Electrophysiological indices of the Violence Inhibition Mechanism and their associations with physical aggression, callous-unemotional traits, and dietary omega-3

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Abstract

Each year, aggressive behaviour contributes to a substantial number of criminal offenses - resulting in severe personal, social, and financial ramifications. As such, there is importance for understanding the underlying mechanisms of aggression and how it might best be managed. The violence inhibition mechanism, thought to be dysfunctional in individuals characterised by aggressive behaviour and associated callous-unemotional traits, is used to explain how the perception of facial distress might inhibit an ongoing aggressive act. Despite a sizeable literature characterising the violence inhibition mechanism, to date, empirical research has overlooked this investigation on an electrophysiological level.

This thesis developed a novel facial-affect stopping task in order to tease apart the distinct stages of face processing and distress-induced motor extinction using electroencephalography. Results suggested that whilst callous-unemotional traits, specifically uncaring traits, were associated with electrophysiological indices of structural/featural face processing (N170, P200), aggressive traits, specifically physical aggression, were associated with electrophysiological indices of distress-induced motor extinction (stop-N200, stop-P300). Furthermore, in light of a growing literature suggesting a benefit of omega-3 dietary intake for both aggressive and callous-unemotional traits, correlational analysis suggested an association between omega-3 and physical aggression/distress-induced motor extinction, but not callous-unemotional traits/face processing.

These results have theoretical implications for understanding and investigating the violence inhibition mechanism on an electrophysiological level, as well as practical utility for better understanding how omega-3 might benefit aggressive traits and motor extinction. Specifically, the importance of distinguishing between both [1] aggressive and callous-unemotional traits and [2] face processing and motor extinction ability, when investigating the violence inhibition mechanism.

Ethical Approval

The Nottingham Trent University College Research Ethics Committee granted ethical approval for this thesis:

No. 2012/01 – 9th January 2012

- Modeling central inhibitory mechanisms in relation to aggression and impulsivity

No. 2012/09 – 23rd February 2012

- Developing a correlation perspective of Omega-3 intake, Aggression, Depression and markers of prenatal testosterone

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- Effect of Omega-3 supplementation on neuro-correlates: A mechanism study

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List of Abbreviations

ADJAR	-	Adjacent response technique
AHR	-	Adrenocorticotropic hormone release
AQ	-	Aggression Questionnaire
AUDIT	-	Alcohol Use Disorder Identification Test
ALA	-	Alpha-linoleic acid
ANOVA	-	Analyses of variance
ACC	-	Anterior cingulate cortex
ASPD	-	Antisocial personality disorder
APSD	-	Antisocial Process Screening Device
AA	-	Arachidonic acid
ADHD	-	Attention deficit hyperactivity disorder
BPD	-	Borderline personality disorder
BPAQ	-	Buss and Perry Aggression Questionnaire
CD	-	Conduct disorder
CU	-	Callous-unemotional
CUDIT	-	Cannabis Use Disorder Identification Test
CRFs	-	Corticotropin-releasing factors
DHA	-	Docosahexaenoic acid
dACC	-	Dorsal anterior cingulate cortex
dlPFC	-	Dorsolateral prefrontal cortex
dmPFC	-	Dorsomedial prefrontal cortex
EPA	-	Eicosapentaenoic acid
EEG	-	Electroencephalography
EFAs	-	Essential fatty acids
ERP	-	Event-related potentials
FAST	-	Facial Affect Stop Task
FFQ	-	Food Frequency Questionnaire
(f)MRI	-	(Functional) magnetic resonance imaging
GAM	-	General aggression model
HRM	-	Horse race model
HPA-axis	-	Hypothalamus, pituitary gland, and adrenal axis
IFG	-	Inferior frontal gyrus
IES	-	Integrated emotion system
ISI	-	Inter-stimulus intervals
IED	-	Intermittent explosive disorder
ICU	-	Inventory of Callous–Unemotional Traits
lHyp	-	Lateral hypothalamus
LA	-	Linoleic acid
LCPUFA	-	Long-chain polyunsaturated fatty acids
MEG	-	Magnetoencephalography
MAPPA	-	Multi-Agency Public Protection Arrangement
OFA	-	Occipital face area
OFC	-	Orbitofrontal cortices

PAG	-	Periaqueductal gray
PUFAs	-	Polyunsaturated fatty acids
Pre-SMA	-	Pre-supplementary motor area
PFC	-	Prefrontal cortex
PPI–R	-	Psychopathic Personality Inventory - Revised
PCL-R	-	Psychopathy Checklist - Revised
RNA	-	Ribonucleic acid
SNR	-	Signal to noise ratio
SCP	-	Social-cognitive processing model
SST	-	Stop-signal task
STG	-	Superior temporal gyrus
STS	-	Superior temporal sulcus
vACC	-	Ventral anterior cingulate cortex
vmHyp	-	Ventromedial hypothalamus
vmPFC	-	Ventromedial prefrontal cortex
VPP	-	Vertex positive potential
VIM	-	Violence inhibition mechanism

Foreword

Aggression is a natural construct adaptive to the requirements of social context (Nelson & Trainor, 2007; Rosel & Siever, 2015). Under certain circumstances, aggression provides a means of successfully negotiating threat, protecting resources, and acquiring mates. However, persistent and extreme aggression can be detrimental to oneself and problematic for wider society (Anderson & Bushman, 2001; Buydens-Branchey & Branchey, 2008; Nelson & Trainor, 2007).

Each year, an estimated 1.3 million people die as a result of violence (World Health Organisation, 2014), a term used to denote extreme, physical aggression (Anderson & Bushman, 2002; Reidy, Shelley-Tremblay, & Lilienfeld, 2011). In England, though lower than its peak in 1995, aggression was associated with 634,462 criminal offences between 2013 and 2014 (Office for National Statistics: Crime Statistics, 2014). Clearly, there is need to better understand the underlying mechanisms contributing to aggression, and how they are best managed.

One such mechanism, the violence inhibition mechanism (VIM; Blair, 1995), suggests that humans are typically able to extinguish antisocial and aggressive behaviour following the perception of facial distress in others. Thought to be a function of empathy, the VIM appears dysfunctional in psychopathic individuals, characterised by aggressive and callous-unemotional (CU) traits (Blair, 2001; Hare, 1991). Furthermore, due to the growing interest in using diet, specifically omega-3, as a moderator of aggressive behaviour (Buydens-Branchey & Branchey, 2008), there is reason to expect omega-3 intake might moderate the VIM response. For the first time, this thesis explores the VIM in relation to physical aggression, CU traits, and omega-3 intake.

Chapter one delineates motivations underpinning, and manifestations of, aggressive behaviour, before outlining key evolutionary and cognitive theories of aggression. In this chapter, aggression is framed around the core stages of the VIM (i.e. face processing and distress-induced motor extinction), with supporting

knowledge drawn from forensic, clinical, and community samples characterised by aggressive and CU traits, in which the VIM is considered dysfunctional. Chapter two focuses discussion on reactive aggression - expanding on the cognitive models outlined in chapter one - using evidence obtained through neuroimaging (e.g. and lesion studies). Here, neurobiological underpinnings of reactive aggression, distress perception, and motor extinction are delineated.

Chapter three explains how the neurobiological mechanisms described in chapter two might be indexed via temporally accurate electrophysiological measurements, and suggests how they might manifest in samples characterised by aggressive and CU traits. Chapter four draws these multidisciplinary themes together and suggests a role of diet, particularly omega-3 fatty acids, in the moderation of physical aggression, CU traits, and distress-induced motor extinction. Furthermore, primary and secondary research aims are delineated in chapter five, before outlining/justifying the key research methods used throughout the thesis.

Six experimental chapters follow. Chapter six identifies baseline associations between physical aggression and CU traits in a large community cohort. Chapter seven documents the development of the Facial Affect Stop Task (FAST) used to investigate how distinct face processing and motor extinction stages of the VIM might be indexed on an electrophysiological level. Chapters eight and nine correlate physical aggression and CU traits with FAST-evoked ERPs and chapters ten and eleven examine these ERP/psychometric associations in the context of omega-3 dietary intake. Specifically, chapter ten explores baseline correlations between omega-3 intake and self-reported measures of physical aggression and CU traits, and chapter eleven explores associations between omega-3 intake and the electrophysiological indices of the FAST.

Finally, chapter twelve draws together the experimental findings by discussing their theoretical and practical utility, and how said results might provide a baseline for future investigation. Subsequently, task-specific and global research limitations are addressed before summarising the novel contributions of the thesis.

"Let's sit down and have a discussion about this queer animal," the second blind man said, breaking the silence.

"A very good idea. Very good." the other two agreed for they also had this in mind. Without waiting for anyone to be properly seated, the second one blurted out, "This queer animal is like our straw fans swinging back and forth to give us a breeze. However, it's not so big or well made. The main portion is rather wispy."

"No, no!" the first blind man shouted in disagreement. "This queer animal resembles two big trees without any branches."

"You're both wrong." the third man replied. "This queer animal is similar to a snake; it's long and round, and very strong."

How they argued! Each one insisted that he alone was correct. Of course, there was no conclusion for not one had thoroughly examined the whole elephant. How can anyone describe the whole until he has learned the total of the parts."

(Kuo & Kuo, 1976, pp. 85)

Chapter One: Conceptualisation and cognitive explanations of aggression

1.1 Introduction

As with the parabolic elephant described on page xvii, the many facets of aggression have complicated adoption of a universal definition (Barratt, Stanford, Kent, & Felthous, 1997) and have lead to extensive debate of its conceptualisation. Leading contemporary definitions classify aggression as the threat of, or actual behaviour towards another, with the intention to cause harm, and a belief that the other is motivated to avoid said harm (Anderson & Huesmann, 2003; Bushman & Anderson, 2001; Paschall & Fishbein, 2002). From an evolutionary perspective, successful traits that increase gene distribution and solve environmental problems are preserved and reproduced (Buss & Shackelford, 1997; Darwin, 1895). A lack of outbreeding for humans to exhibit the capacity to aggress implies prior and on going utility for aggression (Archer, 2009; Raine, 2013).

Understanding the multifarious nature of aggression and its neurobiological underpinnings has benefit for identifying neurobiological markers of aggression and potential treatment measures. This chapter begins by delineating the motivations underpinning, and manifestations of aggression, before exploring how aggression might be explained through cognitive theory.

1.2 Motivation and Manifestation of Aggression

Aggression is a complex phenomenon often discussed through specific dichotomies. As shown in Table 1 (page 4), whilst some dichotomies emphasise the motivations behind an aggressive act (*reactive/instrumental*; Baron & Richardson, 1994), others indicate how aggression might manifestation (*covert/overt, direct/indirect*; Bendig, 1962; Berkowitz, 1993).

1.2.1 Motivation

Emerging from an earlier classification of proactive and reactive behaviour (Feshbach, 1964), motivation to aggress is commonly considered to fall within the reactive/instrumental dichotomy (Baron & Richardson, 1994).

1.2.1.1 Reactive Aggression

Reactive aggression is an unregulated behavioural outburst without intention, goal, or profit (Raine, 2013; Reidy et al., 2011) that likely evolved as a strategy for defending resources, social status, and oneself (Wallner & Machatschke, 2009). In most instances, reactive aggression is accompanied by physiological changes such as heart pounding, a racing pulse, shallow breathing, and muscle tension (Wahlund & Kristiansson, 2009). In animal models, reactive aggression is referred to as affective defence or defensive rage (Blair, 2002; Gregg & Siegel, 2001; Meloy, 2006; Wahlund & Kristiansson, 2009), and features behaviours such as kicking, pawing, and baring teeth (Hess et al., 1928; Hess & Brugger, 1943; Hunsperger, 1956; Kruk et al., 1998; Nakao, 1958).

Reactive aggression is driven by negative affective states, such as anger or anxiety (Feshbach, 1964; Raine, 2013; Spielberger, Reheiser, & Sydeman, 1995), and serves to abolish aversive feelings such as threat (Averill, 1977; Baron & Richardson, 1994; Tyson, 1998, *see row 1 of Table 1*). Threat can be conveyed through many visual and/or auditory modalities, but in humans, is commonly indicated through angry facial expressions (Öhman, Flykt, & Esteves, 2001; Pourtois et al., 2004). Whilst moderate provocation (i.e. a challenge over resources) might evoke anger-induced aggression, a more severe, life-threatening challenge might result in an unrestrained, fear-induced, aggressive response (Blanchard & Blanchard, 1984). At its extreme, homicide might reflect an evolutionary mechanism for solving dangerous, gene-damaging conflicts (Daly & Wilson, 1988).

When used in certain contexts, such as the workplace or when outnumbered, aggression may be detrimental to social status and/or gene survival (Buss &

Shackelford, 1997). In such contexts, aggression might be negatively perceived, and so the loss of social stake from inhibiting aggression is likely outweighed by the benefits of inhibiting an aggressive act (Cashdan & Downes, 2012). Whilst contradicting instinct theories, which frame aggression as a natural instinct manifesting invariantly of context (Buss & Shackelford, 1997), the ability to adapt behaviour to the requirements of social context is vital for gene survival.

In some literature, the term reactive aggression has been used interchangeably with hostility (Feshbach, 1964; Spielberger et al., 1995; Tsytsarev & Grodnitzky, 1995). More accurately, hostility denotes a negative evaluation, accompanied by a desire to inflict harm (Buss, 1961). The conceptual framework of reactive and hostile aggression has been related to impulsivity in both clinical and empirical literature (Atkins et al., 1993; Barratt et al., 1997; Lee & Coccaro, 2001; Moeller et al., 2001). Impulsivity is a multifaceted concept, characterised by acting without thinking (Smith, 1952), an inability to delay gratification (Best, 2002; Logan, Schachar, & Tannock, 1997; Logue, 1988), and poor inhibition of behaviour or thought (Frick & Hare, 2001; Logan et al., 1997). Impulsive aggression frames the use of aggression as a rapid response born from an agitated state (Barratt, 1991; Connor, 2002).

1.2.1.2 Instrumental Aggression

Opposed to impulsive, affect-driven, reactive aggression (Meloy, 2000; Stanford et al., 2003), instrumental aggression represents cold-blooded, controlled behaviour, used to achieve a purpose (Baron & Richardson, 1994; Reidy, et al., 2011; Stanford et al., 2003). Instrumental aggression may manifest following a cooling-off period (Wahlund & Kristiansson, 2009), and as evidenced in Table 1, row 2, might be used to gain advantage over rivals by accruing social status (*see cells A2 and B2*), resources (*see cell C2*), or sexual mates (*see cell D2*).

Table 1.
Examples of scenarios associated with motivations for and manifestations of aggression

	Covert		Overt	
	Direct (A)	**Indirect (B)**	**Direct (C)**	**Indirect (D)**
Reactive (1)	A wife *angers her husband* by accusing him of having an affair. In response, the man yells at his wife – suggesting that he works hard to support their family and that he is hurt by her accusations. The wife is **manipulated to feel guilt.**	A man is told by his wife's friends that he is suspected of having an affair. *Angered by these accusation,* and in the hope that they will tell his wife, the man confides in these friends that he is hurt by the accusations and only wants the best for his family. The wife is **made to feel guilt.**	At a busy bar, a man knocks into another man, subsequently spilling his drinks. With no word of an apology for the act, the second man *feels anger* and **swears** at the male before **punching** him to the ground.	A man learns from a group of friends that another friend is having an affair with his wife. Without the friend present to confront, *anger drives him* to **spread unfavourable and character-damaging rumours** about the friend as retribution. The group exclude the absent friend in the future.
Instrumental (2)	An individual asks advice from a co-worker when rehearsing for a job interview. Unbeknown to them, their co-worker is applying for the same job and tries to *gain an advantage* by giving poor advice about what to say during the interview. This **causes the candidate to fail.**	Two co-workers are interviewing for the same position. During the first candidate's interview, they **mention the other candidate is aggressive and a poor team player.** The employer is manipulated into taking a less favourable view on the second candidate. Ultimately, the *first candidate is hired.*	A man is stopped in the street late at night by an attacker with a knife. The attacker demands the victim's phone and wallet by **threatening harm**, before *escaping with their possessions*.	A male has long been in love with his female friend, who is currently in a relationship with another man. *With the aim of achieving a relationship* with this friend, the male **tells her that her partner is cheating.** This results in the female leaving her partner and finding comfort with her friend, eventually *leading to a relationship.*

Note. Information relating to **Covert**/**Overt** manifestations is emboldened, Direct/Indirect manifestations are underlined, and *Reactive/Instrumental* motivations are italicised.

Resources, including food and shelter, are pivotal to survival (Raine, 2013) and facilitate the acquisition of sexual mates (Archer, 2009). From an evolutionary perspective, genes might benefit from others failing to gather resources (Dawkins, 2006) and so aggression might present a successful strategy for gene preservation (Raine, 2013). However, although winning conflicts benefits gene survival, frequent acts of aggression increase the risk of failure – possibly resulting in harm, retaliation, or the shunning of sexual mates.

Perceived by some as risky and detrimental (Farthing, 2005), the use of aggression might project bravery and dominance during sexual mate acquisition (Ferguson & Dyck, 2012; Kelly & Dunbar, 2001; Krämer, Jansma, Tempelmann, & Münte, 2007). Dominance has been described as a vehicle for reproductive success (Elliot, 1995) and might facilitate the retention of sexual mates by warding off current and future challengers (Buss & Shackelford, 1997; Raine, 2013). Intra-sexual aggression is observed less frequently in females (Campbell, 1995), and may partially account for the evolutionary development of increased body size and strength in males (Archer, 2009).

Instrumental aggression is discussed in conjunction with proactive, premeditated, and predatory aggression. Whilst both proactive and premeditated aggression encapsulate goal-driven behaviours used to detriment another, only proactive aggression occurs solely without provocation (Connor, 2002; Feshbach, 1964). In animals, predatory aggression might be defined through hunting and survival behaviours such as stalking and neck biting (Kruk, 2014; Kruk et al., 1998; Wahlund & Kristiansson, 2009; Wasman & Flynn, 1962), with instrumental aggression in humans thought to include stalking behaviour, and at the extreme, rape (Apostolou, 2013).

1.2.2 Manifestation

Affect- or goal-driven aggression manifests both verbally and physically, as a function of context, sex, and individual differences (Buss & Perry, 1992). This section delineates overt/covert and direct/indirect manifestations of aggression.

1.2.2.1 Overt/Covert Aggression

The overt/covert dichotomy of aggression was coined in Bendig (1962), and later discussed in Loeber & Schmaling (1985). Covert aggressors are predominantly female (Campbell, 1999), and seek to manipulate social hierarchy through charm, guilt, and rumormongering (Björkqvist, Österman, & Kaukiainen, 1992; Crick & Grotpeter, 1995, *see columns A and B of Table 1 for examples*). Typically, covert behaviours are used in an attempt to aggress unnoticed, whilst decreasing the likelihood of counterattack (Björkqvist et al., 1992; Ramirez & Andreu, 2006).

In contrast, overt aggressors use outward, face-to-face aggression against individuals or objects with the intention of causing harm or damage (Baron & Richardson, 1994; Bendig, 1962; Berkowitz, 1993). Overt aggression comprises verbal or physical responses (Alderman, Knight, & Morgan, 1997; Bendig, 1962; Kassinove & Sukhodolsky, 1995; *see cell C1 of Table 1*), and is closely associated with direct aggression (Garandeau & Cillessen, 2006). Though overt aggression might manifest indirectly, such as through spreading character-damaging rumours or secret telling, it is likely to reveal the aggressor's involvement and so might be considered detrimental.

1.2.2.2 Direct/Indirect Aggression

Covert aggression has been associated with indirect aggression (Coyne, Archer, & Eslea, 2004; Garandeau & Cillessen, 2006) whereby damage is caused through the destruction of resources and the elicitation of negative reactions/disapproval of others (Archer, 2001; Berkowitz, 1993; Buss, 1961; Richardson & Green, 2003). For example, negative discourse about an individual to their peers might result in shunning and subsequent harm to their social relationships. Indirect aggression develops during later stages of development and carries only minimal personal risk (Björkqvist et al., 1992; Björkqvist, Österman, & Lagerspetz, 1994). In contrast, direct aggression refers to actual aggression directed towards a target. As shown in Table 1, direct aggression may be threatened through a reactive manner in response to

perceived provocation (*see cell C1*), or through an instrumental manner in order to obtain resources (*see cell C2*).

1.3 Stages of a Reactive Aggressive Encounter

The following section focuses discussion on reactive aggression, and delineates key cognitive theories that might underpin the stages of a reactive aggressive response. First, processes associated with the perception of threat will be outlined. As noted in section 1.2.1.1 (page 2), threat in humans is commonly conveyed through angry facial expressions and so this overview will be restricted to the perception of facial affect, as opposed to other situational (e.g. environment, presence of a weapon) or interpersonal (e.g. voice, body posture) manifestations of threat. Subsequently, cognitive processes associated with threat- and anger-induced negative affect will be discussed along with decision making processes that might moderate the manifestation, or inhibition of aggressive behaviour. Finally, theory thought to underpin the activation of the VIM will be delineated. In particular, how the perception of social cues, specifically facial distress, might engage an empathic response resulting in the halting of aggressive behaviour.

1.3.1 Face Processing

Humans explore the world and attend to environmental stimuli in both a conscious and unconscious manner (Ögmen & Breitmeyer, 2006). Faces are *special* to human perceivers as they capture attention, convey intention, and facilitate the formation of relationships (Apicella, Sicca, Federico, Campatelli, & Muratori, 2013; Meaux, Roux, & Batty, 2014; Stenberg, Wiking, & Dahl, 1998; Vuilleumier, 2000). Efficient face processing is required to rapidly extract key social information such as sex, ethnic origin, gaze direction, and emotional state (Fei-Fei, Iyer, Koch, & Perona, 2007).

Facial expressions impart emotionally salient information (Buck, 1984; Darwin, 1872; Izard, 2007) and are critical for adaptive functioning and the avoidance of

adverse experiences (Anderson, Christoff, Panitz, de Rosa, & Gabrieli, 2003; Hartley & Phelps, 2009; Hinojosa, Mercado, & Carretié, 2015). Humans have evolved a core set of primary emotions, namely fear, anger, sadness, disgust, happiness, and surprise (Ekman, 1993) and are able to decode and understanding these emotions by adulthood (Calder et al., 1996). Following the initial structural processing of faces, facial identities and expressions are thought to process along two parallel, yet independent pathways (Bruce & Young, 1986). Originally conceptualised by the social cognitive information-processing model (Crick & Dodge, 1994), emotion processing is thought to develop through socialisation, whereby information relating to the face is reinforced by social knowledge (Cusi, Nazarov, Holshausen, Macqueen, & McKinnon, 2012; Ekman, 1999; Green et al., 2008).

In particular, angry facial expressions denoting threat demand the rapid reallocation of attention and are rated more arousing than neutral faces (Blanchette, 2006; Fox, Griggs, & Mouchlianitis, 2007; Nummenmaa, Hyönä, & Calvo, 2010; Öhman et al., 2001; Pourtois et al., 2004; Sawada, Sato, Uono, Kochiyama, & Toichi, 2014; Vuilleumier & Schwartz, 2001). From an evolutionary perspective, a propensity to orient attention to threat benefits harm avoidance and protection of social stake (Holmes, Mogg, de Fockert, Nielsen, & Bradley, 2014; LeDoux, 1996; Öhman, Flykt, & Lundqvist, 2000). This preferential response, the threat-superiority effect (Gilbert, 2005; Gilbert, Price, & Allan, 1995), can be thought of as a self-protection system; alerting organisms to potential threat or danger. Here, over-, as opposed to under-reaction, is favourable for survival (Anderson & Carnagey, 2004; Gilbert, 2005). In community samples, angry, relative to happy and neutral facial expressions are processed faster, with fewer errors, when presented both individually or among a crowd of faces (Fox et al., 2000; Hansen & Hansen, 1988; Horstmann & Bauland, 2006; Pinkham, Griffin, Baron, Sasson, & Gur, 2010; Sawada et al., 2014; Williams, McGlone, Abbott, & Mattingley, 2005).

Perception of facial threat is moderated by aggressive traits. Although global face processing deficits have been reported in a moderately large cohort of incarcerated, relative to non-incarcerated male adolescents during a forced-choice paradigm (McCown, Johnson, & Austin, 1986), threat-specific face processing errors have been reported elsewhere (Best et al., 2002; Sato, Uono, Matsuura, & Toichi, 2009). In Sato et al. (2009), incarcerated, relative to non-incarcerated males were more likely to misattribute facial disgust as anger, even when presented with several response options. Similarly, patients with, relative to those without, intermittent explosive disorder (IED), a disorder characterised by recurrent acts of reactive aggression disproportionate to actual provocation (Coccaro, 2003), evidenced anger processing deficits and a greater likelihood of miscategorising neutral facial expressions as being negatively valenced (Best et al., 2002).

Taken together, angry facial expressions are considered a key vehicle in conveying threat, and demand the rapid reallocation of neuronal resources as a function of their social and biological importance. This importance is exemplified by response biases towards threat in typically developing humans. However, in individuals characterised by aggressive traits, this threat perception bias can present atypically – possibly impacting wider face processing.

1.3.2 Generation of Negative Affect

Aggression is often preceded by anger, a negative affective state that serves to index the propensity to aggress (Buss & Warren, 2000). Anger can be short-lived, or persistent, and is thought to play a role in mood disorders, anxiety, and reactive aggression (Wilkowski & Robinson, 2010). Furthermore, anger can be elicited to environmental triggers, memories, and rumination (Berkowitz, 1993; Eckhardt & Deffenbacher, 1995) and is known to disrupt communication, cognition, inhibition, and physiological change (Berkowitz, 1993; Berkowitz & Harmon-Jones, 2004; Demenescu, Kortekaas, den Boer, & Aleman, 2010; Kassinove & Sukhodolsky, 1995; Melcher, Born, & Gruber, 2011; Pillutla & Murnighan, 1996; Tsytsarev & Grodnitzky, 1995). Over an extended period of

time, anger can be detrimental to health and social relationships (Baron et al., 2007; Bettencourt, Talley, Benjamin, & Valentine, 2006; Shorey et al., 2011).

Cumulative effects might also affect anger. Physiological arousal associated with anger dissipates slowly, allowing anger to last over long periods of time. As suggested in the excitation transfer theory (Zillmann, 1983), stimuli presented shortly after the induction of an anger state might be misattributed as hostile and further disrupt cognition. In Cohen, Eckhardt, & Schagat (1998), following the induction of anger in the form of a participant-directed insult, individuals took longer to respond to target words when surrounded by non-target anger-related distractors, suggesting an attentional bias towards angry stimuli. This effect was augmented in individuals with high trait anger. In contrast, using a different, yet qualitatively similar anger induction paradigm, Eckhardt & Cohen (1997) did not observe a bias towards anger-related words. However, although this investigation controlled for word-specific factors (e.g. length), it did not quantify state anger prior to anger induction, and so cannot conclusively state that anger was induced.

Consolidating several cognitive theories of aggression, the general aggression model (GAM) outlines how social stimuli in the form of personal (e.g. personality traits, attitudes, biological predispositions) and situational (e.g. environmental triggers, social cues) inputs might moderate affect, arousal, and cognition (Anderson & Bushman, 2002). On a cognitive level, aggressive thoughts, feelings, emotions, and behaviours are thought to interconnect – forming a composite knowledge structure associated with aggression (Berkowitz, 1993; Guerra & Huesmann, 2004). This structure, elaborated on in section 1.3.3 (page 11), is thought to respond to social cues, resulting in the retrieval of anger-/aggression-related information and in turn sensations of anger (Anderson & Carnagey, 2004). A greater yield of aggressive memories, thoughts, and feelings might be gained as a function of aggressive personality traits, increased attention, and stimulus salience (Berkowitz, 1993; Guerra & Huesmann, 2004)

1.3.3 Executive Control

Through the use of executive functioning, humans are typically able to divert their attention away from task-irrelevant stimuli and regulate their affective feelings in order to inhibit an aggressive response (Chen, Muggleton, & Chang, 2014; Davidson, Putnam, & Larson, 2000; Holmes et al., 2014; Lewis et al., 2008; Raaijmakers et al., 2008; Schachar & Logan, 1990; Vilà-Balló, Hdez-Lafuente, Rostan, Cunillera, & Rodriguez-Fornells, 2014). Prior to the execution of a behavioural response, behavioural scripts, defined as an expected series of behaviours for a given situation, are consciously evaluated against one another (Anderson & Carnagey, 2004). As outlined in the social-cognitive processing model (SCP; Huesmann, 1998), behavioural scripts are acquired through direct and indirect social learning, reinforcement, and punishment. Disruption to this learning process, possibly as a function of social exclusion and/or the absence of positive role models (Maughan & Rutter, 2001), is likely to increase the risk of antisocial and aggressive behaviour (DeWall, Anderson, & Bushman, 2011).

Pro-aggressive scripts (i.e. scripts that promote the use of aggression) are formed, and subsequently reinforced, when aggression is observed to benefit the acquisition of resources, sexual mates, and social status, or as a successful means of defence. An increased availability of pro-aggressive scripts has been inversely associated with cognitive resources (Finkel, DeWall, Slotter, Oaten, & Foshee, 2009), and likely facilitates incidents of aggression (Lochman & Dodge, 1994). Importantly, access to pro-aggressive scripts does not necessarily determine whether aggressive behaviour will ensue. A well-developed cognitive system generates both pro- and non-aggressive responses, and applies them appropriately depending on the given context (Guerra & Huesmann, 2004). As discussed in section 1.3.2 (page 9), the ability to suppress aggressive behaviour is vital for long-term organism survival (Guerra & Huesmann, 2004).

When a response is not instantly required, these aforementioned scripts are considered, evaluated, and re-appraised in accordance with normative beliefs and self-schemas (Crick & Dodge, 1994). Whilst normative beliefs encapsulate

beliefs about what is socially acceptable in the given context (Anderson & Carnagey, 2004; DeWall et al., 2011), self-schemas describe how an individual wants to be perceived (Guerra & Huesmann, 2004). Although limited to self-reports in the absence of behavioural measures, increased physical and verbal inter-partner aggression has been associated with positive attitudes towards inter-partner violence in students (Fincham, Cui, Braithwaite, & Pasley, 2008).

Behavioural responses are further filtered through the assessment of both the consequences of, and actual ability to, enact a certain script (Anderson & Huesmann, 2003). Although the benefit of using aggression as an effective strategy for warding off the advances of others has been discussed in section 1.2.1.2 (page 3), aggression may be personally detrimental. For example, aggressing against a dangerous opponent may lead to repercussions (DiGiuseppe, 1995). Conceptualised within the frustration-anger-displacement theory (Dollard, Doob, Miller, Mowrer, & Sears, 1939), frustration is displaced elsewhere (i.e. an object or another) when it is considered detrimental to aggress against the original source of frustration. However, this theory fails to acknowledge why, and indeed how, subsequent targets are selected.

1.3.4 Inhibition by Cues of Distress

Typically, aggressive behaviour results in a social response, such as facial distress (e.g. fear, sadness). This section explores how the facial distress of another might evoke an inhibitory response. Previously, animal models have considered the role of distress and submission in relation to aggression inhibition. An innate aggression control mechanism was proposed in Eibl-Eibesfeldt (1970) and Lorenz (1966, 1981), suggesting same-species aggression might terminate following victim submission. A similar mechanism, the VIM (Blair, 1995; Blair et al., 1995, 1999), may exist in humans.

The VIM assumes socially acceptable behaviours, which allow individuals to function within society, are formed through instrumental learning and aversive conditioning (Blair, 2003). Over time, positive reactions help forge stimulus-

reward associations with a given behaviour, resulting in increased use of said behaviour (Baxter & Murray, 2002). Conversely, negative reactions may make individuals '*feel bad*' and so are likely to result in behaviours being used less frequently (Blair, 1995). When negative reactions succeed antisocial behaviours, individuals begin to learn moral (i.e. victim-based) and conventional (i.e. social order-based) transgressions (Blair, 2006). Negative reactions to moral transgression create stimulus-punishment associations (Baxter & Murray, 2002), reducing the likelihood of said behaviours being used in the future for fear of negative consequences (Blair et al., 2006; Raine, 2013). However, if bad behaviour goes unpunished, or is rewarded (e.g. through material gain), behaviour attenuation is not effectively learned and might even override previously forged, negative associations (Blair, 1995; Mineka, Davidson, Cook, & Keir, 1984).

In particular, the VIM is thought to respond to the perception of distress, including tears (Blair, 1993) and fearful and sad facial or vocal expressions (Blair et al., 1997; Decety & Chaminade, 2003). In contrast to angry faces, which convey direct threat, facial distress conveys ambiguous threat and the need for avoidance (Blair, 1995; Ewbank et al., 2009; Mineka & Cook, 1993). Distress likely inhibits aggression by activating cognitive withdrawal schemas (Blair, 1993; Nichols, 2001), which result in individuals learning to avoid behaviour that evokes pain and distress in others (Blair, 1993, 1995, 2004; Blair et al., 1999). Distress cues stabilise social interactions among healthy functioning individuals (Marsh & Ambady, 2007), with children as young as four showing reduced stealing behaviour after associating said behaviours with sad facial responses (Camras, 1977).

Salient negative social cues form a malleable database of VIM triggers capable of guiding behaviour across contexts (Blair, 1995, 2003; Blair et al., 1997). Poor responses to distress-related social cues may result in a failure to associate distress with negative affect (Blair, 2001, 2003, 2005) and subsequent failure to trigger the VIM (Blair, 2005). As evidenced in both children and adults, deficits

in distress recognition have been associated with inappropriate response choices, conduct problems, and violent behaviour (Marsh & Blair, 2008; Perry et al., 2001).

Distress-induced behavioural inhibition is likely underpinned by empathy (Decety & Jackson, 2004; Eisenberg, 2000; Marsh & Ambady, 2007). Empathy is a multidimensional phenomenon defined as an ability to understand the feelings and emotional states of others in reference to the self (Decety & Jackson, 2004; Decety & Moriguchi, 2007; Hoffman, 2000; Preston & de Waal, 2002). Empathy is thought to develop through socialisation and moral transgressions (Morton & Frith, 1993).

Empathy comprises both affective and cognitive components (Blair, 2013; Carr & Lutjemeier, 2005; Davis, 1983). Sometimes referred to as empathic concern (Davis, 1983), affective empathy is the vicarious emotional experience of the internal emotional state of another (Bryant, 1982) coupled with the ability to verbally describe said state (Blair, 2013). Affective empathy is said to emerge from the recognition and representation of emotion (Blair, 1995; Feshbach & Feshbach, 1982; Hoffman, 1987). The ability to identify and respond to an emotional state is a vital aspect of everyday interaction (Siedel et al., 2013). In contrast, cognitive empathy, sometimes referred to as perspective taking (Batson, Fultz, & Schoenrade, 1987; Davis, 1983), is the ability to recognise and comprehend the feelings of others as being separate from one's own (Decety, 2011; Decety & Jackson, 2004; Decety, Michalska, Akitsuki, & Lahey, 2009).

Empathic dysfunction is considered detrimental to the VIM because of its associations with deficient processing of fearful faces (Carr & Lutjemeier, 2005). Self-reported cognitive and affective empathic responses to the suffering of others are inversely related to physical aggression. Increased empathy has been associated with pro-social behaviour and moral reasoning (Batson, 1991; Decety & Meyer, 2008), and decreased indirect aggression, verbal aggression,

assault, and irritability (Eisenberg, 2000; Richardson, Hammock, Smith, Gardner, & Signo, 1994). However, this association may only last under moderate, but not intense provocation (Blair, 2005; Davidson et al., 2000; Frick et al., 2003; Miller & Eisenberg, 1988; Zahn-Waxler, Cole, Welsh, & Fox, 1995). Under intense provocation, cognitive inhibitors become ineffective as a result of acute impairment of cognitive functioning (Zillmann, 1988).

The VIM is dysfunctional in psychopathy (Blair, 1995, 2001; Blair et al., 2004), a developmental disorder first described in Cleckley (1941, 1976). Psychopathy is thought to occur in less than 1% of the general population (Hare, 2003), and worsens with age (Kubak & Salekin, 2009). Alongside machiavellianism and narcissism, psychopathy forms one of the components of the Dark Triad (Paulhus & Williams, 2002), and is characterised by a constellation of affective, interpersonal, and behavioural malfunction (Cleckley, 1976; Frick, O'Brien, Wootton, & McBurnett, 1994; Hare, 2003; Hare & Neumann, 2006; Harpur, Hare, & Hakstian, 1989). Specifically, the affective component of psychopathy comprises a lack of guilt, low empathy, callousness, and unemotional and uncaring traits (Hare, 1991, 2003), with the interpersonal component encapsulating manipulative behaviours, such as lying, cheating, and superficial charm (Harpending & Sobus, 1987; Raine, 1993). The behavioural component manifests through impulsive actions and sensation seeking with disregard for the completion of long-term goals (Barry et al., 2000; Dawel, O'Kearney, McKone, & Palermo, 2012; Hart & Dempster, 1997).

The affective dimension of psychopathy is encapsulated by CU traits, and is prominent in most conceptualisations of psychopathy (Cleckley, 1976; Hare, 1993). Although CU traits are frequently used as a proxy measure of psychopathy (Dawel et al., 2012), with their presence in childhood predictive of psychopathic development and poor treatment outcomes (Frick, Ray, Thornton, & Kahn, 2014; Frick & White, 2008), CU traits have only recently been accepted as a diagnostic criterion of antisocial behaviour disorders, such as conduct

disorder (CD) (American Psychological Association, 2013; Waller, Gardner, & Hyde, 2013).

