue Light Enhances Responses to Emotional Stimuli

Light is an ever present feature of the environment, which exercises broad effects besides vision (Vandewalle et al., 2010), including hormone secretion, body temperature, sleep, alertness, cognition, and emotion regulation (Brainard & Hanifin, 2005; Cajochen, 2007; Dijk & Archer, 2009; Lockley & Gooley, 2006; Price & Drevets, 2010; Vandewalle, Maquet, & Dijk, 2009). A major milestone in our understanding of the non-visual influence of light came with the discovery of melanopsin retinal ganglion cells (Provencio, Jiang, de Grip, Hayes, & Rollag, 1998) a type of photoreceptor in the eye that is uniquely sensitive to blue light (Gamlin et al., 2007). Vandewalle et al. (2010) have explained that these non–image-forming responses to light are mediated through a non-classical photoreception system that is maximally sensitive to blue light (≈ 480 nm) but not to the classical photopic luminance visual pathways, which are maximally sensitive to green light (≈ 550 nm). Melanopsin is highly sensitive to blue wavelengths and the finding that patients with seasonal affective disorders have deficient melanopsin genes (Roecklein et al., 2009) has encouraged the use of blue light as a therapeutic intervention for that condition. However, the mechanisms by which these cells communicate with the brain are complex (Bailes and Lucas, 2010), the melanopsin cells provide signals to the suprachiasmatic nucleus (SCN), the brain's body clock (Holzman, 2010) and other regions of the brain.

For instance, the study of Arie et al. (2010) assessed brain activity of healthy participants as they listened to "angry voices" and "neutral voices" in the presence of blue or green light. Blue light enhanced emotional stimuli in the Broca Area and in the hippocampus. Blue light also promoted interaction between suprachiasmatic nucleus of the hypothalamus, amygdala and the Broca Area. These findings inform our understanding of the mechanisms by which changes in lighting environment could improve mood. However, the changes occur at a subconscious level at the hypothalamus site, and hedonic emotion regulations were not accounted for. Vandewalle et al. (2010) demonstrated that blue light proved superior to other wavelengths with respect to increased activity in the left frontal and parietal cortices during

a working memory task. As activation of the left frontal site has also been implicated in electrocortical responses to positive valenced emotional stimuli (Davidson, 1995). It is suggested that exposure to blue light may influence the processing of emotional stimuli. Nonetheless, the endocrine pathways from the hypothalamus have been found to communicate circadian and photic information to the adrenal glands (Jung, Khals, Scheer, Cajohen, Lockley, Czeisler et al., 2010). In this case, the light exposure could exert an effect on the adrenal glands, on cortisol levels. However, the results of the search for an existence of a mechanism by which photic information can acutely influence the human adrenal glands were not congruous. Light exposure has been reported to decrease (Jung et al., 2010), increase (Leproult et al., 2001) or have little effect (Rüger et al., 2006) on cortisol levels. The inconsistent findings could be explained by differences among studies including the intensity of light (~500 to 5500 lux), duration of the light exposure (15 min to 4 h), and circadian phase of light exposure; all of which affect cortisol levels.

One light colour more often associated with variations in cortisol levels is blue (Cajochen et al., 2004; Gordijn et al., 2005). Some studies found arousing properties of blue light (Berson et al., 2002; Brainard et al., 2001; Mills et al., 2007) whereas other studies (e.g., Varkevisser et al., 2011) did not find that blue light increases arousal. Nonetheless, in Varkevisser et al.'s study (2011), arousal was assessed subjectively by means of self-report manikins (Bradley & Lang, 1994), and correlated with cardiac reactivity. What they found was a cardiac effect (heart rate increase) for a light condition where blue light was combined with red light; but not when the blue light was combined with green light. The findings indicated that subjective appraisal of colour's properties was in contradiction to cardiac effects. However, the cardiac effects were not always conclusive and overt interpretation of colour could have been the factor influencing the strength of the cardiovascular responses. Also, the low illuminance levels (45-195 Lux) used in their study probably had limited effect on cardiovascular responses (Govén et al., 2010). The absence of heart rate variability in conditions of different illuminance levels was also reported in other studies (Leproult et al., 2001; Rüger et al.,

2005). Perhaps, the subjective appraisal of colour was more important than the illuminance or the experimental set up evoked a specific vagal withdrawal, possibly related to the mental stressors and not to the different lighting conditions.

Such contrasting findings regarding the arousing properties of blue light could be attributed to different contextual experimental set-ups. For example, different video contexts exercised various reactions. The work of Kim et al., (2013) could be mentioned here. They proposed an emotionally interactive lighting system that enhances affective experiences while watching video content. The emotional lighting system was activated according to the individual's emotional state; a blue light indicated relaxation, whereas a red light indicated high arousal. Their emotion recognition system used three different physiological signals (photoplethysmography, skin temperature, and galvanic skin response), an emotion lighting control system, and an emotion ambient lighting system. The findings indicated that a blue light stimulus increased photoplethysmography signals (a photoplethysmogram was obtained by using a pulse oximeter which illuminated the skin and measured changes in light absorption) when watching a relaxing video. In contrast, the frequencies of the photoplethysmography signals decreased significantly following a red light stimulus when arousing video content was played. The results indicated that red and blue light could be classified as effective manipulators of emotional arousal and relaxation experiences during displays of arousing and relaxing video content, respectively. However, the conclusions are based on a reduced number of participants and the measures were obtained post-task which reduced the physiological responses. It could be highlighted that it is crucial to objectively measure the users' emotional changes in real time. In addition, a certain amount of compensation for the individuality of emotional regulation should be applied.

7.1.2. Blue Light as an Environmental Cue for Emotion Regulation

Adaptive functioning necessitates effective emotion regulation (Hefner Verona & Curtin, 2016). Attenuation of anger, in particular, has direct benefits for health by reducing the likelihood of coronary heart diseases

(Koslov et al., 2011). The regulation of emotion may be attained using top-down processes, including reappraisal in which the person might re-evaluate the perceived relevance of a stimulus (Ochsner, Ray, Cooper, Robertson, Chopra, Gabrieli et al., 2004). Contextual information (i.e., light) can facilitate emotion regulation, such as when a task seems less threatening in the presence of blue light, whereby such light colour is associated by the individual with a relaxation state. Such contextual light-related cues are capable of conveying a sense of security despite the presence of other apparent threats, and have been labelled “safety signals” (e.g., Maier, Hill, Elliot, & Barton, 2015). When used successfully, safety signals can effectively regulate negative emotional reactions and support constructive behavioural responses to aversive situations.

Recent research suggests that distinct colours may implicitly signal the presence or absence of danger and therefore influence emotional regulation and motivational disposition. Elliot et al. (2007) proposed that the colour red signals danger in achievement contexts and implicitly evokes a motivation to avoid threats. As a consequence, red light narrows selective attention to the specific threats. Elliot and Maier's research (Elliot et al., 2007; Maier, Elliot, & Lichtenfeld, 2008) has primarily focused on the effects of red light as a danger cue, nonetheless their conceptual framework served as the basis for a new theory (Maier et al., 2015) that when colours signal safety (e.g., blue) they tend to expand rather than constrict the scope of attention. Therefore, blue colour as a safety-conveying colour promotes task performance in a manner opposite that of red. Mehta and Zhu (2009) indeed observed that the colour blue is typically associated with relaxation and should therefore function as a cue for a benign situation. Nonetheless, the cue used in their analysis was rather implicit and acted in the absence of conscious emotional experience. It was suggested that implicit “benign situation” cues tend to broaden the attention, and implicit “threatening situation” cues to narrow the attention. A substantial number of research findings involving a diverse set of such implicit affective cues (e.g., enactment of approach and avoidance behaviours: Mehta and Zhu, 2009; incidental exposure to colours signaling safety vs. danger; Elliot et al., 2007) supports this proposition. Based on this

interpretation, ambient light could act as a contextual cue (Friedman & Forster, 2010), and enable the individual to self-regulate in accord with the demands of the task. For example, blue has been considered a signal for safety (Maier et al., 2015). Consequently, blue light stimuli could be appraised as favourable and moderate the motivation in the absence of conscious emotional effort. While the consensus of the literature supports the construct that safety signals (e.g., blue light) broaden while threat cues (e.g., red light) constrict involvement in the task (Friedman & Förster, 2010), little is known about the psychophysiological underpinnings of this process.

The proposition that blue / red colours convey different signals about the nature of the current situation has received substantial empirical support. As an example, Elliot et al. (2009) found that participants equipped with body motion sensors demonstrated tendency to lean away from a test cover to a greater degree when it was coloured red compared with green or gray. This tendency toward physical avoidance is consistent with the premise that red implicitly signals danger. Similarly, Mehta and Zhu (2009) observed that participants exposed to blue were less concerned with avoiding mistakes, therefore blue implicitly tends to promote a construal of the task as benign. In contrast, when participants were exposed to red colour they expressed greater concern with avoiding mistakes than when exposed to an achromatic environment, therefore reinforcing that red elicits a construal of the experimental task as somewhat threatening (cf. Elliot et al., 2007). Cumulatively, these results support the argument that blue elicits attentional broadening and is an indicator of safety compared to red that elicits attentional narrowing and the signalling of danger. However, labelling of colour as safety versus danger could be culturally dependent, a consequence of a learnt effect (Madden, Hewett, & Roth, 2000). Studies found that blue is perceived as cold and evil in East Asia (Schmitt, 1995), but stands for warmth in The Netherlands, coldness in Sweden, death in Iran and purity in India (Schiffman et al., 2001). It denotes masculinity in western cultures – e.g., France, Sweden, UK, and the USA (Neal, Quester, & Hawkins, 2002). Blue means high quality, trustworthy and dependable in the USA, Japan, Korea and China (Jacobs, Keown, Worthley, & Ghymn, 1991).

In sum, learnt meanings of blue colour could influence the way ambient blue colour is used as an environmental cue. Hence, the present study aimed (1) to investigate whether blue light affects psychophysiological reactivity to anger as an environmental cue, and (2) to compare effects of blue light as an ambient sensory intervention with effects of blue light as a form of psychological intervention whereby the participant is primed as to the psychological meaning of the colour.

The present study was designed as a pseudo-biofeedback manipulation that consisted of exploring the effectiveness of ambient light interventions in the context of anger induction. The goal of the study was to explore whether cardiovascular markers of anger are mediated by the effects of ambient light when overt priming is introduced. In this case, blue light will be pared with relaxation prior to the task and acts as an explicit cue for the down-regulation of anger (BL1 group). Every colour has its own properties that can invoke emotions. There are for example universally valid colour associations. Red is seen as an activating colour, blue is described as having a calming effect. In experiments carried out by Visweswaraiah (2006) a blue room was found to calm the senses. Apart from meeting participants expectations of having to associate blue light with calmness (more ecologically valid application), there is also empirical evidence to support the usefulness of blue light in manipulating physiological changes. The effects of blue light as an explicit cue will be compared with the effect of blue light solely as a form of sensory stimulation with no overt priming (BL2 group). Both blue light interventions are compared to a control group who experienced the same anger induction but with no sensory stimulation. It was hypothesised that self-reported anger state will be lower in BL1 compared to the other groups. During a light induction phase, the contrast between control and BL1 & 2 will investigate the effects of overt priming and sensory stimulation vs. sensory stimulation alone on subjective and psychophysiological manifestation of anger (fEMG, BP, HR, CO). As a secondary hypothesis, anger induction is achieved using two simulated traffic jams, one that occurs early in the journey (challenge) and a second that takes place during the final phase of the simulated driver

(threat). It was also hypothesised that it is easier to relax (for BL1 or BL2) during challenge rather than threat.

7.2. Method

7.2.1. Participants

Thirty healthy volunteers of 18-41 years of age took part in the experiment. Ten participants (5 female) were assigned to three experimental groups (Table 7.1). The mean age of the participants was 23.4 years (SD = 3.6 years; see Table 7.1).

Table 7.1: Age of participants on each light condition.

	Age (yrs)	
Light Colour	**Mean**	**SD**
BL1	24.3	6.0
BL2	22.9	2.1
C	23.0	2.4

BL1: Blue Light 1, BL2: Blue Light 2, C: No light.

The participants had no history of psychiatric illness or cardiovascular problems, were not currently taking any medication, and scored below the 80th population percentile on the Trait Anger Expression Inventory of the STAXI2 (Spielberger, 1999). All procedures for participant recruitment and data collection were approved by the University Ethical Committee prior to commencement of the study.

7.2.2. Experimental design

A mixed design was employed. Exposure to light was a between-participants manipulation and exposure to experimental phases (Baseline, Light

induction, TJ1 and TJ2) was a within-participants variable. Subjective measures and cardiovascular reactivity to both traffic jams were compared to a baseline condition and a light induction phase, whereby data were collected in the presence of the different light induction procedures:

- BL1 (Blue Light 1 group): exposure to blue light plus overtly primed to associate blue light with relaxation;
- BL2 (Blue Light 2 group): exposure to blue light without priming of the effects of blue light;
- C (No light group): without exposure to blue light.

The BL1 condition involved overt priming, hence participants were told explicitly to relax during light induction phase in the presence of blue light. In the BL2 condition, participants were not informed about any possible benefits of blue light or the significance of the blue light when it was presented. This was meant to be a condition where participants were not overtly primed but experienced the same sensory stimulation with respect to blue light. In the control condition, participants were not told about the effects of blue light and were not exposed to ambient light during the drive.

7.2.3. Simulator trial procedure

The procedural sequences of events and collection of self-reported questionnaire data were replicated from study 4 and consisted of the following: *practice trial* (5 minutes); *attachment of psychophysiological apparatus* (10 minutes), *baseline period* while watching a neutral video (6 min; Piferi et al., 2000); *ambient light induction* (6 minutes); and *simulated drive* (12 minutes). Participants were asked to complete the STAXI after baseline, after the light induction, and after the drive. On completion of the drive, participants were given a debriefing form and a money voucher. The procedures for anger induction through a simulated driving task were explained in detail in the Methodology section of the thesis. In sum, participants were instructed to take a child to school within 8 minutes respecting traffic regulations. If they broke the traffic regulations they would have been penalised £1 for each violation of the traffic rules. Deception was

required to maintain the motivation for task involvement; however, participants were fully reimbursed for their participation in the study.

The simulated drive started with a 3 minutes low traffic density, after which, participants encounter a first traffic jam (TJ1) which lasted for 3 minutes followed by a low density traffic again for approximately 3 minutes and ending with another traffic jam (TJ2) for 4 minutes. The combined delay introduced by both traffic jams made it impossible for the participants to reach the destination within the required 8 min.