CU traits are associated with behavioural aspects of psychopathy. Neatly summarised by a meta-analysis of 24 studies sampling children and adolescents, CU traits predict future delinquency and aggression severity (Frick & Dickens, 2006). Specifically, presence of CU traits during childhood and adolescence positively correlates with future incidents of physical aggression (Coid et al., 2009; Frick et al., 2003; Krischer & Sevecke, 2008; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Woodworth & Porter, 2002), violent sexual offending (Caputo, Frick, & Brodsky, 1999), and long-lasting patterns of antisocial behaviour (Essau et al., 2006; Frick & White, 2008).

Although psychopathic populations frequently aggress in an instrumental manner (Cornell et al., 1996; Meyer-Lindenberg et al., 2006), they also engage in reactive aggressive behaviour (Frick et al., 2003). Such aggression and antisocial behaviour manifests more diversely, and of a greater severity, than presented in non-CU counterparts (Christian, Frick, Hill, Tyler, & Frazer, 1997; Frick et al., 2003; Kruh, Frick, & Clements, 2005; Lynam, 1997). However, some findings (e.g. Kruh et al., 2005) are based on self-report assessments, which are inherently open to inflation, and as restricted to male samples in most cases, should be discussed with caution.

Psychopaths, and children evidencing psychopathic-like traits, are thought to have difficulty understanding the emotional significance, but not the meaning, of emotion (Hervé, Hayes, & Hare, 2003), and evidence both a decreased sensitivity to punishment (Fisher & Blair, 1998) and a lack of victim-based moral reasoning (Blair, 2003; Montagne et al., 2005). Such deficits are likely facilitated by an inability to [1] process stimuli, [2] form cue associations, and [3] condition an emotional response (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002). Specifically, psychopaths express insensitivity (Blair et al., 2004) and low

physiological responses to basic emotions (Verona, Patrick, Curtin, Bradley, & Lang, 2004).

Although a general face processing deficit spanning sadness, fear, disgust, and anger has previously been reported in violent, relative to non-violent offenders and controls (Robinson et al., 2012; Seidel et al., 2013), two meta-analyses support distress-specific face processing deficits in individuals characterised by antisocial behaviour (e.g. psychopaths, criminals, delinquents, and individuals displaying high trait aggression) (Marsh & Blair, 2008; Wilson, Juodis, & Porter, 2011). Distress recognition deficits extend to children with psychopathic-like traits who also exhibit deficits in processing body distress (Blair, Colledge, Murray, & Mitchell, 2001; Dadds et al., 2006; Stevens, Charman, & Blair, 2001). Recognition of body distress is also poor in adult psychopaths (Blair et al., 1997). Whilst a key analysis in the distress processing literature, Wilson et al. (2011) also offer some evidence for an association between antisocial behaviour and deficits in processing non-distress emotions, though this could be an artefact of the use of lenient alpha levels (.10). During aggressive encounters, deficient processing of distress may lead to a prolonged, uninhibited aggressive response (Marsh & Ambady, 2007).

Processing of fearful and sad faces is thought to share similar neurobiological underpinnings (Adolphs & Tranel, 2004, *see chapter two, section 2.5 [page 29]*). Although deficits in sadness recognition have been reported elsewhere (Dolan & Fullam, 2006; Eisenbarth, Alpers, Segrè, Calogero, & Angrilli, 2008; Fairchild, van Goozen, Calder, Stollery, & Goodyer, 2009, 2010; Fullam & Dolan, 2006; Hastings et al., 2008; Woodworth & Waschbusch, 2008), CU traits have been predominantly associated with fear recognition deficits both visually (Blair, 2000; Blair et al., 2004; Dadds et al., 2008; Fairchild et al., 2009; Iria & Barbosa, 2009; Leist & Dadds, 2009; Muñoz, 2009) and vocally (Stevens et al., 2001). Whilst poor fear recognition has been attributed to ambiguity of facial fear as a whole (Elfenbein & Ambady, 2002; Rapcsak et al., 2000; Russell, 1994; Stockdale, Morrison, Kmiecik, Garbarino, & Silton, 2015), intact

recognition of facial fear in healthy controls suggests a specific association with CU traits (Blair et al., 2004).

Deficits in distress recognition do not always coincide with the presence of CU traits. Psychopaths and individuals with psychopathic traits have shown an intact ability to process verbal and facial fear, alongside anger and happiness (Eisenbarth et al., 2008; Fairchild et al., 2010; Glass & Newman, 2006; Hansen, Farivar, Thompson, & Hess, 2008; Hastings et al., 2008; Hiatt, Lorenz, & Newman, 2002; Kosson, Suchy, Mayer, & Libby, 2002). In some cases, psychopathy has been associated with *increased* fear recognition (Del Gaizo & Falkenbach, 2008), with similar findings reported at trend level in children with psychopathic traits (Woodworth & Waschbusch, 2008). However, these results might reflect variation in methodology, including stimuli used, frequency thereof, and measures recorded (*see Glass & Newman, 2006*).

An attempt to understand the disparity of results relating to distress processing was undertaken in Dawel et al. (2012). Impairments in processing positive and negative emotions were found across both facial and vocal modalities. Adults, children, and adolescents exhibited emotion recognition deficits for fear, happiness, sadness, and surprise, but not anger or disgust. Whilst previous meta-analyses considered psychopathy as a united construct (Marsh & Blair, 2008; Wilson et al., 2011), Dawel et al. separated out processing impairments associated with the affective factor of psychopathy only; implicating poor distress, but not threat, recognition in association with CU traits.

There have been mixed results when investigating associations between CU traits and empathy. Although inversely related to CU traits in some child samples (Anastassiou-Hadjicharalambous & Warden, 2008; Dadds et al., 2009; Pasalich, Dadds, & Hawes, 2014), cognitive empathy has been shown to remain intact in adults with psychopathy, and children and adolescents with psychopathic-like traits (Hare, 2003; Jones, Laurens, Herba, Barker, & Viding, 2010; Richell et al., 2005; Schwenck et al., 2012). Conversely, an inverse

association between CU traits and affective empathy has been evidenced in adults (Seidel et al., 2013), as well as children and adolescents (Anastassiou-Hadjicharalambous & Warden, 2008; Dadds et al., 2009; Jones et al., 2010; Pasalich, 2014; Schwenck et al., 2012). Although the results reported in Pasalich et al. were based on parent reports, the stability of this finding across samples suggests this is a reliable measure. Taken together, whilst individuals characterised by CU traits display atypical responses to the emotion of others, their overall cognitive understanding of emotion, in most cases, remains intact (Hare, 2003).

The inability to experience and generate an empathic response to distress likely impedes avoidance learning and subsequent halting behaviours (Bird & Viding, 2014; Blair, 2004). An absence of inhibitory factors might facilitate prolonged aggression (Blair, Morton, Leonard, & Blair, 2006; Hare, 1991, 2003; Richardson et al., 1994). Poor empathy may account for the greater number, and severity of crimes committed by psychopathic, relative to non-psychopathic individuals (Frick et al., 2003; Hare, 2003; Marshall et al., 1995)

1.3.5 Behaviour Modulation

Activation of the VIM is thought to result in the suppression of a pre-initiated motor response (Hughes, Fulham, Johnston, & Michie, 2012). Whilst individuals may not always halt their motor behaviour, successful stopping relies on cognitive control, defined as the stopping or overriding of a mental process with or without intention (MacLeod, 2007; Miyake et al., 2000). Cognitive control represents a cluster of processes underpinning the adaption and planning of actions within an ever-changing social context (Enriquez-Geppert et al., 2010; Oldenburg et al., 2012). On a cognitive level, inhibitory control is modelled using the horse race model (HRM; Logan, 1994), whereby independent go and nogo responses are thought to compete prior to execution (Logan & Cowan, 1984).

Greater motor inhibition is required to suppress a motor response when imminent motor inhibition is required. During target detection tasks where

individuals are asked to respond to target stimuli, but inhibit a response to similar non-target stimuli, motor responses are inhibited prior to elicitation. Alternatively, stop-signal (SST) and stop-go paradigms (Lappin & Eriksen, 1966; Logan & Cowan, 1984), which require motor suppression of a pre-potent, or pre-initiated motor response (Aron, 2007; Bekker, Kenemans, Hoeksma, Talsma, & Verbaten, 2005), require greater inhibition due to the increased cognitive load required to successfully suppress a response (Johnstone et al., 2007; Rubia, Smith, Brammer, & Taylor, 2003). Whilst target detection paradigms require automatic action, stop paradigms require greater cognitive control (Verbruggen & Logan, 2008). Successful stopping is referenced by the HRM (Logan, 1994) and reflects the outcome of competition between independent go and stop operations (Boucher, Palmeri, Logan, & Schall, 2007).

The ability to suppress a planned or on going motor response as a function of environmental stimuli is a prerequisite for socially adaptive behaviour (Huster, Pilis, Lavallee, Calhoun, & Hermann, 2014; Oldenburg et al., 2012). Stopping times are shortened by impulsive traits (Marsh et al., 2002) and emotional imagery (Pessoa, Padmala, Kenzer, & Bauer, 2012). In relation to the VIM, there is currently a dearth of investigation into behavioural extinction sampling from aggressive individuals or using distress stimuli as a stopping agent. In a single study where individuals were asked to classify, but not respond to, angry and neutral facial expressions, individuals characterised by high self-reported aggressive traits exhibited longer stopping times than their low aggressive trait counterparts (Pawliczek, Derntl, Kellermann, Gur, & Schneider, 2013). Although facial affect was incorporated into this paradigm, it was not used as a stopping stimulus. Instead, a stop response was indicated via a change of colour to the image frame. As aggressive and CU traits are associated with poorer distress processing, it is likely further detriments would be observed if facial affect, particularly distress, signalled the need to stop.

1.4 Chapter Summary

This chapter delineated motivations underpinning, and manifestations of aggression in humans, before tailoring discussion to how various stages of a reactive aggressive response might be explained through cognitive theory. In particular, facial anger was identified as core means of conveying threat in humans because of its utility to capture attention and facilitate a negative state of affect. Regulation of this affective state was described by executive functioning, whereby individuals are typically able to select an appropriate behavioural response for a given situation, based on prior learning and contextual information. Moreover, increased attention to threat, and executive functioning deficits are evidenced in samples characterised by aggressive traits. The latter part of the chapter attended to the VIM, and how facial distress, through generation of an empathic response, might allow individuals to inhibit antisocial and aggressive responses in order to better function within society. Chapter two delineates the known neurobiological underpins of facial affect processing and distress-induced motor extinction, and how these networks might show dysfunction in samples characterised by aggressive and CU traits.

Chapter Two: Neurobiology of reactive aggression

2.1 Introduction

In chapter one, facial expressions were described as vehicles for both reactive aggressive responses to threat (e.g. angry faces) and inhibitory responses to distress (e.g. fearful and sad faces) (Ögmen & Breitmeyer, 2006). Chapter two builds on the cognitive perspective of aggression described in chapter one, through delineation of key neurobiological networks thought to be involved in the generation/inhibition of an aggressive response. Cortical networks of face processing are outlined, before discussing how facial threat and distress, in particular, might process along fast subcortical routes as a function of their biological significance (Amaral, Price, Pitknen, & Carmichael, 1992). Moreover, this chapter describes how anger is generated through the activation of core stress systems, and how distress might engage frontal-limbic networks involved in executive functioning, behavioural inhibition, and motor suppression.

2.2 Face Processing

In humans, faces evoke an immediate, and automatic process consisting of visuospatial and attentional networks - comprising both cortical (i.e. dorsolateral prefrontal, anterior and posterior cingulate, inferior/middle occipital gyri, fusiform gyrus, insula, primary and secondary visual cortices) and subcortical (i.e. thalamus, basal ganglia, pulvinar, putamen, amygdala, hippocampus) brain regions (Palermo & Rhodes, 2007; Vuilleumier & Driver, 2007, *but see Fusar-Poli et al., 2009 for a review*).

Initially, retinal information is transferred to the lateral geniculate nucleus, a core relay centre in the thalamus (Fox, 2008; Morris, Öhman, & Dolan, 1999), before being projected to the occipital (i.e. primary and secondary visual cortices, inferior/middle occipital and lingual gyri) and temporal (i.e. fusiform, superior temporal) lobes (Adolphs, 2002; Gauthier et al., 2000; Haxby, Hoffman, & Gobbini, 2000; Vuilleumier & Driver, 2007). At occipital sites, the inferior

occipital gyrus houses the occipital face area (OFA), a region shown to represent facial aspects such as the eyes and mouth (Nichols, Betts, & Wilson, 2010; Pitcher et al., 2007). The OFA shares connections with the middle occipital gyrus (Renier et al., 2010) and posterior parietal cortex (Potts & Tucker, 2001) where spatial information is processed. Renier et al. reported increased middle occipital gyrus activation - as measured by functional magnetic resonance imaging (fMRI) - in participants tasked with comparing the location of geometric shapes between trials. However, as this region also responded to tactile and auditory stimuli, it is not limited to visual processing.

In the temporal lobe, though responsive to non-facial stimuli such as houses (Haxby et al., 2001), activation of the fusiform gyrus is maximal to faces (Apicella et al., 2013; Vuilleumier et al., 2001). The fusiform gyrus is functionally and anatomically connected to the superior temporal sulcus (STS), where facial detail is extracted (Haxby et al., 2000; Haxby, Hoffman, & Gobbini, 2002; Puce & Perrett, 2003) in order to process social relevance and intent (Adolphs, 2002; Allison, Puce, & McCarthy, 2000; Nummenmaa, Hyönä, & Calvo, 2010). Attenuated activation of the fusiform-STS pathway is associated with facial affect recognition deficits (Sato, Toichi, Uono, & Kochiyama, 2012; Schultz et al., 2003). However, as fusiform gyrus volume is unchanged (Pierce et al., 2001) or even enlarged (Waiter et al., 2004) in autistic samples characterised by poor affect recognition, disruption to this pathway might be driven by a poorly developed STS (Boddaert et al., 2004).

The fusiform gyrus and STS share direct (Amaral et al., 1992; Fairhall & Ishai, 2007; Puce & Perrett, 2003; Pujol et al., 2009), and indirect (Petrides & Pandya, 2007), connections with the amygdala. The amygdala is present in both mammalian and non-mammalian creatures (e.g. reptiles, birds, fish) (Gross, 2007; Haxby et al., 2000; Janak & Tye, 2015), and in humans comprises three interconnected nuclei thought to play a core role in emotion processing (Fox, Oler, Tromp, Fudge, & Kalin, 2015; Sah, Faber, Lopez, & Power, 2003). During face processing, the amygdala encodes emotionally valenced facial features

(Adolphs, 2010; Fitzgerald et al., 2006; Sergerie, Chochol, & Armony, 2008; Winston, Gottfried, Kilner, & Dolan, 2005; Zald, 2003; Zink et al., 2008) and feeds this information back into the fusiform-STS pathway (Allison et al., 2000; Pessoa & Adolphs, 2010; Sabatinelli, Bradley, Fitzsimmons, & Lang, 2005; Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). This feedback mechanism serves to both increase the cellular response to facial affect and aid the resolution of visual ambiguity (Adolphs, 2001). Amygdala activation likely explains increased fusiform-STS connectivity when viewing high intensity/emotional, relative to static/neutral, facial stimuli (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003; Fusar-Poli et al., 2009; Winston et al., 2003a).

2.3 Facial Threat

Angry facial expressions denote threat and signal provocation (Blair, 2003; Blair et al., 1999; *but see chapter one, section 1.3.1 [page 7]*). On a neurobiological level, threat-relevant information is transferred from the thalamus to the amygdala, along a fast, yet coarse subcortical pathway (Amaral et al., 1992; LeDoux, 1996; Öhman & Mineka, 2001; Pontius, 2002; Vuilleumier, Armony, Driver, & Dolan, 2003). This pathway, comprising the superior colliculus and pulvinar, has been shown to activate in response to angry facial stimuli - masked in order to inhibit cortical processing (Morris et al., 1999). Furthermore, case studies of patients with bilateral (Adolphs, Tranel, Damasio, & Damasio, 1994, 1995; Calder et al., 1996), but not unilateral (Adolphs et al., 1995) amygdala lesions have reported impaired recognition of facial and vocal expressions of anger. However, whilst this network has evolutionarily benefit for harm avoidance (Helmut, 2003; Vuilleumier et al., 2003), these patients also evidenced impaired fear processing, and so it is not threat-specific.

Being able to appropriately respond to threat is essential for everyday social interaction (*see chapter one, section 1.3.3 [page 9]*). Atypical amygdala structure and activation has been evidenced in populations characterised by aggressive traits. In Matthies et al. (2012), bilateral amygdala volume was

smaller in individuals with high, relative to low, lifetime histories of aggression. This effect survived after controlling for anxiety and depression; disorders related to reduced amygdala volume (Hamilton, Siemer, & Gotlib, 2008). Similarly, relative to non-clinical controls, smaller amygdala volumes were observed in male (Soloff, Nutche, Goradia, & Diwadkar, 2008) and female (Rüsch et al., 2003; Soloff et al., 2008) patients with borderline personality disorder (BPD), a disorder characterised by intense, anger-driven behaviour (American Psychological Association, 2013). Furthermore, bilateral amygdala volume was inversely associated with current and childhood aggressive traits in individuals characterised by a range of aggression-related psychopathologies (Pardini, Raine, Erickson, & Loeber, 2014). Moreover, after correcting for baseline aggression, low amygdala volume in this sample was predictive of aggressive incidents over a three-year follow up period.

Elsewhere, relative to controls, males with IED (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Coccaro, Sripada, Yanowitch, & Phan, 2011) and BPD (Donegan et al., 2003; Thomas et al., 2013) have evidenced increased amygdala activation to angry facial expressions. However, these investigations are not without limitations. Amygdala activation in Coccaro et al. (2007) was calculated by contrasting haemodynamic responses to angry facial expressions with those evoked during rest periods - not to neutral facial expressions. Whilst amygdala responses to angry faces were positively correlated with lifetime histories of aggression, group contrasts might have been augmented as a function of not subtracting the residual amygdala activation to faces during the analysis. Furthermore, in the BPD samples, amygdala hyperactivation was also reported to positive, negative, and neutral expressions (Donegan et al., 2003) as well as fearful and neutral faces (Thomas et al., 2013), suggesting amygdala hyperactivity is not anger-specific in BPD.

Mixed findings have been observed in samples characterised by CU traits. Though not threat-specific, psychopathic offenders, relative to non-psychopathic offenders and non-offender controls, have evidenced attenuated

left amygdala activity during an emotional word memory task (Kiehl et al., 2001). Conversely, Müller et al. (2003) reported *increased* right hemispheric amygdala activity to negative images in a small cohort of psychopathic offenders, relative to non-offender controls. Although Müller et al. argue increased activation might reflect task modality, left hemispheric amygdala hypoactivity to negative images has been observed in adolescent males with CD, relative to age-matched controls, using a comparable paradigm (Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2005). It should be noted that none of these investigations used facial threat as stimuli.

Together, these results suggest that aggressive and CU traits might differentially associate with amygdala reactivity to threat. Whilst amygdala hyperactivity might contribute to an over generation of negative affect, amygdala hypoactivity suggests impaired processing of threat-relevant social cues (Davidson et al., 2000).

2.4 HPA-axis

Facial threat contributes to negative affective states, such as anger (*see chapter one, section 1.3.2 [page 9]*). On a neurobiological level, anger is generated as a function of threat-relevant information being projected from the amygdala to the hypothalamus, pituitary gland, and adrenal axis (HPA-axis). Within this network, threat information projects via the stria terminalis, which activates as a function of threat intensity (Somerville, Whalen, & Kelley, 2010).

Though not fully understood in humans (Toth et al., 2010), stimulation of the feline hypothalamus has long been understood to elicit an aggressive response (Hess, 1928). As reviewed in Kruk (2014), current feline models of aggression suggest neuronal convergence from the amygdala to the ventromedial (vmHyp) and lateral (lHyp) sections of the hypothalamus initiates defensive rage and predatory aggression, respectively (Delville, de Vries, & Ferris, 2000; Greenberg et al., 1984; Gregg & Siegel, 2001; Summers & Greenberg, 1994, 1995). These pathways are mutually inhibitive (Gregg & Siegel, 2001). During

defensive rage, the vmHyp projects to the dorsal midbrain periaqueductal gray (PAG) and brainstem (Gregg & Siegel, 2001; Siegel & Victoroff, 2009), resulting in the manifestation of physical and verbal aggression (Sewards & Sewards, 2003). This aggression is promoted over time as a function of PAG-hypothalamic feedback (Gregg & Siegel, 2001).

The HPA-axis is considered the central stress system in humans (Hawes, Brennan, & Dadds, 2009). Here, threat-induced hypothalamic activation evokes the release of corticotropin-releasing factors (CRFs), which enter the anterior pituitary blood supply and facilitate adrenocorticotropic hormone release (AHR). AHR prepares the release of adrenaline, used to boost energy and physical ability, and so facilitates the preparation of a behavioural response (Fox, 2008). The HPA-axis is regulated by the hippocampus, which decreases CRF output (Gregg & Siegel, 2001; Raine, 2013) and aids affect processing by signalling the declarative fact of threat (Blanchard, Hynd, Minke, Minemoto, & Blanchard, 1991; Raine, 2013; Williams et al., 2001).

Amygdala hyperactivity to facial threat might detriment the amygdala-HPA network by over stimulating the hypothalamus (Davidson et al., 2000; Vyas, Mitra, Shankaranarayana, & Chattarji, 2002). Frequent threat-induced activation of the hypothalamus likely habituates the hypothalamic response, and increases the threat-arousal threshold required to engage hippocampal modulation (Kruk et al., 1998; Strauss et al., 2005; Viau, 2002). Consequently, deficits in hippocampal inhibition likely contribute to increased instances of threat-induced aggressive behaviour (Delville, David, Taravosh-Lahn, & Wommack, 2003; Lopez-Duran, Kovacs, & George, 2009; van Goozen & Fairchild, 2008).

HPA-axis efficacy is typically indexed by cortisol, with increased cortisol related to increased adrenalin output (Hawes et al., 2009). In rodent samples, though not evidenced in Oakes & Coover (1997), low basal cortisol (i.e. cortisol measured over several days) has been associated with increased aggression

(Kruk, Halasz, Meelis, & Haller, 2004), suggesting an inverse relationship between HPA-axis functionality and aggressive behaviour. The null result reported in Oakes & Coover (1997) might reflect low baseline aggression scores restricting variance in the data.

In humans, though based on a small effect size, a meta-analysis of 82 investigations between 1978 and 2006 proposed an inverse relationship between basal cortisol and antisocial behaviour (Alink et al., 2008). This small effect size might reflect an amalgamation of positive and negative relationships with the literature. For example, whilst resting state cortisol has been negatively associated with antisocial behaviour in some investigations (Dolan, Anderson, & Deakin, 2001; McBurnett et al., 2000; Oosterlaan et al., 2005; Pajer, Gardner, Rubin, Perel, & Neal, 2001; van Goozen et al., 1998; Virkkunen, 1985), positive associations with reactive aggression have been reported elsewhere (van Bokhoven et al., 2005).

More recently, basal, but not acute (i.e. current/short term) cortisol, has been inversely associated with paradigm-induced aggression, indexed by the duration and intensity of aversive blasts of white noise (Böhnke, Bertsch, Kruk, & Naumann, 2010). Additionally, whilst not reaching statistical significance, lower cortisol levels have been observed in adolescent offenders detained for property, drug, and violent crimes, relative to non-offender controls (Feilhauer, Cima, Korebrits, & Nicolson, 2013). These findings suggest individuals with low basal HPA-axis activation might be more sensitive to provocation and more vulnerable to aggressive reactions.

Inconsistent results have also been observed in relation to cortisol levels and CU traits. In an adolescent cohort, basal cortisol levels were lower in males with high, relative to low parent-rated CU traits, after controlling for conduct problem severity (Loney, Butler, Lima, Counts, & Eckel, 2006). Similarly, resting state cortisol levels were lower in subgroups of offenders reporting high, relative to low psychopathic traits (Cima, Smeets, & Jelicic, 2008). However, measures of

basal cortisol did not correlate with CU traits in adolescent (Azar et al., 2004; Poustka et al., 2010) or adult (Feilhauer et al., 2013) samples. Feilhauer et al. failed to observe associations between basal cortisol and either callousness, uncaring, unemotional, or narcissistic traits. Variation in findings might reflect population heterogeneity and diverse definitions of antisocial behaviour (Hawes et al. 2009).

2.5 Facial Distress

Facial distress is thought to activate a similar subcortical pathway to that described in section 2.3 (page 24) (Amaral et al., 1992; LeDoux, 2000; Morris et al., 1996; Pessoa et al., 2002; Williams et al., 2001). Bilateral amygdala augmentation has been observed to fearful (Fusar-Poli et al., 2009; Vuilleumier et al., 2001) and sad (Fusar-Poli et al., 2009), relative to neutral facial expressions. Furthermore, in relation to threat, greater amygdala activation has been observed to fearful, relative to angry, facial expressions (Breiter et al., 1996; Phillips et al., 1997, 1998, 2004, *but see Murphy, Nimmo-Smith, & Lawrence, 2003 for a review*), and eyes (Morris, deBonis, & Dolan, 2002; Whalen et al., 2004), suggesting a greater amygdala bias towards distress.

Distress-evoked amygdala activation is central to the integrated emotion system (IES) associated with the VIM – reliant on intact amygdala functioning to extinguish antisocial behaviour (Blair, 2005). Patients with amygdala lesions evidence impaired recognition of fearful and sad (Adolphs et al., 1995, 1999; Blair, 1995, 2006; Calder, Lawrence, & Young, 2001; Lykken, 1957; Vuilleumier et al., 2004), but not happy (Adolphs et al., 1999) facial expressions. Furthermore, as the ability to label afraid body postures remains intact following amygdala lesioning (Adolphs & Tranel, 2003; Atkinson, Heberlein, & Adolphs, 2007), amygdala damage may impair recognition of *facial* distress, specifically.

Amygdala hypoactivation to fearful faces has been observed in adolescents exhibiting CU traits (Blair, 2003; Dadds, El Masry, Wimalaweera, & Guastella, 2008; Jones, Laurens, Herba, Barker, & Viding, 2009; Sterzer et al., 2005;

Sylvers, Brennan, & Lilienfeld, 2011), patients with antisocial personality disorder (ASPD) (Birbaumer et al., 2005), and schizophrenia (Dolan & Fullam, 2009) scoring high on psychopathy measures. Amygdala hypoactivation is also observed in children with conduct problems and attention deficit hyperactivity disorder (ADHD) scoring high on CU traits, relative to controls (Marsh et al., 2008). One recent investigation failed to observe differences in fear-evoked amygdala activation between adolescents with and without conduct problems (Lozier, Cardinale, Vanmeter, & Marsh, 2014). However, *post hoc* analysis, controlling for externalisation, reported an inverse correlation between CU traits and right hemispheric amygdala activity to fearful faces in the overall cohort - suggesting importance for teasing apart aggressive and CU traits.

As described in chapter one, section 1.3.4 (page 12), the successful perception of distress typically evokes an empathic response, critical to the activation of the VIM. On a neurobiological level, empathy is related to activation of the ventromedial prefrontal cortex (vmPFC) and inferior frontal gyrus (IFG). The vmPFC is a composite of the medial frontal and orbitofrontal cortices (OFC) (Bechara, Damasio & Damasio, 2000; Damasio, 1994) and is considered the limbic portion of the prefrontal cortex (PFC) (Pietrini, Guazzelli, Vasso, Jaffe, & Grafman, 2000). During face processing, the OFC, in particular, moderates amygdala activity by providing meaning to stimuli when resolving ambiguity (Ploghaus et al., 2000; Raine, 2013).

The vmPFC is a key brain region involved in the emergence of emotional self-reflection (Blair, 2013; Goubert et al., 2005; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004; *but see van der Meer, Johnson, Schmitzer-Torbert, & Redish, 2010 for review*). Lesioning of the vmPFC is related to deficits in cognitive empathy (Shamay-Tsoory, Aharon-Peret, & Perry, 2009; Shamay-Tsoory, Tomer, Berger, & Aharon-Peretz, 2003), with affective empathy/emotion recognition left intact (Shamay-Tsoory et al., 2009). Conversely, lesioning of the IFG, a region that receives projections from the fusiform gyrus and amygdala (Fairhall & Ishai, 2007), is associated with

impaired affective empathy and emotion recognition (Shamay-Tsoory et al., 2009). Self-reported affective empathy has been shown to predict IFG activation in response to affective facial stimuli (Jabbi, Swart, & Keysers, 2007). The IFG is typically activated during the passive viewing or imitation of emotional faces (Catmur, Walsh, & Heyes, 2009; Dapretto et al., 2006; Gallese, 2007; Heiser, Iacoboni, Maeda, Marcus, & Mazziotta, 2003; Schulte-Ruther, Markowitsch, Fink, & Piefke, 2007; Seitz et al., 2008), as well as tasks involving empathising with the suffering of others (Nummenmaa et al., 2008).

2.6 Executive Functioning

Although this chapter has framed the amygdala as a key brain region involved in affect-driven reactive aggression, humans are typically able to regulate aggressive behaviour through higher-order cognitive systems underpinned by prefrontal networks (Moffitt, 1993; Nauta, 1971). PFC activation is observed during response preparation, sustained motivation, self-control, and affect regulation (Giancola, Mezzich, & Tarter, 1998; Perdeci et al., 2010; Sotres-Bayon, Cain, & LeDoux, 2006; Tranel, Bechara, & Denburg, 2002) and so is thought to contribute to the containment of affect-driven reactive aggression (*see Davidson et al., 2000 for review*).

Previously, composite PFC hypoactivation has been observed in samples of murderers (Raine et al., 1994, 1998; Raine, Venables, & Mednick, 1997), ASPD (Anckarsater, 2006; Bassarath, 2001; Brower & Price, 2001; Hoptman, 2003; Pridmore, Chambers, & McArthur, 2005) and psychopaths exhibiting reactive, but not instrumental, aggression (Kiehl, 2006). Furthermore, use of diffusion tensor MRI, a method of imaging white matter connectivity, has shown dysfunctional fronto-limbic connectivity in antisocial individuals (Sundram et al., 2012). However, it is important to disentangle the underlying frontal networks to better understand their impact on aggression and CU traits. This section delineates the role of the anterior cingulate cortex (ACC), OFC, and IFG in executive functioning and inhibitory processes.

Typically functioning humans show considerable anatomical and functional connectivity between the OFC and amygdala (Amaral & Price, 1984; Freese & Amaral, 2009; Marsh et al., 2011; Pietrini et al., 2000). This network likely underpins script theory (Huesmann, 1988) and the appraisal mechanism described in the GAM (Anderson & Bushman, 2002*, see chapter one, section 1.3.2 [page 9]*). Specifically, the OFC is thought to moderate affect-driven amygdala responses by providing contextual meaning to social cues (Bechara et al., 2000; Damasio, 2003; Davidson, Jackson, & Kalin, 2000; Davidson, Putman, et al., 2000; Etkin et al., 2011; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Rolls, 1999; Saddoris, Gallagher, & Schoenbaum, 2005; Schiller & Delgado, 2010). OFC lesioning has been associated with increased reactive aggression, socially deviant behaviours, and poor affect recognition (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Grafman et al., 1996; Winstanley, Theobald, Cardinal, & Robbins, 2004). Contralateral amygdala-OFC connectivity during resting state inversely associated with trait anger (Fulwiler, King, & Zhang, 2012) and aggression (Hoptman et al., 2010).

Although OFC activation has been observed when experiencing anger in Dougherty et al. (2004), other evidence suggests healthy functioning individuals require OFC *deactivation* in order to imagine aggressive behaviour and to aggress in a video game setting (Pietrini et al., 2000). Baseline OFC hypoactivation might indicate vulnerability to poor affect regulation and antisocial tendencies (Antonucci et al., 2006; Beyer, Münte, & Krämer, 2014; Blair, 2001; Blair et al., 2002; Raine, Fung, & Lam, 2011) and has been observed in samples with comorbid schizophrenia/ASPD (Joyal, Dubreucq, Gendron, & Millaud, 2007), BPD (Goyer et al., 1994; Soloff et al., 2003), IED (Coccaro et al., 2007; Izquierdo & Murray, 2004; Izquierdo, Suda, & Murray, 2004; New et al., 2009), comorbid depression/aggression (Dougherty et al., 2004), as well as both adolescent (Marsh et al., 2011) and adult (Dolan & Fullam, 2009) samples with psychopathic traits. Furthermore, OFC activity evoked to angry facial expressions has been found to inversely correlate with

aggression - measured by the delivery of an aversive noise - in a community sample (Beyer et al., 2014).

The OFC has dense projections to the ACC (Bush, Luu, & Posner, 2000), a region thought to be involved in attention, conflict detection, motor inhibition, and affect regulation (Botvinick et al., 2001; Botvinick, Cohen, & Carter, 2004; Braver et al., 2001; Brown et al., 2006; Bush et al., 2000; Cardinal, Parkinson, Hall, & Everitt, 2002; Devinsky, Morrell, & Vogt, 1995; Garavan, Ross, Murphy, Roche, & Stein, 2002). Whilst the dorsal section of the ACC (dACC) is thought to be involved in decision-making processes such as conflict monitoring (Holroyd & Coles, 2002), choice selection, and performance monitoring (Frith, Friston, Liddle, & Frackowiak, 1991; van Veen & Carter, 2002), the ventral section of the ACC (vACC) is thought to be involved in representing and making judgements on stimuli, as well as regulating emotional behaviour (Bush et al., 2000; Devinsky et al., 1995; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008).

The ACC regulates affect via connections to the amygdala (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Ghashghaei et al., 2007). As such, ACC activation has been observed in response to facial threat (Blair et al., 1999) and distress (Blair et al., 1999; Morris et al., 1998), and during states of anger (Dougherty et al., 1999). Dougherty et al. evoked anger in a community sample using anger-inducing scripts, and found increased cerebral blood flow in the right dACC during affect regulation. However, although anger was confirmed using a psychometric assessment, typical physiological changes such as those described in chapter one, section 1.2.1.1 (page 3) were not observed.

ACC dysfunction has been reported in patients with schizophrenia (Reske et al., 2007), BPD (Tebartz van Elst et al., 2003), and CD (Stadler et al., 2007; Sterzer et al., 2005), characterised by aggressive traits. In both Sterzer et al. (2005) and Stadler et al. (2007), dACC deactivation was observed in aggressive children and adolescents, in response to negative affective images. Together, these

results suggest ACC dysfunction might predict vulnerability for poorer affect regulation, and in turn, antisocial and aggressive behaviour.

In response to a letter-based go/nogo paradigm, where participants are tasked with responding to one stimulus but not another, motor inhibition has been associated with neuronal transmission between the ACC, IFG, basal ganglia, and pre-motor regions (Steele et al., 2014). In addition to its role in the generation of empathy, the IFG has been shown to drive attention (Corbetta & Shulman, 2002), stop-signal detection (Garavan, Hester, Murphy, Fassbender, & Kelly, 2006; Mars et al., 2009; Rubia, Smith, Brammer, & Taylor, 2003; Schall et al., 2002), and motor impulse suppression (Antonucci et al., 2006; Aron et al., 2003). Though evident in the left hemisphere during simple inhibitory tasks (Konishi et al., 1999; Menon, Aldeman, White, Glover, & Reiss, 2001; Swick, Ashley, & Turken, 2008), IFG activity is maximal in the right hemisphere during tasks requiring extinction of a pre-initiated motor response (Aron, Robins, & Poldrack, 2004).

The IFG extracts stop-relevant information and uses it to both update working memory (Mars et al., 2008) and signal the need for motor extinction (Mostofsky & Simmonds, 2008; Verbruggen et al., 2010). As a function of pre-initiated behaviour being more difficult to disrupt than planned behaviour, IFG activity is thought to be greater to stop than nogo stimuli (Hughes, Fulham, Johnston, & Michie, 2012). During behaviour extinction, the IFG directly stimulates the superior temporal gyrus (STG) in order to quickly interrupt on going motor signals (Aron, Behrens, Smith, Frank, & Poldrack, 2007; Nambu et al., 2002). Subsequently, the IFG suppresses the basal ganglia, a region involved in motor behaviour (Aron, 2007; Aron & Poldrack, 2006; Rubia et al., 2001). As measured by a subdural electrode array, successful, relative to unsuccessful stopping is associated with greater activation of the right IFG (Swann et al., 2009).

The role of the IFG in motor extinction has been compounded across lesioning studies as well as task-based and structural MRI. Swick et al. (2008) reported a greater number of inhibitory errors in individuals with, relative to without IFG lesions. Furthermore, in samples with schizophrenia, characterised by inhibitory deficits, IFG activation has been inversely related to stopping times in response to auditory tones (Hughes et al., 2012). IFG grey matter has also been inversely associated with schizophrenic symptom severity (Suga et al., 2010).

Additional to IFG suppression of the basal ganglia, a comparison of haemodynamic responses to nogo and stop stimuli suggests a comparable network whereby the IFG moderates regions of the dorsomedial prefrontal cortex (dmPFC) such as the pre-supplementary motor area (Pre-SMA; Dum & Strick, 2005; Rubia et al., 2001). The Pre-SMA is critical for motor extinction (Aron et al., 2003, 2007; Chamberlain et al., 2008; Chambers et al., 2006; Floden & Stuss, 2006; Li, Huang, Constable, & Sinha, 2006). In particular, the dorsal Pre-SMA is associated with suppression of arm movements in monkeys (Mirabella, Pani, & Ferraina, 2011) and motor responses in humans (Aron et al., 2007; Floden & Stuss, 2006; Garavan et al., 2006; Nachev, Wydell, O'Neill, Husain, & Kennard, 2007; Rubia et al., 2003; Schall, Stuphorn, & Brown, 2002).

The dmPFC is thought to mediate executive control functions such as motor attention, conflict monitoring, and response selection (Rubia et al., 2001). A motor suppression task requiring the classification of angry and neutral faces was used to demonstrate increased Pre-SMA activation in samples of male students characterised by low (lower 15^{th} percentile), relative to high (upper 15^{th} percentile) aggressive scores (Rubia et al., 2003). However, stop signals in this task were not affect-driven, and instead relied upon a change in stimulus frame colour. Additionally, individuals exhibiting high, relative to low trait aggression evidenced Pre-SMA activation attenuation and longer reaction times when asked to classify angry and neutral facial expressions - inhibiting said response when followed by a change of frame colour (Pawliczek et al., 2013).

2.7 Chapter Summary

At its core, chapter two has framed reactive aggression as an interaction between a hyperactive amygdala-HPA axis response to threat and a hypoactive amygdala-PFC response to facial distress (McEllistrem, 2004; Morgan & Lilienfeld, 2000; Raine, 2013; Slever, 2002). However, this response is far from linear and not always guaranteed to result in the manifestation of aggression (Golden, Jackson, Peterson-Rohne, & Gontkovsky, 1996). Informed by the VIM, chapter two used supporting evidence from community, forensic, and clinical samples to describe how aggressive and CU traits might differentially modulate the amygdala responses to threat/distress, and in turn, how this might result in the disengagement of prefrontal brain networks associated with motor extinction. Still, key to the VIM, it remains to be seen how facial distress might moderate motor extinction when used as a stopping mechanism. Chapter three describes how these neurobiological networks might be indexed on an electrophysiological level, and how these markers might differ in samples characterised by aggressive/CU traits.

Chapter Three: Electrophysiological indices of aggression

3.1 Electroencephalography and Event-Related Potentials

Chapter three builds on the VIM-related neurobiological networks discussed in chapter two. Specifically, it describes how face processing, both in general and in response to facial threat/distress, as well as distress-induced motor extinction, might be indexed on an electrophysiological level using event-related potentials (ERP).

First described in humans by Berger (1929), amplified extracellular activity surrounding postsynaptic neurons can be measured at the scalp using electroencephalography (EEG). EEG serves as an inexpensive, non-invasive, and easily implementable method of indexing neuronal processes (Delle-Vigne, Wang, Kornreich, Verbanck, & Campanella, 2014). Compared to neuroimaging techniques reliant upon haemodynamic measures of brain activity, such as MRI, EEG commands high temporal - but low spatial - resolution and reflects the response from brain networks, rather than precise brain structures (Luck, 2012). High temporal resolution allows investigation of dynamic changes and neural network connectivity (Luck, 2005).

For effective measurement of EEG, neural generators need to be orientated in the same direction and share similar excitatory/inhibitory inputs. A collection of positively and negatively orientated generators would result in voltage being cancelled out to near zero. Therefore, summated voltage likely occurs in pyramidal cells; the main input-output cells of the cortex, perpendicularly aligned to the cortical surface (Luck, 2005, 2012).

Stimulus-evoked electrical voltage within a given generator travels through the brain, via the least resistant pathway. Upon reaching the skull, electrical activity spreads laterally as a function of high electrical resistance (Luck, 2005). Like a

voltmeter, EEG is measured between an active electrode and reference site, which can be adjusted for optimal voltage contrast.

EEG that is time-locked to the presentation of a stimulus or behavioural response - the ERP - has been mapped onto distinct information processing stages (Coles et al., 1990; Connor, 2002; Halgren, Marinkovic, & Chauvel, 1998). Components of the ERP encompass stimulus-related processing, alongside background electrical activity from baseline brain function. Several components make up an ERP waveform (Luck, 2006, 2013) and are visualised as negative or positive deflections around a baseline, which can be assessed for latency (measured in milliseconds) and amplitude (measured in millivolts).

Components temporally overlap, such that any given component may visually influence subsequent waves (Luck, 2006, 2013). This influence is predominant across earlier components and likely results in amplitude attenuation or augmentation, as well as latency elongation. By contrasting two experimental conditions, ERPs can be used to determine how specific cognitive processes might be influenced by a given experimental variable and so are useful assays of cognitive dysfunction (Donchin, 1979; Luck, 2013; Rugg & Coles, 1995).

Due to low signal to noise ratio (SNR), it is difficult to discriminate task-specific components from resting state EEG using individual trials (Luck, 2006). Noise is defined as variation in the data, which is not explained by a statistical model, and so should be reduced where possible. As noise is considered constant between trials, averaging data across several trials reduces trial-by-trial background noise to near-zero voltage (Halgren et al., 1998; Luck, 2005). Although trial-by-trial variation is lost, averaging acts to reduce noise and boost statistical power by removing unwanted fluctuations in the data. The residual ERP, mapped over multiple trials, sums task-related signal coherently, and results in a single task-specific response (Luck, 2006).

Typically, the first 50 ms of a given ERP waveform is thought to index basic brainstem activity, with electrophysiological activity between 50 and 100 ms

representing the processing of sensory perceptual information evoked in the primary sensory and frontal cortices. This is followed by the incorporation of primitive limbic processing at around 100 to 300 ms, before higher cognitive processing after 300 ms (Connor, 2002). The following sections discuss ERP components associated with the processes underpinning reactive aggressive behaviour (e.g. face processing, executive function, and distress-induced motor extinction), in addition to how they might be moderated by negative affect and aggressive/CU traits. Where known, ERP generators are related to the neurobiological underpinnings of reactive aggression discussed in chapter two.

3.2 Face Processing

Due to their high temporal resolution, ERPs provide a powerful tool to measure the cognitive efficiency of face processing, and allow for the capture of emotional and attentional processing at several stages (Chai, Castañón, Ongür, & Whitfield-Gabrieli, 2012; de Pascalis et al., 2005; Luck & Kappenman, 2013; Vuilleumier & Pourtois, 2007). Conscious processing of visual stimuli predominantly occurs between 150 and 800 ms, and is indexed by N170, P200, N200, and P300 ERP components. This section maps these components onto brain networks associated with face processing.

3.2.1 N170

Early ERP investigation reported large positive responses to visual stimuli at central-dorsal points of the scalp (i.e. the vertex) between 140 and 180 ms (Bötzel & Grüsser, 1989; Jeffreys, 1989). Coined the vertex positive potential (VPP), this positivity was accompanied by a negative deflection, the N170 (Bentin et al., 1996), at bilateral, occipito-temporal sites with origins in the temporal cortex (Jeffreys, 1996). Jeffreys suggested earlier ignorance of occipito-temporal ERPs was an artefact of EEG output being referenced to the mastoids, which attenuated occipito-temporal voltage. More recently, this claim has been supported by a meta-analysis of studies evoking N170 responses to facial expressions (Hinojosa et al., 2015). Here, Hinojosa et al. reported greater

effect sizes when referencing to the common average, relative to mastoid sites. Measurement of the N170 is preferential to the VPP as [1] electrodes are placed in closer proximity to generating structures, and [2] bilateral electrode placement allows for observation of lateralisation effects (Luck & Kappenman, 2013).

The neural origin of the N170 is thought to lie within the OFA, STS, and lateral and posterior (Deffke et al., 2007; Rossion, Joyce, Cottrell, & Tarr, 2003; Shibata et al., 2002) sections of the fusiform gyrus (Adolphs, 2002; Itier & Taylor, 2004; Nummenmaa, Hyönä, et al., 2010; Rossion et al., 2003; Sadeh et al., 2010; Watanabe, Kakigi, & Puce, 2003). The fusiform gyrus, in particular, is considered critical during face processing (Vuilleumier & Pourtois, 2007). Furthermore, intracranial recordings within the right OFA and fusiform gyrus have shown activation during the N170 time-window (Jonas et al., 2012; Parvizi et al., 2012).

Further support for the involvement of the fusiform gyrus and STS in N170 generation is provided by magnetoencephalography (MEG) and fMRI. Generation of the M170, a magnetic counterpart of the N170, has been localised within the fusiform gyrus (Deffke et al., 2007; Halgren, Raij, Marinkovic, Jousmäki, & Hari, 2000; Linkenkaer-Hansen et al., 1998). Additionally, positive correlations between N170 amplitude and activation of both the fusiform gyrus (Horovitz et al., 2004; Nguyen & Cunnington, 2014; Sadeh et al., 2010) and STS (Nguyen & Cunnington, 2014; Sadeh et al., 2010; Sato, Kochiyama, Uono, & Yoshikawa, 2008) have been measured using concurrent EEG and fMRI in response to neutral faces.

The N170 is evoked to visual stimuli including words, objects, and faces. In particular, faces, compared to objects such as houses, increase postsynaptic activity (Luck & Kappenman, 2013) and evoke larger N170 deflections (Allison et al., 1999; Crist, Wu, Karp, & Woldorff, 2008; Holmes et al., 2005). N170 augmentation is observed to a variety of facial stimuli including line drawings,

cartoons, schematic representations, gray-scale, and isolated eyes (Bentin et al., 1996; Carmel & Bentin, 2002; Eger, Sterzer, Russ, Giraud, & Kleinschmidt, 2003; Krombholz, Schaefer, & Boucsein, 2007; Rossion, 2014; Rossion & Jacques, 2011) and interacts positively with increased stimulus intensity (Sprengelmeyer & Jentzsch, 2006). The face-evoked N170 is characterised by right hemispheric dominance (Bentin et al., 1996; Hinojosa et al., 2015; McCarthy, Puce, Gore, & Allison, 1997; Rossion et al., 2003; Stockdale, Morrison, Kmiecik, Garbarino, & Silton, 2015), regardless of the presence of eyes (Magnuski & Gola, 2013), valence (Polich & Herbst, 2000), or whether the face is static or dynamic (Caharel, et al., 2013).

The N170 can be modulated through experimental manipulations. Presentation of stimuli altered through contrast reversal (Itier & Taylor, 2002), inversion (Bentin et al., 1996; Eimer, 2000b; Jacques, d'Arripe, & Rossion, 2007; Rossion et al., 2000), degradation of quality (Holmes et al., 2005), and the scrambling of facial features (Zion-Golumbic & Bentin, 2007), both prolong and augment the N170 response. Conversely, although not always evident (Huddy et al., 2003; Mnatsakanian & Tarkka, 2004), repeatedly presenting the same facial stimulus acts to attenuate the N170 response (Campanella et al., 2000; Itier & Taylor, 2002). N170 attenuation is also observed in tasks where stimuli are presented over varying sizes and modalities (Jacques et al., 2007). Taken together, these findings suggest that a greater number of neural resources are recruited when faces are more difficult to identify (Rossion et al., 2000).

3.2.2 P200

Directly following the N170, a positive deflection occurs in the ERP waveform around 200 ms post stimulus onset - the P200. The P200 is thought to index the encoding of task salient features (Potts & Tucker, 2001) and is evoked to both visual and auditory stimuli (Potts, Dien, Hartry-Speiser, McDougal, & Tucker, 1998; Potts, Liotti, Tucker, & Posner, 1996). Although distributed over anterior electrode sites during target detection tasks (Carretié et al., 2004; Potts, 2004; Potts & Tucker, 2001), the P200 shows an occipito-temporal distribution, with

maximal activation over the right hemisphere during face processing (Farah, 1990; Watanabe, Kakigi, Koyama, & Kirino, 1999).

Neural generators of the face-evoked P200 are largely unknown (González-Roldán et al., 2011). However, one investigation has measured brain activity of epilepsy patients using depth electrodes implanted into temporal, occipital, and parietal brain regions (Halgren et al., 1994). Approximately 180 ms post stimulus onset, Halgren et al. reported increased bilateral activation in the fusiform gyrus in response to faces. Although the colour and size of facial stimuli were not controlled for, fusiform gyrus activation was observed in all six patients. Two patients showed fusiform gyrus activation exclusively for faces, and three patients evidenced increased fusiform gyrus activation to faces, relative to words, letters, or symbols. Furthermore, whilst not specific to facial stimuli, activation was recorded in the posterior cingulate, as well as supramarginal, posterior superior and middle temporal, and parahippocampal gyri, between 140 and 280 ms post stimulus onset.

Previously, P200 augmentation has been observed in response to affective pictures (Carretié et al., 2001, 2004; Delplanque, Lavoie, Hot, Silvert, & Sequeira, 2004), words (Kanske & Kotz, 2007; Kissler et al., 2006; Schapkin, Gusev, & Kuhl, 2000) and facial expressions (Ashley, Vuilleumier, & Swick, 2004; Eimer et al., 2003; Schulz, Kaufmann, Kurt, & Schweinberger, 2012), relative to respective neutral or non-affective controls. As such, the P200 might reflect the enhanced activation of the amygdala (Vuilleumier & Pourtois, 2007) - sensitive to distinct features associated with emotion portrayal (Begleiter et al., 1979; Carretié & Iglesias, 1995). Specifically, in response to faces, the P200 likely reflects processing of second order, configural facial features (Itier & Taylor, 2004; Latinus & Taylor, 2006).

3.2.3 N200

The N200 is a negative deflection in the ERP that is maximal over right anterior sites between 175 and 300 ms post stimuli onset (Bokura, Yamaguchi, & Kobayashi, 2001; Bruin & Wijers, 2002; Heinze, Munte, Gobiet, Niemann, & Ruff, 1992; Strik, Fallgatter, Brandeis, & Pascual-Marqui, 1998). Among other processes associated with executive functioning (*see section 3.4.1 [page 50]*), the N200 is thought to index automatic alerting, stimulus classification, and template matching (Botvinick et al., 2001; Heinze et al., 1992; Luck, 2005; Sumich et al., 2013; van Veen & Carter, 2002; Yeung et al., 2004, *but see Folstein & van Petten, 2008 for review*).

In response to faces, the N200 likely reflects involvement of the vACC when integrating amygdala-projected emotionally salient information (Carretié et al., 2004; Kanske & Kotz, 2010b). Additionally, N200 generation has been localised to other frontal-temporal brain regions including the lateral OFC, dorsolateral prefrontal cortex (dlPFC), hippocampus, and superior temporal cortex (Bekker, Kenemans, & Verbaten, 2005; Bokura et al., 2001; Nieuwenhuis, Yeung, & Cohen, 2004; van Veen & Carter, 2002). During maturation of the brain, connections within these networks are pruned in order to increase inter-cellular communication (Chandrasekaran, Plas, Gonzalez, & Crair, 2005). As evidenced in community and ADHD samples, this pattern of maturation, indexed by N200 attenuation, is inversely correlated with age (Sumich et al., 2012).

3.2.4 P300

The final component of interest, the P300, is a positive deflection in the ERP occurring around 300 ms post stimulus onset (Bauer & Hesselbrock, 1999). The P300 is thought to reflect processes involved during information processing (Hillyard & Kutas, 1983; Polich & Herbst, 2000), such as the effortful/automatic orientation of attention and evaluation of incoming stimuli against pre-existing working memory representations (Donchin & Coles, 1988; Ito, Larsen, Smith, & Cacioppo, 1998; Nieuwenhuis, Aston-Jones, & Cohen, 2005; Polich & Kok, 1995). Although largest at posterior sites overall (Baumeister et al., 2014), task

condition modulates the P300, such that responses are maximal at central posterior sites in response to target/go stimuli (Luck, 2005) and central anterior sites in response to nogo stimuli (Baumeister et al., 2014; Bokura et al., 2001; Fallgatter et al., 2005).

During face processing, the P300 is thought to index voluntary evaluation of facial emotion (Halgren et al., 1994) and cognitive processing capacity (Donchin & Coles, 1988; Polich, 2003). Whilst P300 augmentation suggests that fewer neural resources are required to attend to environmentally/motivationally relevant stimuli (Bauer & Hesselbrock, 1999; Branchey, Buydens-Branchey, & Liever, 1988; Harmon-Jones, Barratt, & Wigg, 1997; Polich, 2007), P300 attenuation likely reflects unorganised resource allocation, poor attention to facial stimuli, and deficient cognitive functioning (Meier, Perrig, & Koenig, 2012).

The P300 is generated by a large neuronal network, which spans several cortical and subcortical regions (Duncan et al., 2009). On account of their roles in attention, perception, and memory, the cingulate, as well as bilateral frontal, parietal, and limbic cortices have been associated with P300 generation (Volpe et al., 2007). Although source localisation of the P300 to the ACC is considered highly validated (Pizzagalli, Oakes, & Davidson, 2003), more recent evidence suggests additional generation within the insula, OFC, as well as the frontal gyrus, hippocampus, and dlPFC of the right hemisphere (Bocquillon et al., 2011; Campanella et al., 2013; O'Connell et al., 2012).

3.3 Facial Threat and Distress Processing

In section 3.2 (page 39) the N170, P200, N200, and P300, were discussed both in terms of how they might index stages of face processing, and how they might map onto associated neural networks described in chapter two. With facial affect thought to alter the posterior EEG signal as early as 80 to 100 ms post-stimulus onset (Hillyard, Mangun, Woldorff, & Luck, 1995), the following section explores how ERP responses to faces might be moderated as a function of facial threat and distress.

3.3.1 N170

It has been suggested that electrophysiological findings supporting N170 insensitivity to facial affect are relatively concurrent (Rellecke, Sommer, & Schacht, 2013). Previously, although not measured at optimal temporo-occipital sites in some investigations (e.g. Chai et al., 2002), amplitude variation has not been evidenced when comparing the N170 response to neutral, happy, fearful, and disgusted facial stimuli in community (Ashley et al., 2004; Bobes, Martin, Olivares, & Valdes-Sosa, 2000; Chai et al., 2012; Eimer et al., 2003; Eimer & Holmes, 2002, 2007; Halgren et al., 2000; Herrmann et al., 2002; Krolak-Salmon, Fischer, Vighetto, & Mauguiere, 2001; Stockdale et al., 2015) or psychopathic (Eisenbarth et al., 2013; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002) samples. Such findings lead some to consider the N170 as a neural index of early structural and configural face encoding - preceding the identification of emotion (Ashley et al., 2004; Eimer, 2000; Eimer & Holmes, 2002, 2007; Holmes, Kiss, & Eimer, 2006; Ioannides, Liu, Kwapien, Drozdz, & Streit, 2000; Polich & Herbst, 2000).

Recently, debate surrounding the influence of facial affect on the N170 has been expanded via a meta-analysis of fifty-seven investigations (Hinojosa et al., 2015). This analysis sought to delineate whether facial affect moderated the N170 response, and if so, whether this moderation was emotion-specific or uniform across facial expressions. Results suggested that whilst N170 responses to faces depicting sadness and disgust did not differ from those evoked to neutral facial stimuli, N170 responses to angry, fearful, and happy faces were augmented. This finding conflicts the independent face processing model discussed in Bruce & Young (1986, *see chapter one, section 1.3.1 [page 7]*) in that rather than identity and expression being processed over separate routes, the N170 might better index an integrated face processing mechanism. Although the emotion-specific hierarchy of N170 activation observed in Hinojosa et al. (2015) might be partially explained by a dearth of investigations using sad and disgusted facial stimuli, the authors explained these findings in

terms of fearful, angry, and happy facial expressions having increased biological significance.

Angry and fearful facial expressions are negatively valenced, which, relative to neutral and positively valenced stimuli, have been shown to augment the N170 (Smith, Cacioppo, Larsen, & Chartrand, 2003). Angry faces have elicited N170 augmentation across three different visual paradigms. First, in an emotion classification task, though not replicated in their schizophrenic sample, Ibáñez et al. (2012) concluded angry, relative to happy facial expressions, evoked larger N170 amplitudes in a non-clinical cohort. Second, similar findings were observed using schematic facial stimuli, comparing negatively- (e.g. angry), to positively- (e.g. happy) valenced and neutral face drawings (Eger et al., 2003). Third, when priming schematic faces with affect-based words, larger N170 amplitudes were evoked to angry, compared to happy faces, irrespective of the valence of the prime word (Krombholz et al., 2007).

Similarly, N170 augmentation (Ashley et al., 2004; Batty & Taylor, 2003; Campanella, Quinet, Bruyer, Crommelinck, & Guérit, 2002; Ramos-Loyo, Gonzalez-Garrido, Sanchez-Loyo, Medina, & Basar-Eroglu, 2009; Rotschtein et al., 2010; Shannon, Patrick, Venables, & He, 2013; Williams et al., 2006) and shorter latencies (Dawson, Webb, Carver, Panagiotides, & McPartland, 2004; Taylor et al., 2004) have been observed to fearful, relative to neutral, angry, and happy facial expressions. These findings suggest that whilst both angry and fearful facial stimuli augment the N170, possibly via similar threat-specific networks discussed in chapter two, sections 2.3 (page 24) and 2.5 (page 29), distress might elicit an additive response.

Some investigators propose affect-driven N170 responses might be a function of voluntary attention (Holmes et al., 2003) and/or visual priming (Krombholz et al., 2007). Previously, though not observed in paradigms requiring participants to classify faces from objects, or males from females (Boucsein, Schaefer, Sokolov, Schroder, & Furedy, 2001), N170 augmentation to affective, relative to

neutral expressions, has been observed in paradigms requiring emotion classification (Eimer, 2000c; Ibánez et al., 2012). In support of this, N170 responses to facial stimuli were augmented when primed with affectively valenced words (Krombholz et al., 2007). In such cases, priming is thought to orientate voluntary attention to the affective elements of stimuli, resulting in augmentation of the N170 response.

3.3.2 P200

When contrasting P200 responses between emotionally valenced (i.e. pleasant and unpleasant), and neutral stimuli, P200 augmentation has been observed to unpleasant stimuli, across task modalities (Carretié et al., 2004; Delplanque et al., 2004; Gerdes et al., 2013; Olofsson & Polich, 2007). Whilst the P200 reported in Carretié et al. (2004) was measured at frontal sites during a target detection task, P200 augmentation has been observed at occipital-parietal sites during a repetition task (Olofsson & Polich, 2007), and when the affective stimuli were either accompanied by a concurrent auditory stimulus (Gerdes et al., 2013), or characterised by low arousal levels (Delplanque et al., 2004).

When evoked to facial expressions, although P200 augmentation has been observed in response to neutral faces in one investigation (Tortosa, Lupiáñez, & Ruz, 2013), P200 augmentation is typically considered a function of threat intensity (Gonzalez-Roldan et al., 2011). Previously, angry facial expressions have evoked larger P200 amplitudes, and shorter peak latencies, in a large community sample during an emotional oddball paradigm (Chai et al., 2012). As P200 augmentation is also observed in videogame settings in response to pistol-laden targets, compared to those without weapons (Correll, Urland, & Ito, 2006), it is likely that the P200 is responsive to biological relevant social cues (Carretié et al., 2004).

Personality and affective priming might modulate enhanced positivity around 200 ms. Previously, individuals reporting anxious personality traits, characterised by hyper-attention to threat (Zhang, Li, & Zhou, 2008) and

individuals with depression, known to orientate their attention to distress (Dai & Feng, 2012; Gotlib & Joormann, 2010), have shown larger P200 amplitudes to facial threat and sadness, respectively. Furthermore, as observed for the N170 (*see section 3.3.1 [page 45]*), P200 amplitude might function as an interaction between facial affect and priming. In a paradigm whereby facial expressions were preceded by affective words, acting to orientate attention towards facial affect, P200 responses to fearful facial expressions were significantly larger than for neutral faces (Shannon et al., 2013). Together, P200 augmentation to biologically salient stimuli, such as threat and distress, suggests the P200 might index a threat attention bias and preferential processing of biologically significant stimuli.

3.3.3 N200

In respect to the N200, the perception of angry, relative to neutral and happy facial expressions has been associated with shorter N200 latencies (Feldmann-Wüstefeld, Schmidt-Daffy, & Schübo, 2011; Weymar, Low, & Hamm, 2011) and augmented N200 amplitudes in child (Lewis, Todd, & Honsberger, 2007; Nelson & Nugent, 1990; Todd, Lewis, Meusel, & Zelazo, 2008) and adult (Holmes et al., 2014) samples. Similarly, N200 augmentation has been observed in response to fearful faces (Dennis & Chen, 2007). Although not observed in Thomas, Johnstone, & Gonsalvez (2007), larger N200 amplitudes have been elicited in response to negatively valenced, relative to neutral words (Kanske & Kotz, 2010). The null finding in Thomas et al. (2007) might be explained by the N200 being captured over a long epoch that possibly encompassed multiple positively and negatively deflected components, in addition to the N200 - thus attenuating the effect of valence.

Threat-driven N200 augmentation likely reflects threatening social cues capturing attention and commanding greater neural resources in order to redirect attention away from threat during conflict resolution. Recently, Holmes et al. (2014) evidenced enhanced N200 responses to angry facial expressions after depleting cognitive resources using a complex number recall paradigm. In

this investigation, depletion of cognitive resources was thought to detriment the ability to divert attention away from angry facial expressions and so additional resources, indicative of an enhanced N200 response, were required to resolve this process.

3.3.4 P300

Though predominant in response to negative stimuli (Yee & Miller, 1987), P300 augmentation has been observed to positively and negatively valenced, relative to neutral, non-facial stimuli (Delplanque, Silvert, Hot, & Sequeira, 2005; Keil et al., 2002; Mardaga & Hansenne, 2009). When evoked to faces, angry, relative to happy faces are thought to augment the amplitude (Lang et al., 1990) and elongate the latency (Morita, Morita, Yamamoto, Waseda, & Maeda, 2001) of the P300 response. These patterns are indicative of the biological significance of threat demanding a stronger neuronal response.

Similarly, P300 amplitude augmentation is observed to facial distress. Relative to neutral facial stimuli, sad (Ehlers, Walls, Garcia-Andrade, & Phillips, 2001) and fearful (Campanella et al., 2013) faces have elicited P300 augmentation in samples of Mission Indians and individuals from the community, respectively. Interestingly, whilst Ehlers et al. reported P300 amplitude augmentation to sad, relative to happy faces, the P300 evoked to happy faces reported in Campanella et al. was equal to that evoked to fearful faces. Therefore, although fear, relative to sadness might augment N170/P200 responses (*see sections 3.3.1 [page 39] and 3.3.2 [page 41]*), it is possible that sadness better moderates the P300.

3.3.5 Threat/Distress Processing Summary

Taken together, as indexed on an electrophysiological level, there is strong evidence to suggest that facial threat (e.g. anger) and distress (e.g. fear, and to some extent sadness) might moderate face processing across several distinct stages. As a function of their biological and social significance (*see chapter*

one, section 1.3.1 [page 7]), facial threat and distress appear to [1] command greater neural resources during early stages of structural and affect processing (i.e. N170, P200), [2] capture attention (i.e. N200), and [3] yield a stronger neural response during stimulus evaluation (i.e. P300).

3.4 Executive Function

As discussed in chapters one (*see section 1.3.3 [page 11]*) and two (*see section 2.6 [page 31]*), humans are typically able to moderate their thoughts and behaviour through executive functioning. This section describes the N200 and P300 responses evoked during experimental conditions whereby the inhibition, or extinction of a motor response is required. Motor extinction, in particular, is a core aspect of the VIM and so associated ERP responses likely serve as an index of motor extinction efficiency in response to stop stimuli.

3.4.1 N200

Evoked to stimuli denoting the need to inhibit (nogo-) or extinguish (stop-) a motor response, the N200 is thought to reflect pre-motor cognitive control (Huster et al., 2013; Kopp, Rist, & Mattler, 1996; Patel & Azzam, 2005), and is associated with response selection (Jodo & Kayama, 1992; Kok, 1986; Schmajuk et al., 2006). During inhibitory conditions, competing go and nogo/stop responses create conflict and result in the recruitment of compensatory attentional mechanisms in order to assist response selection (Botvinick, 2007; Botvinick, Braver, Barch, Carter, & Cohen, 2001; Kok, 1986; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). This mechanism is evidenced on an electrophysiological level by augmentation of the N200 response during inhibitory, relative to go conditions (Baumeister et al., 2014; Eimer, 1993; Enriquez-Geppert et al., 2010; Falkenstein, Hoormann, & Hohnsbein, 1999; Folstein & van Petten, 2008; Liu et al., 2015; van Boxtel, van der Molen, Jennings, & Brunia, 2001).

Conflict monitoring efficiency during motor inhibition tasks might be moderated by stimulus frequency. With the exception of Pfefferbaum, Ford, Weller, & Kopell (1985), decreasing the frequency of task-relevant go and nogo stimuli is thought to augment the N200 response (Nieuwenhuis et al., 2003), irrespective of whether an overt (e.g. button press) or covert (e.g. mental counting) response is required. The null finding reported in Pfefferbaum et al. (1985) might be explained by [1] the use of word strings as go/nogo stimuli, instead of letters, symbols, or auditory tones used elsewhere (*see Nieuwenhuis et al., 2003*), and/or [2] the observation of a short P300 latency. Pfefferbaum et al. argued that the short P300 latency, which was inversely correlated with N200 amplitude, restricted full development of the N200 response.

On a neurobiological level, the inhibitory N200 is thought to reflect activation within the superior- and medial-frontal gyri, OFC, and ACC (Amodio, Master, Yee, & Taylor, 2008; Enriquez-Geppert et al., 2010) - an inhibitory network discussed throughout chapter two, section 2.6 (from page 31). This generation is further supported by EEG source localisation of a letter-based go/nogo oddball paradigm (Nieuwenhuis et al., 2003). Though based on a small sample, Nieuwenhuis et al. explained most of the variance in anterior N200 generation by a dipole based in the ACC during nogo trials. When inhibition is successful, activation of frontal executive networks manifests in large, anterior scalp distributions (Falkenstein et al., 1999; Jodo & Kayama, 1992). However, although limited to the investigation of ADHD samples, when inhibition is unsuccessful, the inhibitory N200 manifests over a bilateral, inferior-posterior scalp distribution (Dimoska et al., 2003; Pliszka, et al., 2000). This latter distribution is thought to represent a preparatory response to the presentation of stop stimuli in the absence of concurrent motor extinction (Pliszka, et al., 2000).

3.4.2 P300

The P300 is thought to index aspects of executive function (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000), such as response inhibition when evoked during nogo and stop conditions (Bruin & Wijers, 2002; Bruin, Wijers, &

Staveren, 2001; Burle et al., 2004; Fallgatter et al., 2005; Pfefferbaum et al., 1985; Rubia et al., 2001). Maximal at anterior midline sites (De Jong et al., 1990; Dimoska, Johnstone, & Barry, 2006; Kok, Ramautar, de Ruiter, Band, & Ridderinkhof, 2004), the P300 response is larger during inhibitory, relative to go trials (Bekker et al., 2004; Polich, 2007) and augments further following successful, relative to unsuccessful response inhibition (Bekker et al., 2005; De Jong et al., 1990; Hughes et al., 2012; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; Overtoom et al., 2002). Whereas P300 augmentation during successful inhibition is indicative of an efficient motor extinction mechanism (Dimoska et al., 2006), the P300 evoked during unsuccessful stops might better index error related, and/or conflict processing (Bekker et al., 2005).

P300 responses to nogo and stop trials are thought to recruit similar underlying networks (van Boxtel et al., 2001), involving the IFG, insula, and ACC (Bae, Kim, Im, & Lee, 2011). As delineated in chapter two, section 2.6 (page 31), the IFG is thought to play a stronger role in motor extinction than motor inhibition tasks (Enriquez-Geppert et al., 2010; Mattia et al., 2012). Enriquez-Geppert et al. tasked participants with categorising coloured squares with infrequent stop signals. Results suggested the stop-P300 was driven by bilateral IFG activation.

3.5 Negative Affect

Throughout chapter two, the generation of negative affect was localised to subcortical/limbic networks. However, as EEG is restricted to the study of cortical generators (Luck, 2005), investigation into the associations between anger and ERPs is scarce (O'Connell et al., 2012). This section delineates the existing anger/ERP literature in terms of both trait and state anger.

Trait anger, the disposition to experience anger, has been associated with augmentation of the P300 response in some (Liu et al., 2015; Stewart et al., 2010), but not all (Bond & Surguy, 2000; Harmon-Jones et al., 1997) investigations. Using an emotional-word Stroop paradigm (Stroop, 1992), Stewart et al. (2010) evidenced a positive association between the parietal

P300 evoked to negative stimuli and trait anger. Similar results have been obtained using a visual go/nogo paradigm (Liu et al., 2015). In a contrast between high- and low-scoring respondents on a psychometric assessment of outward anger, Liu et al. (2015) evidenced P300 augmentation and faster reaction times in the high-anger group, during the go condition. However, the inverse was observed in the nogo condition, such that lower P300 amplitudes were reported in the high-, relative to low-anger group. Together, these results suggest that individuals with high trait anger show hyperarousal to task-relevant stimuli and are less efficient at recruiting inhibitory resources. Conversely, when evoked to auditory stimuli, no correlation was observed between trait anger and P300 amplitude in child (Harmon-Jones et al., 1997) and adult (Bond & Surguy, 2000) samples.

ERPs have also been modulated through state anger. Although not a direct measure of anger, Stockdale, Morrison, Kmiecik, Garbarino, & Silton (2015) evoked an increase in negative mood and a decrease in positive mood, using a violent film. Subsequently, Stockdale et al. reported a bilateral decrease in N170 amplitude, and a fronto-midline decrease in P200 amplitude, in response to both fearful and happy facial expressions. This finding suggests exposure to violence results in acute decrement to face processing overall. Conversely, the P200, recorded over midline-parietal sites in Bertsch, Böhnke, Kruk, & Naumann (2009), was augmented in response to fearful and angry faces following the induction of anger by way of aversive blasts of white noise. This investigation suggests anger induction evokes hypervigilance to facial threat. However, as anger was induced using varying methodologies, and the P200 was evoked and measured using different tasks and scalp sites, these two investigations are not comparable.

Anger-induced moderation of the N200 and P300 has been observed in investigations where adolescents were tasked with a visual go/nogo paradigm that induced anger via a sudden loss of ‘points’ (Lewis et al., 2006; Stieben et al., 2007). In Lewis et al., following anger induction, both inhibitory N200 and

P300 responses measured over fronto-midline sites were augmented. Similarly, in a comparison between adolescent samples characterised by externalising, or both externalising and internalising traits, relative to non-clinical controls, Stieben et al. evidenced nogo-N200 augmentation in the mixed trait - and in turn externalising - cohorts following anger induction. This investigation did not report findings for the P300. Furthermore, augmentation of inhibitory N200 and P300 responses were reported in Stockdale et al. (2015), after viewing a negative mood-inducing film (*see above for paradigm specifications*).

Taken together, anger, both at state and trait level, might moderate electrophysiological indices of face processing and motor inhibition. Specifically, it appears that anger is associated with fewer cognitive resources being spent on processing target stimuli, which in the context of simple behavioural paradigms, might reduce conflict and facilitate inhibition. However, as observed in chapter one, section 1.2.1.1 (page 2), more intense episodes of anger can result in an unrestrained behavioural response. Further investigation, using clear quantifications of anger, should be carried out in order to better understand associations between anger induction and ERPs.

3.6 CU and Aggressive Traits

EEG provides a useful tool for investigating how CU and aggressive traits moderate distinct cognitive processes associated with facial affect recognition and motor extinction. The use of electrical activity when investigating aggressive traits can be traced back to Hill & Sargant (1943), who evidenced electrical abnormalities in a male who murdered his mother for no identifiable reason. This section considers how the N170, P200, N200, and P300 might manifest in samples characterised by aggressive and CU traits. Where appropriate, these findings are discussed in the context of the VIM.

3.6.1 N170

Individuals characterised by aggressive and CU traits evidence disruption to the fusiform-STS-amygdala network (*see chapter two, section 2.3 [page 24]*), which in turn, might reflect on an electrophysiological level through N170 attenuation (*see section 3.2.1 [page 39]*). Therefore, the N170 represents a useful index for investigating the roles of aggression and CU traits on early stages of face processing and emotion classification. However, this association has only been investigated in young adults with poor emotional expression (Meaux et al., 2014), and clinical samples with schizophrenia (Ibáñez et al., 2012), bipolar disorder (Degabriele, Lagopoulos, & Malhi, 2011), and high, relative to low psychopathic traits (Eisenbarth et al., 2013). Whilst these samples are characterised by emotional bluntness and aggression, to date no investigation has specifically associated the N170 with aggressive or CU traits.

Meaux et al. (2014) presented young adults with facial expressions depicting sadness, fear, anger, disgust, surprise, and happiness. Though not a function of emotion - possibly resulting from referencing to bilateral mastoid sites (*see section 3.2.1 [page 39]*) - the average N170 response was inversely correlated with emotional expressivity, a qualitatively similar concept to CU traits (Kimonis et al., 2008). Furthermore, although not supported by behavioural data, N170 responses were lower in samples of schizophrenia (Ibáñez et al., 2012) and bipolar (Degabriele et al., 2011) patients, relative to controls. In these investigations, the N170 was evoked to happy, angry, and sad facial expressions, and was not modulated by affect.