The blue light was activated during the light induction procedure in the BL1 and BL2 groups and during TJ1 and TJ2 for the light condition groups (BL1 and BL2). In the control condition, participants sat in a dark room during the light induction phase. Photic stimulation procedure was replicated from study 4 with the amendment that only blue light tubes were used. The luminance was set at 500 Lux during all the blue light interventions. As in previous studies, the early traffic jam TJ1 served as a manipulator of *challenge* motivation with the participants still having the time resource to complete the task whereas the timing of the late traffic jam TJ2 represented a source of *threat*, since participants effectively had no opportunity to complete the journey within the required 8 min. schedule.

7.2.4. Self-report measures

The State Anger Expression Inventory 2 (Spielberger, 1999) was used to measure the differences in anger levels between light conditions at baseline, post-light induction, and post-driving. See general methodology section.

7.2.5. Psychophysiological measures

Physiological measures included: systolic/diastolic blood pressure, mean arterial pressure, heart rate, cardiac output, and total peripheral resistance– see the General Methods section for a detailed description of the methodology. The methodology for fEMG and RMS normalization of the Corrugator supercilii was explained in the General Methods section of the thesis.

7.2.6. Statistical analysis

All statistical analyses were conducted using SPSS using multivariate statistical testing (MANOVA). The alpha level for significance was $p < .05$ and violations of sphericity were examined using the Mauchly's Test. In cases when the Mauchly's Test was significant the degrees of freedom for the corresponding ANOVA test were adapted via the Greenhouse–Geisser adjustment. Effect sizes were also included calculated as eta-squared, which provides a representation of the amount of variance in the data explained by the effect; i.e., 0.2 = 20% of variance. The Bonferroni test was used for post hoc analyses of significant main effects. All data were examined for the presence of outliers (defined as any value ± 3 SD from the mean) and outliers were omitted from statistical testing (Tabachmick & Fidell, 2001). The number of participants for each conditions after removing the outliers is presented in Table 7.2.

7.3. Results

7.3.1. Subjective measures

The protocol consisted of reducing anger levels in the participants who were exposed to blue light and had knowledge that the blue light was a trigger for relaxation. State anger was captured using the STAXI in three sub-scales: (a) feelings of anger, (b) feel like expressing anger verbally, and (c) feel like expressing anger physically. Change scores were calculated by subtracting post-baseline STAXI scores from post-induction scores to capture the effect of light induction procedure only; i.e., a positive number is associated with increased anger during light induction procedure. All the three subscales were subjected to 3 x 3 MANOVA (Group x State anger). The analysis showed a significant effect for Group with respect to the feelings of anger sub-scale $F(2,27) = 4.82$, $p < .05$, $\eta^2 = .26$). Post-hoc tests indicated stronger feelings of anger in the control group compared to the BL1 group only $p < .05$ (Fig. 7.1). The sub-scales of the STAXI relating to verbal and physical expression of anger did not significantly differentiate between the three experimental groups.

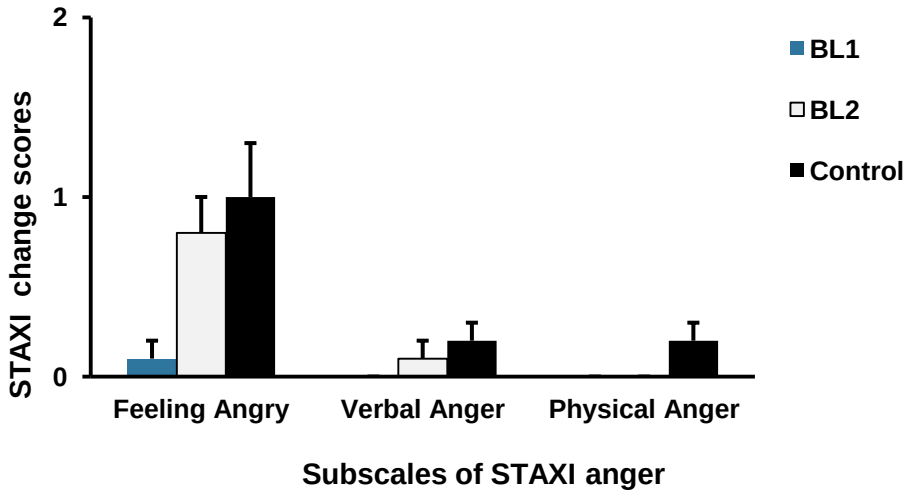


Fig. 7.1: Means and SE for three sub-scales of the STAXI based on difference scores (post-light induction minus baseline) between conditions (BL1 = Blue Light condition 1; BL2 = Blue Light condition 2; Control = No light condition). N = 30.

7.3.2. Facial Electromyography (fEMG)

Muscle activity from the corrugator supercilii was captured and normalised (via RMS transformation) prior to analysis and the baseline subtracted for the three stages of the experiment (light induction, TJ1 and TJ2). The data was baselined due to significant variations between groups at the baseline phase (Tabachmick & Fidell, 2001). A 3 x 3 ANOVA (Group x Experimental Stage) was performed. The analysis of corrugator supercilii revealed significant interaction Group between Stage ($F(3.33, 4.30) = 2.69$, $p = .05$). Post-hoc analyses indicated that participants in both *blue light* conditions exhibit reduced levels of corrugator activity compared to the control group [$p < .05$]. It was observed that participants in the light conditions had an opposing reactions at TJ1 with participants in the BL1 showing less Corrugator muscle reactivity than participants in BL2 condition. This effect can seen in Fig. 7.2.

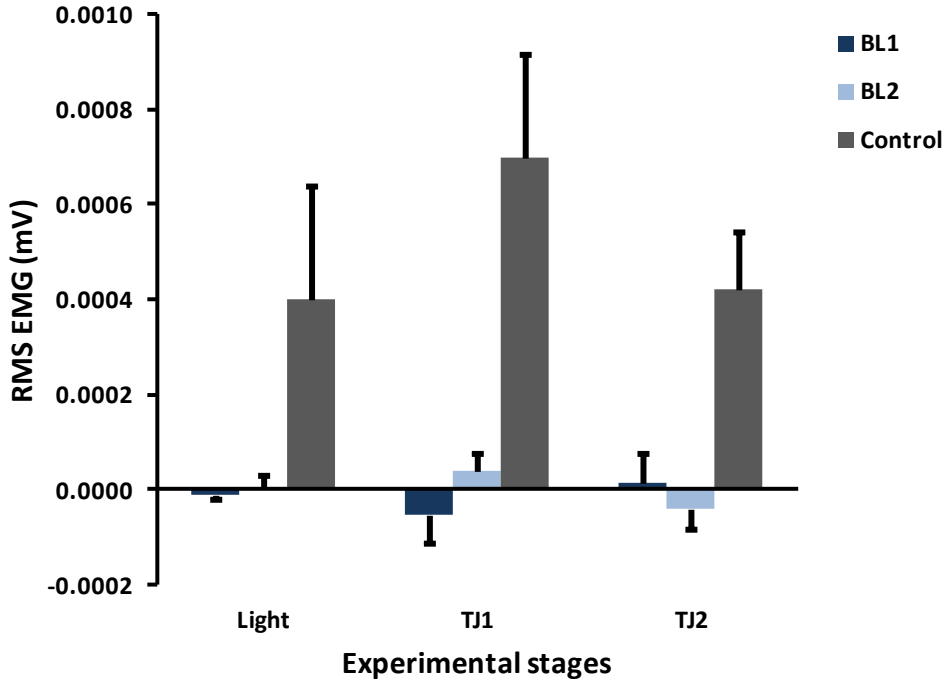


Fig. 7.2: Group mean and SE for baselined Corrugator muscle activity within experimental stages (1 = light induction; 2 = Traffic jam 1; 3 = Traffic jam 2). Note: Condition No light (control), Blue Light 1 (BL1) and Blue Light 2 (BL2). N = 30.

7.3.3. Cardiovascular measures

Cardiovascular responses were tested at baseline between the three experimental groups using a MANOVA analysis. There was a significant difference between light conditions in relation to PEP [$F(3, 36) = 2.93$, $p < .05$, $\eta^2 = .43$]. Hence, it was necessary to baseline the data for systolic BP (Tabachmick & Fidell, 2001). For reasons of consistency all other cardiovascular variables were also baselined (i.e, Systolic BP, Diastolic BP, CO, MAP, HR; see also Table A4 in Appendix 8). In the statistical analysis, a 3 (light groups) x 3 (light induction, TJ1, TJ2) ANOVA model was applied to all cardiovascular data.

The analysis of SBP revealed a significant main effect for group ($F(2,23) = 5.04$, $p < .05$, $\eta^2 = .31$). Post-hoc tests revealed a significant increase of SBP for the Control group compared to both blue light groups (BL1, BL2).

Diastolic BP was significantly different between groups [F(2, 52 = 9.63, p < .05, η^2 = .27] with highest values at the TJ1 in BL1 and control group and the lowest in BL2 group (Table 7.2). There was a main effect for stages [F(4, 52) = 5.98, p < .001, η^2 = .32]; DBP significantly increased from light induction (M = .27, SE = .59) in the TJ1 (M= 2.5, SE = .85). There was no significant Group effect for the analysis of MAP, however there was a significant effect for experimental stages [(F(2,52) = 6.53, p < .05, η^2 = .20].

The ANOVA on HR revealed a significant main effect between the three experimental phase [F(2,52) = 10.07, p < .001, η^2 = .30] with highest HR in TJ1 (M = 4.04, SE = 1.03) when compared to induction period (M = .78, SE = .55) and TJ2 (M = 2.22, SE = .96) [p < .05].

There was a PEP effect F(2,48) = 4.08, p < .05, η^2 = .14) however the differences within participants across experimental stages and the differences between conditions (Table 7.2) were not significant.

In terms of the stroke volume, there was a significant interaction between experimental stages and conditions (F(4, 52) = 4.53, p < .05, η^2 = .26 with participants in the control condition (no light) having significant higher SV responses (M = 11.32, SE = 6.85) compared to BL2 condition (M = -16.22, SE = 6.49) (p < .05).

Regarding the CO responses, the interaction Phase x Group was significant (F(4, 52) = 3.20 p < .05, η^2 = .20). Participants in the BL2 condition experienced high blood circulating volume during blue light induction (Table 7.2) and then a decrease through the stages of the experiment (TJ1 and TJ2). Nonetheless, the multiple comparisons adjusted for error via Bonferroni did not identify significant post-hoc differences between groups (p > .05).

Table 7.2: Mean and standard error for baselined cardiovascular measures and impedance results during light induction and both traffic jams. Note: Heart rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Pre-Ejection Period (PEP), Left Ventricular Ejection Time (LVET), Stroke Volume (SV) and Cardiac Output (CO).

		Light Induction	TJ1	TJ2
SBP (mmHg)	BL1 (10)	-1.50 [1.34]	.400 [2.5]	-2.6 [1.9]
	BL2 (7)	1.57 [1.6]	4.43 [2.45]	5.00 [2.27]
	Control (9)	3.00 [1.41]	7.33 [2.12]	5.56 [2.01]
DBP (mmHg)	BL1 (10)	.22 [1.07]	1.56 [1.52]	-.33 [1.37]
	BL2 (9)	1.20 [1.01]	1.1 [1.45]	-3.1 [1.3]
	Control (10)	-0.6 [1.01]	5.00 [1.45]	2.6 [1.3]
HR (bpm)	BL1 (9)	.001 [.95]	1.00[1.79]	.22 [1.66]
	BL2 (9)	1.33 [.95]	4.56 [1.79]	2.89 [1.66]
	Control (9)	1.00 [.95]	6.56 [1.8]	3.56 [1.66]
MAP (mmHg)	BL1 (10)	-1.5 [1.14]	1.2 [1.8]	-.90 [1.17]
	BL2 (9)	.78 [1.21]	.89 [1.90]	-1.11 [1.86]
	Control (10)	.2 [1.14]	5.00 [1.8]	2.8 [1.76]
PEP (ms)	BL1 (9)	.001 [.007]	-.006 [.009]	.001 [.008]
	BL2 (9)	.001 [.007]	-.023 [.009]	-.009 [.008]
	Control (9)	-.006 [.007]	-.014 [.009]	-.011 [.008]
LVET (ms)	BL1 (10)	-.004 [.006]	.001 [.011]	-.004 [.008]
	BL2 (8)	.001 [.007]	-.003 [.013]	-.016 [.009]
	Control (9)	-.008 [.007]	.014 [.012]	.005 [.009]
SV (ml)	BL1 (10)	-8.61 [7.56]	12.05 [9.61]	3.07 [7.15]
	BL2 (10)	-.35 [7.56]	-21.06 [9.61]	-27.24 [7.15]
	Control (9)	2.4 [7.97]	16.14 [10.13]	15.43 [7.54]
CO (L/min)	BL1 (9)	-.5 [.73]	.69 [1.05]	.46 [.96]
	BL2 (10)	1.03 [.73]	-.52 [1.05]	-.99 [.98]
	Control (10)	-.25 [.77]	.84 [1.1]	.85 [1.03]

Condition: Blue Light 1 (BL1), Blue Light 2 (BL2) and No light (Control).
Number of participants in round brackets.

In sum, BL1 was successful in attenuating subjective feelings of anger compared to the control group, fEMG and SBP marked expressive anger in the control group more than in the light groups.

7.4. Discussion

The experimental protocol aimed to reduce anger levels in participants who were exposed to ambient blue blight and utilised blue blight as an external explicit cue for relaxation (BL1), compared to participants exposed to blue light only (BL2) and to a control group. In sum, BL1 was successful in attenuating subjective feelings of anger compared to the control group in the light induction stage; but, both light conditions were equally effective compared to the control condition in reducing fEMG, BP and self-appraisal responses to anger inducting driving scenario. Although, the heart rate did not differentiate between groups, the effects observed at TJ1 and TJ2 were in congruence with the threat /challenge model proposed by Blascovich and Tomaka (1996) in that there was a reduced increase in HR in the TJ2 (anger/threat) condition compared to TJ1 (anger/challenge) relative to the baselined induction phase. It could be considered that the induction phase acted as baseline for the actual test. The differences found, hence, are relative to an experimental stage and by no means, are indication of absolute threat and challenge entities.

Since feelings of anger were significantly lower in the BL1 group relative to the control group, it could be claimed that BL1 participants were actively processing the presence of light stimuli and memorising its effects which comes in line with Vandewalle et al.'s findings (2010) that blue light could enhance responses in the left frontal brain activity, part of the brain also involved in processing positive valenced emotional stimuli during a working memory task (Davidson, 1995). In this context, relaxation was the positive valence emotion and the working memory was necessary to associate blue light with positive emotion. However, because the study measured the impact of light on anger, the relaxation was considered the opposite of anger state measures (the less angry, the more relaxed). Therefore, the assumption that relaxation opposed anger should be the subject of future

work. In sum, blue light with overt priming made BL1 participants more relaxed when there was nothing else to distract them.

The fact that fEMG and BP marked expressive anger in the control group more than in both light groups indicated either a reduce effect of the manipulation of primings or a sole effect of the blue light. The first explanation that could be brought forwards could be that overt priming had no effect because the appearance of blue light in BL2 was sufficient to prompt relaxation due to the biological effects of blue light wave on melanopsin receptors (Holzman, 2010). However, the mechanisms by which these cells communicate with the brain are complex (Bailes & Lucas, 2010), Since melanopsin cells sends messages to the SCN of the hypothalamus (Arie et al, 2010), which in turn communicates with endocrine pathways to reduce cortisol (Jung et al., 2010), perhaps the biological regulation occurs due to the blue light wave properties on their own. Other explanation could be a cultural association between blue light and states of low psychological arousal (Schiffman et al., 2001). Even participants not primed to the effects of blue light expressed similar reduced corrugators' activity. It could also be that the overt priming used in BL1 had no effect on actual relaxation during TJ1/TJ2; i.e., overt priming was superfluous due to biological effects or cultural associations.

A further explanation points to a flaw with the methodology. Sitting in the dark during light induction for the control participants caused fear and apprehension, which increased subjective, fEMG and SBP markers during that phase and distorted the results. Moreover, the effects of ambient light was strongest in the light induction phase when it was the sole source of light compared to the simulator phases where it was a secondary light source. Although the present study showed various increasing trends between BL1 and BL2 during the drive compared to the light induction, the statistical power of such findings was limited given the reduced scale of the comparison groups. At this stage, an extra control condition could be suggested where participants are not introduced to any ambient light prior to the task and exposure to ambient light throughout the drive could have been appropriate.

The relative discrepancy between the subjective experiences and cardiovascular responses, could be related to a dishabituating effect from the induction period given that a physical stimulus, in this case blue light, needs to reach a high level of salience after a longer expose which might not always be possible in a laboratory environment (Gendolla, 2000). Within the driving simulator the light exposure was in the context of a dark room which may have resulted in desynchrony between the biological clock and the external environment (Bedrosian, Galan, Vaughn, Weil, & Nelson, 2013a), which may lead to altered mood (Healy, Minors & Waterhouse, 1993). In the present study, the ambient light exposure was in a dark room with a black display screen and participants in the control condition had no additional ambient light which could have caused feelings of threat or anxiety presiding physiological reactions. Moreover, participants exposed to the blue light may have undergone a different mood alternation since sources of light at night that contain blue wavelengths may also be particularly disruptive to the circadian system, potentially contributing to altered mood regulation (Bedrosian, Vaughn, Galan, Daye, Weil, & Nelson, 2013). A future research direction would be a longitudinal study to allow for a classic conditioning of blue light as a physiological regulator factor. Future work should expand the investigation of the relationship between cardiovascular reactivity and different categories of negative emotional state. There is need for a new control variable whereby the blue light will be activated for the 3 conditions (BL1, BL2 and control) during TJ1 and TJ2. The blue light activated during challenge (TJ1) will have different effects on the light conditions groups (blue light with overt priming, blue light without priming, no light). Blue light activated during the threat phase of the test (TJ2) may have different effects across light conditions groups.

Future research should also address the photometry of vehicle interior lighting that offers the possibility to self-regulate based on an on-board biofeedback-system and that improves driver's health in situations of negative emotional reactions. A biofeedback investigation could be proposed to more deeply understand the importance of exposure to pervasive light in

modulating cardiovascular activity. Affective computing has had an important influence on physiological computing (Picard, 1997) in which principles of psychophysiology are implemented to determine different emotional states (Kapoor et al., 2007). The idea is for the system to respond to the emotional state of the user in order to challenge or help the user using principles of adaptive computing (Dekker & Champion, 2007; Fairclough, 2007; Gilleade & Dix, 2004). Brain-Computer Interfaces can also take advantage of volitional changes in psychophysiology to command intentional input control to computer systems (Allison, Wolpaw, & Wolpaw, 2007; Wolpaw, Birbaumer, McFarland, Pfurtscheller, & Vaughan, 2002; Kim et al., 2011, 2013). Hence, awareness of emotion is an important step forwards in the design of computer interfaces. In particular, different types of interactive lighting systems could be proposed when user can increase immersion and trigger specific emotions (Shi et al*.,* 2010) by altering the interpretation of environmental cues (i.e., blue light being interpreted as a relaxation cue).

In conclusion, blue light when accompanied by conscious appraisal of its relevance as an external cue for relaxation reduced feelings of anger in the absence of other stimuli and reduced systolic blood pressure and fEMG within an anger inducing. However, ambient blue light had limited impact on HR and MAP when dealing with a demanding task. Future work could be proposed to incorporate systolic blood pressure measures into biofeedback platforms for self-regulation of negative emotions.

CHAPTER 8
GENERAL DISCUSSION

The discussion addresses the aim of the research which was twofold: 1. to investigate how a motivational context (challenge vs. threat) influences the cardiovascular system and frontal EEG asymmetry during anger induction protocols and 2. to analyse the efficacy of ambient interventions (music, light) to reduce the impact of anger on cardiovascular responses. The general discussion evaluates the five experimental studies and addresses the primary hypotheses of the thesis relating to anger/motivational context and ambient interventions. There are two main sections within the General Discussion, these are anger in the motivational context and ambient interventions. This section ends with an outline of methodological limitations and suggestions for future studies.

8.1 Anger in the Motivational Context

8.1.1 Cardiovascular markers of threat vs. challenge

The core investigation of the thesis concerned the effects of anger induction within the motivational contexts of challenge/threat on cardiovascular responses following the model proposed by Blascovich and his team (Blascovich & Tomaka, 1996; Blascovich & Mendes, 2000; Blascovich et al, 2001). It was hypothesised that anger in the context of challenge would be manifested by a SAM response, i.e. increased HR, SV, CO, LVET but reduced TPR and low BP in combination with greater activation of left frontal cortex. In contrast, anger in the context of threat would be associated with a PAC response, i.e. increased TPR and BP; increases in HR, SV, CO, LVET (but at a lower intensity compared to a SAM response) and greater activation of the frontal right cortex.

Investigation of these hypotheses began with a laboratory-based study (Study 1) where participants were exposed to a Stroop Number computer task. A state of anger was manipulated via an experimental effect (rude vs. polite experimenter) and motivational states of control and no control were achieved by correct functioning and malfunctioning of the computer,

respectively. The four experimental groups (anger/no control, anger/control, neutral/no control, neutral/control) were compared to test for differences in psychophysiological variables. The control state was operationalised as an indicator of a challenge context, whereas the no control state was considered a threat context. In the subsequent studies (Studies 2-5), anger was induced by exposing participants to traffic delays in a simulated driving route. The driving scenario was selected in order to induce anger within a real-world scenario that would be a more potent source of anger. The first traffic delay in the driving scenario was considered a challenge context, whereas the second traffic delay acted as a threat context. The differences between Study 1, and the follow up studies consisted in increased motivation levels by introducing real life consequences for the performance in the task (i.e., financial disincentives from their participation reward voucher). Study 2 was a repeated measures study, and Studies 3-5 were concerned with differences between participants to investigate the impact of ambient interventions on anger-motivation contexts.

The results of the cardiovascular variables in this thesis are shown in Table 8.1. The changes in CV responses were assessed relative to baseline or to a baselined mood induction stage (mood induction scores were subtracted from baseline). Some differences were found between anger/challenge vs. anger/threat conditions in ambient intervention studies (Studies 3-5).

BP increased relative to baseline with the exception of Study 3 where decrease of SBP and DBP was noted in the anger/threat condition (TJ2) relative to anger/challenge (TJ1). The expected highest increase in BP in an anger/threat situation was found in Study 1. However, a word of caution, the highest increase was relative to baseline when comparing the 4 experimental groups. There was some support for the hypothesis that BP would increase in an anger/threat situation in Study 1 and some contradiction of the hypothesis was found in Study 3. We expected that BP results to follow the same increase-decrease trends as for the TPR results. TPR increased relative to baseline in one study (Study 2) in an anger/challenge context.

Table 8.1: Cardiovascular results across experimental studies 1-5: Systolic Blood Pressure (SBP); Diastolic Blood Pressure (DBP) Heart Rate (HR); Total Peripheral Resistance (TPR); Cardiac Output (CO).

	Study 1		Studies 2-5	
	Anger/Control	Anger/No control	TJ1 (Anger/Challenge)	TJ2 (Anger/Threat)
SBP		↑ relative to baseline (1)	↑ relative to baseline (2) ↑ relative to mood induction (3,4)	↑ relative to baseline (2) ↓ relative to TJ1 (3)
DBP	↑ relative to baseline (1)	↑ relative to baseline (1)	↑ relative to baseline (2) ↑ relative to mood induction (3,5)	↑ relative to baseline (2) ↓ relative to TJ1 (3)
HR			↑ relative to baseline (2) ↑ relative to mood induction (3,4,5)	↑ relative to baseline (2) ↑ relative to mood induction but less ↑ compared to TJ1 (4,5)
TPR			↑ relative to baseline (2)	
CO			↓ relative to mood induction (3)	↑ relative to mood induction (4)

Within the same context, a decrease was observed when comparing participants who were exposed to high activation negative valence to participants in the high activation positive valence group in Study 3. Hence, the TPR results were contradictory between studies. In terms of HR, we found some support in Studies 4 and 5 with a reduced increase in HR in the anger/threat condition. All the other studies found an increase in HR relative to baseline or to a baselined mood induction stage in all experimental conditions. With respect to CO, we found a decrease in an anger/challenge context in the music study and an increase in the anger/threat condition in an ambient light study. All these fluctuations were relative to mood induction stages. In sum, CV variables that differentiated between anger/challenge and anger/threat conditions were BP and HR, with lower BP (SBP and DBP) and HR.

Although the majority of studies did not differentiate between challenge and threat, BP was higher in both contexts of traffic jams (Studies 2-5) relative to baseline indicating that the induction of anger by reducing chances to complete the task on time increased BP. This finding may suggest the

influence of rumination about the task on blood pressure. For example, thinking about the consequences of performance, which had real life implications (financial penalties) was present throughout the drive, and perhaps this factor explained the high SBP values in both contexts (threat/challenge) relative to baseline which is consistent with other studies where higher systolic blood pressure was associated with rumination as opposed to reappraisal of a task (Pedersen et al., 2011; Ray et al., 2008). A differentiation between anger/challenge (TJ1) and anger/threat (TJ2) was also noted, although not in the expected direction with lower SBP in TJ2 (anger/threat). Such findings contradicted the theory that SBP should increase in an anger/threat condition (Blascovich et al., 2001) and point against the importance of conscious rumination of the consequences.

Changes in BP values should be interpreted in relation to TPR values. We expected that a challenge situation would release adrenaline into the blood stream (which will decrease the TPR and decrease SBP (Herrald & Tomaka, 2002)), whereas a threat situation releases cortisol that increases TPR and BP (Herrald & Tomaka, 2002). However, the present findings contradicted the model where decreased TPR is associated with a low BP (Blascovich et al., 2001; Blascovich & Tomaka, 1996; Dienstbier, 1989). The findings of the current thesis are in line with other studies (Ray et al., 2008, Montoya et al., 2005; Lavoie et al, 2011) that found the opposite effect, where decreased TPR was associated with increased SBP during anger induction. This reduction of TPR could be attributed to decreased vasoconstriction and active vasodilation in the muscle and arteries (Courboulin, Tremblay, Barrier, Meloche, Jacob, Chapolard, Bisserier, Paulin, Lambert, Provencher, & Bonnet, 2011; Herrald & Tomaka, 2002). Vasoconstriction is related to decreased sympathetic arousal and habituation; whereas, active vasodilation has been associated with the release of adrenaline from the sympathetic nervous system (Dart et al., 2002). The pattern of cardiovascular activity observed for the anger conditions suggests active vasodilation. It is proposed that the reduced TPR caused increased blood flow back to the heart (Montoya et al., 2005; Kassam & Mendes, 2013). For example, an increase in BP and decrease in TPR was observed in the anger/challenge condition in

the music study, but only in the High Activation/Negative Valence group which could be a direct effect of music, rather than an influence of anger within motivational context. These findings indicate that the induction protocol related to music had an enormous influence over the precise autonomic manifestation of anger (Stemmler et al., 2007) rather than the existence of various sub-forms of anger, differentiated by motivational direction that influences the heart rate and α-adrenergic response. In this case, the sensory and cognitive characteristics of anger induction scenario override any influence due to motivational context. However, the TPR values could also be gender dependent which could explain different results across studies and reverse effects. For example, it is known that males show enhanced vasoconstriction responses to stimulation compared to women (Dart et al., 2002). Given that the participants were males in the study 2, it could explain the high TPR values in the challenge context, and the TPR was one of the measures that differentiated between threat/challenge although in the opposition direction. There remains some scope for specific emotional manipulations in relation to gender and manipulation checks.

Continuing with the analysis of challenge/threat model (Blascovich et al., 2001; Blascovich & Tomaka, 1996; Dienstbier, 1989), we found some support for the heart rate measures and contradictory results on CO when ambient factors (i.e., music, light) were introduced. Although in the original model HR did not differentiate between challenge and threat (Blascovich et al., 2001; Blascovich & Tomaka, 1996; Dienstbier, 1989) trends in the data and significant results in one study (Table 8.1) indicated a possible distinction with an increase trend for HR and MAP in the challenge context. Therefore, cardiovascular reactivity was enhanced when success was perceived as possible in consonance with the threat/challenge model (Blascovich et al., 2001) as both HR and MAP increased at TJ1 compared to TJ2. However, these interpretations are based on the implied consequences of TJ1 and TJ2, and subjective assessment of challenge/threat could add more support for this position in future studies (Mendes, Blascovich, Major, & Seery, 2001). However, CO did not differentiate between threat/challenge conditions. In fact, CO decreased in a challenge context and increased

during threat compared to the induction experimental phases. These findings do not align to the proposition that challenge represents the perception that resources exceed demand (Blascovich et al., 2001). The conflicting trends between CO responses, in fact, point to an interpretation that anger could motivate the individual to appraise the situation as a challenge rather than threat even when the resources are not available (Lerner & Keltner, 2001; Mackie et al., 2000). The difficulty in separating cause from effect is founded on previous research. On the one hand, motivational theories (Blascovich and Tomaka, 1996) emphasise the overpower of threat/challenge motivational triggers on generating positive or negative affective states, while on the other hand previous research provided support for anger provoking an alerting response channeled by a pattern similar to defense reflex occurring in a threat situation (Lerner & Keltner, 2001; Mackie et al., 2000). However, a pure distinction between emotions and motivation is difficult because emotions include a cognitive, a motor, as well as a motivational component (Stemmler et al., 2007). Hence, it is important to consider each emotion such as anger with motivational facets on a reciprocal connection. Subjective definitions of challenge/threat might be different from operationalised definitions and there remains some scope to assess individual appraisals of resources and demand. For example, as described by Mauss *et al*. (2007), compared to individuals who are low on reappraisal (not efficient at decreasing negative emotional experience), high reappraisers (successful at down-regulating negative emotion) display relatively adaptive responses; that is, approach when they have control, and avoidance when they lack control. Hence, the inclusion of subjective differences in the appraisal skills should be considered in the future studies.