In contrast to the findings above, differences in N170 amplitude were not observed between groups of female psychopaths scoring high and low on the Psychopathy Checklist - Revised (PCL-R; Hare, 1991) in response to fearful, angry, and happy facial expressions (Eisenbarth et al., 2013). This null result could be explained through task parameters. Although all paradigms required face processing, Ibáñez et al. and Degabriele et al. asked participants to view and respond to target faces irrespective of emotion, and Eisenbarth tasked

participants with empathising with the target stimuli. The latter might have implicated an alternative processing mechanism.

3.6.2 P200

Similar to the N170 (*see section 3.6.1 [page 55]*), to date, the P200 has not been investigated in association with aggressive or CU traits, specifically. Previously, P200 responses to neutral facial expressions presented in parallel with an aversive odour have been observed to be lower in small samples of psychopathic, relative to non-psychopathic males (Flor et al., 2002). Furthermore, in young adults derived from the community, P200 amplitudes were inversely correlated with emotional control, a key factor in regulating the emotional urges that precede an aggressive response (Meaux et al., 2014, *see chapter one, section 1.3.2 [page 9]*).

Whilst similar findings have not been replicated in a comparison between individuals with, and without bipolar disorder (Degabriele et al., 2011), this null finding might be an artefact of the P200 being measured from a composite of left and right occipito-parietal sites. This method might mask subtle hemispheric effects. Nevertheless, these findings suggest P200 attenuation to facial stimuli might be observed in samples characterised by aggressive/CU traits, and warrants more specific, hypothesis-driven investigation.

3.6.3 N200

In contrast to the limited number of investigations associating aggressive/CU traits with the N170 (*see section 3.6.1 [page 55]*) and P200 (*see section 3.6.2 [page 56]*), these personality traits have been investigated more frequently, using a variety of paradigms and stimuli, in association with the N200 and P300.

Although not observed in two samples of male offenders, relative to non-offender controls (Barratt et al., 1997; Munro et al., 2007), inhibitory N200

responses have shown attenuation in forensic samples and samples characterised by externalising traits. Over a series of oddball and Flanker paradigms, which used simple keyboard characters/arrows as stimuli, male offenders characterised by high impulsivity and violent tendencies, relative to age-matched controls, evidenced smaller anterior midline inhibitory N200 responses (Chen et al., 2005, 2008, 2014). Furthermore, in a sample of juvenile male offenders, incarcerated for extreme and violent crimes, relative to controls, exhibited attenuated N200 responses during motor inhibition trials (Vilà-Balló et al., 2014). Such findings suggest that externalising and aggressive traits might detriment preparation of an inhibitory response. Similarly, in non-forensic samples, the inhibitory N200 has been attenuated in cohorts of children with, relative to without, ADHD (Dimoska et al., 2003; Pliszka et al., 2000). Whilst Pliszka et al. (2000) observed right anterior N200 attenuation in response to visual stop signals, Dimoska et al. (2003) observed midline anterior N200 attenuation to auditory stop signals.

Variation in N200 amplitude has also been observed as a function of CU traits (Kiehl, Bates, Laurens, Hare, & Liddle, 2006; Sumich et al., 2012). In Kiehl et al., the N200 evoked to auditory tones at midline sites, was larger in male inmates scoring high, relative to low, on measures of psychopathy. Furthermore, although an inverse relationship between age and N200 amplitude has been observed in young males with, and without ADHD, this association was not replicated in a cohort of males with CD (Sumich et al., 2012). In this study, the N200 was evoked to letters and was measured in response to background stimuli in order to better index cortical maturation without drawing on networks involved in executive functioning. Together, these findings suggest that CU traits might be associated with delayed cortical remodelling and poorly developed brain function as described in section 3.2.3 (page 43) (de Brito et al., 2009; Sumich et al., 2012).

In contrast to these findings, one investigation has reported N200 attenuation in offenders with psychopathy, relative to offenders with and without schizophrenia

(Kiehl, Smith, Hare, & Liddle, 2000). Although N200 augmentation was expected, this result could be an artefact of either aggressive traits not being controlled for, a lack of comparison to a non-offender cohort, and/or the use of an equal ratio of go/nogo stimuli. Stimulus frequency has been shown to moderate N200 amplitude (*see section 3.4.1 ([page 50]*), and a 50:50 ratio of stimuli has been thought to attenuate the inhibitory N200 response (Kok, 1986). Additionally, associations between CU traits and N200 amplitudes were not observed in samples of comorbid ADHD and CD outpatients (Du et al., 2006) or aggressive offenders with psychopathic traits (Munro et al., 2007), relative to controls. However, these findings might be explained by the N200 being measured over a large 200 ms epoch in Du et al. and baseline aggressive traits not being controlled for in Munro et al.

3.6.4 P300

First reported in alcohol dependency (Porjesz, Begleiter, & Garozzo, 1980; *see Polich et al., 1994 for review*), early P300 investigations found reduced P300 amplitudes in alcohol- (Bauer, O'Connor, & Hesselbrock, 1994), cocaine- (Bauer, 1997), and substance- (Iacono, Carlson, Malone, & McGue, 2002) dependent patients with, relative to without, antisocial personality traits. In these investigations, the P300 was evoked using visual (e.g. letter, light, schematic faces) oddball paradigms, and showed specific attenuation over anterior midline sites.

More recently, variation in P300 amplitude has been shown to index a wide spectrum of externalising disorders characterised by poor inhibitory control (Hicks et al., 2007; Iacono, Malone, & McGue, 2003; Pandey et al., 2012). For example, using variants of the visual oddball paradigm, parietal P300 amplitude attenuation has been observed in large cohorts of individuals with severe ADHD symptoms (Brandeis et al., 2002), and has been confirmed through meta-analysis (Szuromi, Czobor, Komlósi, & Bitter, 2011). Similarly, the auditory P300 is attenuated in schizophrenic patients, relative to individuals presenting schizophrenic-like symptoms, and in turn, controls (Trestman et al., 1996), with

the visual inhibitory P300 attenuated in patients with BPD, relative to age- and sex-matched controls (Ruchsow et al., 2008). However, these results might be better explained by a single externalising factor. Patrick et al. (2006) observed an inverse correlation between parietal P300 amplitude, evoked using a visual oddball paradigm, and the breadth and severity of externalising symptoms.

This point being made, there exists a large literature documenting associations between aggressive/CU traits and the P300. In one three-way comparison of two inmate groups characterised by reactive/instrumental aggression and a cohort of non-forensic controls, visual P300 attenuation was observed in both inmate cohorts, at frontal sites (Barratt et al., 1997). Furthermore, after correcting for age, intelligence, response accuracy, and reaction time, P300 responses to schematic faces were attenuated in a large forensic cohort comprising reactive/non-reactive aggressors and non-violent offenders (Bernat, Hall, Steffen, & Patrick, 2007). However, whilst this finding suggests an effect of aggression on the P300 within a forensic setting, unlike findings reported in Barratt et al., this finding is restricted to offenders due to the absence of non-incarcerated controls.

Similar attenuation of the P300 has been observed in community samples characterised by impulsive aggressive tendencies, relative to non-aggressive controls (Gerstle et al., 1998). Gerstle et al. evidenced frontal P300 attenuation during an auditory oddball paradigm, but noted that half of their aggressive cohort reported extensive brain damage, which may have accounted for some variation in data. An inverse relationship between the visual P300 and aggression has also been evidenced in students (Bartholow, Bushman, & Sestir, 2006). Bartholow et al. suggested that P300 amplitude was inversely correlated with both experience of violent video games and laboratory-measured aggression.

Though some variance in P300 amplitude might be attributed to trait impulsivity, this position has been clarified in non-forensic samples. Using a three-stimulus

rotating-head paradigm, whereby the third factor was affective stimuli, Venables et al. (2011) reported an inverse relationship between impulsivity and anterior-midline P300 amplitude. However, as this finding only held when controlling for self-reported aggression, impulsivity might only moderate the P300 via trait aggression.

Conflicting findings have been reported in response to nogo stimuli. An absence of visual inhibitory P300 amplitude variation has been observed between samples of reactive- and non-aggressive offenders (Chen et al., 2005), alcohol dependant individuals with and without comorbid antisocial behaviour (Costa et al., 2000), and violent and non-violent juvenile offenders (Vilà-Balló et al., 2014). Similar findings have been reported between samples of aggressive and non-aggressive ADHD patients and controls using a facial affect paradigm whereby a motor response was required to a successive train of angry faces, but not if succeeded by a happy face (Meier et al., 2012). In this investigation, although nogo-P300 attenuation was evident in both delinquent and non-delinquent patients, relative to controls, P300 amplitude did not differ between aggressive and non-aggressive patients. However, it is possible that P300 differences might have been masked by ADHD symptoms, medication, and/or the samples including offenders characterised by both reactive and instrumental aggression.

With few exceptions (Bauer & Hesselbrock, 1999; Du et al., 2006), individuals characterised by instrumental aggression exhibit P300 amplitudes similar to (Patrick, 2008; Raine & Venables, 1987, 1988), or greater than (Bernat et al., 2007) the general population. Psychopaths (Flor et al., 2002**;** Raine, 1989), and individuals presenting CU traits such as fearlessness (Carlson & Thái, 2010) also evidence P300 augmentation. However, as the P300 of individuals with psychopathic traits, relative to controls, shows attenuation when evoked to nogo stimuli (Kim & Jung, 2014), CU traits might differentially moderate electrophysiological responses as a function of task. P300 attenuation during

motor inhibition might reflect individuals characterised by CU traits requiring greater neuronal resources to inhibit motor responses.

Compounding the inverse association between P300 amplitude and reactive aggression, P300 augmentation is absent in psychopaths during negative-affect-driven interpersonal encounters (Hicks et al., 2007; Kiehl, Hare, Liddle, & McDonald, 1999) and individuals reporting high self-centred impulsivity (Carlson, Zayas, & Guthormsen, 2009). Furthermore, P300 attenuation is only found to associate with antisocial behaviour when controlling for psychopathic traits (Gao & Raine, 2009). However, these findings fail to explain why P300 attenuation was observed in instrumental aggressors in Barratt et al. (1997).

Evidently, the P300 has utility for indexing CU traits, antisocial behaviour, and aggression (Krueger, 1999; Patrick, 2008). Nonetheless, whilst the stop-P300 is a key index of the effects of facial distress on motor inhibition in regards to the VIM, although extensively investigated during go and nogo trials, the stop-P300 has not been associated with aggressive or CU traits. Absence of such knowledge warrants further investigation. Once established, it would be possible to assess how aggressive and CU traits might moderate resulting electrophysiological responses.

3.7 Chapter Summary

In chapters one and two, the VIM was framed as a cognitive mechanism reliant on the ability to process facial distress in order to extinguish antisocial and aggressive behaviour. Chapter three used electrophysiological literature to index the distinct neurobiological stages of face processing and motor extinction described in chapter two, on an electrophysiological level. Specifically, structural, task salient, categorical, and evaluative stages of face processing were teased apart using N170, P200, N200, and P300 ERP responses, prior to discussing how threat- and distress-specific facial expressions might command greater mobilisation of neuronal resources. Moreover, N200 and P300 responses were discussed in terms of motor inhibition efficiency. To date, no

paradigm has sufficiently indexed the VIM, namely indexing electrophysiological response to distress-induced motor extinction. As mirrored on a cognitive and neurobiological level, electrophysiological indices of distress processing and motor extinction are deficient in populations characterised by CU and aggressive traits, and so the use of a distress-induced motor extinction paradigm might have utility for indexing aggressive and CU traits on an electrophysiological level. Chapter four accumulates these concepts, and discusses how the VIM, as indexed on a cognitive, neurobiological, and electrophysiological level, might be moderated by diet, specifically dietary intake of omega-3 polyunsaturated fatty acids.

Chapter Four: Dietary impact on aggression

4.1 Introduction

Thus far, this thesis has discussed the possible cognitive, social, and biological underpinnings of reactive aggression, their associations with aggressive and CU traits, and how they might be indexed on an electrophysiological level. Chapter four delineates how dietary intake, more specifically omega-3 polyunsaturated fatty acids (PUFAs) intake, might moderate personality traits (i.e. aggression, CU) and their underpinning neurobiological networks.

4.2 Dietary Modulators of Aggression

A nutritious diet, including daily intake of micronutrients such as zinc, iron, and lithium, is essential for healthy development of the central nervous system (Portillo-Reyes, Pérez-García, Loya-Méndez, & Puente, 2014; Raine, 2013; Serfaty & de Velasco, 2014). Malnourished diets, defined by a lack of protein, fatty acids, vitamins, and nutrients (Tofail et al., 2008), might contribute to the emergence of psychopathologies, characterised by poor cognition and aggression (Georgieff, 2011; Grantham-McGregor et al., 2007; Pollitt, 2000).

For example, zinc deprived rodents (Takeda et al., 2008), and their offspring (Halas, Reynolds, & Sandstead, 1977) have evidenced increased aggression, relative to non-zinc deprived controls. In human samples, lower blood concentrations of zinc and copper have been reported in violent, relative to non-violent patients with schizophrenia (Tokdemir, Plota, Acik, Gursu, & Cikim, 2003), with increased ratios of copper to zinc reported in individuals presenting frequent incidents of assaultive behaviour, relative to a small cohort of non-violent controls (Walsh, Isaacson, Rehman, & Hall, 1997). Additionally, supplementation of lithium has been shown to decrease the duration and intensity of aggression in community (Campbell et al., 1995; Malone, Delaney, Luebbert, Cater, & Campbell, 2000; Malone, Luebbert, Pena-Ariet, Biesecker, &

Delaney, 1994) and forensic (Sheard, 1971; Sheard, Marini, Bridges, & Wagner, 1976) populations.

Fluctuations in dietary glucose metabolism have also been associated with impaired cognition and aggression (Gailliot & Baumeister, 2007). Previously, increased self-reported trait aggression has been reported in two student samples presenting comorbid hypoglycaemia - a medical condition associated with diminished glucose content (Benton, Kumari, & Brain, 1982; Donohoe & Benton, 1999). However, although assessed in a small sample, differences in blood glucose have not been observed when contrasting aggressive and non-aggressive patients with brain injury after controlling for weight, age, and time since injury (Stanislav, Crismon, & Childs, 1998). Elsewhere, this glucose/aggression association has only been observed to manifest under mild provocation (Denson, von Hipple, Kemp, & Teo, 2010).

4.3 Polyunsaturated Fatty Acids

Essential fatty acids (EFAs) benefit brain function during childhood and early adulthood (Bhatia et al., 2011, Luchtman & Song, 2012; Yehuda, 2003) and are thought to play a key role in the development of cognitive and emotional function (Marszalek & Lodish, 2005). Between .25 and 3 g EFA intake is recommended daily for healthy development (Food and Agriculture Organisation, 2010).

4.3.1 Composition, Intake, and Cell-level Importance

The two main families of EFAs - omega-3s and -6s - comprise 30 to 35% of total brain fatty acids in humans (Luchtman & Sog, 2012; SanGiovanni & Chew, 2005). Following dietary consumption, EFAs are desaturated and elongated via the addition of double bonds and [*two*] carbon units (SanGiovanni & Chew, 2005). This procedure produces long-chain polyunsaturated fatty acids (LCPUFA), which are structurally classified by [1] the number of carbon units,

[2] the number of double bonds, and [3] the location of the first double bond relative to the methyl omega terminal of the chain (SanGiovanni & Chew, 2005).

Around 50% of the dry brain weight of adults is made up of lipids, 35% of which are LCPUFAs (Serfaty & de Velasco, 2014). Parent LCPUFAs include the omega-6 linoleic acid (LA, 18:2n-6) and the omega-3 alpha-linoleic acid (ALA; 18:3n-3). LA contributes to the synthesis of the second order omega-6 fatty acids arachidonic acid (AA, 20:4n-6) and adrenic acid (22:4n-6), which are prominent in grey and white matter, respectively. ALA is synthesised into eicosapentaenoic acid (EPA, 20:5n-3) and docosahexaenoic acid (DHA, 22:6n-3). EPA is the precursor to DHA metabolism (SanGiovanni & Chew, 2005), and accounts for between 15% (Aid, Vancassel, Poumes-Ballihaut, Chalon, Guesnet, & Lavialle, 2003; Benton, 2007) and 30% (Innis, 2007; Salem Jr., Litman, Kim, & Gawrisch, 2001) of fatty acids in grey matter.

Conversion of ALA to EPA and DHA is thought to be less than 5% in humans (Brenna, 2002; Holub, 2002), and may be decremented further by, among other variables, alcohol consumption (Serfaty & de Velasco, 2014). During metabolism, omega-3s and -6s undergo cyclooxygenase enzyme competition (Arbuckle, MacKinnon, & Innis, 1994; Hrboticky, MacKinnon, & Innis, 1990), whereby EPA and DHA act to reduce AA levels (Endres et al., 1989; SanGiovanni & Chew, 2005). Omega-6 augmentation follows omega-3 deficiency (Calder & Grimble, 2002; Salem et al., 2001).

Through the absence of delta-15 and -12 desaturase enzymes, vertebrates are unable to synthesize LCPUFA *de novo* (Gadoth, 2008; SanGiovanni & Chew, 2005; Sinn, Milte, & Howe, 2010) and so LCPUFAs must be obtained through dietary omega-3 and -6 (McNamara et al., 2007; Owen, Rees, & Parker, 2008; Smith, 2008; Su et al., 1999). Omega-6s are predominantly derived from poultry, eggs, and vegetable oils (Luchtman & Sog, 2013), which are found in processed foods that often undergoing hydrogenation and the removal of omega-3s in order to prolong food preservation (James, Montgomery, &

Williams, 2011; Singh, 2005). Omega-3s are derived from seeds (e.g. flaxseed and rapeseed), grains, nuts (e.g. walnuts), green leafy vegetables, fatty fish, and shellfish (Innis, 2008; James, et al., 2011; Kris-Etherton et al., 2000; Marszalek & Lodish, 2005). Independent contributions of DHA and EPA from fish are measured as a function of the age, diet, species, and farming location of the fish, as well as the season they were caught (Kolakowska, Zienkowicz, Domiszewski, & Bienkiewicz, 2006).

For optimal psychological and physical well-being, humans are expected to consume a 4:1 diet of omega-3 and -6 (Gomez-Pinilla, 2008; Simpoulos, 2002). Recent decades have marked a reduction in omega-3 consumption in Western diets, which is now thought to comprise 5 to 20 times higher omega-6s than -3s (Ailhaud et al., 2006; Appleton et al., 2007; Bazan, 2005). In particular, Hallahan & Garland (2004) suggest that the modern Western diet mainly consists of meat and saturated fats, with comparatively little fresh vegetables and fish consumed.

Heightened omega-6 intake, at the expense of omega-3, is associated with health deficiencies such as cancer (Anderson & Ma, 2009; Schram et al., 2007) and cardiovascular diseases (Hibbeln Jr., Ferguson, & Blasbalg, 2006; Kris-Etherton, Harris, & Appel, 2002; Mozaffarian & Wu, 2011; Ryan et al., 2010; Sinclair, Begg, Mathai, & Weisinger, 2007), as well as the development of psychiatric disorders characterised by emotional deregulation (Conklin et al., 2007; Freeman et al., 2006; Peet & Horrobin, 2002; Simopoulos, 2002). Additionally, increased dietary intake of omega-6 is comorbid with zinc deficiency (Bourre, 2006), highlighted in section 4.2 (page 63) to associate with aggression.

Omega-3 selectively accumulates in the foetal central nervous system between the final trimester (Green, Glozman, Kamensky, & Yavin, 1999) and up to two years after birth (Martinez, 1992). Omega-3s are essential for cell membrane development (Riemer, Maes, Chrisophe, & Rief, 2010). In particular, whilst

EPA, via eicosanoids production, is important for anti-inflammation, blood clotting, and neuroimmunology (Sinclair et al., 2007; Sinn & Howe, 2008), DHA moderates membrane properties such as fluidity, thickness, plasticity, and cell permeability (Jump, 2002; Jumpsen & Clandinin, 1997; Tanabe et al., 2004) and protects cells from death (Carlson, 2001; Crawford, 2006; Innis, 2000; Itomura et al., 2005; Marszalek & Lodish, 2005; Michael-Titus & Priestley, 2014; Uauy & Dangour, 2006). Membrane changes influence how cells incorporate proteins (Bazan, 2005; Luchtman & Sog, 2013), and so DHA-rich membranes help shape brain circuitry by increasing molecular information processing (Michael-Titus & Priestley, 2014; Serfaty & de Velasco, 2014). In essence, DHA promotes inter- and intra-cellular communication (Bazan, 2005; Litman, Niu, Polozova, & Mitchell, 2001; Uauy & Dangour, 2006).

Foetal accruement of fatty acid coincides with synaptic development during both prenatal and postnatal growth (Carlson, 2001; Crawford et al., 2003; Green et al., 1999). DHA availability decreases with age (Soderberg, Edlund, Kristensson, & Dallner, 1991) and contributes to cognitive decline and the breakdown of cell communication (Danthiir, Burns, Nettelbeck, Wilson, & Wittert, 2011). Fatty acids are vital for brain growth and development (Conklin et al., 2007), and benefit early stages of brain modelling (Chen & Su, 2012; de Velasco et al., 2012). In particular, DHA facilitates neuronal growth through dendritic branching, synaptic formation, and myelination (Calderon & Kim, 2004; Sinn & Howe, 2008; Yehuda, Rabinovitz, Carasso, & Mostofsky, 2002). Early omega-3-deficient cell membranes contribute to synaptic degradation (Suzuki, Manabe, Wada, & Crawford, 1997) and dysfunction in synaptic pruning (Carlson, 2001). As described in chapter three, section 3.6.3 (page 57), synaptic pruning is essential for cell specialisation. However, at least in rodents, effects of neonatal omega-3 deprivation on cell growth appear reversible through DHA and EPA supplementation during infanthood (de Velasco et al., 2012).

4.3.2 Polyunsaturated Fatty Acids and Aggression

Throughout the last quarter of a century, omega-3 deficiency and low omega-3:-6 ratios have been associated with suboptimal child development and behavioural pathologies (Crawford, Hassam, & Williams, 1976; Fiennes, Sinclair, & Crawford, 1973). More recently, review publications have not only linked low omega 3 intake/blood measures to impulsive, aggressive behaviours, but have suggested these behaviours may be resolved through dietary supplementation (Garland & Hallahan, 2006). This section highlights how omega-3 might moderate aggression at baseline and through supplementation, in animal and human samples.

4.3.2.1 Correlational Studies

Whilst not establishing cause and effect, correlating two variables together within, or across a given population(s) indicates the relationship strength between those variables. Previously, a cross-national analysis of 26 countries has suggested an association between low fish consumption (omega-3s), and high rates of homicide mortality (Hibbeln, 2001; The World Health Organisation, 1995). Although this result survived after correcting for population, as some of the countries included in the sample reported both high omega-3 intake and high homicide rates, it is important to consider the importance of omega-3 intake in the context of co-variates.

Elsewhere, outward aggression (Meyer et al., 2015), and self-harm (Garland et al., 2007) have been inversely correlated with blood levels of omega-3. In Meyer et al. (2015), though only significant at trend level, omega-3 was inversely correlated with self-reported physical aggression in a large cohort of males from an offender correctional facility. In Garland et al. (2007), plasma concentrations of omega-3, as well as individual levels of EPA and DHA, were inversely associated with incidents of self-harm in patients presenting at an accident and emergency unit. These correlations held after correcting for age, sex, substance use (i.e. alcohol and nicotine), and economic status.

In particular, DHA has been inversely associated with both psychometric-assessed aggression in a sample of patients derived from a forensic mental health clinic (Zaalberg et al., 2015), and hostility in a larger sample of young males taking part in a longitudinal health assessment (Iribarren et al., 2004). However, whilst additional associations were reported between aggression and omega-3:-6 ratios in Zaalberg et al. (2015), a comparative association was not observed after correcting for age, sex, race, education, employment, BMI, and substance use in Iribarren et al. (2004).

In contrast, very few investigations have assessed the relationship between omega-3 intake and CU traits. One such investigation, Gow, Vallee-Tourangeau et al. (2013), reported an inverse association between psychometric-assessments of callousness, uncaring, and unemotional traits, and total blood measures of omega-3, EPA, and DHA at trend-level significance. However, in this investigation, [1] the CU/omega-3 association was only observed in children with, but not without ADHD, and [2] unlike the other investigations noted above, omega-3 was not associated with psychometric-based trait aggression in either ADHD or control group. Elsewhere, although not a direct measure of CU traits, omega-3 intake has been shown to be inversely associated with unfair offers of remuneration in a large student sample (Emanuele, Brondino, Bertona, & Gerald, 2009).

4.3.2.2 Group Comparison Studies

Group comparison analysis has been used to explore relationships between LCPUFA and aggression in both animal and human samples. In rodents, reduced omega-3 (DeMar et al., 2006) and increased omega-6 (Raygada, Cho, & Hilakivi-Clarke, 1998) has been associated with more frequent episodes of blocking behaviours, over longer durations, during resident-intruder paradigms. Whereas DeMar et al. compared Long-Evans rat pups fed an omega-3 deficient, relative to adequate, diet for 15 weeks, Raygada et al. contrasted offspring of mothers fed either a standard, or omega-6-rich diet during pregnancy. Similarly, in a canine sample, aggressive, relative to non-aggressive

German Shepherd dogs presented low omega-3 levels, and sub optimal omega-3:-6 ratios (Re, Zanoletti, & Emanuele, 2008). However, this association was specific to DHA, not EPA.

In humans, omega-3 deficiency has been associated with externalising behaviours. For example, in samples of cocaine addicts, recruited from a substance abuse clinic, patients with, relative to those without, histories of aggression were found to have lower serum levels of total omega-3 and DHA (Buydens-Branchey, Branchey, McMakin, & Hibbeln Jr., 2003). Although this finding suggests omega-3 deficiency might facilitate aggression, this result should be discussed in light of the aggressive group consisting of only 6 patients, and the nonaggressive group containing some patients who evidenced low omega-3 serum levels. Moreover, after separating a cohort of adolescent males on the basis of their omega-3 intake, those reporting low omega-3 evidenced increased parent- and teacher-rated behavioural and learning problems, including hyperactivity, impulsivity, and poor conduct control (Stevens, Zentall, Abate, Kuczek, & Burgess, 1996). Elsewhere, although omega-3 has been measured in males derived from the community both with, and without histories of inflicting physical harm (Umhau et al., 2006), this investigation only assessed relationships between DHA and folic acid, not intergroup variation.

In a forensic sample, low levels of omega-3, and high levels of omega-6 were found in violent offenders characterised by frequent, alcohol-fuelled episodes of aggression, relative to non-aggressive controls without prior alcohol use problems (Virkkunen, Horrobin, Jenkins, & Manku, 1987). Furthermore, EPA deficiency has been associated with trait aggression in individuals with major depressive disorder with, but not without, comorbid substance abuse (Beier et al., 2014). However, as alcohol consumption is thought to compromise the structural composition of fatty acids (Pawlosky, Bacher, & Salem, 2001), controlling for alcohol use when assessing the relationship between fatty acids and aggression has importance.

4.3.2.3 Supplementation Studies

Whilst correlational and group studies are useful for identifying associations between omega-3 intake/blood measures and aggression at baseline and in contrast to nonaggressive controls, supplementation studies have utility for assessing the direct impact of omega-3 on behaviour, traits, and underlying neurobiological networks. This section critically evaluates the effectiveness of omega-3 supplementation on aggression in human samples.

Recently, the synergistic effects of mineral and DHA supplementation on aggression have been assessed using a randomised-controlled design (Long & Benton, 2013). In adult males without previous histories of aggression, daily supplementation of approximately 670 mg DHA over a twelve-week period was observed to reduce the use of aggressive dialogue in a picture-frustration paradigm, as well as impulsivity as indexed by a visual number-based stop-go task. However, there was no effect of DHA supplementation on either self-reported verbal or physical aggression, and DHA did not show additive benefit when supplemented alongside minerals. In samples with ADHD, DHA-rich supplementation over a two- (Hamazaki & Hirayama, 2004 [510 mg DHA; 120 mg EPA]) and four- (Stevens et al., 2003 [480 mg DHA; 80 mg EPA]) month period has been shown to improve parent- and teacher-rated aggression and oppositional/defiant behaviour, respectively. However, as ADHD diagnosis was not verified in Stevens et al., this particular result might better reflect DHA-driven alleviation of ADHD symptoms.

Interestingly, though twice the dose delivered in Hamazaki & Hirayama (2004) and Stevens et al. (2003), neither a DHA- (1032 mg DHA; 264 mg EPA), nor EPA-rich (108 mg DHA; 1109 mg EPA) supplement, in contrast to a sunflower oil placebo, improved oppositional behaviour in an ADHD sample over a four-month period (Milte et al., 2012). An absence of improvement to ADHD symptoms was also reported in a large sample of children, following daily supplementation of 345 mg DHA, over a four-month period (Voigt et al., 2001). Moreover, DHA-rich supplementation (510 mg DHA; 120 mg EPA) to the food of

166 children was in fact observed to *increase* task-based measures of aggression towards others, as measured by a picture-frustration task (Itomura et al., 2005). This effect was only observed in male, not female participants, and could possibly be an artefact of low aggression scores at baseline.

Null effects of DHA supplementation have been reported elsewhere. In Hamazaki et al. (1996), a small sample of students were administered either a large daily dose of DHA (1500-1800 mg) or a soybean placebo over a three-month period. Whilst no effect of DHA was observed, students administered the soybean placebo evidenced increased rates of aggression, as indexed by aggressive dialogue in a picture-frustration task. As this experiment was conducted over an exam period, Hamazaki et al. explained the results in terms of DHA possibly having protective properties during stressful situations. As discussed in section 1.3.1 (page 7), environmental triggers such as stressors may precede aggression.

In respect to the effect of EPA supplementation on aggression, there exists very little literature. One such example, Zanarini & Frankenburg (2003), reported that relative to a placebo, daily supplementation of 1000 mg EPA over a two-month period reduced both verbal and physical aggression in female, BPD patients. Further support of the effect of omega-3 on aggression comes from investigations using a mixed EPA and DHA supplement.

Incidents of aggression have been reduced in two clinical trials using a composite omega-3 supplement; one based in the United Kingdom (Gesch et al., 2002) and one based in the Netherlands (Zaalberg, Nijman, Bulten, Stroosma, & van der Staak, 2010). Although only a small dose of daily supplementation was administered (44 mg DHA; 80 mg EPA) between 142 to 172 days, Gesch et al. (2002) observed a 26.3% reduction in violent incidents in a large sample of male offenders. However, as the active supplement also included 13 vitamins and 12 minerals, it is unclear how much variance in behaviour was explained by omega-3 alone. Previously, there have been mixed

reports as to the effectiveness of mineral and vitamin supplementation on aggression. Whereas Long & Benton (2013) found no effect of mineral supplementation, Schoenthaler et al. (1997) elicited a 28% decrease of incidents of violence in imprisoned juveniles following supplementation of 12 vitamins and 11 minerals, relative to placebo. In the Dutch trial, Zaalberg et al. (2010) found a 34% reduction in aggression and rule-breaking behaviour using a much larger omega-3 supplement than Gesch et al. (400 mg DHA; 400 mg EPA).

Recently, a randomised-controlled trial assessed the impact of omega-3 supplementation on aggressive and CU traits (Raine, Portnoy, Liu, Mahoomed, & Hibbeln, 2015). In this trial, a large sample of children aged between 8 and 16 years received a daily fruit juice drink (1000 mg), supplemented with either omega-3 (300 mg DHA; 200 mg EPA) or a placebo matched for weight, colour, and taste. In the active group, child-reported reactive aggression, parent-reported antisocial behaviour (e.g. CU traits, narcissism, and impulsivity), and psychopathic traits (e.g. cold-heartedness and fearlessness) were reduced over a six-month period.

Although clinical trials seem to indicate an omega-3-evoked reduction in aggression assessed using psychometric measures, simple behavioural paradigms and both parent and teacher ratings, there are some inconsistent findings. For example, in a sample of 47 self-harmers, although a large, composite omega-3 supplement (900 mg DHA; 1200 mg EPA) improved symptoms of depression and suicidality over a four-month period, there was no reduction in aggression or impulsivity (Hallahan, Hibbeln, Davis, & Garland, 2007). Moreover, there was no difference in parent- and teacher-rated aggression in a sample of 40 children with ADHD following a DHA-rich (500 mg DHA; 100 mg EPA), relative to olive oil-rich dietary plan matched for taste and smell, over a two-month period (Hirayama, Hamazaki, & Terasawa, 2004). However, this investigation assessed aggression using only two questions

requiring congruent answers from parents and teachers and so likely limited the variance in data.

4.3.2.4 Fatty Acids/Aggression Summary

Though not conclusive, baseline aggression in animals and humans might be a function of omega-3 blood content, as evidenced in correlational and group comparison studies. Furthermore, *de novo* omega-3 supplementation might benefit the reduction of aggression under certain circumstances, such as presence of a stressor. Although stressors were not implicitly outlined in studies conducted in forensic settings (Gesch et al., 2002; Zaalberg et al., 2010), stress might result from unfavourable physical and environmental conditions. Whilst omega-3 might reduce aggression, possibly through moderation of neurotransmission (Parletta, Milte, & Meyer, 2013), it remains unclear whether this effect is global, or influential at a specific point during the reactive aggressive processing route outlined throughout this thesis. The following sections suggest how omega-3 might modulate stages of '*face processing'*, '*negative affect*', and '*executive functioning*'.

4.4 Face Processing

DHA is concentrated in sensory and vascular retinal cells and comprises around 20% of retinal fatty acids, which enrich membrane disks in the outer photoreceptors (Innis, 2008; SanGiovanni & Chew, 2005). Prenatal omega-3 deficiency has been shown to detriment long-term primate retinal function (Anderson, Neuringer, Lin, & Connor, 2005), with specific DHA deficit in humans associated with visual system dysfunction (SanGiovanni & Chew, 2005; Uauy et al., 2001) and impaired visual attention (Fedorova & Salem, 2006). Postpartum, omega-3 supplementation of infant formula benefits developmental needs such as visual acuity (Birch et al., 2010; Uauy et al., 2000; Uauy & Castilo, 2003).

On a biological level, although omega-3 has been shown to be rich in brain regions involved in face processing, such as the temporal lobe (McNamara, 2010), to date, no studies have associated omega-3 concentrations in specific brain networks (e.g. fusiform gyrus-STS-amygdala) in regards to face processing efficacy. However, on an electrophysiological level, EPA and DHA have been shown to moderate distinct components of the face-evoked ERP, in samples of children and adolescents with ADHD symptoms.

Though not generalisable to non-ADHD, Gow, Matsudaira, et al. (2009) evidenced differential effects of EPA and DHA on the N170 in a sample of ADHD children. Here, red blood cell lipid levels of DHA were positively correlated with the right temporal N170 evoked to a 10 ms presentation of facial fear. This finding was argued to reflect activation of underlying neural networks associated with empathy (*see chapter two, section 2.5 [page 29]*), and suggests a benefit of DHA for improving neural transmission of these networks in response to facial distress. Conversely, EPA, but not DHA, positively associated with N170 and P300 responses to happy and happy minus fearful/sad facial expressions, respectively, at anterior midline sites. This finding suggests orientation of attention towards happy faces over several face processing stages. Additionally, whilst not concurrent with the notion that fatty acids benefit face processing, EPA was inversely associated with N170 amplitude over left temporal and bilateral occipital sites to happy and sad faces, respectively, and so warrants further investigation.

Whilst effects of omega-3 on face-evoked ERPs have also been observed in Gow, Sumich et al. (2013), this investigation did not report correlations between blood fatty acid levels and N170, P200, and N200 responses, in either ADHD or control samples. However, had this association been made, results would have been limited due to ERPs being measured over midline, and not bilateral, temporal sites as suggested in chapter three, sections 3.2.1 (page 39) and 3.2.2 (page 41). No correlations between omega-3 fatty acid levels and the

P300 responses to facial expressions reached statistical significance in either ADHD or control sample.

4.5 Negative Affect

As discussed in section 1.3.2 (page 9), negative affect plays a role in the antecedence of an aggressive response. This section describes the associations between omega-3 and mood in general, before noting correlational and supplementation studies that have investigated how similar underlying processes might moderate anger, specifically.

The role of omega-3 in the moderation of affective processes of rodents, children, adolescents, and adults is well documented (*see Freeman et al., 2006 for review*), and is likely a function of omega-3-specific inflammatory effects (Raison & Miller, 2013). Omega-3 intake is thought to benefit affective processes, such as depression (Nemets, Nemets, Apter, Bracha, & Belmaker, 2006), and is inversely related to the severity of depressive symptoms across several age ranges (Freeman et al., 2006; Owen et al., 2008; Montgomery & Richardson, 2008; Tiemeier, Breteler, van Popele, Hofman, & Witteman, 2003). Furthermore, DHA (Edwards, Peet, Shay, & Horrobin, 1998; Fedorova & Salem, 2006) and EPA (Zanarini & Frankenburg, 2003) are reduced at baseline in individuals reporting depressive traits. Although daily supplementation of 1000 mg EPA (Peet & Horrobin, 2002) and 9600 mg DHA (Su et al., 2003) has been shown to reduce depression symptoms, Su (2009) suggests EPA, relative to DHA, might have greater treatment benefit. Omega-3 supplementation did not reduce depression in Marangell et al. (2003) or Silvers, Woolley, Hamilton, Watts, & Watson (2005).