Based on the CV results in the present thesis, it can be concluded that there was little support for challenge/threat model of Blascovich (Blascovich & Tomaka, 1996; Blascovich et al., 2001). Only the HR values increased as predicted by the model in challenge/threat contexts with a lesser amplitude of increase in the threat condition. However, this pattern was observed only in the studies were ambient light was an additional stimulus. Based on the results of the present thesis, it cannot be assumed that challenge and threat

are discrete, nongraded states (Wright & Kirby, 2001). In fact, the present work assessed for relative differences between challenge and threat patterns but the current work did not test for group means against zero to verify whether the pattern for the group was consistent with a challenge profile, threat profile, or neither (Mendes et al., 2001). Nonetheless, the nature of challenge and threat along an anger axis indicated relative differences to a baseline state and not absolute differences between challenge and threat. Also, there were findings that did not fit either challenge/threat patterns of cardiovascular responses. The question remains whether there is a problem at a theoretical level or methodological boundaries prevented yet identifying the full set of CV indexes as described by Blascovich's research team (1996, 2000, 2001). Since there was limited support from the studies incorporated in this thesis, it could be assumed that the model is not bidirectional, and instead challenge/threat CV responses fluctuate in response to motivational dispositions to either approach or avoid the task.

8.1.2. EEG Markers of Approach and Avoidance

In addition to CV markers of threat/challenge, the thesis also explored a hypothesis that motivational disposition (approach vs. avoidance) based on measures of EEG frontal asymmetry would provide context for anger induction. The hypothesis was only tested in studies 1 and 2. The first study made a between-participants comparison (anger/control; anger/no control; neutral/control; neutral/no control) and the second within-participants study compared threat/challenge as indicated by traffic jam1 and traffic jam 2 during a simulated drive. It was anticipated that right activation would be indicative of an anger/avoidance and a left activation will indicate an anger/approach state. Following Harmon-Jones model (2004a), avoidance was defined as a state associated with low control whereas approach was associated with a sense of control over circumstances. A laterality difference score was calculated in order to examine hemispheric asymmetry in each region. The asymmetry score was calculated as an Ln difference (ln power (right hemisphere)) – (ln power (left hemisphere)). Positive values on this metric represented greater relative left hemisphere activation and vice versa (Davidson et al., 2000).

Results from the analysis of frontal EEG asymmetry found some support for the hypotheses in study 1 but results were inconclusive in study 2. In particular, a distinction was made between the states of anger/avoidance and anger/approach at the FP1/FP2 frontopolar sites during study 1, i.e. increased right-side activation in the anger/no control condition, indicating the expected trend in the direction of avoidance motivation. An earlier Harmon-Jones & Allen's study (1998) also found effects at the FP1/ FP2. However, not many other studies concerning EEG asymmetry and emotion (i.e., Buss et al., 2003; Jackson et al, 2003) have reported activity at FP1/FP2 sites (for a review, see Coan & Allen, 2004). Within the literature, the topography of the frontal asymmetry effect tends to identify activity at midfrontal sites; i.e., F3-F4 (Waldstein et al., 2000) or anterior frontal sites AF3-AF4 (Miller & Tomarken, 2001). Nonetheless, the present analysis at the F3/F4 and AF3/AF4 sites showed a contradictory pattern; i.e. greater left side activation in the anger/no control condition. Difference reference schemes have been found to measure psychometrically distinct properties of brain activity (Allen et al. 2004). It is a matter of debate which reference scheme has the greatest predictive validity regarding motivation emotion. The results should reflect the activity at specific electrode sites and not at the reference lead. For this purpose, the research in the thesis used relatively inactive reference (linked ears) as it has been found to be less prone to residual variances than Cz reference (Hagemann et al., 2001). In fact, Cz reference has the limitation of potential errors in estimating activity at target sites (Hagemann 2004). Asymmetry scores using Cz reference may have more irrelevant variance regarding asymmetry and could explain inconsistencies across studies investigating asymmetry patterns with respect to motivation/emotion models. Difference reference schemes have been found to measure psychometrically distinct properties of brain activity (Allen et al. 2004). It is a matter of debate which reference scheme has the greatest predictive validity regarding motivation emotion. The results should reflect the activity at specific electrode sites and not at the reference lead. For this purpose, the research in the thesis used relatively inactive reference (linked ears) as it has been found to be less prone to residual variances than Cz reference (Hagemann et al., 2001). In fact, Cz reference has the limitation of

potential errors in estimating activity at target sites (Hagemann 2004). Asymmetry scores using Cz reference may have more irrelevant variance regarding asymmetry and could explain inconsistencies across studies investigating asymmetry patterns with respect to motivation/emotion models. Further analysis of frontal EEG asymmetry revealed a reduction of left hemisphere activation at F3/F4 during both situations of approach/avoidance in relation to traffic jams. Changes in frontal EEG asymmetry during the first traffic jam indicated a significant reduction of left frontal activation/approach motivation when an increase of left activation had been anticipated. These findings do not support Harmon-Jones's (2004a) theory which postulates that asymmetric left activation is observed only in an approach state. However, the induction protocol could have influenced the findings. On one hand, in agreement with Miller & Tomarken's findings (2001), the manipulations of the outcome expectancies (reducing the possibility to end the task on the time schedule by means of traffic jams) resulted in changes in activity at mid frontal sites. On the other hand, it could be that both traffic jams induced a loss of control because the participant was trapped in traffic and helpless - hence there was reduced left-side activation or a bias towards right side activation. Other studies have also reported inconsistent findings. For example, Wacker et al. (2003) found that anger with a withdrawal state presented asymmetric left frontal EEG activation. Waldstein et al. (2000) found comparable left and right activation during states of anger, however only right activation correlated with other physiological changes. Asymmetric mid frontal right EEG activation was found to enhance BP reactivity, without a significant change in heart rate (Witling, 1990; Waldstein et al., 2000). Considering that systolic BP was also sensitive to the anger/avoidance state, it could be suggested that the right activation at the frontal sites increases vascular resistance. However, the frontal peripheral site showed the expected electroencephalographic right-brain activation in the anger/no control condition and not the mid-frontal as found in previous literature (Waldstein et al., 2000). Nonetheless, in a male only study (study 2), the findings indicated reduced mid frontal left activation and an increase in BP which might be explained by a gender bias with respect to interhemispheric regulation of blood pressure (Witling, 1990).

The conflicting trends in the frontal asymmetry data may point to the fundamental bivariate structure of positive and negative evaluative substrates. This interpretation is consistent with the hypothesis that the left hemisphere is associated with parasympathetic activity related to nourishment, positive affect; whereas the right hemisphere is more linked to sympathetic regulation and negative affect, aversive reactions and defensive behaviours (Craig, 2005). Alternatively, it could be a case of adaptive responses, wherein higher level neural systems permitted “avoidance” responses as a goal strategy (Rutherford & Lindell, 2011). In this situation it could be expected to find an association between lower left frontal asymmetry and a maladaptive cardiovascular response (threat) in support of Koslov et al.’s study (2011). However, there was no direct support for this position as none of the cardiovascular variables showed any significant association with frontal EEG asymmetry during either study, in fact, all correlations were in a negative direction, i.e. greater left activation = reduced CO. In hindsight, the present findings could caution against a fundamental hemispheric coding of approach/avoidance behaviours and emphasise the need to better characterise the multidimensionality of anger and motivation.

8.1.3. Summary

In summary, the cardiovascular and EEG results of the present thesis point to a model of anger with quadruplet facets along cardiovascular responses to challenge/threat contexts in conjunction with approach/avoidance tendencies (Fig. 8.1) where right frontal activation and increased TPR define a state of challenge and increased CO defined a threat. The circumplex model derived from the present findings moves along the horizontal axis defined by activation levels (avoidance-approach) and the motivational levels (challenge-threat). A threat motivation with avoidance was indexed by increased SBP, DBP and CO and by greater right frontal activation. The difference in the approach-threat responses will be the activation of the left hemisphere. The challenge-avoidance state was defined by increased TPR, SBP, HR, MAP and greater right frontal hemisphere activation. No frontal asymmetric index was identified was identified in the challenge-approach, but increased TPR, SBP, HR and MAP were observed. The findings point

the physiological differences observed in an anger state within a threat context to indicate a non-healthy pattern of physiological responses that could be damaging to health in the long term (Mathews, 2005). Nonetheless, the methodological differences between studies make it hard to ascertain the specificity of each dimension in relation to the cardiovascular and EEG findings.

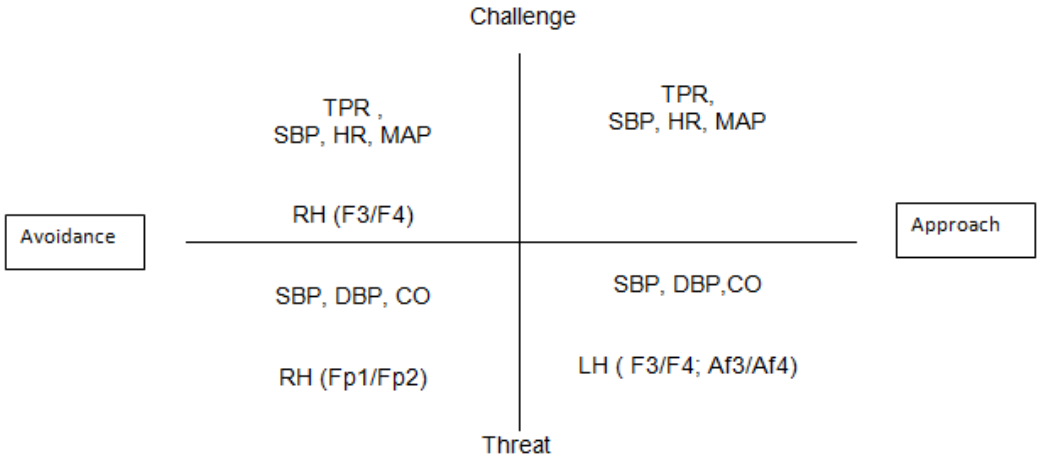


Fig. 8.1: A circumplex model of anger. Note: horizontal axis represents activation levels and vertical axis represents motivational dimension. Note: Systolic Blood Pressure (SBP); Diastolic Blood Pressure (DBP) Heart Rate (HR); Mean arterial pressure (MAP); Cardiac Output (CO); Total Peripheral Resistance (TPR); Left hemisphere activation (LH); Right hemisphere activation (RH).

8.2. Ambient Interventions

The second general hypothesis was related to the effectiveness of ambient mood interventions on cardiovascular correlates of challenge/threat as a form of affective computing. In study 3, the primary hypotheses were whether ambient intervention in the form of music could influence CV markers of anger and whether music modulated changes in the CV system in context of both anger/challenge and anger/threat. Study 4 explored whether coloured lights can have similar effectiveness to music during anger induction. Study 5 investigated whether CV markers of anger responded to blue ambient light as a sensory intervention or an information cue for relaxation. The results from all three studies indicated that music mitigated the influence of anger on

the CV system during certain scenarios and that blue ambient light had a psychophysiological effect on anger only when functioning as an information cue for relaxation. These data are summarised in Table 8.2.

Table 8.2: Cardiovascular differences mitigated by ambient interventions.

Variable	Study	Groups	Group Comparisons	Study	Groups	Group Comparisons
SBP	3	LA/PV HA/PV LA/NV HA/NV No music	↓ LA/PV ↓ LA/NV (compared to HA/NV and no music groups)	5	BL1 BL2 No light	↑ No light (compared to BL1 & BL2)
DBP	3	LA/PV HA/PV LA/NV HA/NV No music	↓ LA/PV ↓ LA/NV (compared to HA/NV and no music groups)	5	BL1 BL2 No light	↑ No light (compared to BL1 & BL2)
HR	3	LA/PV HA/PV LA/NV HA/NV No music		5	BL1 BL2 No light	
CO	3	LA/PV HA/PV LA/NV HA/NV No music	↓ LA/PV (compared to HA/NV in TJ1)	5	BL1 BL2 No light	↑ No light (compared to BL2)
TPR	3	LA/PV HA/PV LA/NV HA/NV	↓ HA/NV (compared to HA/PV in TJ1)			

HA = High activation, LA = Low activation, PV = Positive valence, NV = Negative valence, BL1 = Blue light with information prime for relaxation, BL2 = Blue light without information prime, TJ1 = Traffic jam 1.

The findings of the music intervention study (study 3) suggested that prior exposure to ambient intervention characterised by high activation, negative valence (HA/NV) during the induction session primed the cardiovascular

system to enhance blood flow during the subsequent traffic jams and that factor could explain the high systolic reactivity observed in the traffic jams for this type of music. LA music reduced systolic reactivity but only relative to HA/NV and control (no music). The sensitivity of systolic blood pressure to the activating properties of the music suggested that the observed effect was mediated by beta-adrenergic activation (Richter & Gendolla, 2009). Since systolic blood pressure was reduced in both low activation groups (LA/NV and LA/PV), the study demonstrated that effects of music on CV measures were linked to the stimulating or activating qualities of the music, rather than being influenced by the qualities of music in inducing positive or negative emotion. Systolic reactivity is a function of myocardial contraction and it is governed primarily by the level of beta-adrenergic activity in the autonomic system (Ganong, 2005). Although not reaching statistical significance, the same trend was observed in the PEP data, which can be considered the most robust marker of beta-adrenergic activation (Sherwood et al., 1990; Richter & Gendolla, 2009). Also, another indication of beta-adrenergic activity was the fact that SBP presented a pattern that differed from diastolic reactivity and TPR (the impact of beta-adrenergic activity may be masked (Richter, 2010).This fact supports the concept that mood manipulation using low activation music can moderate anger during driving (Wiesenthal et al., 2003). Nonetheless, the effectiveness of low activation music to reduce SBP could also be an indication of the propensity of music to distract or to stimulate during boredom of the traffic jam. Given that self-report STAXI scores related to the experience of the music were incongruent with the SBP findings, it could be concluded that ambient effects of music occured at an unconscious level (Lovallo, 2005; Brosschot, 2010). Based on the findings, there is scope for the Ambient Intelligence (AmI) interfaces that support the activity of the user through the use of information and communication technologies distributed pervasively in the environment (Morganti and Riva, 2004) with personalised adaptable environment to the emotional state of the user. Such personalised environment was created in the music study following an earlier procedure (Skowronek et al., 2006) and the results were validated. Specifically, the mood music was calibrated to the preferences of the individual in advance. The advantage of such approach was that

individual differences were catered for, whereas the disadvantage was that experimental control was diminished (because participants heard different types of music).

With respect to the studies of ambient light as an intervention, it was proposed that red may activate states of threat whereas blue was associated with challenge. The cardiovascular markers did not provide strong support for the red/threat and blue/challenge colour theories (Elliot et al., 2007; Williams & Noyes, 2007; Friedman & Förster, 2010; Koch et al., 2008). However, self-reports advocated the expected colour –state linkage indicating a social conditioning of the meaning of colour (Mehta & Zhu, 2009). It is worth noting that within the coloured light environment (Table 8.2), SBP increased in challenge scenario (traffic jam 1) compared to induction stage, and CO increased in threat context (traffic jam 2) compared to induction. Higher CO in threat context might indicate that TJ2 was not perceived as a threat, but rather as a challenge despite the fact that it was impossible for participants to complete the journey on time at this point. There were therefore conflicting trends between CO and SBP responses that may suggest a counter reactivity to light, a form of reappraisal using light as a cue. In support for this argument, the results from study 5 indicated that the systolic BP was at a lowest level in the condition where participants used the light as an environmental cue for relaxation. During light indication, blue light reduced anger with respect to self-report and effectively regulated negative emotional reactions and supported constructive behavioural responses to aversive situations, in this case, anger inducing drive. Hence, the appraisal of environmental cues for relaxation reduced cardiovascular responses, whereby blue light colour was a contextual cue capable of conveying a sense of security despite the presence of challenges and threats. In both situations blue light acted as an explicit safety signal (Maier et al., 2015) effectively regulating negative emotional reactions. In this situation, participants may have ruminated about the consequences of the task but in fact reappraised the task based on the environmental cues. These findings support Ray et al.'s (2008) claims that the physiological response of the participants could be influenced by increased peripheral vasoconstriction

during rumination compared to the reappraisal of condition. Moreover, the cardiovascular reactivity as measured by stroke volume was found to be reduced by reappraisal of environmental cues in study 5. Participants in the BL2 condition experienced lower blood circulating volume during blue light induction. This suggests that ambient light could influence cardiac activity.

Exposure to colour with different psychological attributes had also a significant effect on subjective anger. The significant differences between red and blue light in subjective feelings of anger in study 4 could have been caused by a social learned effect of colour (Elliot et al., 2007); where, red tends to be associated with negative affect. It was assumed that blue light facilitates the connectivity between the amygdala and the hypothalamus (Aries et al., 2010). Thus, the effect was not likely to be perceived consciously by the individual (Lovallo, 2005; Brosschot, 2010) therefore throwing light into the null differences between baseline and light induction stages on the experimental intervention in the participants in the blue condition. The blue light did not yield any significant effect which may indicate that blue light does not provide a social cue in the same way that red light does; i.e., it is not considered salient from a social perspective. Accordingly, some theories support that mood should first pass a certain intensity and saliency before the person can consciously notice its effect (Gendolla, 2000). However, such theories do not explain the differences between the blue light group and the red light group in study 4. The exploration of the unconscious influences of red light on anger is a subject for further research. It may be speculated that even if the effect is at an unconscious level it still produces changes in the nonvisual systems. There is evidence in support of blue having an arousal quality (Cajochen et al., 2004; Gordijn et al., 2005). However, other studies (e.g., Varkevisser et al., 2011) reported that blue light did not increase arousal. Such studies did not investigate the valence component of anger as an emotion characterised by high arousal with negative valence. As found in study 5, participants in the blue light conditions did had lower cardiovascular responses (BP, SV) compared to the control group regardless of conscious appraisal of the blue light as an information cue for relaxation indicating a biological effect

contradicting self-reported anger level. In fact, self-reported anger state was lower in the group of participants exposed to blue light and primed a relaxation cue (BL1) compared to the control group. BL1 participants were actively processing the presence of light stimuli and memorising its effects. These findings are in agreement with Vandewalle et al. (2010) who proposed that blue light enhances responses in the left frontal brain activity, which is the part of the brain also involved in processing positive valenced emotional stimuli during working memory tasks (Davidson, 1995). In this context, relaxation was the positive valence emotion and the working memory was a vehicle to associate blue light with positive emotion. However, in the study relaxation was considered the opposite of anger state (the less angry, the more relaxed). Therefore, the assumption that relaxation opposed anger should be explored in future research. The effect of the blue light with overt priming was that BL1 participants felt relaxed when there was nothing else to distract them.

In summary, the second general hypothesis of the thesis addressed the effectiveness of ambient mood interventions on cardiovascular correlates of challenge/threat as a form of affective computing. The ambient interventions confirmed that psychophysiological correlates of emotion triggered within a driving context could be modulated by environmental stimuli such as music in study 3 in accordance with previous studies (Wiesenthal et al., 2000; Juslin & Sloboda, 2010) or ambient coloured light (Knez, 2001) but only if the light is used an environmental cue (study 5). The results suggested that low activation music mitigated the influence of anger on the CV system; in addition, the appearance of blue ambient light elicited a psychophysiological effect on anger only when functioning as an information cue for relaxation. It was proposed that red may activate states of threat whereas blue was associated with challenge. However, the cardiovascular markers did not provide strong support for the red/threat and blue/challenge colour theories.

8.3. Methodological Issues

8.3.1. Validity of Methodological Protocols

Issues concerning the effectiveness and specificity of the anger induction

was a methodological issue that cut through the whole thesis. The experiments were not designed to discriminate between different categories of negative emotion, such as anger and fear (Stemmler et al., 2001, 2007), and the specificity of the manipulation suffered as a result. For example, the pattern of subjective mood and cardiovascular psychophysiology observed during the driving protocol were consistent with anxiety in response to a perception of failure (being unable to complete the journey on schedule) and the impact of real-world consequences (the threat of not being paid for their participation), which was equally capable of inducing states of anxiety and fear, as well as states closely related to anger such as frustration and annoyance. Designing studies to induce 'pure' emotions or pure 'challenge' or 'threat' is close to impossible given that all emotions encompass the effects of motor responses, cognitive responses (Stemmler et al., 2007).

Creating an authentic anger induction protocol was particularly challenging in study 1 as the task was perceived mundane in nature and the lack of real life consequences could have attenuated cardiovascular responses (Cacioppo et al., 1993). The combination of a computer malfunction and vocal harassment by experimenter used to provoke anger did not differentiate between the two anger conditions (anger/control vs anger/no control) in terms of self-report measures and the corrugator activity underlined the lack of distinction between anger and control groups. Therefore, what was considered a mundane task in study 1 was replaced by a more naturalistic driving task in studies 2-5 because it was more likely to tap into past experiences of anger in real life. Driving is a routine activity that raises negative emotions when roads obstructions are present (Vanlaar, 2008; McGarva, 2005). Links were found between high levels of systolic blood pressure and the appraisal of the impossibility of completing the journey schedule in line with previous studies (Hennessy & Wiesenthal, 1997). Self-reported measures also indicated higher levels of anger post drive compared to pre-drive anger. Overall, the subjective anger was higher for the driving task compared to the subjective anger measured in study 1 where a recorded voice of a rude experimenter was used to manipulate anger while participants performed a computer task with a malfunctioning keyboard. In sum, from a methodological perspective,

the simulated driving task proved to be a more ecologically valid method for anger induction in studies 2 to 5 compared to study 1.

Within the driving simulator, anger was induced using traffic delays in a simulated driving route. The first traffic delay was designed as a challenge context and the second traffic delay presented a threat context. A methodological limitation was the fixed order of traffic jams which could not be easily changed without affecting the experimental manipulations. This fixed order was designed as a progressive inducer of genuine anger responses within the motivational continuous from an initial challenge of TJ1 to a threat of not completing the journey on schedule at TJ2. Future studies could consider a mid-experiment target. Another avenue for research would be testing the possible effects of time on task using a control group who would receive no penalty when late or receive reward rather than punishment. Another disadvantage of using continuous manipulation of motivation was that the motivational directions (challenge/threat) could not be assessed by means of self-report measures during the simulated drive and there was no indication whether the physiological effects reflected subjective appraisal of driving situations as challenge or threat. The indication came from the Corrugator muscle activity which suggested that significant changes in negative affect were observed in both challenge/threat scenarios with a decrease being observed in the challenge scenario (i.e., study 5) and increase in study 2 indicating various levels of anger. However, this finding should be interpreted with caution since corrugator activity could be an ambiguous measure of anger expression via facial musculature; and increased muscle activity could be generated by other events like increased mental information processing (Topolinski et al., 2009). However, the mental demands of the driving task were minimal during the traffic jams; therefore, a high mental information processing can be safely disregarded as an activator of corrugator contraction. Nonetheless, the presence of traffic jams could have been a surprise accompanied with a facial coding that involves the action of the corrugator as an indicator of negative valence (Topolinski & Strack, 2015).

The anger-inducing protocol of the simulator studies were derived from the presence of real-world consequences (payment) associated with failure and a resonance between the simulated traffic jam and participants' actual experience as drivers. The absence of a control condition meant that it was impossible to differentiate between the effects of anger provocation (traffic jams) and real-world consequences (journey schedule/financial penalties) on psychophysiological markers. The real-world consequences and scenario meant that people may have been anxious as well as angry. Hence, the protocol to induce anger may have induced a mixed state of anger and anxiety. Moreover, the way anger manifested itself with respect to the cardiovascular system may be specific to the domain of anger during driving and the expression of anger in a driving scenario may not generalise to the measurement of anger other domains of life such as home or the workplace (Bongard & Al'Absi, 2005). High job stress was found to be associated with greater reports of anger-in and anger-out behaviour suggesting that the way people express anger at their work place might be different from other domains. However, even typically calm, reasonable people in a home or work scenario could turn into high anger-expressing individuals behind the wheel (Deffenbacher, Deffenbacher, Lynch, & Richards, 2003). A high anger driver will also be more predisposed to have more accidents, take more risks on the road, and engage in hostile, aggressive thinking. The domain specific anger is therefore an important factor in determining the impact of anger on cardiovascular health.

Aside from the individual differences that were not considered in the present thesis (e.g., high trait anger vs. low trait anger drivers), there were other possible methodological explanations for the lack of consistent differentiation between cardiovascular reactivity associated with anger in a threat context vs. a challenge context were that the task manipulations were either not powerful enough to induce changes (e.g., study 1), or that the nature of the tasks (TJ1, TJ2) elicited sufficiently large changes, but that the presentation order was a key factor in fluctuating the cardiovascular reactivity to anger. A drawback of the driving simulator studies was the lack of counterbalancing between challenge and threat scenarios, which was unavoidable given the

time-critical definition of both states. It could be that the physiological reactivity to the first traffic jam was the consequence of a surprise, whereas the reactions to the second traffic jam were reduced due to a learned effect. Also, it is possible that the use of financial penalties (i.e., fines for speeding) to prevent participants from undermining the time schedule manipulation could have reduced approach-related anger.

The choice of ambient interventions as countermeasures to anger was based upon a capacity to not distract from the primary driving task, i.e. no direct visual inspection was necessary to detect the intervention. Ambient interventions can influence autonomic markers of emotional states without awareness of the person (Cearreta et al., 2007). The low activation music and blue light were examples of this property where the ambient interventions acted as mediators of cardiovascular parameters. However, there was greater individual variability in responses to light compared to music which could have been a consequence of sample size or individual differences. Participants' prior knowledge, social learning and associations all could have had an influence on their reaction to colour lights. Individual mental representations of colour result from activity in the visual cortex as well as other areas of the brain concerned with the long-term memory (Kosslyn & Thompson, 2003). Hence, the colour could be a consequence of a learning effect (Madden et al., 2000) based on cultural influences. In the ambient light studies, the choice of colours to produce the desired effect on mood was based on western literature and not individualised on cultural grounds. The samples contained also some student participants from an Easter Asian background and each participant's cultural conditioning for the effects of colour light could have resulted in different reactions to the presence of colour. Studies (e.g., Jacobs et al., 1991; Schiffman et al., 2001) found that colour is perceived differently across cultures and such differences should be investigated in future studies in rapport to their effect on human physiology.

The driving simulator was located in a dark room environment (i.e. to maximise visual clarity and context of the simulated driving scene) and this

factor may have caused desynchrony between the biological clock and the external environment (Bedrosian et al., 2013), therefore possibly altering mood (Healy et al., 1993). The simulator room was a dark room which mimicked night time environment and any alteration to the light exposure could have influence the activity of the SNC hypothalamus which could have influenced the mood. Nonetheless, the experimental set up in a dark room could have also caused feelings of threat or anxiety presiding physiological reactions by suggesting potential dangers and risks (Blöbaum & Hunecke, 2005). Other studies (e.g., Li, Mac, Kanga, Qiaoa, Tanga, Qiua, Zhanga, & Lid, 2015) found darkness to be associated with fear but with less impact on cardiovascular responses compared to night time. There is an issue of the darkness influencing physiological effects and future studies should accurately distinguish between “night” and “darkness” influences on self-appraisal of various categories of emotions and specific physiological correlates.