4.5.1 Correlational Studies

Similar to aggression (*see section 4.3 [page 64]*), levels of blood-measured omega-3 have been inversely associated with anger-related traits. Though not a function of specific metabolites, increased composite omega-3 has been

positively associated with self-reported anger and hostility in male offenders (Meyer et al., 2015). Furthermore, in drug naive patients with schizophrenia, red blood cell lipid levels of DHA and EPA were inversely correlated with hostility (Watari, Hamazaki, Hirata, Hamazaki, & Okubo, 2010). However, whilst this investigation sampled patients without histories of malnutrition or drug/alcohol abuse, correlations did not control for current dietary intake and lacked a non-clinical control sample.

In community samples, DHA, but not EPA, serum levels have been positively associated with psychometric-measured agreeableness, after adjusting for age, sex, and race (Conklin et al., 2007). Additionally, though specific for black respondents, Iribarren et al. (2004) evidenced inverse correlations between hostility and retrospective self-reported levels of total omega-3, DHA, and EPA, after controlling for alcohol use. However, as [1] the self-report assessment of omega-3 only took into account intake over the previous month, and [2] due to the nature of the investigation, hostility and omega-3 were reported two years apart, this relationship assumes stability of both omega-3 consumption and trait hostility.

4.5.2 Supplementation Studies

Compared to investigations testing the effect of omega-3 supplementation on self-reported, task-induced, and historical incidents of aggression (*see section 4.3 [page 64]*), there is a paucity of research into the effect of omega-3 on anger. Over two decades ago, reduced hostility was reported in individuals fed a low fat/high carbohydrate and fish diet (high omega-3:-6 ratio), relative to those fed a '*standard Western diet'* (low omega-3:-6 ratio) over a five-year assessment (Weidner, Connor, Hollis, & Connor, 1992). However as this investigation lacked an accurate assay of omega-3 intake, direct group comparisons are limited, with some variance possibly accounted for by other dietary factors.

Elsewhere, following a three-month period of daily EPA-rich supplementation (500 mg DHA; 2250 mg EPA; 250 mg other LCPUFA), relative to a soybean oil placebo, self-reported feelings of anger and anxiety were reduced in a small sample of males recruited from substance abuse clinics (Buydens-Branchey, Branchey, & Hibbeln, 2008). This finding was also replicated using a comparable design, supplement, and sample elsewhere (Buydens-Branchey & Branchey, 2008). In this latter investigation, following the three-month supplementation period, anger scores in a subsample of patients remained attenuated for a further three months post supplementation.

Anger has also been reduced as a function of daily EPA-rich omega-3 supplementation (800 mg DHA; 1600 mg EPA; 400 mg other LCPUFA) over a period as short as 35 days (Fontani et al., 2005a, 2005b). These investigations found no effect of sex, but also evidenced benefit for self-reported anxiety and depression - compounding the possible role of omega-3 in mood. However, in both cases, investigations were limited to small sample sizes. Additionally, although containing a large dose of EPA, DHA-rich (6000 mg DHA; 1200 mg EPA) supplementation of omega-3 over twelve weeks was found to decrease self-reported irritation in a small sample of violent schizophrenic patients (Légaré et al., 2007).

4.5.3 Underlying Mechanisms

Moderation of anger as a function of omega-3 intake suggests an effect of omega-3 on affect-serving regions such as the amygdala (Conklin et al., 2007). Although to date there have been no investigations into amygdaloidal omega-3 concentrations and anger, specifically, chronic omega-3 supplementation has been shown to increase amygdaloidal cell concentrations of EPA and DHA in rodents (Taha et al., 2013). In humans, baseline amygdala omega-3 concentrations have been shown to be equivocal in patients with schizophrenia, bipolar disorder, and MDD evidencing affect and behavioural problems, relative to controls (Hamazaki et al., 2010).

In chapter two, section 2.4 (page 26), reactive aggression was described through amygdala projections to the hypothalamus alongside a disruption to the hippocampal-mediated HPA axis. Omega-3 intake might moderate HPA efficacy by altering oxidant membrane levels (Conklin et al., 2007; Songur et al., 2004). In rodents, hypothalamic cells of rodents fed an omega-3-restricted, relative to a control diet, comprised lower DHA and increased omega-6 concentrations (Chen & Su, 2013; Li et al., 2006). However, it remains unclear as to whether hypothalamic DHA deficiency can be replenished. Whilst omega-3 supplementation over a twenty-four-week period did not fully replenish DHA in Li et al. (2006), full omega-3 replenishment was achieved in Chen & Su (2013) after seven weeks.

Hypothalamic activation moderates the pituitary gland through expression of CRF (Ressler & Nemeroff, 2000), a neuropeptide hyperexpressed in omega-3 deficient domestic abusers (Hibbeln, Bissette, Umhau, & George, 2004). The mechanism underpinning fatty acid-induced increases in CRF expression is thought to be AA metabolites prostaglandin E2 and F2. These metabolites control the release of CRF messenger ribonucleic acid (RNA) (Cambronero, Rivas, Borrell, & Guaza, 1992; Hibbeln et al., 2004), and share a positive correlation with omega-6:-3 ratio (Cambronero et al., 1992; Hoffmann et al., 1986; Lands et al., 1992). Specifically, EPA, relative to DHA, has been shown to suppress greater RNA expression (Mickleborough et al., 2009).

Although DHA and EPA have been shown to moderate the hypothalamus and pituitary gland respectively, it is DHA that seemingly moderates dendritic branching within hippocampal neurons - the final node of the HPA axis (Wurtman, 2008, *see chapter two, section 2.4 [page 26]*). The hippocampus responds to adrenal gland cortisol release, which is increased in primates with greater omega-3 blood concentrations (Laugero et al., 2011). Functionally, DHA deficiency disrupts synaptic fluidity in the hippocampus at a cellular level (Aid et al., 2003; He et al., 2009), and is evident through long-lasting synaptic dysfunction in omega-3 deficient rodents (Patten et al., 2013). There is some

evidence to support DHA supplementation playing a preventative role in stress-induced hippocampal dysfunction in rat pups (Feng et al., 2012).

Structurally, omega-3 deficiency attenuates hippocampal cell body size (Ahmad et al., 2002) and impairs hippocampal cell migration in rat pups fed an omega-3 deficient diet (Yavin, Himovichi, & Eilam, 2009). During foetal development, this reduction in hippocampal outgrowth might predominantly relate to DHA, but not EPA deficiency (Cao et al., 2009). Conversely, chronic supplementation of omega-3 might augment hippocampal volume in rodents fed omega-3 deficient (Venna et al., 2009) and non-omega-3 deficient diets (Calderon & Kim, 2004). Additionally, DHA supplementation might restore cellular growth impairment (Cao et al., 2009; Yavin et al., 2009), though some studies argue only for females (Patten et al., 2013).

4.6 Executive Functioning

Omega-3 has been shown to moderate processes relating to executive functioning, including attention, working memory, and motor inhibition (Branca, 2006; Johnson, Ostlund, Fransson, Kadesjö, & Gillberg, 2009; Karr, Alexander, & Winningham, 2011; Sinn & Bryan, 2007). As discussed in chapter one, section 1.3.3 (page 11), executive functioning typically moderates reactive aggressive behaviour in humans.

Attention, the ability to orient towards target stimuli is poor in ADHD, a disorder first related to EPA deficiency in Colquhoun & Bunday (1981). Subsequently low omega-3 levels have been reported in both child (Bekoroglu et al., 1996) and adult (Young, Conquer, & Thomas, 2005) samples with ADHD. EPA-rich supplementation has been found to improve attention over a four-week period in a sample derived from the community (Fontani et al., 2005, 2009 [800 mg DHA; 1600 mg EPA; 400 mg other LCPUFA]) and over a four-month period in two samples of children evidencing ADHD symptoms with and without oppositional traits (Gustafsson et al., 2010 [500 mg EPA]). Elsewhere, some (McNamara et al., 2010b; Milte et al., 2012), but not all (Voigt et al., 2001)

investigations suggest DHA might benefit attention as well. However, as ADHD patients, relative to non-clinical controls, show reduced blood DHA concentration at baseline (Burgess et al., 2000), it is possible that this disparity is an artefact of individuals characterised by poor executive functioning having a decremented ability to metabolise EPA into DHA.

On a neurobiological level, EPA-rich (600 mg DHA; 1800 mg EPA), relative to DHA-rich (1650 mg DHA; 550 mg EPA) supplementation over a four-week period has been shown to better improve cognitive efficiency, as evidenced by lower activation of the left ACC during a spatial working memory paradigm (Bauer et al., 2014). Previously, working memory, as indexed by digit span and letter number sequencing, has been shown to benefit from EPA-rich supplementation (600 mg EPA; 120 mg DHA), relative to an olive oil placebo, over a sixteen-week period (Widenhorn-Müller, Schwanda, Scholz, Spitzer, & Bode, 2014). However, this finding is limited to children diagnosed with ADHD.

4.7 Chapter Summary

This chapter outlined the possible benefit of omega-3 on health in general, before highlighting investigations that have used correlational analysis, group comparisons, and supplementation to indicate relationships between omega-3 and reactive aggression/CU traits. Although some conflicting findings have been presented, it is apparent that nutrition, specifically omega-3 dietary intake, might benefit individuals characterised by aggressive and antisocial behaviour. In relation to the VIM, the role of omega-3 was discussed in association with threat and distress processing, negative affect, and executive control. However, whilst omega-3 might facilitate face processing and its associated neurobiological underpinnings, to date, there is no documented investigation into the role of omega-3 fatty acids on distress-induced motor extinction, and very little understanding of how these processes might reflect on an electrophysiological level. As EEG is a useful tool for indexing efficacy of neuronal networks underpinning aggression and CU traits, expansion of this literature has both theoretical and practical importance.

Chapter Five: Thesis aims and the delineation and justification of methods

5.1 Introduction

This chapter is split into two sections. The first delineates the aims of this thesis and the second details the self-report measures used in conjunction with EEG methodology to fulfil these aims.

5.2 Thesis Aims

This thesis aims to:

1. Explore associations between physical aggression and CU traits in an adult community sample
2. Develop a sensitive experimental paradigm for characterising electrophysiological indices of different processing components of the VIM
3. Explore associations between physical aggression/CU traits and electrophysiological responses to face processing (i.e. N170, P200), and distress-induced motor extinction (i.e. stop-N200, stop–P300)
4. Explore associations between omega-3 intake, physical aggression, and CU traits
5. Explore associations between omega-3 intake and electrophysiological responses to face processing (i.e. N170, P200), and distress-induced motor extinction (i.e. stop-N200, stop–P300)

To date, though there exists a relatively comprehensive literature associating psychopathy-related personality traits (e.g. aggressive, CU traits) with atypical activation of the VIM - and their possible benefit from an omega-3-rich diet – investigation of these associations on an electrophysiological level appears to have been overlooked. Development of a novel EEG paradigm that allows for the simultaneous assessment of the distinct processing components of the VIM

not only facilitates investigation into how such traits might differentially moderate particular stages of the VIM, but also allows for delineation of more temporally accurate effects that might otherwise be difficult to identify on a behavioural level. Moreover, with increasing interest into the effect of omega-3 dietary intake on both aggressive and CU traits (Buydens-Branchey & Branchey, 2008; Gow, Vallee-Tourangeau et al., 2013; Raine et al., 2015), questions asked within this thesis will benefit understanding of how omega-3 might not only associate with VIM-related traits, but also distinct processing components of the VIM as measured using EEG.

5.3 Psychometric and Quantitative Measures

This section describes the scales used throughout this thesis to index physical aggression, CU traits, omega-3 intake, and alcohol/drug use. The design, history, and validation of each measure are discussed, alongside their respective limitations. Development and piloting of the FAST used in conjunction with these measures is described in chapter seven (from page 102).

5.3.1 Aggression Questionnaire v.2

The Aggression Questionnaire (AQ) Version 2 (Buss & Warren, 2000) measures self-perceived aggression and anger - the propensity to aggress (e.g. “I have threatened people I know“). The AQ is written using accessible language, can be completed by children and adults, and is suitable for use in several populations (e.g. community, clinical, forensic, military). The 34-item scale is scored on a five-point Likert scale ranging from 1 (‘*Not at all like me*’) to 5 (‘*Completely like me*’), and can be completed both on paper and online. Both mediums are used in this thesis. Total scores range from 34 to 170, with higher scores indicative of greater aggression. Twelve item pairs index inconsistent responding, with responses containing five or more pairs differing by more than one point removed.

The AQ is split into the five subscales: physical aggression, verbal aggression, anger, hostility, and indirect aggression. Whereas physical and verbal aggression account for the manifestation of an aggressive response, anger and hostility index the affective and cognitive components of aggression, respectively (Buss & Perry, 1992; Buss & Warren, 2000). Indirect aggression represents the instrumental component of an aggressive response. Total scale reliability is high (r = .94), with subscale reliabilities as follows: physical aggression (r = .88), verbal aggression (r = .76), anger (r = .78), hostility (r = .82), and indirect aggression (r = .71) (Buss & Warren, 2000).

Responses are standardised across age (9 to 18, 19 to 39, and 40 to 88), with physical and verbal aggression scores standardised across sex. Standardisation levels are based on a sample of 2,138 Americans (1,252 female) aged 9 to 88, recruited from schools, churches, and community centres (Buss & Warren, 2000). Racial backgrounds of the baseline sample consisted of Asian (1%), Black (15%), Hispanic (8%), Native American (<1%), White (72%), and Other (3%). Validity-wise, the AQ is comparable to existing measures of anger, hostility, and pro-aggressive attitudes ('*Children's Inventory of Anger*', Nelson & Finch, 2000; '*Novaco Anger Scale and Provocation Inventory*', Novaco, 2003; '*Attitudes Toward Guns and Violence Questionnaire*', Shapiro, 2000) with correlation coefficients ranging between .37 and .74.

The AQ is a revision of the Buss and Perry Aggression Questionnaire Version 1 (BPAQ; Buss & Perry, 1992), which itself is the successor to the Buss-Durkee Hostility Inventory (Buss & Durkee, 1957). The 29-item BPAQ is similar to the AQ in that it taps into cognitive (8 items) and affective (7 items) motivational components of aggression, as well as physical (9 items) and verbal (5 items) behavioural manifestations, but does not measure indirect aggression. Similar to the AQ, items are scored on a five-point Likert scale ranging from 1 ('*Extremely unlike me*') to 5 ('*Extremely like me*') and show internal consistency ranging from .70 to .88 for physical aggression, hostility and the total scores, and from .53 to .67 for verbal aggression and anger.

5.3.2 Inventory of Callous-Unemotional Traits

The Inventory of Callous–Unemotional Traits (ICU; Frick, 2003) measures the occurrence and intensity of CU traits, and consists of 24-items (e.g. “I do not show my emotions to others”) measured on a four-point Likert scale from 0 (‘*Not at all*’) to 3 (‘*Definitely true*’). The ICU can be completed using self-report, interview, or using parent-/teacher-ratings. For this thesis, self-reported values are used. Total scores are calculated by reverse scoring positively worded items prior to summation.

Confirmatory factor analysis on a large (n = 1,443) sample of German adolescents aged 12 to 18 suggested a three-way factor structure consisting of callousness, uncaring, and unemotional traits (Essau et al., 2006). Confirmation of this structure is provided in Fanti, Frick, & Georgiou (2009), Kimonis et al. (2008), and Roose, Bijttebier, Claes, Decoene, & Frick (2010). Eleven items load onto the callousness subscale (e.g. “The feelings of others are unimportant to me”), denoting callous attitudes towards others, and a further eight items (e.g. “I hide my feelings from others”) load onto the uncaring subscale, characterised by a lack of concern for one's own performance and the well-being of others. The final five items load onto the unemotional subscale (e.g. “I try not to hurt others’ feelings”) and denote a lack of emotion expression. ICU stability has been confirmed over one year in Australian cohorts aged between 4 to 9 (r = .55; Dadds, Fraser, Frost, & Hawes, 2005) and 17 to 24 years old (r = .60; Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006).

The ICU has three key limitations. First, though internal consistency is high in total ICU (.74 to .85) and callous and uncaring (.53 to .81) subscales, the unemotional subscale has low internal consistency (Essau et al., 2006; Frick, 2006; Kimonis et al., 2008). Low internal consistency might be an artefact of only five items loading onto the unemotional subscale (Essau et al., 2006; Fanti et al., 2009; Kimonis et al., 2008). Therefore, conclusions drawn from this scale should be considered with caution (Berg et al., 2013; Guelker, Barry, Barry, & Malkin, 2014). Second, though the ICU contains positively- and negatively-

worded items (Essau et al., 2006), these are not distributed evenly across subscales. The callousness scale consists mainly of negatively-worded items and the uncaring scale consists mainly of positively-worded items (Feilhauer, Cima, & Arntz, 2012). Third, although the ICU is thought to remain valid in adults (P. Frick, personal communication, December 12, 2011), it has predominantly been tested in adolescents (Essau et al., 2006; Fanti et al., 2009; Kimonis et al., 2008; Roose, et al., 2010).

The use of the ICU in this thesis is justified over other measures of CU traits such as the PCL-R (Hare, 2003) and the Psychopathic Personality Inventory – Revised (PPI–R; Lilienfeld & Widows, 2005). The PCL-R encompasses two factors - emotional detachment and antisocial deviance, which can be further divided into affective, interpersonal, lifestyle, and behavioural components (Hare, 2003; Hare & Neumann, 2005). However, this assay is time consuming and requires a combination of structured interview and historical information. Furthermore, the PCL-R only contains four items assessing CU traits, specifically, and so might not be an accurate conceptualisation (Feilhauer et al., 2012).

The PPI-R is an assay of personality characteristics as opposed to behaviours associated with psychopathy and can be used in forensic, clinical, and community samples (Gonsalves, McLawsen, Huss, & Scalora, 2013). However, the PPI-R is both time-consuming with 154 self-report items, and fails to tap into distinct CU traits, with the two key subscales indexing self-centred impulsivity and fearless dominance.

The ICU emerged from the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), and is a 20-item assay of psychopathic traits in children and adolescents, which can be completed by parent-, teacher-, or self-report. The APSD has a three-factor structure comprising a narcissism (7 items; e.g. “You brag a lot about your abilities, accomplishments, or possessions”), impulsivity (5 items; e.g. “You act without thinking of the consequences”), and CU dimension

(6 items; e.g. “You are concerned about the feelings of others”). Clinic and community samples of preadolescent boys and girls (Frick et al., 2000) and a detained sample of adolescent boys and girls (Vitacco, Rogers, & Neumann, 2003) confirm this three-way factor structure.

Although the APSD is a common measure of CU traits, having been used solely in twenty-six out of thirty studies, and in combination in a further two studies between 1997 and 2013 (Waller, Gardner, & Hyde, 2013), the internal consistency of the CU subscale is only moderate (*alpha* = .63; Bijttebier & Decoene, 2009). Poor internal consistency likely results from [1] only six items belong to the CU subscale, to which five are positively worded, and [2] each item being rated on a three-point Likert scale ranging from 0 (*‘Not at all’*) to 2 (*‘Definitely true’*), effectively prompting midline responses (Essau et al., 2006; Kimonis et al., 2008). Additionally, measures of CU traits do not delineate between subtypes of CU traits. The ICU was developed to overcome these limitations - providing a more extended assessment of CU traits.

5.3.3 Food Frequency Questionnaire

The Food Frequency Questionnaire (FFQ; Sublette et al., 2011) was developed as a means of rapidly assessing omega-3 intake in mood disorder research. Consisting of 21 items assessing intake of commonly accessible fish and seafood over a six-month period, as well as intake of sushi, walnuts, flaxseed, flaxseed oil, cod liver oil, and canola oil, the FFQ allows the calculation of independent DHA, ALA, and EPA consumption. Omega-3 contributions are calculated as a function of fish type, portion sizes (i.e. small, medium, large), consumption frequency (i.e. monthly, weekly, daily), and sex of responder. Dietary supplements are also assessed.

The FFQ was validated with omega-3 blood content in adults aged 18 to 73 years old with (n = 34) and without (n = 27) major depressive disorder (Sublette et al., 2011). After controlling for participants reporting no omega-3 intake, DHA (r = .50, p < .001) and EPA (r = .47, p < .001) correlated significantly with their

respective plasma levels. Furthermore, although no significant association was reported between FFQ- and plasma-measured ALA ($r = .22$, $p = .09$), ALA is not assessed in this thesis.

Although the briefness of this measure limits the distinction of wild versus farmed fish and excludes measurement of omega-3-rich sources such as eggs, its use is justifiable. Whilst there are other assays of omega-3 intake available (Fawzi, Rifas-Shiman, Rich-Edwards, Willett, & Gillman, 2004; Garland et al., 1998; Hunter et al., 1992; Parra et al., 2002), these consist of more than 100 items and are time-consuming to complete (Sublette et al., 2011). Additionally, although brief 14- and 15-item measures are available (Laviolle et al., 2005; Svilaas et al., 2002) these do not solely assess omega-3 intake and so do not provide an accurate assessment of omega-3 intake.

5.3.4 Alcohol Use Disorders Identification Test

The AUDIT (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) is a 10-item measure designed to help practitioners identify individuals who present alcohol dependence and drinking behaviours that are detrimental to themselves or others. This measure can be administered via interview or self-assessment and can be completed either online or offline. Items are scored between 0 and 4, and are summated to produce a total between 0 and 40. Scores of 0-7 suggest a possible benefit for "*alcohol education*", and scores of 20-40 indicate a need for "*referral to specialists for diagnostic evaluation and treatment*"). Questions 1 to 3 assess alcohol consumption, questions 4 to 6 assess alcohol dependence, and questions 7 to 10 pertain to alcohol-related problems (Babor, Higgins-Biddle, Saunders, & Monteiro, 2006). Internal reliability is high in control (.80) and alcohol-dependent (.80) samples (Moussas et al., 2009).

5.3.5 Cannabis Use Disorders Identification Test

The CUDIT (Adamson & Sellman, 2003) is a 10-item assay of cannabis use, formulated from the AUDIT (*see section 5.3.4 [page 88]*) by replacing the word

“alcohol” with “cannabis”. The CUDIT assesses the subdomains of cannabis consumption, using behaviours, adverse psychological reactions, and problems. The measure can be administered via interviewer or self-assessment and can be completed either on- or offline. Each item is scored from 0 to 4, giving a minimum possible score of 0 and a maximum possible score of 40, which can be used to inform clinical opinion (i.e. whilst scores of 0-7 suggests benefit for “*cannabis education*”, scores of 20-40 indicate a need for “*referral to specialists for diagnostic evaluation and treatment*”). Internal reliability and predictive value was found to be high (.84) in a moderately large sample of alcohol dependent outpatients (Adamson & Sellman, 2003).

Chapter Six: Correlations between physical aggression & callous-unemotional traits

6.1 Introduction

Physical aggression and CU traits manifest in disorders characterised by affective, interpersonal, and behavioural dysfunction (American Psychological Association, 2013), and as such are commonly used as proxy measures of psychopathy (Dawel et al., 2012). Discussed throughout chapter one, section 1.3.4 (page 12), the association between aggression and CU traits is well established (*see Frick & Dickens, 2006 for review*), with CU traits thought predict long-lasting patterns of antisocial behaviour (Frick & White, 2008), increased aggression severity (Frick & Dickens, 2006), and poor treatment outcomes (Frick et al., 2014). When presented together, CU traits facilitate the use of aggression as a means of acquiring resources and social-status through decreased social inhibition and the reorientation of attention away from the negative consequences of aggression towards the social rewards there of (Guelker et al., 2014; Pardini & Byrd, 2012; Raine, 2013).

CU traits comprise callousness (i.e. the poor affective response towards the feelings of others), uncaring (i.e. a lack of concern for one's own performance and the wellbeing of others), and unemotional (i.e. a lack of emotion expression) components (Fanti et al., 2009; Frick, 2003; Kimonis et al., 2008; Roose et al., 2010), which might differentially associate with aggressive traits and related behaviours. Whilst both callousness and uncaring traits have been positively associated with self-reported aggression and offending behaviour (Guelker et al., 2014; Kimonis et al., 2008), uncaring traits, specifically, have been found to better predict delinquency (Essau et al., 2006; Guelker et al., 2014; Kimonis et al., 2008; Roose et al., 2010). Conversely, unemotional traits are considered poor predictors of behavioural problems (Guelker et al., 2014).

Although a key medium for the manifestation of aggressive behaviour (Buss & Perry, 1992), to date, only one investigation has assessed the relationship between CU traits and measures of *physical* aggression in particular (Feilhauer et al., 2012). Sampling adolescents within community, clinical, and forensic settings, Feilhauer et al. documented positive, albeit weak, correlations between physical aggression, and all three components of the ICU. However, such inferences are solely derived from child and adolescent samples, and so their relationship in adults remains unclear. As considerable developmental changes occur between adolescence and adulthood (Bryd, Kahn, & Pardini, 2013), delineating relationships between CU traits and physical aggression within an adult cohort would provide valuable insight into how such relationships vary as a function of age.

Scores on psychometric measures of physical aggression, as measured by the AQ (Buss & Perry, 1992), and CU traits, as measured by the ICU (Frick, 2003), have shown to vary as a function of sex. In regards to the AQ, robust sex differences have been observed in the physical aggression subscale, with males consistently reporting higher physical aggression than females in adult samples (Bernstein & Gesn, 1997; Buss & Perry, 1992; Williams, Boyd, Cascardi, & Poythress, 1996). This pattern is also observed in the elderly (Morales-Vives & Vigil-Colet, 2010).

In regards to the ICU, although sex differences have not been observed in some analyses (Guelker et al., 2014; Roose et al., 2010), others have documented higher scores for males, relative to females, both uniformly across the three CU components (Essau et al., 2006) and for the unemotional dimension, specifically (Kimonis et al., 2008). Additionally, although not a true index of physical aggression, callousness has been shown to predict problematic behaviour in both male and female children, with uncaring traits only predictive of problematic behaviour in males (Essau et al., 2006). Possibly indicative of a more inclusive relationship between aggressive and CU traits in males, the authors explain this finding through males, relative to females,

reporting consistently higher scores on composite measures of psychopathy - as indexed by the PCL-R, *see chapter five, section 5.3.2 [page 84]*) - during both adolescence and adulthood (Vitale & Newman, 2001).

The current study sought to delineate the relationships between self-reported physical aggression, callousness, uncaring, and unemotional traits, in an adult cohort derived from the community. Physical aggression was hypothesised to correlate positively with callousness, uncaring, and to a lesser extent, unemotional traits in males, but only with callousness in females. Furthermore, it tested whether these traits, and their bivariate relationships, would vary as a function of sex. Informed by literature targeting these associations in child/adolescent samples, physical aggression and all three CU traits were hypothesised to be higher in males than females.

6.2 Methods

6.2.1 Participants

A total of 184 participants (136 female; M age = 20.46; SD = 2.42) were recruited via online, snowball sampling. Participants consented to take part in a brief online survey after responding to an advertisement disseminated via social media.

6.2.2 Measures

Participants completed a battery of demographics and online versions of the AQ and ICU (*see chapter five, sections 5.3.1 [page 83] and 5.3.2 [page 84], respectively).* For this investigation, only the physical aggression subscale of the AQ was used.

6.2.3 Procedure

Participants were briefed and provided informed consent. On average, the questionnaire battery took less than 10 minutes to complete, and respondents

were provided with means of redistributing the survey through social media, if they wished. The final section of the online battery debriefed respondents and provided them with a means of contacting the lead investigator (DF).

6.2.4 Statistical Analysis

Incomplete responses were removed. Scores for the physical aggression subscale of the AQ, and the callous, uncaring, and unemotional subscales of the ICU were reverse scored and calculated into an R Statistics v.2.15 dataset (R Core Team, 2012). Physical aggression scores underwent *t*-transformations as a function of age and sex. Four *t*-tests were conducted to outline baseline sex differences between psychometric responses. Six Pearson's partial-correlations were performed, holding age (demeaned), sex (demeaned), and scores on the psychometric measures *not* included in the specific analysis, constant. For example, a correlation between physical aggression and callousness controlled for scores on uncaring and unemotional measures, in order to find the unique variance shared by physical aggression and callousness. *Post hoc*, a further twelve Pearson's partial-correlations were performed, in addition to six *z*-difference comparisons in order to test differences in correlation effect sizes as a function of sex. Significance values were corrected for multiple comparisons (*see Benjamini & Hochberg, 1995*).

6.3 Results

Twenty-nine incomplete responses were removed, leaving 155 participants (M age = 20.28; SD = 2.36). Of the complete responses, 123 were female (M age = 20.25; SD = 2.79) and 32 were male (M age = 20.29; SD = 2.25).

6.3.1 Psychometric Measures

Means and standard deviations for scores on physical aggression, callousness, and uncaring and unemotional traits, for males, females, and the total sample are exhibited in Table 2. Relative to females, males responded significantly higher on measures of *callousness* ($t = 2.73$, df = 153, $p = .01$, *padj* = .03), but

not *physical aggression* (t = 1.04, df = 153, p = .86, *padj* = .86), *unemotional* traits (t = 1.99, df = 153, p = .05, *padj* = .14), or *uncaring* traits (t = 1.12, df = 153, p = .27, *padj* = .53). Comparisons of psychometric responses between sexes are presented in Figure 1.

Table 2.

Means and standard deviations of psychometric measures

	P. Aggression	Callousness	Uncaring	Unemotional
Male	52.84 (8.40)	6.22 (3.33)	8.28 (3.50)	8.31 (3.01)
Female	51.04 (8.82)	4.46 (3.23)	7.49 (3.60)	7.08 (3.14)
Total	51.41 (8.73)	4.82 (3.32)	7.65 (3.58)	7.34 (3.14)

Note. **P. Aggression = Physical Aggression**

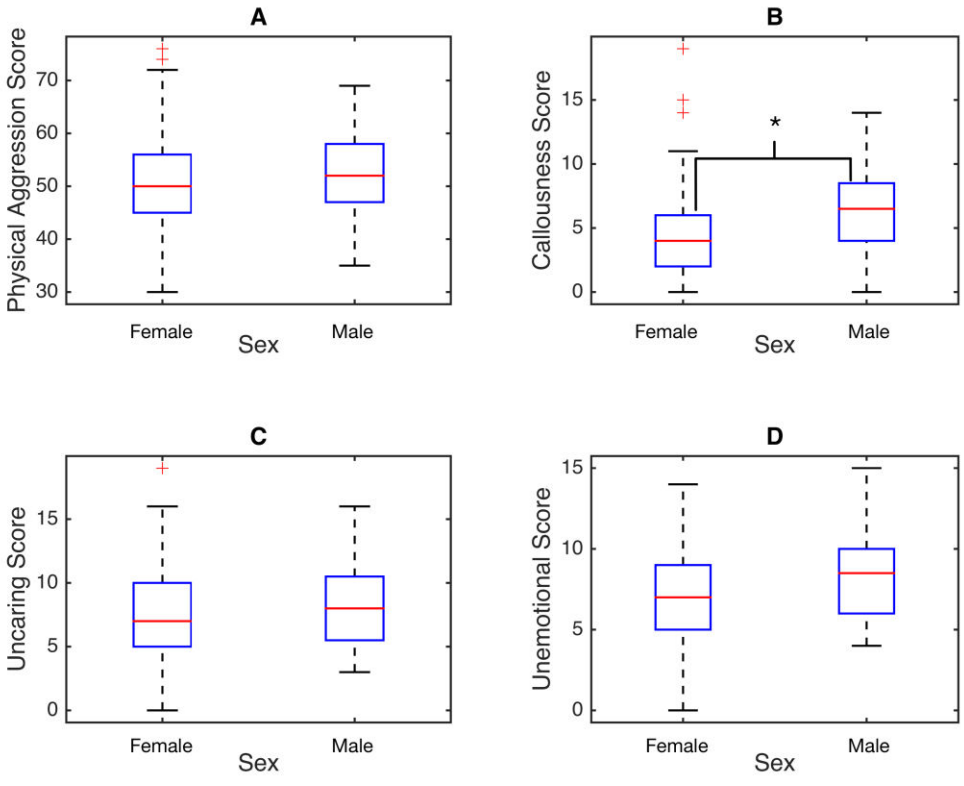

Figure 1. Means and standard deviations of psychometric responses (A = physical aggression, B = callousness, C = uncaring, D = unemotional) for males and females.
Note. *** p < .05**

6.3.2 Correlations

As presented in Table 3, bivariate partial correlations are reported for physical aggression, callousness, uncaring, and unemotional traits in the total sample, as well as for males and females separately. Correlations controlled for age, sex, and scores on psychometric measures *not* included in the specific analysis (*see section 6.2.4 [page 93] for an example*). All correlations underwent multiple comparison correction.

Table 3.

Bivariate correlation coefficients between psychometric measures and sex

		P. Aggression	Callousness	Uncaring	Unemotional
Male	P. Aggression	1	.29**	.03	.10
	Callousness	-	1	.10	.11
	Uncaring	-	-	1	.01
	Unemotional	-	-	-	1
Female	P. Aggression	1	.25**	.12	.17
	Callousness	-	1	.32***	.18
	Uncaring	-	-	1	.05
	Unemotional	-	-	-	1
Total	P. Aggression	1	.28**	.08	.15
	Callousness	-	1	.33***	.12
	Uncaring	-	-	1	.06
	Unemotional	-	-	-	1

***Note.* * $p < .05$, ** $p < .005$, *** $p < .001$, P. Aggression = Physical Aggression**

6.3.2.1 Total Sample

In the total sample, *physical aggression* was significantly correlated with *callousness* (r = .28, p < .001, *padj* < .05), but not *uncaring* (r = .08, p = .33, *padj* = .45) or *unemotional* (r = .15, p = .07, *padj* = .26) traits. *Physical aggression* accounted for 7.6% of the variance in *callousness* in the total sample. Between the components of the ICU, *callousness* was significantly correlated with *uncaring* traits (r = .33, p < .001, *padj* < .001), explaining 10.9% of variance in the total sample, but was not significantly correlated with *unemotional* traits (r = .12, p = .14, *padj* = .43). There was no statistically significant correlation between *uncaring* and *unemotional* traits (r = .06, p = .45, *padj* = .45). Correlations between physical aggression and callousness (subplot C), and callousness and uncaring traits (subplot G) in the total sample are displayed in Figure 2.

6.3.2.2 Males

In males, *physical aggression* significantly correlated with *callousness* (r = .29, p < .001, *padj* < .05), but not *uncaring* (r = .03, p = .70, *padj* = .88) or *unemotional* (r = .10, p = .22, *padj* = .66) traits. *Physical aggression* accounted for 8.4% of the variance in *callousness* in males. Between the components of the ICU, *callousness* was not significantly correlated with either *uncaring* (r = .10, p = .22, *padj* = .66), or *unemotional* (r = .11, p = .17, *padj* = .66) traits. There was no significant correlation between *uncaring* and *unemotional* traits (r = .01, p = .88, *padj* = .88). Correlations between physical aggression and callousness (subplot A), and callousness and uncaring traits (subplot D) in males are displayed in Figure 2.

6.3.2.3 Females

In females, *physical aggression* significantly correlated with *callousness* (r = .25, p < .05, *padj* < .05), but not *uncaring* (r = .12, p = .13, *padj* = .26) or *unemotional* (r = .17, p = .04, *padj* = .12) traits. *Physical aggression* accounted for 6.3% of the variance in *callousness* in females. Between the components of

the ICU, *callousness* was significantly correlated with *uncaring* traits (r = .32, p < .001, *padj* < .001), explaining 10.5% of variance in females, but was not significantly correlated with *unemotional* traits (r = .18, p = .03, *padj* = .11). There was no significant correlation between *uncaring* and *unemotional* traits (r = .05, p = .51, *padj* = .51). Correlations between physical aggression and callousness (subplot B) and callousness and uncaring traits (subplot E) in females are displayed in Figure 2.