The limited moderator properties of colour lights could be explained on the design of the intervention room. It could be that light as mood moderator needs a more thorough control of its individual properties. The ambient light within the driving simulator room could have been impaired by the luminosity of the display screen, in that different coloured lights might have generated variations of tones and nuances which in turn generated different cardiovascular responses. For example, red light with blue tones from the display monitor elicited different reactions compared to red light on its own. It was noticed that the effects of ambient light were strongest in the light induction phase when ambient light was the sole source of light compared to the simulator phases in which case the ambient light become a secondary light source. It could be claimed that the ambient light had less effect on the task and on the physiological reactions because it exerted an effect on peripheral vision while the foveal vision was under high demand during the driving tasks (Löcken et al., 2013). The lack of differences between ambient colour interventions could be attributed to specific quality exerted by the blue light on its own, either generated by the monitor display or by the ambient light tubes behind the monitor in the blue light group. Participants exposed to

the blue light may have undergone a different mood alternation since sources of light at night (dark room in the present studies) that contain blue wavelengths may also be particularly disruptive to the circadian system, potentially contributing to altered mood regulation (Bedrosian et al., 2013). Based on the results of the last study that contained only blue light groups there were differences evidenced between conditions. Hence, a future research direction would be designing matched ambient light/monitor display interventions to allow for comparisons between discrete colour lights.

In summary, the main methodological issues around the independent variables were the difficulties to create authentic anger and challenge/ threat induction protocols due to either the mundane nature of the task or to other associate unaccounted for feelings of anxiety and fear generated by the darkness of the simulator.The strict order effect was another limitation of the designed simulated scenario along with the light interferences which could have reduced the effect of individual colour lights. Although self-reports captured anger-related information, subjective variability between studies could be a reflection of sample size effect or individual differences.

8.3.2. Dependent Variables

Subjective state anger was measured using a general score on STAXI 2 (Spielberger, 1999) or subscales scores of anger (feelings, verbal, physical anger). Although STAXI-2 (Spielberger, 1999) is one of the most widely used anger measures in both clinical and research settings (Novaco & Taylor, 2004), and there is psychometric support for its use with different cultures, including Asian (Ghos & Sharma 2006) and Western populations (Lindqvist et al., 2003), in the present thesis it was noticed that participants were more inclined to self-report feelings of anger rather than verbal or physical anger. Such findings could indicate demand characteristics as to what it is considered a more socially acceptable behaviour. In Spielberger's original research (1999), state anger correlated negatively with social desirability (-.33) showing that there is a relationship between self-report of anger levels and perceived social norms. Future studies should advocate the need for a self-report of desirability questionnaire to test its influence on the reporting of

anger levels.

Other methodological limitations relate to the large array of apparatus used to measure BP and the cardiovascular indicators of SAM and PAC which may have led to a lack of consistency of significant findings. The analyses of the cardiac impedance measures employed different algorithms across studies (see Method section) which allow limited flexibility for comparisons across studies. The detection of significant points in the ICG wave (e.g., Q, B, X) and ECG (e.g., R-R interval) proved challenging in study 1 directing to the development of a new detection algorithm based on existing research (e.g., Sherwood et al., 1990; Lozano et al., 2007). It was observed that due to heavy movement artifact or respiration the points were not always well defined and, in some cases, were completely absent from the ICG signal. Therefore, the new method proposed allowed for correction on time band as well as correlations with the R peak. The C point was defined as the maximum point in the dz/dt signal in a time window 60–200 ms from the R peak (Gratze et al., 1998), the X point was defined as the minimum point over the course of the cycle after the C point whereas B was set as the maximum derivative of the dz/dt signal in a time window 150–100 ms before the C point. However, there was no control whether the B and X corresponded exactly to the opening and closing of the aortic valve. Studies (e.g., Shyu, Lin, Lu & Hu, 2004) using synchronised echocardiography with ICG suggests that the definitions of B and X points do not correlate well to the exact opening and closing instants of the aortic valve which could explain some discrepancies between impedance and cardiac measures. Moreover, the algorithm was only validated within the present thesis and future investigations are required in other research domains.

On a final note, movement artifacts were more difficult to control in a driving simulator that required steering of a wheel. Whereas the impedance measures or the EEG analysis allowed for motion artifact filtering via algorithm and BESA software respectively, the BP measure remained susceptible to movement artifacts. While the cuff was used in the

nondominant arm and the participants were asked to stay still any slight movement could have interrupted the air flow in the deflating cuff (Lim, Ng, Jassim, Redmond, Zilany, Avolio, Lim, Tan, & Lovell, 2015). Suggestions for improvement could be detecting noise in the blood pressure signals using additional sensing devices such as acceleration and capacitive sensors (Choi, Park & Lee, 2007); alternatively morphological comparison with good-quality reference pulses may be utilised (Charbonnier, Siche, & Vancura, 2000).

8.4. Future research

Anger in a driving scenario was provoked using simulated traffic jams within the context of a demand to complete the journey on a strict schedule and avoid a financial penalty. Anger was induced via exposure to short-term boredom/displeasure via traffic jams and delays in the driving task. There are several avenues for future research including: 1) assessment of the relative contribution of each factor (i.e., time schedule, outcome expectancies, traffic jams) to the cardiovascular manifestation of anger, 2) investigation of how anger influences psychophysiology in the presence of a reward rather than punishment since variations in monetary incentives could result in changes in anterior frontal alpha asymmetry(AF3/AF4) (Miller & Tomarken, 2001), and 3) confirmation of whether the present findings with the respect to impact of ambient interventions on anger generalise to other contexts of anger. In addition, studies 2-5 took place in a simulated environment that represented a simplification of the real driving environment. Hence, the ecological validity was reduced - especially when comparing with a modern driving environment where it is possible for drivers to distract themselves with music or mobile devices when caught in traffic. Also, a night drive on an unlit road was the closest naturalistic environment for the simulator. Hence, a big topic for further investigation for the ambient light intervention is how well it would work in naturalistic lighting conditions (bright sunlight, cloudy). Since data were extracted from wearable sensors, the cardiovascular responses could be assessed in traffic jams on real-road at different times of the day.

A future research direction would be a longitudinal study to allow for a classic

conditioning of ambient factors (e.g., music, light) as physiological regulator factors. The questions are whether the affective ambient intervention is time bias, whether users habituate to its effects in which case the stimulus may have to change. It could be purposed an interactive system that identifies physiological expressions of emotion and offers biofeedback in order to raise self-awareness as a strategy for reducing the experience of a negative emotion with beneficial consequences for health (Mauss et al., 2007). Given the association between anger and cardiovascular diseases such as hypertension (Everson et al., 1997; Kiecolt-Glaser et al., 2002; Stewart et al., 2008), there is a clear need to further understand the physiological components of this emotion. In such, the present findings indicate that negative emotions such as anger must be defined multi-dimensionally in relation to motivational contexts. Future studies should test for group means against zero to verify whether the pattern for the group is consistent with a challenge profile, threat profile, or neither (Mendes et al., 2001). Self-report measures of challenge/threat during that actual task should be considered in future work. An emotional model in any biocybernetic system that could be used for health monitoring should, therefore, include a measure of affect within motivational contexts. This model could be applied to a simple biofeedback (cue/relax) system that may prove beneficial in self-adapting BP. Although some reviews (i.e., Greenhalgh, Disckson, & Dundar, 2010) on the usefulness of biofeedback for hypertension are inconclusive, the results of the present thesis point to a possible benefit of a simple model of biofeedback (light cue/relaxation response) on temporary increases of BP.

The findings could have also implications for how the photometry of vehicle interior lighting should be arranged in order to improve driver's health in situations where negative emotional reactions, such as anger in a traffic jam, may increase cardiac rhythm to acute levels. Future research should further quantify the psychophysiological effects of an automotive interior lighting system and offer the possibility to self-regulate based on an on-board biofeedback-system. Testing of the suitability of the system in the bright sunlight is necessary as well as the various locations of the ambient light within the interior of the vehicle (e.g., dashboard, upper screen display

behind the wheel or pervasively).

8.5. Summary

The cardiovascular and EEG results of the present thesis point to a circumplex model of anger with quadruplet facets along cardiovascular responses to challenge/threat contexts in conjunction with approach/avoidance tendencies (Fig. 8.1) where right frontal activation and increased TPR define a state of challenge and increased CO defined a threat. A threat motivation with avoidance was indexed by increased SBP, DBP and CO and by greater right frontal activation. The difference in the approach-threat responses will be the activation of the left hemisphere. The challenge-avoidance state was defined by increased TPR, SBP, HR, MAP and greater right frontal hemisphere activation. No frontal asymmetric index was identified in the challenge-approach, but increased TPR, SBP, HR and MAP were observed. The ambient intervention results suggested that cardiovascular responses (SBP) could be reduced by low activation music or blue ambient light.

CHAPTER 9
CONCLUSIONS

According to the findings of the present thesis it can be concluded that increased BP during unachievable targets coupled with high levels of anger could be a warning sign of possible health hazard if negative experiences are sustained and that music and blue light appraised as a relaxation cue appeared to reduce the sympathetic response to anger, which may be a positive adaptation in terms of long-term health. For example, in Study 1, SBP and frontal peripheral brain site were indicators of anger in combination with the perception of a state of no control. Hence, further research is required to explore the specific contribution of motivational parameters within the anger state to cardiovascular reactivity and frontal brain asymmetry. The results of Study 2 demonstrated increased cardiovascular reactivity and reduced approach motivation in response to anger in the context of a simulated traffic jam. Further studies are needed to understand the connection between frontal EEG asymmetry and cardiovascular correlates of anger in order to create a broad framework for the analysis of anger in a motivational context. On the basis of Study 3, music may mediate the subjective experience of anger and alter the magnitude of the psychophysiological response to anger. We suggest that music serves two effects in the context of driving simulation: firstly, music acted as an overt source of distraction from negative events in the environment and, secondly, music exerted a subconscious effect on psychophysiology. People may use music to distract themselves from a monotonous situation (as in a traffic jam) and to divert themselves from negative thoughts and feelings. With respect to psychophysiology, categories of music with low levels of energy (regardless of valence) appeared to reduce the sympathetic response to anger, which may be a positive adaptation in terms of long-term health. The study has demonstrated that music can mediate the effects of anger in a simulated environment. The study demonstrated that low activation music, regardless of valence, was found to reduce systolic reactivity associated with a state of high-activation and negative valence albeit in a simulated driving environment. Further research is required to explore whether this finding can

be replicated in the field and in other domains of anger induction. Study 4 showed that red ambient light increased anger at a subjective level but the traffic jams increased physiological reactions on a subliminal level. Thus, a future investigation of the influence of colour on emotional behaviour using learned association would be appropriate. According to the findings in Study 5, sensors of BP, HR and facial expressions can detect the driver's psychophysiological state and provide feedback by means of ambient light when a negative emotional state reaches undesirably high levels. The results indicated that systolic BP could be attenuated by an ambient light illumination and by self-regulating using blue light as a facilitator.

The environmental cues served two effects in the context of driving simulation: firstly, they acted as an overt source of distraction from negative events in the environment (e.g., low activation music was used to distract during boredom of the traffic jam and stimulated in a way that reduced SBP) and, secondly, music and light exerted a subconscious effect on psychophysiology. However, when conscious appraisal is present, the effect is even more noticeable. People may use music to distract themselves from a monotonous situation (as in a traffic jam) and to divert themselves from negative thoughts and feelings. The intervention studies have demonstrated that music can mediate the effects of anger in a simulated environment and light only mediated the effect of anger when participants were actively using the light as cue for relaxation. The findings could be implemented in the development of Ambient Intelligence areas by offering a model of physiological reactions that can be measured and an indication of how ambient environment (i.e., music, light) could be used as a way of providing feedback to the user and as a counter measure. For example, the photometry of vehicle interior lighting could be arranged in order to allow the possibility to self-regulate based on an on-board biofeedback-systems that quantifies the physiological effects.

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APPENDICES

Appendix 1a – Advertising Campaign

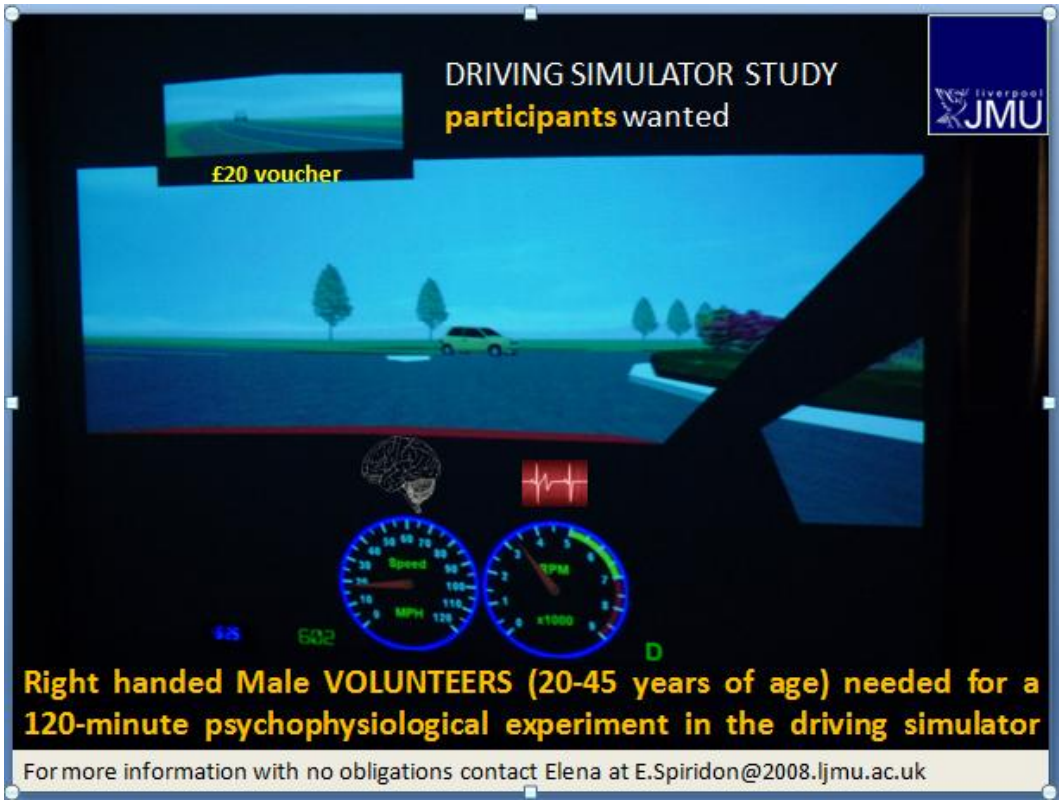

Fig. A1: Advertisement for the recruitment of participants.

Appendix 1b – Block email Used for the Recruitment of Participants

LIVERPOOL JOHN MOORES UNIVERSITY
School of Natural Sciences and Psychology

Dear all,

I am a PhD student cordially asking for **Volunteers** to take part in an investigation pertaining **driving simulation and psychophysiological reactions.**

The study involves a screening questionnaire (5 minutes) which will condition the invitation to the driving simulator laboratory. A pre-test (3 minutes) in the simulator will assess your general comfort in order to identify any potential signs of motion sickness. If you are found to be comfortable with the use of the simulator then a set of physiological measures will be taken while you perform two simulated drives (for approximately 120 minutes).

Nothing complicated
Completely anonymous
No skills required

Suitability: Must be male; right handed; 20-40 years of age; in good mental and physical health, especially no history of motion sickness.

Participants will be compensated with a £20 voucher for their time.

For further details with NO obligation e-mail:
E.Spiridon@2008.ljmu.ac.uk
Thank you,

Elena Spiridon

Fig. A2: Block email for the recruitment of participants.

Appendix 2 – Screening for Health

LIVERPOOL JOHN MOORES UNIVERSITY
SCREENING FORM

Title of Project: *Individual psychophysiological differences during a simulated drive*

Name of Researcher and School/Faculty: *ELENA SPIRIDON*
School of Natural Sciences and Psychology
Faculty of Science

Please read the following questions and highlight the answer that best applies to you:

(3)Are you left handed? YES NO

(4)Do you suffer or have you suffered from depression or anxiety in the last 5 years? YES NO

(5)Do you have high blood pressure? YES NO

(6)Are you currently taking prescribed medication? YES NO

(7)Have you ever suffered from a neurological condition or head trauma? YES NO

(8)Do you have any history of psychiatric illness? YES NO

(9)Do you usually experience motion sickness when travelling? YES NO

(10)Do you have a heart abnormality / heart disease / wear a pacemaker? YES NO

(11)Are you aged over 45? YES NO

If you answered YES to any of the above questions please accept my apologies for not being able to accept your kind offer to take part in the study.

If you answered NO to all of the questions, you'll be invited to the Driving Simulator Laboratory where the experiment will take place.

Thank you!

_____	_____	_____
Name of Participant	Date	Signature
_____	_____	_____
Name of Researcher	Date	Signature

Appendix 3 – Example of Information Sheet for Participants

LIVERPOOL JOHN MOORES UNIVERSITY
PARTICIPANT INFORMATION SHEET 1

Title of Project: *Individual psychophysiological differences during a simulated drive*

Name of Researcher and School/Faculty:
ELENA SPIRIDON
School of Natural Sciences and Psychology
Faculty of Science

You are cordially invited to take part in an approximately 120-minute study carried out in the driving simulator at LJMU. Before you decide to take part it is important that you understand why the research is being done and what it involves. Please take your time to read the following information. Ask if there is anything that is not clear or if you would like more information. Take your time to decide if you want to take part or not. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

1. What is the purpose of the study?
This project is being undertaken as part of the requirements for a post-graduate degree and it aims to obtain an accurate measure of physiological reactions during the performance of a simulated drive.

2. Do I have to take part?
No. It is up to you to decide whether or not to take part. If you do, you will be given this information sheet and asked to sign a consent form. You are still free to withdraw at any time and without giving a reason. A decision to withdraw will not affect the payment of the £20 voucher.

3. What will happen to me if I take part?

	Time (min)	*PROCEDURE*
Introduction	*5*	*You will be reading the information sheet and consent form and signing if you agree to take part*
Screening protocol	*2*	*You will receive a set of questionnaires that will condition the participation in the laboratory experiment*
Personality questionnaires	*5*	*You will be completing 2 questionnaires as quickly as possible*
Familiarisation	*3*	*On the day of the experiment, you will have a chance to tell us how you feel after a short 3-minute practice drive in the simulator; if you do not feel well, we'll stop the experiment and thank you for your participation*
Cardiovascular equipment (e.g., heart rate monitor)	*5*	*Cardiovascular equipment will be attached to your body*
Electrodes attachment to the scalp	*30*	• *an allergy test of the gel used for the electrodes will be carried out* • *a cap will be placed over your scalp* • *gel will be poured through the 32 holes of the cap* • *electrodes will be attached to the cap*
External electrodes and sensors	*15*	*8 external electrodes will be placed around the eyes and on the ear lobes; sensors will be attached to the fingers of the non-dominant hand.*
INSTRUCTIONS	*5*	• *you are kindly asked not to move at all during the task* • *you need to complete the task on the simulator* • *you will receive precise instructions for the task*
Practice trial	*5*	*You will have a chance to practice using the simulator*
TEST	*2x 12*	*You will be performing 2 simulated drives and answer a set of pre- and post-test questionnaires*
Debriefing	*10*	*You will be explained what the experiment was about and what results are expected.*

4. Are there any risks / benefits involved?

Risks

- You might find it restricting the fact that you have to stay still, but it is for only a few minutes and you'll get breaks in between trials.
- You might develop an allergy from the gel we use; however, an allergy test will be performed in advance and it is very unlikely that anyone develops any reactions since the gel is highly hypoallergenic.
- You might experience simulator sickness (a form of motion sickness) but a prior short drive will ensure you are comfortable with the simulator.
- You might experience emotional reactions but they won't exceed the normal emotions you feel on a daily basis.

Benefits

- You will get an insight into psychological research as you'll be fully debriefed at the end of the experiment.
- You are contributing to research in the field.

5. Will my taking part in the study be kept confidential?

The results of this project may be published but you will be only identified by a number, not your name. The data collected will be securely stored in such a way that only the researchers below will be able to gain access to them. At the end of the project any personal information will be destroyed immediately except that, as required by the University's research policy, any raw data on which the results of the project depend will be retained in secure storage for five years, after which it will be destroyed. You are most welcome to request a copy of the results of the project should you wish.

Contact Details of the Researchers

If you have any questions about our project, either now or in the future, please feel free to contact either:

Elena Spiridon –PhD student or Dr. *Stephen Fairclough - Supervisor*

Tom Reilly Building
School of Natural Sciences and Psychology
Liverpool John Moores University
Byrom Street
Liverpool
L3 3AF

E.Spiridon@2008.ljmu.ac.uk *S.Fairclough@ljmu.ac.uk*

Appendix 4 - Consent Form

LIVERPOOL JOHN MOORES UNIVERSITY
CONSENT FORM

Title of Project: *Individual psychophysiological differences during a simulated drive*

Name of Researcher and School/Faculty:
ELENA SPIRIDON
School of Natural Sciences and Psychology
Faculty of Science

1. I confirm that I understood the information provided for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving a reason and that this will not affect my legal rights. ☐
3. I understand that any personal information collected during the study will be anonymised and remain confidential. ☐
4. I agree to take part in the above study. ☐

Name of Participant	Date	Signature
_____	_____	_____
Name of Researcher	Date	Signature
_____	_____	_____
Name of Person taking consent *(if different from researcher)*	Date	Signature
_____	_____	_____

Appendix 5 – Example of Experiment Debriefing

LIVERPOOL JOHN MOORES UNIVERSITY DEBRIFING SHEET

Version 4

Title of Project: *Individual psychophysiological differences during a simulated drive*

Name of Researcher and School/Faculty:
ELENA SPIRIDON
School of Natural Sciences and Psychology
Faculty of Science

Thank you for taking part in the study. It is our duty to fully inform you as to the real nature of the experiment. We are investigating the natural biological reactions to an anger situation when the individual has control compared to when the individual has no control over the situation.

Anger was manipulated during the second drive by pre-test instructions to reach the destination without any driving errors in a set time and with the encouragement of a double payment if the journey was completed in time. Please let us reassure you that regardless of the driving performance you will receive your payment.

Throughout the drive you encountered sections of difficult driving conditions. Half of these situations allowed you to continue driving (you had control of the situation) and in the other half there was nothing you could have done to continue the drive in order to reach your destination in time (you did not have control of the situation). We are hoping to find that in the anger/no control situation the participants will display stronger biological reactions than in anger/control condition. The reason for investigating 'anger in context' is that, in real life, when we feel angry our bodies respond in an unhealthy manner without us being aware of any biological changes. Repetitively feeling angry may have serious consequences for our health. Also, in a driving context

feelings of anger in association with a feeling of uselessness could jeopardize the safety of the road users. In order to test whether music reduces feelings of anger, you listen to the song that you rated as the most relaxing.

If you would like more details regarding the results of the experiment or you decide to withdraw your data, please do not hesitate to contact either:

Elena Spiridon – PhD student or Dr. *Stephen Fairclough - Supervisor*

Tom Reilly Building
School of Natural Sciences and Psychology
Liverpool John Moores University
Byrom Street
Liverpool
L3 3AF

E.Spiridon@2008.ljmu.ac.uk *S.Fairclough@ljmu.ac.uk*

Thank you!

Appendix 6 – Tables of the Principal Research Findings

Table A1: Summary of the principal research findings of this thesis.

					Groups					
Study	**Variable**	**Groups**	**Interventions**	**Baseline**	**Challenge (Control)**	**Threat (No control)**	**Anger / control (challenge)**	**Anger / no control (threat)**	**Theory for Challenge**	**Theory for Threat**
1 Stroop number test	SBP	Control No control Anger / control Anger / No control	Challenge (control) Threat (no control) Anger / control Anger / No control Challenge: Control of keyboard Threat: No control keyboard	Yes				Highest ↑ relative to baseline (Significant) **"Support for threat theory"**	↓ SBP	↑ SBP **"Support for theory"** Note: theory based on threat only, does not include anger
1 Stroop number test	DBP	Control No control Anger / control Anger / No control	Challenge (control) Threat (no control) Anger / control Anger / No control Challenge: Control of keyboard Threat: No control keyboard	Yes		↑ increase relative to baseline (Significant) **"Support for threat theory"**	↑ increase relative to baseline (Significant) **"No support for challenge theory"**	↑ increase relative to baseline (Significant) **"Support for threat theory"**	↓ DBP **"No support for theory"**	↑ DBP **"Support for theory"** Note: theory based on threat only, does not include anger

Table A1: Summary of the principal research findings of this thesis (cont.).

Study	Variable	Baseline	Groups		Hypothesis for Challenge	Hypothesis for Threat
			TJ1 Anger / control (challenge)	TJ2 Anger / no control (threat)		
2 Driving simulator (repeated measures)	SBP	Yes	↑ Increase relative to baseline (Significant) **"No support for challenge hypothesis"**	↑ Increase relative to baseline (Significant) **"Support for threat hypothesis"**	↓ SBP **"No support for hypothesis"**	↑ SBP **"Support for hypothesis"**
2 Driving simulator (repeated measures)	DBP	Yes	↑ Increase relative to baseline (Significant) **"No support for challenge hypothesis"**	↑ Increase relative to baseline (Significant) **"Support for threat hypothesis"**	↓ DBP **"No support for hypothesis"**	↑ DBP **"Support for hypothesis"**
2 Driving simulator (repeated measures)	HR	Yes	↑ Increase relative to baseline (Significant) **"Support for threat hypothesis"**	↑ Increase relative to baseline (Significant) **"Support for threat hypothesis"**	↑ HR **"Support for hypothesis"**	↑ HR (less increase) **"Support for hypothesis"**
2 Driving simulator (repeated measures)	TPR	Yes	↑ Increase relative to baseline (Significant) **"No support for challenge hypothesis"**		↓ TPR **"No support for hypothesis"**	↑ TPR

Table A1: Summary of the principal research findings of this thesis (cont.).

				Conditions					
Study	**Variable**	**Groups**	**Group Comparisons**	**Baseline**	**Mood Induction**	**TJ1 Anger / control (challenge)**	**TJ2 Anger / no control (threat)**	**Hypothesis for Challenge**	**Hypothesis for Threat**
3 Driving simulator (between groups)	SBP	LA/PV HA/PV LA/NV HA/NV No music	↓ LA/PV ↓ LA/NV (compared to HA/NV and no music groups) (Significant)	All data baselined		↑ Increase relative to mood induction (Significant) **"No support for challenge hypothesis"**	↓ Decrease relative to TJ1 (Significant) **"No support for threat hypothesis"**	↓ SBP **"No support for hypothesis"**	↑ SBP **"No support for hypothesis"**
3 Driving simulator (between groups)	DBP	LA/PV HA/PV LA/NV HA/NV No music	↓ LA/PV ↓ LA/NV (compared to HA/NV and no music groups) (Significant)	All data baselined		↑ Increase relative to mood induction (Significant) **"No support for challenge hypothesis"**	↓ Decrease relative to TJ1 (Significant) **"No support for threat hypothesis"**	↓ DBP **"No support for hypothesis"**	↑ DBP **"No support for hypothesis"**
3 Driving simulator (between groups)	HR	LA/PV HA/PV LA/NV HA/NV No music		All data baselined		↑ Increase relative to mood induction (Significant) **"No support for challenge hypothesis"**		↓ HR **"No support for hypothesis"**	
3 Driving simulator (between groups)	PEP	LA/PV HA/PV LA/NV HA/NV No music		All data baselined		↓ Decrease relative to mood induction (Significant) **"Support for challenge hypothesis"**	↓ Decreased but less than TJ1 relative to mood induction (Significant) **"No support for threat hypothesis"**	↓ PEP **"Support for hypothesis"**	↑ PEP **"No support for hypothesis"**

Table A1: Summary of the principal research findings of this thesis (cont.).

3 Driving simulator (between groups)	CO	LA/PV HA/PV LA/NV HA/NV No music	↓ LA/PV (compared to HA/NV in TJ1) (Significant)	All data baselined	↓ Decrease relative to mood induction (Significant) **"No support for challenge hypothesis"**	↑ CO **"No support for hypothesis"**	↑ CO (less increase)
3 Driving simulator (between groups)	TPR	LA/PV HA/PV LA/NV HA/NV No music	↓ HA/NV (compared to HA/PV in TJ1) (Significant)	All data baselined		↓ TPR **"Support for hypothesis"**	↑ TPR

Table A1: Summary of the principal research findings of this thesis (cont.).

				Conditions					
Study	**Variable**	**Groups**	**Group Comparisons**	**Baseline**	**Mood Induction**	**TJ1 Anger / control (challenge)**	**TJ2 Anger / no control (threat)**	**Hypothesis for Challenge**	**Hypothesis for Threat**
4 Driving simulator (between groups)	SBP	Red Blue White No light		All data baselined		↑ SBP at TJ1 (compared to induction (Significant) **"No support for hypothesis"**		↓ SBP **"No support for hypothesis"**	↑ SBP
4 Driving simulator (between groups)	CO	Red Blue White No light		All data baselined			↑ CO at TJ2 (compared to induction (Significant) **"Support for hypothesis"**	↑ CO	↑ CO (less increase) **"Support for hypothesis"**
4 Driving simulator (between groups)	HR	Red Blue White No light		All data baselined		HR ↑ more at TJ1 (compared to induction (Significant) **"Support for hypothesis"**	HR ↑ less at TJ2 (compared to induction (Significant) **"Support for hypothesis"**	↑ HR **"Support for hypothesis"**	↑ HR (less increase) **"Support for hypothesis"**
4 Driving simulator (between groups)	MAP	Red Blue White No light		All data baselined		MAP ↑ more at TJ1 (compared to induction (Significant) **"Support for hypothesis"**	MAP ↑ less at TJ2 (compared to induction (Significant) **"Support for hypothesis"**	↑ MAP **"Support for hypothesis"**	↑ MAP (less increase) **"Support for hypothesis"**

Table A1: Summary of the principal research findings of this thesis (cont.).