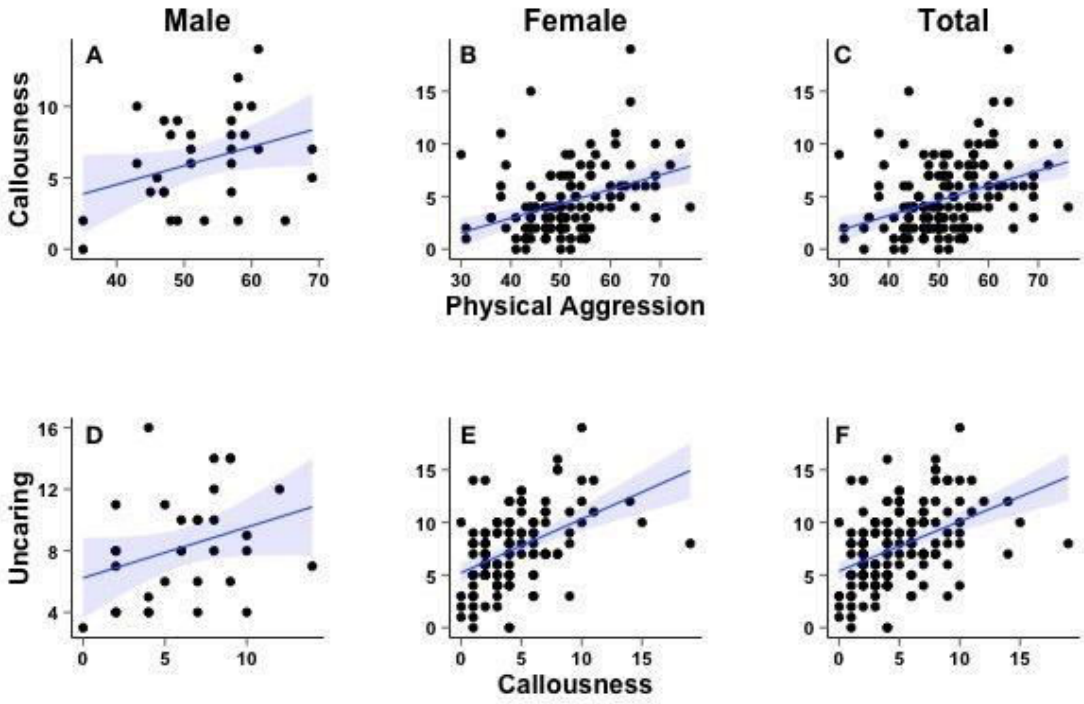

Figure 2. Scatter plots of partial correlations between physical aggression vs. callousness (subplots A - C), and callousness vs. uncaring traits (subplots D - F), for males, females, and total sample. Males - physical aggression vs. callousness (subplot A, *r* = .29), callousness vs. uncaring (subplot D, *r* = .10). Female - physical aggression vs. callousness (subplot B, *r* = .25), callousness vs. uncaring (subplot E, *r* = .32). Total sample - physical aggression vs. callousness (subplot C, *r* = .28), callousness vs. uncaring (plot F, *r* = .33). Shaded areas denote 95% confidence interval areas along linear associations.

6.3.2.4 Sex Differences

A comparison of each bivariate partial correlation between males and females revealed no statistically significant difference (*z*-difference) in effect size after controlling for sample size.

6.4 Discussion

This investigation is the first to delineate the relationships between self-reported physical aggression, callousness, uncaring, and unemotional traits, in an adult cohort. It was hypothesised that [1] psychometric measures of physical aggression and CU traits would be uniformly higher in males than females, and [2] physical aggression would be positively correlated with callousness, uncaring, and unemotional traits in males, but only with callousness in females.

In contrast to expectations, males only scored higher than females on scores of callousness, with measures of physical aggression, uncaring, and emotional traits yielding similar scores across sexes. Relating to physical aggression, the current findings diverge from those observed elsewhere that sampled from adolescent (Reyna, Ivacevich, Sanchez, & Brussino, 2011), adult (Bernstein & Gesn, 1997; Buss & Perry, 1992; Williams et al., 1996), and elderly populations (Morales-Vives & Vigil-Colet, 2010). In these investigations, males reported higher scores on comparative measures than females. Possibly an artefact of the current sample consisting predominantly of students, a population considered less prone to generate pro-aggressive ideologies (Finkel, et al., 2009, *see chapter one, section 1.3.3 [page 11]*), males, relative to females, have reported greater scores on physical aggression traits in a sample of Spanish students (García-León et al., 2002). However, it should be noted that akin to this investigation, the sample reported García-León et al. also comprised predominantly of females.

In respect to CU traits, male adolescents have been shown to score higher across all three subscales of the ICU (i.e. callousness, uncaring, unemotional), compared to female counterparts (Essau et al., 2006). Whilst not specific to the ICU, males are thought to score higher than females during both adolescence and adulthood, on a wide array of psychopathic traits measured across behavioural, interpersonal, and affective dimensions (Vitale & Newman, 2001). In the current study, only scores on callousness (i.e. the indifference to the feelings of others) were greater for males than females. It is possible that a low

rate of male respondents contributed to the lack of statistical difference across all psychometric traits between sexes in this investigation. Although the variance in data for each psychometric was comparable between sexes, the male cohort made up only a quarter of the overall sample, and after removal of incomplete responses, yielded just 32 datasets.

The second key finding of this investigation was that regardless of sex, physical aggression correlated with callousness, but not uncaring or unemotional traits, after controlling for age and CU/aggressive traits not included in the specific analysis. This finding contrasts previously reported relationships between psychometric-based measures of physical aggression and CU traits (Feilhauer et al., 2012). Although established in adolescents, not adults, Feilhauer et al. documented positive associations between AQ-measured physical aggression, and all three components of the ICU - compounding the well-established association between CU traits and behaviours relating to aggression (Frick & Dickens, 2006).

In this investigation, an association between physical aggression and callousness, but not uncaring or unemotional traits, might signal differential relationships between CU traits and physical aggression across age. Whilst useful for identifying the possibility of age-specific relationships, further validation is needed in the form of direct group comparisons and/or longitudinal research. From these findings, it is possible that the relationships between physical aggression and unemotional and uncaring traits, indicative of low emotional reactivity and caring towards others (Ciucci & Baroncelli, 2014), might represent differently in adult and adolescent cohorts.

Bivariate analysis of the ICU suggested that in line with some (Berg et al., 2013; Essau et al., 2006; Fanti et al., 2009) but not all (Guelker et al., 2014) factor analyses of the ICU, callousness was positively associated with uncaring traits in the composite sample. As a function of sex, although callousness and uncaring traits were positively correlated in females, but not males, effect sizes

were comparable after controlling for sample size. Therefore, it is possible that a statistically significant association between callousness and uncaring traits in males might emerge in a larger cohort. However, in contrast to results presented here, positive correlations between unemotional traits and both callousness (Essau et al., 2006; Fanti et al., 2009) and uncaring traits (Berg et al., 2013; Essau et al., 2006; Fanti et al., 2009; Guelker et al., 2014) have been reported in adolescent samples elsewhere. Though potentially an artefact of the sample reported here being derived from an adult cohort, because of the low internal consistency of the unemotional subscale of the ICU (Essau et al., 2006; Fanti et al., 2009; Kimonis et al., 2008), conclusions should be drawn with caution (Guelker et al., 2014).

Results of this investigation should be considered in light of two limitations. First, only associations between CU traits and physical aggression were reported leaving associations with other subscales of the AQ (i.e. verbal and indirect aggression, anger, hostility) unknown. This decision was taken with the understanding that the assessment of other aggressive traits would require a far larger sample size in order to produce sufficient statistical power. Additionally, as the key aims of this thesis involve delineating the relationships between physical aggression/CU traits and the electrophysiological indices of the VIM (*see chapter five, section 5.1 [page 82]*), these specific analyses were computed with the prospect of better understanding results reported elsewhere in this thesis. However, as males and females have long been shown to aggress in qualitatively different manners (White & Kowalski, 1994), there is clear importance of capturing this distinction in relation to CU traits. Second, as noted on page 93, although this analysis controlled for age within the sample, it does not allow for a direct comparison between adolescent and adult cohorts. This is an important avenue of exploration given previous declarations of aggressive and CU traits evidencing the capacity to alter over time, as a function of maturation and intervention (Moffitt, 1993).

In summary, although CU traits are thought to remain stable throughout adolescence and early adulthood (Frick, et al., 2003; Frick & White, 2008; Kruh et al., 2005), current findings suggest their association with physical aggression might not. In this adult cohort, across both males and females, physical aggression was shown to correlate with callousness, but not uncaring or unemotional traits. Discussed in light of study limitations, these results suggest trait-specific relationships between physical aggression and CU traits. Future investigation should observe these associations as a function of age, using group comparisons or longitudinal measures, whilst exploring similar associations with other manifestations and motivational underpinnings of aggression.

Chapter Seven: Baseline assessment of the Facial Affect Stop-Go Task

7.1 General Introduction

The VIM is a social-developmental model thought to underpin the extinction of a behavioural response, as a function of the successful perception of distress (Blair et al., 1995, 1999; Blair, 1995, 2001, *see chapter one, section 1.3.4 [page 12]*). The high temporal resolution of EEG makes it a useful tool for disentangling the time course of facial affect processing and motor extinction stages of the VIM. However, to date, electrophysiological signatures of the VIM have not been identified. In order to achieve this, a novel FAST is required. This chapter is split into two sections. The first documents the development of a FAST through three pilot studies, each accompanied by discussion of paradigm utility and shortcomings. The second uses the final iteration of the FAST to characterise ERPs related to facial affect processing and distress-induced motor extinction.

7.2 Development of the Facial Affect Stop-Go Task

7.2.1 Task Development

In laboratory settings, motor inhibition is typically investigated using go-nogo and SSTs (Aron, 2007, *see chapter one, section 1.3.5 [page 19]*). Whereas go-nogo paradigms allow investigation of pre-motor inhibition, SSTs better reflect motor extinction after the fact (Verbruggen & Logan, 2008). Previously, nogo and stop stimuli have consisted of auditory (e.g. tones, white noise) and visual (e.g. geometric shapes, frames) stimuli, as well as colour changes there of (Huster et al., 2014; Johnstone et al., 2007; Rubia et al., 2003). However, as argued in chapter one, section 1.3.5 (page 19), the use of emotion, specifically distress, as a stop stimulus is absent in existing literature. The use of distress as a stopping mechanism is imperative to the exploration the VIM on an electrophysiological level.

7.2.1.1 Stimuli

Investigation of the VIM using EEG requires a paradigm whereby one stimulus cues the initiation of a motor response, and a second commands the need to halt said response. As the VIM underpins inhibition of aggression (Blair, 1995), which itself might manifest in response to threat (Blanchette, 2006; Fox et al., 2007, *see chapter one, section 1.2.1.1 [page 2]*), angry facial expressions are fitting stimuli to signal a motor response. Likewise, as VIM motor extinction is thought to result from an empathic response to facial distress (Blair, 2001; Decety & Jackson, 2004, *see chapter one, section 1.3.4 [page 11]*), fearful and sad facial expressions are appropriate signals for motor extinction.

The Hillyard Principle (*see Luck, 2005*) dictates that in order to account for as much variance in the data as possible, where appropriate, stimuli should consist of similar physical properties. In the context of the FAST, although necessary to use several facial identities as to not attenuate the N170 response (*see chapter three, section 3.2.1 [page 39]*), trial-specific stimuli should be of the same identity. This approach has additional benefit for increasing task validity. For this paradigm, male and female facial expressions were chosen from the MacBrain NimStim Face Stimulus Set (Tottenham et al., 2009). Facial stimuli with open mouths were chosen in order to increase affect valence.

7.2.1.2 Trials

As discussed in chapter three, section 3.1 (page 37), accurate delineation of ERPs from resting state EEG requires high SNR (Luck, 2005). There is no definitive rule as to the optimum number of trials within a given EEG experiment, and so trial quantity largely depends on the sample, stimulus, environment, and ERP of interest (Luck, 2004, 2005). Although Luck (2004) suggests that as few as 30 trials per condition, per stimulus might be enough to accurately model the P300, as many as 200 trials might be required for comparable accuracy of earlier components. As the FAST seeks to model both early (e.g. N170, P200) and late (e.g. N200, P300) ERP components within a single paradigm, a compromise must be reached. Furthermore, trial quantity

must be considered in light of practicality and participant engagement. For example, whilst the inclusion of a 100 trials per condition might be ideal, responses from dis-engaged/tired participants would likely detriment accurate interpretation of the EEG signal.

7.2.1.3 Stimulus Presentation and Filtering

As ERPs are capable of exceeding the initiation of a subsequent ERP, temporal overlap of ERP components should be minimised (*see chapter three, section 3.1 [page 37]*). Overlapping ERPs detriment waveform interpretation, and are particularly problematic for stop paradigms where shorter inter-stimulus intervals (ISI) are required (Woldorff, 1993). In these instances, stop stimuli need to be presented prior to the completion of a motor response. During a FAST, ERPs evoked to go stimuli (i.e. angry facial expressions) would likely overlap with those evoked to subsequent stop stimuli (i.e. fearful/sad facial expressions).

A case has been made for the partial removal of temporal overlap using high-pass filters (Woldorff, 1993). Whilst ideal for investigation of high-frequency components, such as those used to index early auditory brainstem responses, high-pass filtering distorts long latency, slow-wave components such as the P300, which become attenuated (Luck, 2005).

A second method of offline ERP deconvolution, the two-stage adjacent response technique (ADJAR), reduces temporal overlap through jittering the ISI around the mean (Luck, 2005; Woldorff, 1993). After averaging across trials, stage one of the ADJAR acts as a low-pass filter in order to preserve the time-locked, stimulus-specific ERP signal by averaging out trial-by-trial interactions. For greater effect (i.e. less temporal overlap), the range of the ISI must be sufficiently wide in order to encapsulate overlapping ERP components. Woldorff (1993) suggests a duration that surpasses that of the slowest dominant waveform likely to interact with the subsequent ERP.

The second stage of the ADJAR is applied when temporal overlap is too severe to be attenuated by jittering the ISI alone. In these instances, estimation of the exact overlap between the first and second stimulus is derived over a series of iterations. Iterations are varied across the jittered ISIs with the final estimation of overlap being defined when the model can no longer be manipulated. Subtraction of this estimation from the ERP response to the second stimulus removes any residual EEG signal relating to stimulus one, resulting in a waveform depicting the ERP response to the second stimulus alone. However, the ADJAR is limited to instances whereby a substantial ISI range can be used. In Bekker et al. (2005), a range of 240 ms was not sufficient to completely remove overlapping ERP responses. The following sections outline three pilot studies documenting the initial iterations of the FAST.

7.2.2 FAST v.1

7.2.2.1 Participants

Six undergraduate students (3 females; M age = 19.66; SD = .82) were recruited by opportunity sampling from Nottingham Trent University.

7.2.2.2 Procedure

Data were recorded in a sound-attenuated room. After consenting, participants were sat 60 cm away from a 19" CRT monitor (1024 x 768 resolution; 85 Hz refresh rate). EEG apparatus was shown and attached to participants and stability checks were made. Participants completed version one of the FAST (*see Figure 3*), which consisted of 210 trials (90 background; 60 go; 60 stop). Background stimuli consisted of neutral, surprised, and happy facial expressions, go stimuli consisted of angry facial expressions, and stop stimuli consisted of fearful and sad facial expressions. Stimuli comprised twenty identities (10 females; African- and Caucasian-American; IDs 01, 03, 05, 06, 07, 08, 09, 10, 20, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36).

Participants completed twenty practice trials using two identities not in the experimental trials (1 female; African-American; IDs 13, 38). Following a lead in period of 4000 ms, participants were randomly presented with a background or go stimulus. Participants were asked to respond to go stimuli (200 ms, 60 ms jitter) via a button press. On 50% of go trials, participants were presented with a stop stimulus (200 ms), to which they were asked to not complete their motor response. Trials were separated by a 900 ms inter-trial-interval (100 ms jitter).

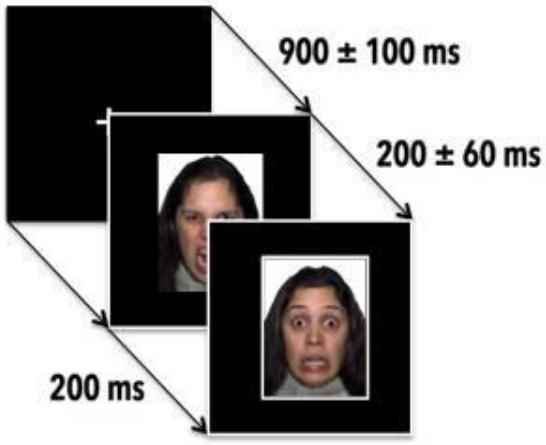

Figure 3. FAST version 1. Participants respond to infrequent go (i.e. angry) stimuli among frequent background (i.e. neutral, surprised, happy) stimuli. Following a response period of between 140 and 260 ms, a stop stimulus (i.e. fear, sadness) denoted the requirement of motor response extinction.

7.2.2.3 Physiological Data Acquisition

EEG activity was recorded using an active-electrode, 64-channel Active-Two acquisition system (BioSemi, Amsterdam, Netherlands), sampled at 2048 Hz and digitised at 24-bits. Impedances were kept below 5 kohms as recommended in Luck (2005). Data were collected using ActiView v.6.05 (BioSemi, Amsterdam, Netherlands). All EEG signals were average referenced on-line. The N170, P200, N200, and P300 were measured in response to background stimuli, and the stop-N200 and stop-P300 were measured in response to stop stimuli.

7.2.2.4 Signal Processing

Vertical and horizontal electrooculography artefacts were identified and corrected (Jung et al., 2000). A high-pass filter of .01 Hz (~ 6 Hz, forward shift) was used to remove slow drifts as described in section 7.2.1.3 (page 104) and a low-pass filter of 35 Hz (~ 24 dB/oct, 0-phase shift) was applied to remove high frequency waves. A 50 Hz notch filter was used to remove electrical noise. Trials were baseline corrected prior to averaging (-200 ms).

The N170 and P200, evoked to both go and background trials, were referenced to the average of all the electrodes in order to avoid attenuating ERPs measured over temporo-parietal sites (*see Joyce & Rossion, 2005 for review of reference-specific attenuation of the N170, as well as chapter three, section 3.2.1 [page 39]*). The N200 and P300, evoked to both go and stop trials, were re-referenced to the linked mastoids (i.e. the bone behind the ear) in order to [1] minimise any spatial distortion resulting from the use of average referencing (Luck, 2005), and [2] increase the generalisability of results as a function of the wide-use of linked mastoids when referencing later perceptual components (Luck, 2005; Woodman, 2010).

7.2.2.5 Results and Discussion

Data inspection and participant feedback revealed two confounding problems. First, participants periodically reported an inability to accurately interpret the emotion depicted by stimulus one. This deficit is likely a function of the relatively brief 140 to 260 ms display period being insufficient to accurately categorise facial affect. Previously, Fox et al. (2000) noted that participants in their investigation reported facial expressions - including those depicting anger thought to command a rapid response (*see chapter one, section 1.3.1 [page 7]*) - were difficult to interpret when presented for 300 ms. This was supported by behavioural data evidencing increased reaction times and error rates for neutral, happy, and angry schematic faces.

Second, ERPs evoked to stimulus one and two still presented extreme convolutions after averaging across jittered trials. Such convolution prevented the accurate interpretation of the P200, N200, and P300 evoked to stimulus one, and also the N170 and P200 evoked to stimulus two. As fearful and sad facial expressions were not used as background stimuli, distortion of N170 and P200 responses to stimulus two means that the FAST v.1 does not enable accurate assessment of N170 and P200 responses to fearful and sad facial expressions.

Although a jitter period of over 300 ms is recommended, such that Woldorff (1993) refers to a 'short jitter' epoch of between 150 to 350 ms, this pilot only used a jitter period of 120 ms around a 200 ms mean (140 to 260 ms). In this paradigm, this jitter period was justified by the need for the stop stimulus to be temporally close to the go stimulus in order to allow successful motor extinction. Elongation of this jitter period would have likely resulted in participants completing go responses prior to the presentation of stop stimuli.

7.2.3 FAST v.2

The second iteration of the FAST (v.2) addressed the problems highlighted in section 7.2.2.5 (page 107), that of [1] participant-reported inability to accurately categorise facial expressions represented in stimulus one, and [2] temporal overlap distorting the P200, N200, and P300 responses to stimulus one and the N170 and P200 responses to stimulus two.

7.2.3.1 Participants

Six undergraduate students (2 females; M age = 19.66, SD = .52) were recruited by opportunity sampling from Nottingham Trent University.

7.2.3.2 Procedure

Data were recorded in a sound-attenuated room. After consenting, participants were sat 60 cm away from a 19" CRT monitor (1024 x 768 resolution; 85 Hz

refresh rate). EEG equipment was shown and attached to participants before stability checks were made. Participants completed the FAST v.2 (*see Figure 4*). Background stimuli consisted of neutral, fearful, and sad facial expressions, go stimuli consisted of angry facial expressions, and stop stimuli consisted of fearful and sad facial expressions of seventeen identities (10 females; African- and Caucasian-American; IDs 01, 03, 05, 06, 07, 08, 09, 10, 20, 21, 23, 25, 26, 32, 34, 35, 36).

Twenty practice trials were given using two identities not in the experimental trials (1 female; African-American; IDs 13, 38). The paradigm consisted of 272 trials, split into 2 counterbalanced blocks. Each block began with a 4000 ms lead-in, with each trial consisting of the random presentation of a background or go stimulus for 250 ms. A black display (500 ms) followed stimulus one, prior to presentation of a go cue (red cross; 200 ms). Participants were asked to respond via a button box press when the go cue followed a go stimulus (i.e. angry face), but not when it followed a background stimulus (i.e. neutral, fearful, or sad face). On 50% of the go trials, participants were presented with a stop stimulus (i.e. fearful or sad faces; 250 ms), upon which they were instructed to halt their motor responses. Trials were separated by a 900 ms (100 ms jitter), inter-trial-interval.

7.2.3.3 Physiological Data Acquisition

Physiological data acquisition was identical to that reported in section 7.2.2.3 (page 106).

7.2.3.4 Signal Processing

Signal processing was identical to that reported in section 7.2.2.4 (page 107).

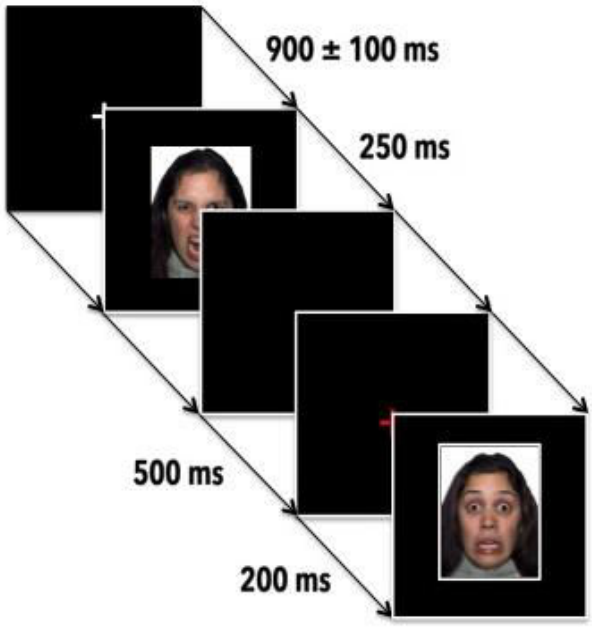

Figure 4. FAST version 2. Participants observe infrequent go (i.e. angry) stimuli amongst frequent background (i.e. neutral, fearful, sad) stimuli for a duration of 250 ms. Following a blank display lasting 500 ms, if stimulus one was a go stimulus, participants were asked to respond via button box when they observed a red cross. A stop stimulus (i.e. fearful, sad) denoted the requirement of motor response extinction.

7.2.3.5 Results and Discussion

The second pilot investigation sought to address the limitations of categorisation and temporal overlap outlined in section 7.2.2.5 (page 107). In the FAST v.1, participants reported the duration of 140 to 260 ms was not sufficient enough to accurately categorise facial expressions. In the second iteration of the FAST, stimulus presentation times were increased to a fixed 250 ms in order to better facilitate stimulus processing. Furthermore, as a means of moderating ERP convolution and distortion of the early face-related ERP responses to stop stimuli, the FAST v.2 included sad and fearful facial expressions as background stimuli. This inclusion provided a means of measuring N170 and P200 responses to distress, without interference from preceding ERP waveforms. As to not elongate the paradigm, the outcome of which is highlighted in section 7.2.1.2 (page 103), surprised and happy facial

expressions were removed as background stimuli. Although the use of fearful and sad facial expressions as both background and stop stimuli had potential to habituate ERP responses to distress (*see chapter three, section 3.2.1 [page 45]*), visual inspection of data recorded during the first and final third of the paradigm did not reveal such habituation to either fearful or sad facial expressions.

Piloting of the FAST v.2 yielded three limitations. First, although the duration of stimulus one was elongated to 250 ms, participants yet again reported low confidence in their responses. To assure validity of this paradigm as a means of investigating the VIM on an electrophysiological level, it is vital that participants have awareness of the stimuli that they are initiating, and extinguishing motor response to. Furthermore, frequent presentation of stimuli within a given trial (i.e. stimulus one, black screen, go cue, stimulus two), over successive trials, led to participants reporting disorientation. Disorientation likely hinders the ability to efficiently categorise facial affect - ultimately manifesting in poorer response accuracy and reduced task validity.

Second, the go cue (i.e. red cross) evoked a slow, low frequency wave between 300 and 700 ms post stimulus onset, which did not return to baseline before presentation of stimulus two. This ERP overlapped with, and distorted, N170 and P200 responses to stimulus two. Although still possible to measure N170, P200, N200, and P300 responses to stimulus one, residual temporal overlap of the slow wave evoked to the red cross would likely distort ERP responses evoked during stop trials. Undistorted measurement of stop-ERPs is key to understanding the affect-specific electrophysiological distinctions during motor extinction.

Third, from a behavioural perspective, the 200 ms ISI between the go cue and stop stimulus was not short enough to prevent completion of a go response. With feedback from the first iteration of the FAST suggesting 200 ms was sufficient to successfully initiate motor extinction, disparity between these two

pilot investigations might be explained through priming. Whilst participants were instructed to initiate a go response directly following stimulus one onset in the FAST v.1, participants were asked to withhold this response until after the presentation of the go cue - some 750 ms post stimulus onset - in the FAST v.2. Priming could decrease motor response times as a function of resolved processing conflict (Kristjánsson & Jóhannesson, 2014).

7.2.4 FAST v.3 and Experimental Methods

The third iteration of the FAST (v.3) addressed the limitations of the FAST v.2 highlighted in section 7.2.3.5 (page 110), that of [1] disorientation to frequent stimulus presentation, [2] a slow wave component evoked to the go cue overlapping N170 and P200 responses to stimulus two, and [3] response priming.

7.2.4.1 Participants

Six undergraduate students (2 females, M age = 19.66, SD = .82) were recruited by opportunity sampling from Nottingham Trent University.

7.2.4.2 Procedure

Data were recorded in a sound-attenuated room. After consenting, participants were sat 60 cm away from a 19" CRT monitor (1024 x 768 resolution; 85 Hz refresh rate). EEG equipment was shown and attached to participants before stability checks were made. Participants completed the FAST v.3 (*see Figure 5*). Go and stop stimuli consisted of neutral, angry, sad, fearful, and surprised expressions from 17 identities (8 females; African- and Caucasian-American; IDs 01, 03, 05, 06, 07, 08, 09, 10, 20, 21, 23, 25, 26, 32, 34, 35, 36). The paradigm consisted of 272 trials, split into 2 counterbalanced blocks. Each block contained 8 affect-pair combinations (Background Pairs: *Fear-Fear; Sad-Sad; Neutral-Sad; Neutral-Fear*, Go Response Pairs: *Anger-Anger; Anger-Surprise,* Stop Response Pairs: *Anger-Fear; Anger-Sad*).

Each block had a 4000 ms lead-in followed by presentation of stimulus one (800 ms; 100 ms jitter), a black screen acting as an ISI (160 ms; 40 ms jitter) and finally presentation of stimulus two (800 ms; 100 ms jitter). A red fixation cross accompanied an inter-trial-interval of 1800 ms (200 ms jitter). Participants were instructed to place their right index finger on a red button box key, moving it to an adjacent green key following the extinction of a go stimulus (i.e. angry face). Participants were instructed to interrupt go responses by returning their finger to the red key, if stop stimuli subsequently appeared. If stimulus one was not an angry facial expression no response was required at all during the trial. Twenty practice trials were given using two identities not in the experimental trials (1 female; African-American; IDs 13, 38).

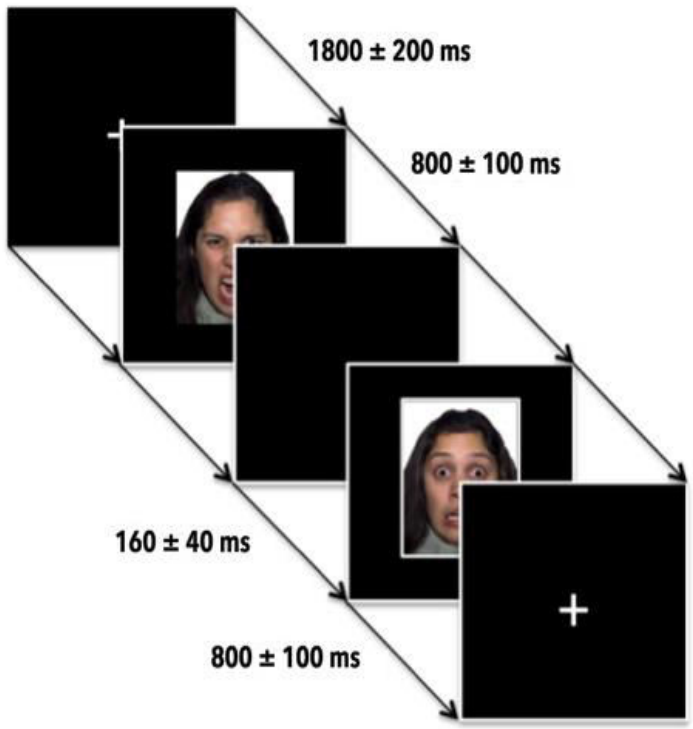

Figure 5. FAST version 3. Participants responded to infrequent go (i.e. angry) stimuli among frequent background (i.e. neutral, fearful, sad) stimuli, presented between 700 and 900 ms, when the go stimulus dissipated. Following a black screen lasting between 120 and 200 ms, a stop stimulus (i.e. fear or sadness) was presented, denoting the requirement of motor response extinction.

7.2.4.3 Physiological Data Acquisition

Physiological data acquisition was identical to that reported in section 7.2.2.3 (page 106).

7.2.4.4 Signal Processing

Signal processing was identical to that reported in section 7.2.2.4 (page 107).

7.2.4.5 Results and Discussion

The aim of the third pilot investigation was to address the limitations highlighted in response to the FAST v.2 as outlined in section 7.2.3.5 (page 110), namely that of disorientation, go cue related slow wave activity, and response priming. First, participant-reported deficits in categorisation efficiency and feelings of disorientation were resolved by extending the presentation duration uniformly across stimuli from 250 ms to 800 ms. In Fox et al. (2000), greater accuracy of facial affect categorisation was observed after increasing the stimulus duration from 300 ms to 800 ms. In this investigation, elongation of stimulus duration facilitated [1] face perception accuracy, and [2] the measurement of deconvoluted ERP waveforms in response to both stimulus one and two. In the main, participants reported accurate affect categorisation.

Second, removal of the go cue, observed in the FAST v.2 to evoke a slow waveform that became convoluted with the ERP evoked to stimulus two, facilitated the accurate measurement of ERP responses to stimulus two. Third, contraction of the ISI between the go and stop stimuli was reduced to 160 ms (40 ms jitter) in order to combat priming effects. Participants reported that this ISI provided a suitable period in which to initiate and extinguish motor responses.

7.3 Testing of the Facial Affect Stop-Go Task

7.3.1 Introduction

Electrophysiological indices of facial affect processing can be split into early (e.g. N170; P200) and late (e.g. N200; P300) components. Evoked to faces, the N170 and P200 are thought to index the integrated processing of structural (i.e. identity) and second-order (e.g. task-salient, face components) facial features, respectively (Apicella et al., 2013; Hinojosa et al., 2015; Itier & Taylor, 2004) and show temporo-parietal dominance over the right hemisphere (Bentin et al., 1996; Hinojosa et al., 2015; Rossion et al., 2003).

Although conflicting results have been reported elsewhere (*see chapter three, sections 3.3.1 [page 45] and 3.3.2 [page 47]*), the N170 and P200 have shown a bias towards negative, over positive or neutral imagery (Chai et al., 2012; Smith et al., 2003). A negativity bias likely reflects reallocation of neuronal resources when processing biologically significant social stimuli. Specifically, the N170 shows augmentation in response to angry, relative to happy facial expressions (Eger et al., 2003; Ibánez et al., 2012; Krombholz, 2007), and in turn shows additive augmentation to fearful expressions (Batty & Taylor, 2003; Blau et al., 2007; Taylor et al., 2004; Ramos-Loyo et al., 2009; Shannon et al., 2013). Similarly, P200 augmentation is observed to facial threat (Chai et al., 2012; Gonzalez-Roldan et al., 2011) and distress (Shannon et al., 2013). Though not previously investigated, as sad facial expressions are indicative of distress, N170 and P200 augmentation resembling that evoked to fear would be expected.

N200 and P300 ERP components are typically evoked prior to motor responses and appear maximal over anterior and parietal midline sites, respectively (Patel & Azzam, 2005; Polich, 2007). Whilst both ERP components index higher order, cognitive control processes (Hillyard & Kutas, 1983), the N200 best encapsulates stimulus classification and conflict monitoring (Folstein & van Petten, 2008; van Veen & Carter, 2002), and the P300 better indexes working

memory and orientation of neuronal resources to motivationally relevant stimuli (Polich, 2007).

During face processing, relative to happy and neutral facial expressions, fearful faces evoke N200 and P300 augmentation (Campanella et al., 2013; Dennis & Chen, 2007), with P300 augmentation also evident to angry (Lang et al., 1990) and sad facial expressions (Ehlers et al., 2001). Such amplitude variation likely denotes threat, in the form of anger and distress, creating processing conflicts that require additional neuronal resources to resolve.

The N200 and P300 can also be evoked to stop stimuli demanding the halting of a motor response, and are maximal over frontal midline sites (Bruin & Wijers, 2002; Dimoska et al., 2006; Kok et al., 2004; Pliszka et al., 2000). Anterior scalp topographies likely index neuronal generators associated with motor extinction such as the superior- and medial-frontal gyri, OFC, and ACC (Amodio et al., 2008; Enriquez-Geppert et al., 2010). Whilst facial affect has previously been used in motor inhibition paradigms (*see chapter three, section 3.4 [page 50]*), neither nogo paradigms nor SSTs have used facial affect as a stopping mechanism. Therefore, the role of facial distress (i.e. fear and sadness) on stop-N200 and stop-P300 responses remains unknown.

This investigation sought to delineate the effect of angry, sad, and fearful facial stimuli on ERPs associated with face processing (N170 and P200) during a FAST. Additionally, it aimed to outline how fearful and sad facial expressions moderate the N200 and P300 during both background and stop conditions. The N170 and P200 were hypothesised to [1] exhibit right hemispheric dominance and [2] augment in response to fearful and sad, and in turn angry facial expressions, relative to neutral faces. The N200 was hypothesised to show [3] an anterior scalp distribution to both background and stop stimuli with the P300 being maximal at [4] parietal sites to background stimuli and [5] anterior sites to stop stimuli. Both the N200 and P300 were expected to [6] show greater augmentation to fearful, relative to sad facial expressions.

7.3.2 Methods

7.3.2.1 Participants

Fifty-seven psychology students (35 females, M age = 19.25, SD = 1.89) were recruited from Nottingham Trent University after replying to an online advertisement. Inclusion criteria required participants to be right-handed, 18 to 65 years old, and without current diagnosis of psychiatric or neurological disorders, or medication that might affect electrophysiological responses. Participants refrained from consuming alcohol for twenty-four hours, and nicotine and caffeine for three hours prior to experimentation. Participants were compensated for their time with research participation credits.

7.3.2.2 Procedure

After written consent was obtained, participants completed the FAST v.3 as described in section 7.2.4.2 (page 112).

7.3.2.3 Physiological Data Acquisition

Physiological data acquisition was identical to that reported in section 7.2.2.3 (page 106). Mean peak amplitudes were measured using BESA Research v.5.3.7 (BESA GmbH, Gräfelfing, Germany) for N170 (150 to 220 ms interval), P200 (180 to 250 ms interval), N200 (210 to 310 ms interval), and P300 (300 to 480 ms interval) responses. On go/background trials, ERP responses were time-locked to the presentation of stimulus one, with ERP responses time-locked to presentation of stimulus two on stop trials. The measurement of mean, over peak, amplitudes mediated ERP attenuation thought to result from averaging multiple trials over various latencies (Luck, 2005).

7.3.2.4 Signal Processing

Signal processing was identical to that reported in section 7.2.2.4 (page 107).

7.3.2.5 Statistics

Average temporo-parietal N170 and P200 responses for each emotion were created using left (P7, PO7, O1, PO3) and right (P8, PO8, O2, PO4) electrodes. The N200 and P300 for both background and stop conditions were measured bilaterally and at midline electrodes at both anterior and parietal sites (F5, Fz, F6, P5, Pz, P6). Mean ERP amplitudes were entered into an R Statistics v.2.15 database (R Core Team, 2012).

For the N170 and P200, analyses of variance (ANOVA) were computed using the within-subjects factors *emotion* (i.e. anger, fear, sadness, neutral) and *hemisphere* (i.e. left, right). For the N200 and P300, ANOVA were computed using the within-subjects factors *emotion* (i.e. fear, sadness), *hemisphere* (i.e. left, midline, right), and *task* (i.e. background, stop). Interaction effects were computed for each ERP. *Post hoc* second order ANOVAs and *t*-tests were used to delineate statistically significant interactions. All *post hoc* tests were corrected for multiple comparisons (*see Benjamini & Hochberg, 1995*). Partial Eta squared is reported for each main effect and interaction as an index of effect size. As the N170 and N200 are negative deflections of the ERP, correlations are inverted. For example, in the context of the N170 and N200, inverse correlations between psychometrics and negative amplitudes suggest a reduction in amplitude, and are presented as positive associations. *T*-tests were used to identify differences in successful motor extinction and associated reaction times between fearful and sad stop stimuli.

7.3.3 Results

7.3.3.1 Behavioural

There was no significant difference in motor extinction success between fearful and sad facial stimuli (fearful [81.58%, SE = 2.48], sad [82.97%, SE = 2.31]; t [56] = 1.37, p = .18, *padj* = .31). However, motor extinction reaction time were shorter to sad (762.29 ms, SE = 26.90) than fearful (822.13 ms, SE = 30.48) stop stimuli (t [56] = 4.20, $p < .001$, *padj* < .001).

7.3.3.2 N170

Means and standard deviations of N170 amplitudes evoked to fearful, sad, angry, and neutral facial expressions are summarised in Table 4.

There was no statistically significant main effect of *hemisphere* (left [2.00 μv, SE = .41]; right [2.04 μv, SE = .38]; F [1, 56] = .02, p = .90, η_p^2 = .00) on N170 amplitude. Neither was there a statistically significant interaction between *hemisphere* and *emotion* (F [3, 54] = 2.43, p = .08, η_p^2 = .09).

Table 4.
Descriptions of N170 amplitudes across emotion and hemisphere to background stimuli

Emotion	Hemisphere	M [μv]	SD
Fear	Left	1.77	3.21
	Right	2.23	3.41
Sad	Left	1.69	3.30
	Right	2.31	3.04
Anger	Left	2.24	3.03
	Right	2.08	3.44
Neutral	Left	1.56	3.10
	Right	2.32	2.64

Note. **Left site comprised an average of P07, P7, P03, and O1 electrodes. Right site comprised an average of P08, P8, P04, and O2 electrodes.**

A main effect of *emotion* on the N170 was identified (F [3, 54] = 12.58, p < .001, η_p^2 = .41). Although Mauchly's test indicated violation of the assumption of sphericity (X^2 [5] = 29.65, p < .001), the effect held after correcting degrees of freedom using Greenhouse-Geisser sphericity estimates (ε = .74, F [2.23, 124.64] = 6.37, p < .05, η_p^2 = .10). The effect of *emotion* on the N170 was explored using six paired-sample t-tests. As evidenced in Figure 6, the effect of *emotion* was driven by larger N170 amplitudes to anger (1.61 μv, SE = .37) than fearful (2.00 μv, SE = .38; t [56] = 2.42, p = .02, *padj* = .04), sad (2.16 μv, SE = .39; t [56] = 3.29, p < .05, *padj* < .05), or neutral facial expressions (2.31 μv, SE = .34; t [56] = 6.02, p < .001, *padj* < .001). There was no statistically significant

difference in N170 amplitude between responses to fearful and sad (t [56] = -.71, p = .48, *padj* = .48), fearful and neutral (t [56] = -1.67, p = .10, *padj* = .15) or sad and neutral facial expressions (t [56] = -1.10, p = .28, *padj* = .33). Main effects are summarised in Table 8 (page 140).

7.3.3.3 P200

Means and standard deviations of P200 amplitudes evoked to fearful, sad, angry, and neutral facial expressions are summarised in Table 5 (page 121).

There was a statistically significant main effect of *hemisphere* on the P200 (F [1, 56] = 12.38, p < .001, η_p^2 = .18) such that the right, relative to left hemispheric P200 was consistently larger across all emotions (fear left [5.03 µv, SE = .37] vs. fear right [6.42 µv, SE = .40]; t [56] = -4.38, p < .001, *padj* < .001; sad left [5.21 µv, SE = .39] vs. sad right [6.29 µv, SE = .47]; t [56] = -2.62, p = .01, *padj* = .01; anger left [5.01 µv, SE = .39] vs. anger right [6.09 µv, SE = .48]; t [56] = -2.83, p = .01, *padj* = .01; neutral left [5.56 µv, SE = .37] vs. neutral right [6.62 µv, SE = .40]; t [56] = -3.46, p < .001 *padj* < .05). This effect is illustrated in Figure 6.

There was also a statistically significant main effect of *emotion* on the P200 (F [3, 54] = 7.66, p < .001, η_p^2 = .30), see Figure 6. Although violating Mauchly's test of sphericity (X^2 [5] = 22.63, p < .001), the effect remained significant after correcting degrees of freedom using Greenhouse-Geisser estimates of sphericity (ε = .79, F [2.36, 132.09] = 3.79, p = .02, η_p^2 = .06). Paired-sample t-tests suggested this effect was driven by larger P200 amplitudes to neutral (6.09 µv, SE = .35), relative to fearful (5.73 µv, SE = 35; t [56] = -2.13, p = .04, *padj* = .04), sad (5.75 µv, SE = .38; t [56] = -2.47, p = .02, *padj* = .05), and angry facial expressions (5.55 µv, SE = .39; t [56] = 4.68, p < .001, *padj* < .001). P200 amplitude did not differ in response to fearful and sad (t [56] = -.09, p = .93, *padj* = .93), fearful and angry (t [56] = .97, p = .34, *padj* = .40), and sad and angry facial expressions (t [56] = 1.28, p = .21, *padj* = .31). There was no statistically

interaction between *hemisphere* and *emotion* for the P200 (F [1, 54] = 1.06, p = .37, η_p^2 = .06). Main effects are summarised in Table 8 (page 140).

Table 5.

Descriptions of P200 amplitudes across emotion and hemisphere to background stimuli

Emotion	Hemisphere	M [µv]	SD
Fear	Left	5.03	2.75
	Right	5.21	2.90
Sad	Left	5.01	2.90
	Right	5.56	2.78
Anger	Left	6.42	3.04
	Right	6.29	3.58
Neutral	Left	6.09	3.70
	Right	6.62	2.98

Note. **Left site comprised an average of P07, P7, P03, and O1 electrodes. Right site comprised an average of P08, P8, P04, and O2 electrodes.**

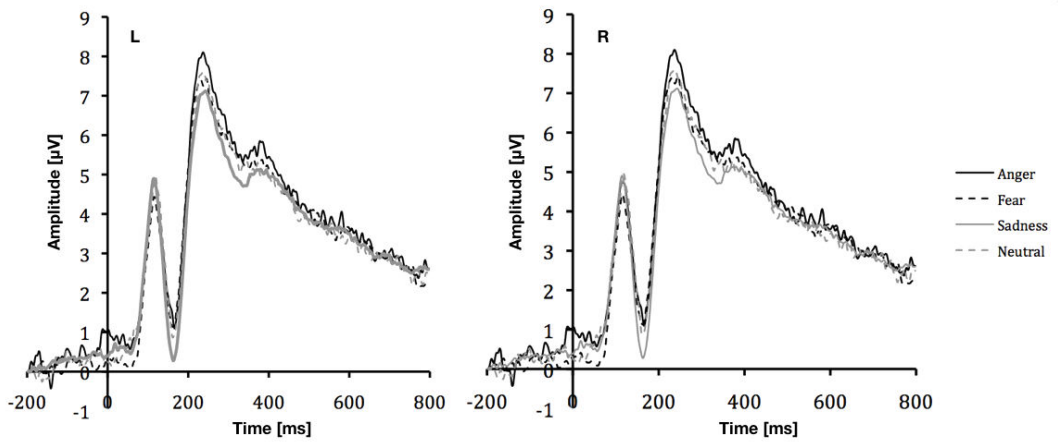

Figure 6. ERP waveforms to fearful, sad, angry, and neutral facial expression. Left ERP is an average of P07, P7, O1, and T7 electrodes. Right ERP is an average of P08, P8, O2, and T8 electrodes. ERP waveform is representative of EEG signal referenced to averaged electrodes.

7.3.3.4 N200

Means and standard deviations of N200 amplitudes evoked to fearful and sad facial stimuli across hemisphere, site, and task are displayed in Table 6 (page 123). Main effects, interactions, and *post hoc* analyses are summarised in Table 8 (page 140).

7.3.3.4.1 Main Effects

There was no statistically significant main effect of *emotion* (F [1, 56] = 2.01, p = .16, η_p^2 = .04) such that the N200 amplitude evoked to fearful facial expressions (.11 μv, SE = .12) did not differ significantly to that evoked to sad facial expressions (.17 μv, SE = .07). Similarly, there was no main effect of *Task* (F [1, 56] = .53, p = .47, η_p^2 = .01) with the N200 in background conditions (.17 μv, SE = .10) yielding similar amplitudes to the N200 in stop conditions (.11 μv, SE = .07).

There were two statistically significant main effects. First, a main effect of *site* (F [1, 56] = 113.06, p < .001, η_p^2 = .67) such that on average, N200 amplitudes were larger over anterior (-1.32 μv, SE = .13) than parietal sites (1.60 μv, SE = .18). Second, there was a main effect of *hemisphere* (F [2, 55] = 13.18, p < .001, η_p^2 = .32). Paired-sample t-tests suggested that on average, the N200 measured over midline sites (-.40 μv, SE = .18) was larger than when measured over both left (.19 μv, SE = .12; t [56] = 2.32, p = .02, $padj$ = .04) and right hemispheres (.63 μv, SE = .15; t [56] = -5.14, p < .001, $padj$ < .001). Although the left, relative to right hemisphere initially indicated larger N200 amplitudes, this did not survive Hochberg correction (t [56] = -2.00, p = .05, $padj$ = .08). These main effects are illustrated in Figure 7 (page 130).

Table 6.

Descriptions of N200 amplitudes across emotion, hemisphere, site location, and task

Emotion	Stimuli	Site	Hemisphere	M [μv]	SD
Fear	Background	Anterior	Left	-2.15	2.16
			Midline	-3.36	1.65
			Right	-2.08	1.84
		Posterior	Left	2.79	2.52
			Midline	1.66	2.78
			Right	4.03	2.89
	Stop	Anterior	Left	-.42	1.80
			Midline	.27	2.16
			Right	-1.02	1.72
		Posterior	Left	.38	1.94
			Midline	-.01	2.33
			Right	1.19	2.46
Sad	Background	Anterior	Left	-2.23	1.84
			Midline	-3.37	1.88
			Right	-1.64	1.71
		Posterior	Left	2.64	2.11
			Midline	1.61	2.83
			Right	4.16	2.99
	Stop	Anterior	Left	.18	1.91
			Midline	.24	1.83
			Right	-.25	1.51
		Posterior	Left	.34	1.77
			Midline	-.23	2.15
			Right	.64	2.25

Note. **Anterior and parietal sites are measured at F and P electrodes, respectively with left, midline, and right hemispheric sites indexed by corresponding electrodes at sites 5, z, and 6, respectively.**

7.3.3.4.2 Interactions

The variables of *hemisphere, task, site,* and *emotion* did not yield a statistically significant four-way interaction (F [2, 55] = 2.79, p = .07, η_p^2 = .09). Furthermore, *emotion* did not interact with *hemisphere* (F [2, 55] = 1.96, p = .15, η_p^2 = .07), *task* (F [1,56] = .13, p = .72, η_p^2 = .01), *hemisphere * task* (F [2,55] = 2.00, p = .15, η_p^2 = .07), or *site * task* (F [1, 56] = 2.74, p = .10, η_p^2 = .05). An initial significant interaction between *hemisphere* and *site* (F [2, 55] = 6.89, p < .05, η_p^2 = .20) violated Mauchly's test of sphericity (X^2 [2] = 9.78, p = .01) and after adjusting degrees of freedom using Greenhouse-Geisser estimates of sphericity (ε = .86), did not survive (F [1.72, 96.33] = 2.70, p = .08, η_p^2 = .05).

There was a significant interaction between *emotion* and *site* (F [1, 56] = 7.51, p = .01, η_p^2 = .12). Larger N200 amplitudes were recorded over anterior, relative to parietal sites, to both sad (anterior [-1.18 μv, SE = .12], parietal [1.53 μv, SE = .17]; t [56] = -10.70, p < .001, *padj* < .001) and fearful facial expressions (anterior [-1.46 μv, SE = .16], parietal [1.68 μv, SE = .20]; t [56] = 9.95, p < .001, *padj* < .001). Though not present at parietal sites (t [56] = 1.75, p = .09, *padj* = .11), fearful, relative to sad, facial expressions augmented N200 amplitudes at anterior sites (t [56] = -2.90, p = .01, *padj* = .01).

A statistically significant three-way interaction between *hemisphere, site* and *task* (F [2, 56] = 3.94, p = .03, η_p^2 = .13) violated Mauchly's test of sphericity (X^2 [2] = 7.22, p = .03) but maintained statistical significance following correction of degrees of freedom using Greenhouse-Geisser estimates of sphericity (ε = .89; F [1.78, 99.73] = 11.23, p < .001, η_p^2 = .17). This interaction was explored using *post hoc* lower order ANOVAs.

7.3.3.4.2.1 Hemisphere * Site | Task

Evoked to background stimuli, there was a statistically significant main effect of *hemisphere* (F [2, 55] = 28.45, $p < .001$, $\eta_p^2 = .51$) such that on average, N200 amplitude was larger over midline sites (-.87 μv, SE = .21), relative to both left (.26 μv, SE = .14; t [56] = 4.25, $p < .001$, *padj* < .001), and right hemispheres (1.12 μv, SE = .18; t [56] = 7.61, $p < .001$, *padj* < .001). N200 amplitude was greater in the left, than right hemisphere (t [56] = 3.48, $p < .001$, *padj* < .001). There was also a significant main effect of *site* (F [1, 56] = 209.58, $p < .001$, $\eta_p^2 = .79$) such than on average, N200 amplitude was larger over anterior, relative to parietal sites (anterior [-2.47 μv, SE = .17], parietal [2.82 μv, SE = .24]). Paired-sample *t*-tests suggested N200 amplitudes were larger over anterior, than parietal sites at left (anterior [-2.19 μv, SE = .24], parietal [2.72 μv, SE = .28]; t [56] = 11.19, $p < .001$, *padj* < .001), right (anterior [-1.86 μv, SE = .21], parietal [4.10 μv, SE = .38]; t [56] = -12.15, $p < .001$, *padj* < .001), and midline sites (anterior [-3.37 μv, SE = 1.63], parietal [1.63 μv, SE = .36]; t [56] = 12.12, $p < .001$, *padj* < .001).

After correcting degrees of freedom using estimates of sphericity (ε = .90) following a violation of the assumption of sphericity using Mauchly's test (X^2 [2] = 6.73, $p = .04$), there was a statistically significant interaction between *hemisphere* and *site* (F [1.79, 100.43] = 3.32, $p = .05$, $\eta_p^2 = .06$). Paired-sample *t*-tests suggested N200 amplitudes evoked to background stimuli over anterior sites were larger at midline sites (-3.37 μv, SE = .21) than both left (-2.19 μv, SE = .24; t [56] = 4.47, $p < .001$, *padj* < .001) and right hemispheres (-1.86 μv, SE = .21; t [56] = 6.46, $p < .001$, *padj* < .001). There was no significant difference between N200 amplitude over left and right hemispheres (t [56] = 1.26, $p = .21$, *padj* = .31). Over parietal sites, N200 amplitudes evoked to background stimuli were larger at midline sites (1.63 μv, SE = .36) than over the right hemisphere (4.10 μv, SE = .38; t [56] = 6.39, $p < .001$, *padj* < .001). A similar effect was observed between midline sites and the left hemisphere but this did not survive multiple comparison correction (2.72 μv, SE = .28; t [56] =

2.50, p = .02, *padj* = .27). Furthermore, N200 amplitudes were larger over the left, than right hemisphere (t [56] = 3.34, p = .002, *padj* = .042).

When evoked to stop stimuli, whilst there was no statistically significant main effect of *hemisphere* (F [2, 55] = .96, p = .96, η_p^2 = .01), there was a significant main effect of *site* (F [1, 56] = 4.18, p = .050, η_p^2 = .07) such that on average, N200 amplitude was larger over anterior, than parietal sites (anterior [-.17 µv, SE = .13], parietal [.39 µv, SE = .17]). Paired-sample t-tests suggested this effect was driven by larger N200 amplitudes at anterior, than parietal sites in the right (anterior [-.64 µv, SE = .19], parietal [.91 µv, SE = .29]; t [56] = 4.19, p < .001, *padj* < .001), but not left hemisphere (anterior [-.12 µv, SE = .22], parietal [.36 µv, SE = .22]; t [56] = 1.59, p = .12, *padj* = .31), or at midline sites (anterior [.26 µv, SE = .25], parietal [-.12 µv, SE = .28]; t [56] = 1.19, p = .24, *padj* = .31).

There was a statistically significant interaction between *hemisphere* and *site* (F [2, 55] = 22.64, p < .001, η_p^2 = .45). Paired-sample t-tests suggested N200 amplitudes evoked to stop stimuli at anterior sites were statistically larger over the right hemisphere (-.64 µv, SE = .19) than midline sites (.26 µv, SE = .25; t [56] = 3.92, p < .001, *padj* < .001) but not the left hemispheres (-.12 µv, SE = .22; t [56] = 1.04, p = .31, *padj* = .31). There was no statistically significant difference in N200 amplitude between left hemispheres and midline sites (t [56] = 1.61, p = .11, *padj* = .31). After correcting for multiple comparisons, N200 amplitudes over parietal sites did not differ between the midline (-.12 µv, SE = .28) and either the right (.91 µv, SE = .29; t [56] = 3.07, p < .05, *padj* = .06) or left hemisphere (.36 µv, SE = .22; t [56] = 1.26, p = .21, *padj* = .31). There was no statistically significant difference in N200 amplitude between left and right hemispheres (t [56] = 1.64, p = .11, *padj* = .31).

7.3.3.4.2.2 Hemisphere * Task | Site

At anterior sites, whilst there was no statistically significant main effect of *hemisphere* (F [2, 55] = 2.26, p = .11, η_p^2 = .08), there was a significant main effect of *task* (F [1, 56] = 241.01, p < .001, η_p^2 = .81) such that on average, N200 amplitude was larger in response to background than stop stimuli (background [-2.47 μv, SE = .17], stop [-.17 μv, SE = .13]). Paired-sample *t*-tests suggested this effect was congruent over both left (background [-2.19 μv, SE = .24], stop [-.12 μv, SE = .22]; t [56] = 7.41, p < .001, *padj* < .001), and right hemispheres (background [-1.86 μv, SE = .21], stop [-.64 μv, SE = .19]; t [56] = 5.19, p < .001, *padj* < .001), and at midline sites (background [-3.37 μv, SE = .21], stop [.26 μv, SE = .25]; t [56] = 13.35, p < .001, *padj* < .001).

There was a statistically significant interaction between *hemisphere* and *task* (F [2, 55] = 25.06, p < .001, η_p^2 = .48). Paired-sample *t*-tests suggested N200 amplitudes evoked to background stimuli over anterior sites were statistically larger at midline sites (-3.37 μv, SE = .21) than over both left (-2.19 μv, SE = .24; t [56] = 4.47, p < .001, *padj* < .001) and right hemispheres (-1.86 μv, SE = .21; t [56] = 6.46, p < .001, *padj* < .001). There was no statistically significant difference in N200 amplitude between left and right hemispheres (t [56] = 1.26, p = .21, *padj* = .31). Evoked to stop stimuli, N200 amplitudes were significantly larger over the right hemisphere (-.64 μv, SE = .19) than midline sites (.26 μv, SE = .25; t [56] = 3.92, p < .001, *padj* < .001) but not the left hemispheres (-.121 μv, SE = .22; t [56] = 1.04, p = .31, *padj* = .31). There was no statistically significant difference in N200 amplitude between left and right hemispheres (t [56] = 1.61, p = .11, *padj* = .31).

At parietal sites, there was a statistically significant main effect of *hemisphere* (F [2, 55] = 17.76, p < .001, η_p^2 = .39), such than on average N200 amplitude was larger at midline sites (.76 μv, SE = .27) than over the right hemisphere (2.51 μv, SE = .29; t [56] = 5.82, p < .001, *padj* < .001). Additionally, larger N200 amplitudes were observed at midline sites relative to the left hemisphere (1.54 μv, SE = .22; t [56] = 2.14, p = .04, *padj* = .31) as well as over the left

relative to right hemisphere (*t* [56] = 3.11, *p* < .05, *padj* = .06). However, neither finding survived multiple comparison correction. A second statistically significant main effect of *task* (*F* [1, 56] = 122.40, *p* < .001, η_p^2 = .69), suggested that on average, N200 amplitude was larger in response to stop than background stimuli (stop [.39 µv, SE = .17], background [2.82 µv, SE = .24]).

There was a statistically significant interaction between *hemisphere* and *task* (*F* [2, 55] = 6.30, *p* < .05, η_p^2 = .19). Paired-sample *t*-tests suggested N200 amplitudes evoked to background stimuli over parietal sites were statistically larger at midline sites (1.63 µv, SE = .36) than over the right hemispheres (4.10 µv, SE = .38; *t* [56] = 6.39, *p* < .001, *padj* < .001). A similar difference was observed between midline sites and the left hemisphere but this did not survive multiple comparison correction (2.72 µv, SE = .28; *t* [56] = 2.50, *p* = .02, *padj* = .27). Furthermore, the N200 to background stimuli was larger over the left than right hemisphere (*t* [56] = 3.34, *p* < .05, *padj* = .04). Evoked to stop stimuli, N200 amplitudes were statistically larger over midline sites (-.12 µv, SE = .28) than the left hemisphere (.36 µv, SE = .22; *t* [56] = 1.26, *p* = .21, *padj* = .31). Again, a similar difference was observed between midline sites and the right hemisphere but this did not survive multiple comparison correction (.91 µv, SE = .29; *t* [56] = 3.07, *p* < .05, *padj* = .06). There was no statistically significant difference in N200 amplitude between left and right hemispheres (*t* [56] = 1.64, *p* = .11, *padj* = .31).

7.3.3.4.2.3 Task * Site | Hemisphere

Over the left hemisphere, whilst there was no statistically significant main effect of *task* (*F* [1, 56] = .65, *p* = .42, η_p^2 = .01), there was a significant main effect of *site* (*F* [1, 56] = 68.43, *p* < .001, η_p^2 = .55) such that on average, N200 amplitude was larger over anterior than parietal sites (anterior [-1.16 µv, SE = .19], parietal [1.54 µv, SE = .22]). There was a statistically significant interaction between *task* and *site* (*F* [1, 56] = 135.97, *p* < .001, η_p^2 = .71). Paired-sample *t*-tests suggested N200 amplitudes evoked to background stimuli over the left

hemisphere were statistically larger over anterior (-2.19 μv, SE = .24) than parietal sites (2.72 μv, SE = .28; *t* [56] = 11.19, *p* < .001, *padj* < .001). No statistical difference in N200 amplitude was observed over the left hemisphere to stop stimuli (anterior [-.12 μv, SE = .24], parietal [.36 μv, SE = .22]; *t* [56] = 1.59, *p* = .12, *padj* = .31). Furthermore, whilst N200 amplitude was statistically larger for background, relative to stop stimuli over anterior sites (*t* [56] = 7.41, *p* < .001, *padj* < .001), the inverse was observed over parietal sites (*t* [56] = 10.09, *p* < .001, *padj* < .001).

Over midline sites, there was a statistically significant main effect of *task* (*F* [1, 56] = 16.05, *p* < .001, η_p^2 = .22), such that on average, N200 amplitude was larger when evoked to background than stop stimuli (background [-.87 μv, SE = .21], stop [.07 μv, SE = .21]). There was a second significant main effect of *site* (*F* [1, 56] = 58.07, *p* < .001, η_p^2 = .51) such that on average, N200 amplitude was larger over anterior than parietal sites (anterior [-1.16 μv, SE = .19], parietal [.76 μv, SE = .27]). There was a statistically significant interaction between *task* and *site* (*F* [1, 56] = 171.18, *p* < .001, η_p^2 = .75). Paired-sample *t*-tests suggested N200 amplitudes evoked to background stimuli over midline sites were statistically larger over anterior (-3.37 μv, SE = .21) than parietal sites (1.63 μv, SE = .36; *t* [56] = 12.12, *p* < .001, *padj* < .001). No statistical difference in N200 amplitude was observed over the left hemisphere to stop stimuli (anterior [.26 μv, SE = .25], parietal [-.12 μv, SE = .28]; *t* [56] = 1.19, *p* = .24, *padj* = .31). Furthermore, whilst N200 amplitude was statistically larger for background, relative to stop stimuli over anterior sites (*t* [56] = 13.35, *p* < .001, *padj* < .001), the inverse was observed over parietal sites (*t* [56] = 5.06, *p* < .001, *padj* < .001).

Over the right hemisphere, there was a statistically significant main effect of *task* (*F* [1, 56] = 31.17, *p* < .001, η_p^2 = .36), such that on average, N200 amplitude was larger when evoked to stop than background stimuli (stop [.14 μv, SE = .16], background [1.12 μv, SE = .18]). There was a second significant main effect of *site* (*F* [1, 56] = 111.10, *p* < .001, η_p^2 = .67) such that on average,

N200 amplitude was larger over anterior than parietal sites (anterior [-1.25 μv, SE = .16], parietal [2.51 μv, SE = .29]). There was a statistically significant interaction between *task* and *site* (F [1, 56] = 78.75, $p < .001$, $\eta_p^2 = .58$). Paired-sample *t*-tests suggested N200 amplitudes evoked to background stimuli over the right hemisphere were statistically larger over anterior (-1.86 μv, SE = .21) than parietal sites (4.10 μv, SE = .38; t [56] = 12.15, $p < .001$, *padj* < .001). A similar relationship was observed to stop stimuli (anterior [-.64 μv, SE = .19], parietal [.91 μv, SE = .29]; t [56] = 4.19, $p < .001$, *padj* < .001). Furthermore, whilst N200 amplitude was statistically larger for background, relative to stop stimuli over anterior sites (t [56] = 5.19, $p < .001$, *padj* < .001), the inverse was observed over parietal sites (t [56] = 8.85, $p < .001$, *padj* < .001).

7.3.3.5 P300

Means and standard deviations of P300 amplitudes to fearful and sad facial expressions collapsed over their respective trials are exhibited in Table 7 (page 133), with associated waveforms exhibited in Figure 7 (page 131). Main effects, interactions, and *post hoc* analyses are summarised in Table 8 (page 140).

7.3.3.5.1 Main Effects

Although there was no main effect of *task* (F [1,56] = .40, $p = .53$, $\eta_p^2 = .01$) or *emotion* (F [1, 56] = 2.15, $p = .15$, $\eta_p^2 = .04$), there was a main effect of *site* on the P300 (F [1, 56] = 156.99, $p < .001$, $\eta_p^2 = .74$). On average, larger P300 amplitudes were recorded over parietal, relative to anterior electrode sites (parietal [1.94 μv, SE = .15], anterior [-.89 μv, SE = .11)]. A main effect of *hemisphere* (F [2, 55] = 6.44, $p < .05$, $\eta_p^2 = .19$), suggested midline P300 amplitudes (.91 μv, SE = .14) were larger than over left (.13 μv, SE = .11; t [56] = -3.61, $p < .001$, *padj* < .05) and right hemispheres (.54 μv, SE = .13; t [56] = 2.22, $p = .03$, *padj* = .04). Additionally, P300 amplitude was larger in the right, relative to left hemisphere (t [56] = -2.15, $p = .04$, *padj* = .05).

7.3.3.5.2 Interactions

Emotion did not significantly interact with *hemisphere, site,* and *task* (F [2,55] = .17, p = .84, η_p^2 = .01) or *hemisphere* and *site* (F [2,55] = 2.29, p = .11, η_p^2 = .08). However, e*motion* formed three-way interactions with *site* and *task* (F [1, 56] = 10.49, p < .05, η_p^2 = .16) and *hemisphere* and *task* (F [2,55] = 5.79, p = .01, η_p^2 = .17). These interactions were explored using *post hoc* lower order ANOVAs.

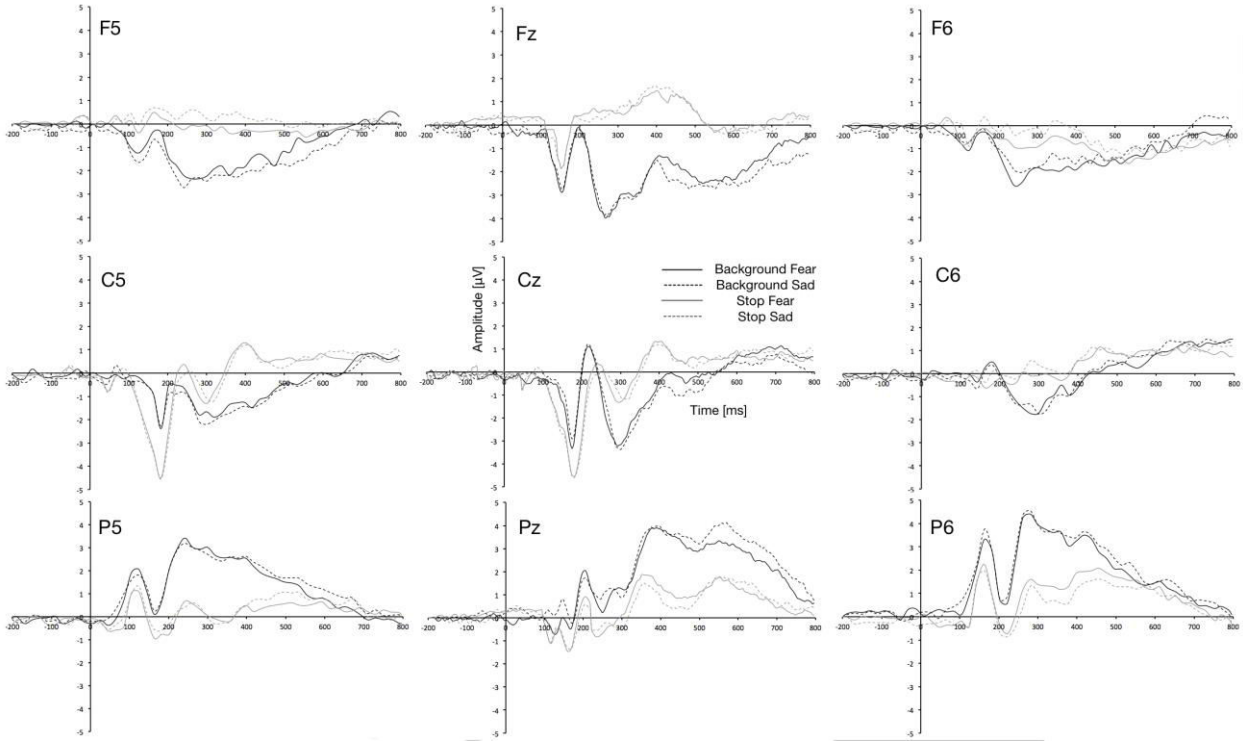

Figure 7. ERP waveforms to fearful and sad stop facial expressions during background and stop task conditions, at bilateral frontal, midline, and parietal electrode sites. ERP waveform is representative of EEG signal referenced to linked mastoid electrodes.

7.3.3.5.2.1 Emotion * Task | Site

Over anterior sites, there was a significant main effect of *emotion* (F [1, 56] = 5.13, p = .03, η_p^2 = .08), such that on average, larger P300 amplitudes were evoked to fearful, relative to sad faces (fearful [-.78 μv, SE = .10], sad [-1.01 μv, SE = .14]). A second main effect of *task* (F [1, 56] = 121.06, p < .001, η_p^2 = .68), suggested that on average, larger P300 amplitudes were evoked to stop, relative to background stimuli (stop [.06 μv, SE = .12], background [-1.84 μv, SE = .16]). Additionally, there was a significant interaction between *emotion* and *task* (F [1, 56] = 11.75, p < .001, η_p^2 = .17), with paired-sample t-tests suggesting this interaction was driven by larger responses to sad, relative to fearful, stop stimuli (sad [.35 μv, SE = .12], fearful [-.23 μv, SE = .17]; t [56] = -3.92, p < .001, $padj$ < .001), with no amplitude difference when evoked to background stimuli (sad [-1.90 μv, SE = .16], fearful [-1.78 μv, SE = .18]; t [56] = .85, p = .40, $padj$ = .93).

Over parietal sites, there was a significant main effect of *emotion* (F [1, 56] = 155.55, p < .001, η_p^2 = .74), such that on average, larger P300 amplitudes were evoked to sad, relative to fearful faces (sad [1.90 μv, SE = .15], fearful [-1.01 μv, SE = .14]). A second main effect of *task* (F [1, 56] = 7.48, p = .01, η_p^2 = .12), suggested that on average, larger P300 amplitudes were evoked to background, relative to stop stimuli (background [.63 μv, SE = .12], stop [.27 μv, SE = .10]). Additionally, there was a significant interaction between *emotion* and *task* (F [1, 56] = 118.43, p < .001, η_p^2 = .68). Although not surviving multiple comparison correction, paired-sample t-tests suggested this interaction might be driven by larger responses to fearful, relative to sad, stop stimuli (fearful [1.11 μv, SE = .19], sad [.77 μv, SE = .16]; t [56] = 2.66, p = .01, $padj$ = .31). No amplitude difference was observed when evoked to background stimuli (fearful [2.87 μv, SE = .21], sad [3.04 μv, SE = .22]; t [56] = 1.13, p = .26, $padj$ = .93).

Table 7.

Descriptions of P300 amplitudes across emotion, hemisphere, site location, and task

Emotion	Stimuli	Site	Hemisphere	M [μv]	SD
Fear	Background	Anterior	Left	-1.66	1.82
			Midline	-1.91	2.01
			Right	-1.77	1.82
		Posterior	Left	2.240	2.35
			Midline	3.47	2.22
			Right	2.89	2.19
	Stop	Anterior	Left	-.68	1.93
			Midline	1.00	1.98
			Right	-1.00	1.72
		Posterior	Left	.27	2.01
			Midline	1.39	2.16
			Right	1.67	1.98
Sad	Background	Anterior	Left	-1.94	1.64
			Midline	-2.32	1.75
			Right	-1.44	1.89
		Posterior	Left	2.25	1.89
			Midline	3.60	2.44
			Right	3.25	2.54
	Stop	Anterior	Left	.19	1.64
			Midline	1.21	1.69
			Right	-.35	1.57
		Posterior	Left	.40	1.88
			Midline	.85	1.95
			Right	1.05	1.89

Note. **Anterior and parietal sites are measured at F and P electrodes, respectively with left, midline, and right hemispheric sites indexed by corresponding electrodes at sites 5, z, and 6, respectively.**

7.3.3.5.2.2 Emotion * Site | Task

When evoked to background stimuli, whilst there was no main effect of *emotion* (F [1, 56] = .09, p = .76, η_p^2 = .01), there was a significant main effect of *site* (F [1, 56] = 226.76, p < .001, η_p^2 = .80), such that on average, larger P300 amplitudes were evoked over parietal, relative to anterior sites (parietal [2.95 μv, SE = .20], anterior [-1.84 μv, SE = .16]). There was no significant interaction between *emotion* and *site*.

When evoked to stop stimuli, whilst there was no main effect of *emotion* (F [1, 56] = 3.15, p = .08, η_p^2 = .05), there was a significant main effect of *site* (F [1, 56] = 12.79, p < .001, η_p^2 = .19), such that on average, larger P300 amplitudes were evoked over parietal, relative to anterior sites (parietal [.94 μv, SE = .16], anterior [.06 μv, SE = .09]). Additionally, there was a significant interaction between *emotion* and *site* (F [1, 56] = 14.34, p < .001, η_p^2 = .20). Paired-sample t-tests suggested that whilst P300 amplitudes were larger when evoked to sad, relative to fearful facial stimuli at anterior sites (sad [.35 μv, SE = .11], fear [-.23 μv, SE = .17]; t [56] = 3.92, p < .001, $padj$ < .001), the inverse was observed at parietal sites (fearful [1.11 μv, SE = .19], sad [.77 μv, SE = .16]; t [56] = 2.66, p = .01, $padj$ = .31). However, this latter result did not survive multiple comparison correction.

7.3.3.5.2.3 Site * Task | Emotion

When evoked to fearful facial stimuli, whilst there was no main effect of *task* (F [1, 56] = .85, p = .36, η_p^2 = .02), there was a significant main effect of *site* (F [1, 56] = 133.48, p < .001, η_p^2 = .70), such that on average, larger P300 amplitudes were evoked over parietal, relative to anterior sites (parietal [1.99 μv, SE = .16], anterior [-1.01 μv, SE = .14]). Additionally, there was a significant interaction between *site* and *task* (F [1, 56] = 70.72, p < .001, η_p^2 = .56). Paired-sample t-tests suggested that whilst P300 amplitudes were larger when evoked to fearful stop, relative to background stimuli at anterior sites (stop [-.23 μv, SE = .17], background [-1.78 μv, SE = .18]; t [56] = 7.74, p < .001, $padj$ < .001), the

inverse was observed at parietal sites (background [2.87 μv, SE = .21], stop [1.11 μv, SE = .19]; t [56] = 7.03, p < .001, $padj$ < .001).

When evoked to sad facial stimuli, whilst there was no main effect of *task* (F [1, 56] = .01, p = .91, η_p^2 = .00), there was a significant main effect of *site* (F [1, 56] = 152.71, p < .001, η_p^2 = .73), such that on average, larger P300 amplitudes were evoked over parietal, relative to anterior sites (parietal [1.90 μv, SE = .15], anterior [-.78 μv, SE = .10]). Additionally, there was a significant interaction between *site* and *task* (F [1, 56] = 135.39, p < .001, η_p^2 = .71). Paired-sample t-tests suggested that whilst P300 amplitudes were larger when evoked to sad stop, relative to background stimuli at anterior sites (stop [-.35 μv, SE = .11], background [-1.90 μv, SE = .16]; t [56] = 11.24, p < .001, $padj$ < .001), the inverse was observed at parietal sites (background [3.04 μv, SE = .22], stop [.77 μv, SE = .16]; t [56] = 9.60, p < .001, $padj$ < .001).