				Conditions					
Study	**Variable**	**Groups**	**Group Comparisons**	**Baseline**	**Mood Induction**	**TJ1 Anger / control (challenge)**	**TJ2 Anger / no control (threat)**	**Hypothesis for Challenge**	**Hypothesis for Threat**
5 Driving simulator (between groups)	SBP	BL1/Feedback BL2/Feedback No light	↑ No light (compared to BL1 & BL2)	All data baselined				↓ SBP	↑ SBP
5 Driving simulator (between groups)	DBP	BL1/Feedback BL2/Feedback No light	↑ No light (compared to BL1 & BL2)	All data baselined		↑ DBP (compared to induction (Significant) **"No support for hypothesis"**		↓ DBP **"No support for hypothesis"**	↑ DBP
5 Driving simulator (between groups)	HR	BL1/Feedback BL2/Feedback No light		All data baselined		HR ↑ more at TJ1 (compared to induction (Significant) **"Support for hypothesis"**	HR ↑ less at TJ2 (compared to induction (Significant) **"Support for hypothesis"**	↑ HR **"Support for hypothesis"**	↑ HR (less increase) **"Support for hypothesis"**
5 Driving simulator (between groups)	SV	BL1/Feedback BL2/Feedback No light	↑ No light (compared to BL2)	All data baselined				↑ SV	↑ SV (less increase)

Appendix 7 – Additional Descriptive and Inferential Statistical Findings for Study 3

Table A2: Summary of the descriptive statistics for Study 3

	PL	PE	NL	NO	NE
			SBP (mmg/Hg)		
Induction - Baseline					
Mean [SE]	.82 [.99]	1.23 [1.23]	-.42 [1.04]	3.32 [1.17]	4.18 [1.09]
TJ1 - Baseline					
Mean [SE]	2.33 [1.49]	6.88 [2.37]	3.13 [2.68]	9.43 [2.32]	9.30 [2.17]
TJ2 - Baseline					
Mean [SE]	-.61 [1.56]	-.83 [2.35]	-1.88 [2.04]	5.93 [2.25]	4.30 [1.35]
			DBP (mmg/Hg)		
Induction - Baseline					
Mean [SE]	.82 [.99]	1.23 [1.23]	-.42 [1.04]	3.32 [1.17]	4.18 [1.09]
TJ1 - Baseline					
Mean [SE]	2.33 [1.49]	6.88 [2.37]	3.13 [2.68]	9.43 [2.32]	9.30 [2.17]
TJ2 - Baseline					
Mean [SE]	-.61 [1.56]	-.83 [2.35]	-1.88 [2.04]	5.93 [2.25]	4.30 [1.35]
			MAP (mmg/Hg)		
Induction - Baseline					
Mean [SE]	1.05 [.68]	.96 [.93]	-.27 [.89]	1.55 [.61]	1.98 [.87]
TJ1 - Baseline					
Mean [SE]	3.50 [1.20]	4.53 [1.90]	4.22 [1.97]	5.36 [1.60]	6.60 [1.97]
TJ2 - Baseline					
Mean [SE]	1.15 [1.41]	.66 [1.33]	-1.01 [1.45]	3.13 [1.71]	3.20 [1.35]
N	18	20	20	20	20

Table A2: Summary of the descriptive statistics for Study 3 (cont.).

	PL	PE	NL	NO	NE
			PEP (ms)		
Induction - Baseline					
Mean [SE]	.0022 [.0046]	.0014 [.0019]	.0066 [.0068]	.0033 [.0022]	-.0040 [.0061]
N	11	12	9	9	12
TJ1 - Baseline					
Mean [SE]	-.0145 [.0032]	-.0090 [.0030]	-.0160 [.0062]	-.0151 [.0058]	-.0224 [.0077]
N	8	8	7	7	9
TJ2 - Baseline					
Mean [SE]	-.0130 [.0031]	-.0064 [.0022]	-.0079 [.0046]	-.0123 [.0049]	-.0142 [.0071]
N	8	8	7	7	9
			HR (bpm)		
Induction - Baseline					
Mean [SE]	-.327 [1.274]	.308 [.792]	-3.556 [2.937]	.433 [.780]	1.850 [.826]
N	11	12	9	9	12
TJ1 - Baseline					
Mean [SE]	-2.990 [5.302]	7.675 [3.322]	7.157 [2.502]	4.114 [5.814]	6.633 [3.584]
N	8	8	7	7	9
TJ2 - Baseline					
Mean [SE]	-.063 [4.408]	3.478 [1.240]	5.486 [1.047]	.971 [4.974]	2.911 [3.486]
N	8	8	7	7	9

Table A2: Summary of the descriptive statistics for Study 3 (cont.).

	PL	PE	NL	NO	NE
			CO (L/min)		
Induction - Baseline					
Mean [SE]	.555 [.424]	-.025 [.634]	2.722 [4.247]	-.167 [.263]	.392 [.410]
N	9	12	9	9	12
TJ1 - Baseline					
Mean [SE]	.029 [1.021]	.088 [.364]	.353 [2.939]	-.814 [1.009]	1.222 [1.343]
N	7	8	7	7	9
TJ2 - Baseline					
Mean [SE]	.129 [1.185]	-.713 [.250]	2.100 [1.945]	-1.400 [.788]	.200 [1.046]
N	7	8	7	7	9
			SV (ml)		
Induction - Baseline					
Mean [SE]	6.89 [5.63]	1.58 [8.57]	25.56 [39.64]	-2.29 [3.11]	2.45 [5.30]
N	9	12	9	9	11
TJ1 - Baseline					
Mean [SE]	2.44 [10.16]	-4.66 [3.79]	30.97 [26.81]	-11.57 [8.22]	9.49 [13.24]
N	7	8	7	7	9
TJ2 - Baseline					
Mean [SE]	1.74 [13.61]	-11.99 [3.42]	17.39 [19.13]	-16.94 [7.63]	.52 [10.92]
N	7	8	7	7	9

Table A2: Summary of the descriptive statistics for Study 3 (cont.).

	PL	PE	NL	NO	NE
			LVET (ms)		
Induction - Baseline					
Mean [SE]	-.0125 [.0061]	-.0012 [.0025]	.0010 [.0060]	-.0090 [.0057]	.0011 [.0041]
N	11	12	9	9	12
TJ1 - Baseline					
Mean [SE]	-.0159 [.0168]	.0091 [.0036]	-.0086 [.0143]	-.0130 [.0156]	.0028 [.0076]
N	8	8	7	7	9
TJ2 - Baseline					
Mean [SE]	-.0128 [.0141]	.0056 [.0036]	-.0083 [.0014]	-.0160 [.0111]	-.0022 [.0077]
N	8	8	7	7	9
			TPR (dynes * cm^{-5})		
Induction - Baseline					
Mean [SE]	-74.13 [75.27]	-158.66 [345.55]	81.64 [194.10]	64.74 [85.68]	-240.03 [281.28]
N	8	12	9	9	12
TJ1 - Baseline					
Mean [SE]	290.94 [276.01]	74.55 [151.37]	-17.64 [212.68]	176.64 [290.80]	-311.98 [293.52]
N	7	8	7	7	9
TJ2 - Baseline					
Mean [SE]	293.00 [253.96]	278.81 [81.77]	52.24]219.83]	400.00 [279.61]	-166.54 [310.13]
N	7	8	7	7	9

Table A3: Summary of the inferential statistics for Study 3

Dependent variable	Independent variables	*F*	df	*p*	η^2	Power
	Drive Stages	18.320	2, 186	0.001*	0.165	1.00
	Mood	3.970	4, 93	0.005*	0.146	0.89
SBP (mm/Hg)	Drive stages * Mood	0.886	8, 186	0.529	0.037	0.41
	post-hoc					
	Induction vs. TJ1			0.001*		
	TJ1 vs. TJ2			0.001*		
DBP (mm/Hg)	Drive Stages	9.285	2, 186	0.001*	0.091	0.98
	Mood	0.257	4, 93	0.905	0.011	0.10
	Drive stages * Mood	0.390	8, 186	0.925	0.017	0.18
	post-hoc					
	Induction vs. TJ1			0.001*		
	TJ1 vs. TJ2			0.009*		
HR (bpm)	Drive Stages	5.750	2, 62	0.005*	0.156	0.85
	Mood	1.011	4, 31	0.417	0.115	0.28
	Drive stages * Mood	1.362	8, 62	0.231	0.149	0.57
	post-hoc					
	Induction vs. TJ1			0.036*		
MAP (mm/Hg)	Drive Stages	17.56	2, 186	0.001*	0.159	1.00
	Mood	1.234	4, 93	0.302	0.050	0.37
	Drive stages * Mood	0.436	8, 186	0.898	0.018	0.20
	post-hoc					
	Induction vs. TJ1			0.001*		
	TJ1 vs. TJ2			0.001*		
PEP (ms)	Drive Stages	32.04	2, 62	0.001*	0.508	1.00
	Mood	0.707	4, 31	0.593	0.084	0.20
	Drive stages * Mood	0.677	8, 62	0.709	0.080	0.29
	post-hoc					
	Induction vs. TJ1			0.001*		
	Induction vs. TJ2			0.001*		
	TJ1 vs. TJ2			0.001*		

*** significant**

Table A3: Summary of the inferential statistics for Study 3 (cont.).

Dependent variable	Independent variables	*F*	df	*p*	η^2	Power
CO (L/min)	Drive Stages	0.355	2, 60	0.703	0.012	0.1
	Mood	1.658	4, 30	0.186	0.181	0.45
	Drive stages * Mood	0.127	8, 60	0.998	0.017	0.08
	post-hoc					
	Induction vs. TJ1			0.013*		
SV (ml)	Drive Stages	0.471	2, 60	0.627	0.015	0.12
	Mood	1.751	4, 30	0.165	0.189	0.47
	Drive stages * Mood	0.135	8, 60	0.997	0.018	0.09
	post-hoc					
	TJ1 vs. TJ2			0.010*		
LVET (ms)	Drive Stages	1.680	2, 62	0.195	0.051	0.34
	Mood	1.816	4, 31	0.151	0.190	0.49
	Drive stages * Mood	0.976	8, 62	0.463	0.112	0.410
TPR (dynes * cm^{-5})	Drive Stages	0.939	2, 60	0.397	0.030	0.21
	Mood	0.944	4, 30	0.452	0.112	0.26
	Drive stages * Mood	0.936	8, 60	0.494	0.111	0.39

*** significant**

Appendix 8 – Cardiovascular Measures Including Baseline Condition for Study 5

Table A4: Mean and SE of cardiovascular measures including baseline condition for Study 5

		Baseline	Light	TJ1	TJ2
SBP	BL1	112.1 [2.9]	110.6 [2.6]	112.5 [3.2]	109.5 [2.2]
	BL2	109.1 [3.3]	110.9 [3.1]	115.1 [4.0]	114.2 [3.0]
	Control	102.4 [3.7]	107.8 [2.4]	112.4 [2.7]	110.7 [2.8]
DBP	BL1	75.7 [1.5]	75.9 [1.9]	77.2 [2.5]	77.1 [2.1]
	BL2	73.0 [2.1]	74.2 [2.1]	74.1 [2.2]	69.9 [1.8]
	Control	73.8 [2.6]	73.2 [2.1]	78.8 [2.4]	76.4 [1.6]
CO	BL1	9.1 [0.8]	8.6 [0.7]	9.8 [1.0]	9.6 [0.7]
	BL2	8.2 [1.3]	9.2 [1.4]	7.7 [1.3]	7.2 [1.0]
	Control	6.3 [0.8]	5.6 [0.7]	6.9 [0.9]	6.9 [0.8]
MAP	BL1	83.8 [2.6]	82.3 [2.0]	85.0 [2.3]	82.9 [1.6]
	BL2	79.8 [2.6]	81.3 [2.8]	81.7 [2.8]	79.8 [2.7]
	Control	78.9 [2.1]	79.1 [1.9]	83.9 [2.7]	81.7 [1.7]
HR	BL1	78.1 [4.2]	78.8 [3.5]	80.5 [3.6]	79.7 [3.4]
	BL2	66.6 [3.0]	68.7 [2.6]	72.0 [2.8]	70.3 [2.9]
	Control	70.3 [4.6]	73.3 [3.9]	78.6 [3.9]	75.5 [3.8]

Appendix 9 – List of Publications Derived from the Present Research

2014

Fairclough, S.H., van der Zwaag, M., **Spiridon, E.,** & Westerink, J. (2014). Effects of mood induction via music on cardiovascular measures of negative emotion during simulated driving. *Physiology and Behavior, 129*, 173–180.

2012

Fairclough, S., & **Spiridon**, **E.** (2012). Cardiovascular and Electrocortical Markers of Anger and Motivation during a Simulated Driving Task. *International Journal of Psychophysiology*, *84*, 188-193.

2011

van der Zwaag, M.D., Fairclough, S.H., **Spiridon, E**., & Westerlink, J. (2011). The impact of music on affect during anger inducing drives. *Affective Computing and Intelligent Interaction Lecture Notes in Computer Science, 6974*, 407-416.

2010

Spiridon, E.. & Fairclough, S.H. (2010). Anger in the motivational context of states of control and no control. *International Journal of Psychophysiology*, *77*, 269-270.

2009

Spiridon, E., & Fairclough, S.H. (2009). Detection of anger with and without control for Affective Computing systems. *International Conference on Affective Computing and Intelligent Interaction*, 21st - 24th October, Amsterdam, Netherlands.

Spiridon, E., & Fairclough, S.H. (2009). Cardiovascular and EEG indicators of anger and control/no control states. *Psychophysiology*, *46*(S1), S54.

Appendix 10 – Illustration of Factors Investigated in the Thesis

Fig. A10.1: Investigated emotion. Adapted from Kreibig (2010)

Fig. A10.2: Cardiovascular measures. Adapted from Kreibig (2010)