7.3.3.5.2.4 Emotion * Hemisphere | Task

When evoked to background stimuli, whilst there was no main effect of *emotion* (F [1, 56] = .09, p = .76, η_p^2 = .01), there was a significant main effect of *hemisphere* (F [1, 56] = 3.62, p = .03, η_p^2 = .12). Though not surviving multiple comparison correction, paired-sample t-tests suggested that P300 amplitudes were larger in the right hemisphere (.73 μv, SE = .15; t [56] = 2.48, p = .02, $padj$ = .43) and at midline sites (.71 μv, SE = .16; t [56] = 2.26, p = .03, $padj$ = .67), relative to the left hemisphere (.22 μv, SE = .13). There was no statistical difference between the P300 measured at midline sites and those over the right hemisphere (t [56] = -.09, p = .93, $padj$ = .93). There was no significant interaction between *emotion* and *hemisphere*.

When evoked to stop stimuli, whilst there was no main effect of *emotion* (F [1, 56] = 3.15, p = .08, η_p^2 = .05), there was a significant main effect of *hemisphere* (F [1, 56] = 7.44, p < .001, η_p^2 = .21). Paired-sample t-tests suggested that P300 amplitudes were larger at midline sites (1.10 μv, SE = .20) than over the

left hemisphere (.05 μv, SE = .15; *t* [56] = 3.67, *p* < .001, *padj* < .001). Whilst a similar finding was observed between midline sites and the right hemisphere (.34 μv, SE = .15; *t* [56] = 3.18, *p* < .05, *padj* = .07), this finding did not survive multiple comparison correction There was no statistical difference between the P300 measured over the left or right hemispheres (*t* [56] = 1.18, *p* = .24, *padj* = .93).

Additionally, there was also a significant interaction between *emotion* and *hemisphere* (*F* [1, 56] = 8.31, *p* < .001, η_p^2 = .23). Paired-sample *t*-tests suggested that P300 amplitudes were larger when evoked to fearful, relative to sad facial stimuli over the left (fearful [-.21 μv, SE = .16], sad [.30 μv, SE = .17]; *t* [56] = 4.12, *p* < .001, *padj* < .001), but not right hemisphere (fearful [.33 μv, SE = .16], sad [.35 μv, SE = .17]; *t* [56] = .11, *p* = .91, *padj* = .93) or midlines sites (fearful [1.19 μv, SE = .22], sad [1.03 μv, SE = .20]; *t* [56] = 1.39, *p* = .17, *padj* = .93). Moreover, when evoked to fearful faces, P300 amplitudes were larger over midline sites (1.19 μv, SE = .22), relative to the left hemisphere (-.21 μv, SE = .16; *t* [56] = 4.55, *p* < .001, *padj* < .001). Although P300 amplitudes were also found to be larger over midline, relative to the right hemispheres (.33 μv, SE = .16; *t* [56] = 3.32, *p* < .05, *padj* = .07) and over the right, relative to left hemisphere (*t* [56] = 2.17, *p* = .03, *padj* = .75), these findings did not survive multiple comparison correction. When evoked to sad faces, whilst P300 amplitudes were larger over midline sites (1.03 μv, SE = .20) than both left (.30 μv, SE = .17; *t* [56] = 2.45, *p* = .02, *padj* = .43) and right hemispheres (.35 μv, SE = .17; *t* [56] = 2.55, *p* = .01, *padj* = .36), these findings did not survive multiple comparison correction. There was no statistical difference between the P300 measured over the left or right hemispheres (*t* [56] = .19, *p* = .85, *padj* = .93).

7.3.3.5.2.5 Emotion * Task | Hemisphere

In the left hemisphere, there was no statistically significant main effect of either *emotion* (*F* [1, 56] = 2.89, *p* = .10, η_p^2 = .05) or *task* (*F* [1, 56] = 1.16, *p* = .29, η_p^2 = .02). However, there was a significant interaction between *emotion* and

task (*F* [1, 56] = 8.90, *p* < .05, η_p^2 = .14) such that whilst P300 amplitudes were larger when evoked to sad, relative to fearful stop stimuli (sad [.30 μv, SE = .17], fearful [-.21 μv, SE = .16]; *t* [56] = 4.12, *p* < .001, *padj* < .001), there was no amplitude difference for background stimuli (sad [.16 μv, SE = .15], fearful [.29 μv, SE = .17]; *t* [56] = .75, *p* = .46, *padj* = .93).

At midline sites, there were no main effects of either *emotion* (*F* [1, 56] = 2.25, *p* = .14, η_p^2 = .04) or *task* (*F* [1, 56] = 3.06, *p* = .09, η_p^2 = .05). Additionally, there was no significant interaction between *emotion* and *task* (*F* [1, 56] = .01, *p* = .92, η_p^2 = .00).

In the right hemisphere, whilst there was no statistically significant main effect of *emotion* (*F* [1, 56] = 2.22, *p* = .14, η_p^2 = .04), there was a statistically significant main effect of *task* (*F* [1, 56] = 6.58, *p* = .01, η_p^2 = .10), such that on average the P300 response to background stimuli was larger than that to stop stimuli (background [.73 μv, SE = .15], stop [.34 μv, SE = .15]). There was no statistically significant interaction between *emotion* and *task* (*F* [1, 56] = 3.02, *p* = .09, η_p^2 = .05).

7.3.3.5.2.6 Hemisphere * Task | Emotion

When evoked to fearful facial stimuli, there was a significant main effect of *hemisphere* (*F* [2, 55] = 8.20, *p* < .001, η_p^2 = .23) but not *task* (*F* [1, 56] = .85, *p* = .36, η_p^2 = .02), Paired-sample *t*-tests suggested that P300 amplitudes were larger at midline sites (.99 μv, SE = .16) than in both the left (.04 μv, SE = .13; *t* [56] = 3.94, *p* < .001, *padj* < .001) and right hemispheres (.45 μv, SE = .14; *t* [56] = 2.94, *p* < .001, *padj* < .001). There was no statistical difference between the P300 measured over left and right hemispheres (*t* [56] = 1.88, *p* = .06, *padj* = .93).

Additionally, there was a significant interaction between *hemisphere* and *task* (*F* [2, 55] = 4.18, *p* = .02, η_p^2 = .13). Although not surviving multiple comparison

correction, paired-sample *t*-tests suggested that P300 amplitudes evoked to fearful faces were significantly larger when used as background, relative to stop stimuli in the left hemisphere (background [.29 μv, SE = .17], stop [-.21 μv, SE = .16]; *t* [56] = 2.56, *p* = .01, *padj* = .36). There were no statistically significant differences in the right hemisphere (background [.56 μv, SE = .17], stop [.33 μv, SE = .16]; *t* [56] = 1.36, *p* = .18, *padj* = .93) or at midline sites (background [.78 μv, SE = .19], stop [1.19 μv, SE = .22]; *t* [56] = -1.59, *p* = .12, *padj* = .93). Moreover, when used as background stimuli, there was no statistical difference in P300 amplitudes evoked to fearful faces over midline sites (.78 μv, SE = .19), relative to the left (.29 μv, SE = .17; *t* [56] = 1.89, *p* = .06, *padj* = .93), or right hemisphere (.56 μv, SE = .17; *t* [56] = 1.03, *p* = .31, *padj* = .93). There was also no statistical difference between the left and right hemisphere (*t* [56] = .98, *p* = .33, *padj* = .93). When used as stop stimuli, P300 responses to fearful faces were larger at midline sites (1.19 μv, SE = .22) than over the left hemisphere (-.21 μv, SE = .17; *t* [56] = 4.55, *p* < .001, *padj* < .001). Although P300 differences were observed between midline sites and the right hemisphere (.33 μv, SE = .17; *t* [56] = 3.32, *p* < .05, *padj* = .07) and over the right, relative to left hemisphere (*t* [56] = 2.17, *p* = .03, *padj* = .75), these findings did not survive multiple comparison correction.

When evoked to sad facial stimuli there was a significant main effect of *hemisphere* (*F* [2, 55] = 3.86, *p* = .03, η_p^2 = .12), but not *task* (*F* [1, 56] = .01, *p* = .91, η_p^2 = .00), Although not surviving multiple comparison correction, paired-sample *t*-tests suggested that P300 amplitudes were significantly larger at midline sites (.84 μv, SE = .15) than in the left (.23 μv, SE = .12; *t* [56] = 2.77, *p* = .01, *padj* = .26). No P300 amplitude differences were observed between the right hemisphere (.63 μv, SE = .14) and either midline sites (*t* [56] = 1.08, *p* = .28, *padj* = .93) or the left hemisphere (*t* [56] = 1.97, *p* = .05, *padj* = .93).

Additionally, there was a significant interaction between *hemisphere* and *task* (*F* [2, 55] = 4.42, *p* = .02, η_p^2 = .14). Paired-sample *t*-tests suggested that P300 amplitudes evoked to sad faces were significantly larger when used as

background, relative to stop stimuli in the right (background [.90 μv, SE = .17], stop [.35 μv, SE = .17]; *t* [56] = 2.89, *p* = .01, *padj* = .20), but not left hemisphere (background [.16 μv, SE = .17], stop [.30 μv, SE = .16]; *t* [56] = -.69, *p* = .49, *padj* = .93) or at midline sites (background [.64 μv, SE = .19], stop [1.03 μv, SE = .20]; *t* [56] = -1.56, *p* = .12, *padj* = .93). Moreover, when used as background stimuli, although P300 amplitude was significantly larger in the right (.90 μv, SE = .17), than left hemisphere (.30 μv, SE = .17; *t* [56] = 3.26, *p* < .05, *padj* = .07), this did not survive multiple comparison correction. No P300 amplitude differences were observed between midline sites (.64 μv, SE = .19) and either the right (*t* [56] = 1.07, *p* = .29, *padj* = .93) or left hemisphere (*t* [56] = 1.94, *p* = .06, *padj* = .93). When used as stop stimuli, P300 responses to sad faces were larger at midline sites (1.03 μv, SE = .20) than both left (.30 μv, SE = .17; *t* [56] = 2.45, *p* = .02, *padj* = .43), and right hemispheres (.35 μv, SE = .17; *t* [56] = 2.55, *p* = .01, *padj* = .36). There was no significant difference in P300 amplitude between left and right hemispheres (*t* [56] = .19, *p* = .85, *padj* = .93).

Table 8.

Statistically significant ANOVA summary table for main effects and interactions across ERPs

ERP	Effect	*F*	*df*	η_p^2
N170	Emotion	6.37**	2.23, 124.64	.10
P200	Hemisphere	12.38***	1, 56	.18
	Emotion	3.79*	2.36, 132.09	.06
N200	Site	113.06***	1, 56	.67
	Hemisphere	13.18***	2, 55	.32
	Emotion*Site	7.51**	1, 56	.12
	Hemisphere*Site*Task	11.23***	1.78, 99.73	.17
	Hemisphere {Background}	28.45***	2, 55	.51
	Site {Background}	209.58***	1, 56	.79
	Hemisphere*Site {Background}	3.32*	1.79, 100.43	.06
	Site {Stop}	4.18*	1, 56	.07
	Hemisphere*Site {Stop}	22.64***	2, 55	.45
	Task {Anterior}	241.01***	1, 56	.81
	Hemisphere*Task {Anterior}	25.06***	2, 55	.48
	Hemisphere {Parietal}	17.76***	2, 55	.39
	Task {Parietal}	122.40***	1, 56	.69
	Hemisphere*Task {Parietal}	6.30**	2, 55	.19
	Site {Left}	68.43***	1, 56	.55
	Task*Site {Left}	135.97***	1, 56	.71
	Task {Midline}	16.05***	1, 56	.22
	Site {Midline}	58.07***	1, 56	.51
	Task*Site {Midline}	171.18***	1, 56	.75
	Task {Right}	31.17***	1, 56	.36
	Site {Right}	111.10***	1, 56	.67
	Task*Site {Right}	78.75***	1, 56	.58
P300	Site	156.99***	1, 56	.74
	Emotion*Site*Task	10.49**	1, 56	.16
	Emotion*Hemisphere*Task	5.79**	2, 55	.17
	Emotion {Anterior}	5.13*	1, 56	.08
	Task {Anterior}	121.06***	1, 56	.68
	Emotion*Task {Anterior}	11.75***	1, 56	.17
	Emotion {Parietal}	155.55***	1, 56	.74
	Task {Parietal}	7.48*	1, 56	.12
	Emotion*Task {Parietal}	118.43***	1, 56	.68
	Site {Background}	226.76***	1, 56	.80
	Site {Stop}	12.79***	1, 56	.19
	Emotion*Site {Stop}	14.34***	1, 56	.20
	Site {Fearful}	133.48***	1, 56	.70
	Site*Task {Fearful}	70.72***	1, 56	.56
	Site {Sad}	152.71***	1, 56	.73
	Site*Task {Sad}	135.39***	1, 56	.71
	Hemisphere {Background}	3.62*	1, 56	.03
	Hemisphere {Stop}	7.44***	1, 56	.21
	Emotion*Hemisphere {Stop}	8.31***	1, 56	.23
	Emotion*Task {Left}	8.90**	1, 56	.14
	Task {Right}	6.58*	1, 56	.11
	Hemisphere {Fearful}	8.20***	1, 56	.23
	Hemisphere*Task {Fear}	4.19*	2, 55	.13
	Hemisphere {Sad}	3.86*	2, 55	.12
	Hemisphere*Task {Sad}	4.42*	2, 55	.14

***Note.* * $p < .05$, ** $p < .005$, *** $p < .001$. All statistics are reported after adjusting degrees of freedom using Greenhouse-Geisser estimates of sphericity where necessary**

7.4 Discussion

This investigation is the first to characterise the electrophysiological responses to face processing and distress-induced motor extinction as an index of the VIM, using a novel FAST. Key findings of this investigation are:

1. N170 augmentation was observed in response to angry, but not fearful, sad, and neutral facial expressions.
2. P200 augmentation was observed in response to neutral, but not angry, fearful, and sad facial expressions. P200 responses were larger in the left, relative to right, hemisphere uniformly across emotion.
3. N200 responses were maximal over anterior midline sites when evoked to background stimuli and over right hemispheric anterior sites when evoked to stop stimuli. There was no effect of emotion on either background or stop stimuli.
4. P300 responses to both fearful and sad stop stimuli were larger over anterior, relative to parietal sites, with the inverse observed for background stimuli. Fearful, relative to sad facial expressions evoked larger P300 amplitudes overall. However, whilst P300 amplitudes were larger in response to fearful stimuli over anterior sites, P300 amplitudes were larger in response to sad stimuli over parietal sites.

7.4.1 N170

The N170 was hypothesised to show right hemispheric dominance; augmenting in response to distress (i.e. fear, sad) and in turn to angry, relative to neutral, facial expressions. First, although N170 dominance over the right hemisphere has been reported elsewhere (Bentin et al., 1996; Rossion et al., 2003, Stockdale et al., 2015, *but see Hinojosa et al., 2015 for review*), a main effect of hemisphere was not found in this investigation. It is possible that this null result might be explained through the use of averaged electrode referencing, which was used in order to mitigate attenuation of the temporo-parietal N170/P200 signal (*see Kappenman & Luck, 2012 for review*). Previously, whilst a nasal reference was used in Bentin et al., Rossion et al. referenced EEG signal to the

left mastoid, which might have accounted for right hemispheric dominance as a function of left hemispheric signal attenuation. However as the meta-analysis reported in Hinojosa et al. (2015) highlights both the common use of averaged referencing when measuring the N170 and a predominant right hemispheric N170 distribution, this current finding is atypical.

Second, whilst N170 augmentation was expected in response to both threat (angry) and distress (fear; sad), relative to neutral facial expressions, *post hoc* analysis revealed the main effect of emotion was driven by sensitivity to threat. No amplitude variation was observed between N170 responses to fearful, sad, or neutral expressions. This finding is in line with the threat-superiority effect (Gilbert, 2005; Gilbert et al., 1995), which proposes a self-protection system that directs attention to potential danger. Furthermore, it is supported by investigations suggesting a N170 bias towards negative stimuli (Chai et al., 2012), including angry facial expressions (Eger et al., 2003; Ibánez et al., 2012; Krombholz et al., 2007; Taylor et al., 2004). However, anger-specific augmentation of the N170 contrasts investigations reporting a negativity bias associated with N170 augmentation to fearful facial expressions (Batty & Taylor, 2003; Blau et al., 2007; Taylor et al., 2004; Ramos-Loyo et al., 2009; Shannon et al., 2013). In particular, Taylor et al. (2004) reported fear-evoked N170 augmentation, relative to angry facial expressions.

Conflicting findings might be explained by methodological variation. As discussed in chapter three, section 3.2.1 (page 39), some, but not all (Hinojosa et al., 2015), believe the N170 to reflect early structural face encoding, preceding emotion categorisation (Boucsein et al., 2001; Chai et al., 2012; Dawson et al., 2004; Eimer, 2000; Holmes, 2002). As the FAST requires a motor response to angry facial expressions, anger-specific N170 augmentation reported here might be a function of participations being primed to respond to angry facial stimuli, which unlike fearful, sad, and neutral expressions, were target stimuli. Previously, N170 augmentation has been evoked to actual (Ibánez et al., 2012) and schematic (Eger et al., 2003; Krombholz, 2007) angry

faces when primed with negatively or positively valenced words, but not during un-primed trials (Boucsein et al., 2001; Holmes, 2002) and so supports previous claims that the face-evoked N170 functions through voluntary attention (Holmes et al., 2003). Whilst the FAST does not use affective words to prime responses, task instructions ask participants to attend to the affective elements of stimulus one in order to respond to angry facial expressions.

Measuring the N170 from stimulus two instead of stimulus one might elicit N170 augmentation to fearful and sad facial expressions as participants are directed to respond to these emotions, specifically, during this part of the trial. However, this hypothesis cannot reliably be explored using the current paradigm because of [1] possible convolution of early ERP components and [2] stop stimuli only consisting of fearful and sad expressions. Future investigation should seek to clarify this effect by investigating the role of emotion-specific task instructions on ERPs. Using a FAST, this could be achieved by varying the facial expression used as go and stop stimuli. Although such modifications would alter the paradigm in terms of ecological validity, they are important in order to better understand the role of emotion on the N170 within a FAST.

7.4.2 P200

In contrast to findings relating to the N170, the P200 evoked in this investigation demonstrated right hemispheric dominance. This finding supports classic characterisations of the P200 demonstrating larger P200 amplitudes over right, relative to left, occipito-temporal sites (Farah, 1990; Watanabe et al., 1999).

As an index of second-order processing of facial features such as emotion (Itier & Taylor, 2004; Latinud & Taylor 2006), the P200 was hypothesised to augment in response to facial distress. In contrast to expectations, the current investigation found P200 amplitude was, on average, larger in response to neutral, not affective facial expressions. Additionally, there was no statistically significant amplitude difference in P200 responses to angry, fearful, or sad faces. Previously, although neutral-specific P200 augmentation has been

reported in one investigation (Tortosa et al., 2013), P200 augmentation to affective facial expressions is more commonly observed (Ashley et al., 2004; Chai et al., 2012; Eimer et al., 2003; Schulz et al., 2012) including augmentation to threat and distress, specifically (Gonzalez-Roldan et al., 2011; Shannon et al., 2013).

In this investigation, P200 augmentation to neutral facial stimuli might be an artefact of participants being primed to respond to facial affect. As participants were primed to expect facial affect, P200 augmentation to neutral facial expressions might reflect the recruitment of additional neuronal resources when determining the threat potential of more ambiguous neutral expressions. This theory might help explain the P200 augmentation to neutral expressions reported in Tortosa et al. (2013), where participants were asked to assign reward based on facial expressions. Such conflict might mask subtle effects of emotion and so warrants further investigation in respect to the FAST.

7.4.3 N200

The N200, an electrophysiological index of stimulus classification and conflict monitoring (Fostein & van Petten, 2008; van Veen & Carter, 2002), was hypothesised to present an anterior scalp distribution. As expected, irrespective of facial expression, the N200 was maximal over anterior electrode sites in response to both background and stop stimuli, with stop stimuli showing a slight bias over the right hemisphere. Previously, N200 anterior scalp distributions have been observed in response to background, nogo, and stop stimuli (Bruin & Wijers, 2002; Patel & Azzan, 2005), thought to reflect activation of frontal networks comprising the dlPFC, superior- and medial-frontal gyri, lateral OFC, and ACC (Amodio et al., 2008; Anderer et al., 2004; Enriquez-Geppert et al., 2010; van Veen & Carter, 2002).

Furthermore, findings of this investigation suggested that N200 responses to fearful and sad facial expressions were statistically similar, irrespective of stimulus type (i.e. background, stop). Although N200 augmentation to fearful

facial expressions has been reported in Dennis & Chen (2007), this finding was relative to happy and neutral, but not sad facial expressions, and so cannot be used as a direct comparison to observations reported here. However, as Dennis & Chen interpret their findings as reflecting a distress-driven response, with fear and sadness thought to share similar neural underpinnings (Adolphs & Tranel, 2004, *see chapter two, section 2.5 [page 29]*), it is possible that comparable N200 augmentation might have be evidenced to sad facial expressions.

7.4.4 P300

The P300 was hypothesised to present maximally over parietal and anterior electrode sites when evoked to background and stop stimuli, respectively. Although P300 responses were larger over parietal electrode sites on average, higher-order interaction effects supported this stimulus-/site-specific hypothesis. This pattern of activation supports an extensive literature documenting both the parietal distribution of the P300 response to background stimuli (Baumeister et al., 2014; Luck, 2005, *but see Polich, 2007 for review*) and the anterior distribution of the P300 response to no-go/stop stimuli (Baumeister et al., 2014; Bokura et al., 2001; Dimosaka et al., 2006; Fallgatter et al., 2005; Hillyard & Kutas, 1983; Kok et al., 2004).

Though maximal at anterior midline sites, subsequent analysis suggested a right hemispheric bias of the stop-P300 response, relative to left and midline sites. Right hemispheric dominance might reflect activation of the right IFG, thought to underpin the generation of the stop-P300 (Bae et al., 2011; Enriquez-Geppert et al., 2010; Kim et al., 2011; Mattia et al., 2012, *but see chapter three, section 3.4.2 [page 52]*). This investigation offers a novel finding such that it is the first to characterise the stop-P300 to facial stimuli, in particular facial-distress. It appears that anterior stop-P300 distributions are uniform across stimulus modality.

The second hypothesis relating to the P300, that of larger P300 responses to fearful, relative to sad stop stimuli, was not supported. In this characterisation of

the electrophysiological indices of the FAST, there were no amplitude differences between the P300 evoked to fearful and sad faces during stop conditions. On a neurobiological level, fearful and sad facial expressions are thought to share similar processing networks (Adolphs & Tranel, 2004), and so moderate the P300 as a function of their biological significance (Halgren et al., 1994; Rossignol et al., 2005). However, when P300 responses to background and stop stimuli were combined, P300 amplitudes were, on average, larger in response to fearful, relative to sad, facial expressions. In relation to the VIM, though not specific to stop stimuli, this finding might reflect the predominant influence of fear thought to trigger distress-induced motor extinction (Blair, 2000; Blair et al., 2004; Dadds et al., 2008; Fairchild et al., 2009; Iria & Barbosa, 2009). As this is the first investigation to use facial distress as a stop stimulus, future replication is required in order to test this effect.

7.4.5 Limitations

Though the FAST provides a useful tool for investigated the electrophysiological indices of face processing and distress-induced motor extinction, is important to delineate its constraints. First, fearful and sad facial expressions were selected as stopping stimuli based on their theoretical relevance to avoidance behaviour (Berkowitz, 1993; Blair, 1995). However, it remains unknown how ERP responses reported here compare to non-distress facial expressions, such as neutral, happy, and angry expressions. Evoking stop ERPs to non-distress emotions is a logical and necessary advancement in validating the FAST. If the FAST is to be regarded as a valid index of the VIM, stop-P300 responses to non-distress facial expressions should be attenuated; indicative of non-distressed faces being less biologically significant.

Similarly, although the FAST is useful for delineating electrophysiological responses to sad and fearful facial expressions, currently, it has not been used to index electrophysiological responses to facial distress in the form of pain. Akin to fear and sadness, pain processing recruits a network that includes the amygdala, ACC, and insula (Akitsuki & Decety, 2009; Decety et al., 2008, 2009;

Sewards & Sewards, 2003). Dysfunction of this network has been associated with poor empathic responses (Raine, 2013), which, as described throughout this thesis might detriment activation of the VIM (Blair, 1995). Currently, knowledge of ERP responses to facial pain is fragmentary, with only a single study, sampling fibromyalgia patients, observing an absence of N170 amplitude variation between painful, neutral and angry facial expressions (González-Roldán, Muñoz, Cifre, Sitges, & Montoya, 2013). Painful faces would be expected to evoke similar stop-ERPs to sad or fearful facial expressions.

Second, in its current incarnation, the FAST can only assess the role of *facial* distress on motor extinction. In addition to facial expressions, distress can be imparted through auditory signals such as crying, whimpers, and screaming, and through body posture such as presentation of open palms and cowering (Blair et al., 2001; Dadds et al., 2006; Stevens et al., 2001). In accordance with body posture, other physical characteristics such as height, weight, muscle mass, and body size would likely moderate stopping, and associated electrophysiological responses. Inclusion of stimuli encapsulating physical characteristics might further moderate motor extinction. Although the importance of facial expressions has been delineated in chapter one, section 1.3.4 (page 12), distress presented through other modalities might have an additive effect on distress-induced motor extinction, which in turn might be quantified on an electrophysiological level. Delineating VIM-relevant electrophysiological responses, as a function of stimulus modality, would add a further dimension to the FAST; increasing its utility.

Finally, although the FAST provides an index of distress-induced motor extinction on an electrophysiological level, source localisation of EEG signal and simultaneous fMRI would benefit understanding of the neurobiological underpinnings of this paradigm. FMRI allows for the localisation of haemodynamic changes within the brain and so is useful for attributing task-related regional changes. However, as haemodynamic change can take several

seconds to return to baseline, stopping paradigms such as the FAST might not readily transfer into an fMRI environment.

7.4.6 Summary

In summary, the FAST provides a useful tool for investigating the VIM on an electrophysiological level, and in the most part, evokes comparable ERP responses to those reported elsewhere. However, unlike other paradigms, the FAST has utility for evoking face- and motor extinction-related ERPs within a single paradigm, and for the first time, characterises electrophysiological indices of distress-induced motor extinction. Furthermore, this investigation provides grounding for future manipulations of the FAST in order to better [1] index the VIM in terms of ecological validity and [2] understand the neurobiological underpinnings of distress-induced motor extinction. As the VIM is thought to be dysfunctional in individuals characterised by aggressive and CU traits (Blair, 1995, 2001), it is important to understand how ERPs elicited using the FAST might associate with such traits.

Chapter Eight: Correlations between physical aggression/callousness-unemotional traits and face-evoked ERPs

8.1 Introduction

Observed in both adolescent (Dadds et al., 2008; Sylvers et al., 2011) and adult (Blair, 2000; Blair et al., 2004; Fairchild et al., 2009) samples, aggressive and CU traits are thought to predict poor categorisation of, and atypical responses to, facial distress (Marsh & Blair, 2008; Wilson et al., 2011, *see chapter one, section 1.3.4 [page 11]*). In such samples where distress recognition is reported preserved, results likely reflect variation in methodology. For example, Glass & Newman (2006) presented offenders with multiple response options in a face/affect matching task, and Eisenbarth et al. (2008) allowed females with psychopathy an unlimited amount of time when categorising facial expressions. Such paradigms likely facilitated stimulus processing and contributed to increased face recognition accuracy. Compounding this point, a second task reported in Eisenbarth et al. reported sadness processing deficits when stimuli were presented for a much shorter duration of 33 ms.

A recent case has been made for disentangling aggressive/CU traits when investigating facial affect recognition deficits (Dawel et al., 2012). Although sampling from investigations with small sample sizes using only a few trials per condition, the meta-analysis reported in Dawel et al. identified prevalent face processing deficits across facial expressions. This analysis was constrained to individuals scoring high on the affective (i.e. CU traits), but not antisocial (i.e. aggressive traits), factor of psychopathy and contrasts earlier meta-analyses documenting distress-specific processing deficits in samples characterised by both affective and antisocial traits (Marsh & Blair, 2008; Wilson et al., 2011). As suggested in chapter six of this thesis (from page 90), relationships between

physical aggression and distinct CU traits may be nonlinear in adults, and so each trait might differentially moderate face processing.

Discussed in chapter one, section 1.3.1 (page 7), aggressive traits detriment facial affect recognition by enhancing threat-specific processing and contributing to the misattribution of threat in ambiguous facial expressions. Previously, threat-biased misattribution has been observed in individuals with psychopathy (Eisenbarth et al., 2008), but as this investigation did not tease apart affective and antisocial traits, it is possible that this threat-bias was driven by the antisocial factor of psychopathy. An amplified response to threat combined with deficient processing of distress might result in a failure to trigger the VIM (*see chapter one, section 1.3.4 [page 12]*), and in turn, facilitate prolonged, uninhibited acts of aggression (Blair, 1995; Marsh & Ambady, 2007).

Due to its high temporal resolution, EEG provides a useful tool for observing the neuronal response to faces (Kappenman & Luck, 2012, *but see chapter three, section 3.2 [page 39]*), and so might aid investigation into how aggressive/CU traits moderate distinct temporal stages of face processing. Faces elicit a well-documented electrophysiological response comprising the N170, an index of early structural face encoding, and the P200, an index of second-order feature integration (Eimer & Holmes, 2002; Rossion et al., 2003; Tortosa et al., 2013).

N170 and P200 responses are associated with individual differences in emotional skill (Meaux et al., 2014). Specifically, the N170 has been shown to correlate with a cluster of traits associated with callous and uncaring behaviours. N170 attenuation has been observed in schizophrenic samples reporting blunted affect (Ibáñez et al., 2012), as well as healthy cohorts presenting low emotional sensitivity/expressivity (Meaux et al., 2014) and high fearless dominance (Almeida et al., 2014). Fearless dominance is thought to relate to uncaring traits (Kimonis et al., 2013). Conversely, N170 *augmentation* has been related to cold-heartedness (Almeida et al., 2014), a factor associated

with callous and unemotional traits (Patrick, 2010), and so suggests qualitatively different CU traits might differentially modulate the N170.

Elsewhere, a lack of N170 amplitude variation has been reported in small samples of males (Flor et al., 2002) and females (Eisenbarth et al., 2013) with psychopathy. However, Flor et al. measured a slightly earlier negative component, at non-temporo-parietal sites, during an odour/facial conditioning task, and Eisenbarth et al. measured the N170 using an average of frontal, central, and parietal electrode sites during a mood induction task. In such investigations, inconsistent findings might relate to the use of paradigms that did not solely assess face processing ability and/or the non-optimal measurement of the N170 (*see chapter three, section 3.2.1 [page 39]*)

In contrast, there is only a small literature relating the P200 to emotional skills such as the decoding, understanding, and knowledge-based reasoning of emotion. Not only do such skills have importance for the perception of emotion, but they also facilitate the management of emotion-induced thoughts and behaviours (Meaux et al., 2014). Whereas males with psychopathy have evidenced P200 *augmentation* to neutral facial expressions (Flor et al., 2002), individuals drawn from the community with poor emotional control (i.e. the ability to regulate outward displays of experienced emotion) have shown P200 *attenuation* (Meaux et al., 2014). Whilst not a direct assay of physical aggression, poor control over negative affect is key to the manifestation of physical aggression (*see chapter two, section 2.4 [page 26]*). The apparent differentiation between the effects of CU traits and physical aggression on the P200 warrants further investigation.

Taken together, although the N170 and P200 have been moderated by traits associated with CU behaviours and poor emotional control, a risk factor for physical aggression, associations between N170 and P200 responses with distinct CU and aggressive traits remain unclear - as does the effect of emotion. This investigation delineates these associations in a community sample. CU

traits, particularly uncaring traits, were hypothesised to associate with N170 attenuation (poor emotional sensitivity) and physical aggression was expected to associate with P200 attenuation (poor emotional control).

8.2 Methodology

Core methodology has been documented in chapter seven, section 7.3.2 (page 117). For this investigation, only N170 and P200 responses to stimulus one (comprising neutral, fearful, sad, and angry faces) were reported.

8.2.1 Measures

Participants completed the AQ and ICU (*see chapter five, sections 5.3.1 [page 83] and 5.3.2 [page 84], respectively).* For this investigation, only the physical aggression subscale of the AQ was used.

8.2.2 Statistical Analysis

Psychometric responses and mean ERP amplitudes for each subscale and trial type were calculated and entered into an R Statistics v.2.15 dataset (R Core Team, 2012). Average temporo-parietal N170 and P200 responses for each emotion were created using left (P7, PO7, O1, PO3) and right (P8, PO8, O2, PO4) electrodes. Pearson's partial-correlations were calculated for *trait* (i.e. callousness, uncaring, unemotional, physical aggression)*, ERP* (i.e. N170, P200), *emotion* (i.e. anger, fear, sadness neutral), and *hemisphere* (i.e. left, right), and controlled for age and sex (demeaned). As temporal overlap of ERP components has been reported up to 200 ms post stimulus onset (*see Kappenman & Luck, 2012*), partial correlations involving the P200 also controlled for preceding N170 amplitude. Furthermore, bilateral N170 and P200 responses were correlated with task behavioural results (i.e. stopping reaction time, stopping success). All comparisons were corrected for multiple comparisons (*see Benjamini & Hochberg, 1995*).

8.3 Results

Means and standard deviations for psychometric measures were as follows: *physical aggression* (M = 52.98 [*t*-transformed], SD = 8.61), *callousness* (M = 5.98, SD = 2.97), *uncaring* (M = 8.00, SD = 3.33), and *unemotional* (M = 8.16, SD = 3.06). Mean amplitudes for N170 and P200 responses are reported in Table 4 (page 119) and Table 5 (page 121), respectively.

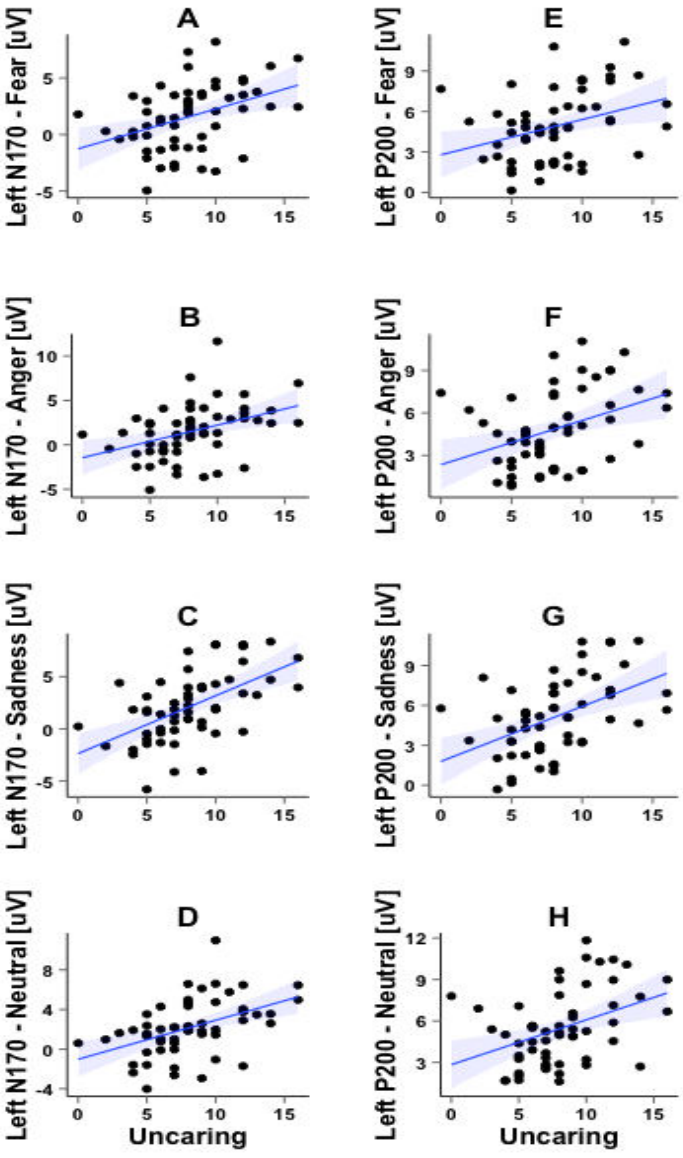

Figure 8. Scatter plots of correlations between ICU-measured uncaring traits and ERPs evoked over left temporo-parietal sites (i.e. PO7, P7, PO4, and O1). Subplots A to D indicate the N170 evoked to fearful (*r* = .41), angry (*r* = .41), sad (*r* = .56), and neutral (*r* = .47) faces. Subplots E to H indicate the P200 evoked to fearful (*r* = .37), angry (*r* = .41), sad (*r* = .51), and neutral (*r* = .44) faces. Shaded areas denote 95% confidence interval areas along linear associations.

8.3.1 Behavioural

Neither N170 nor P200 responses correlated with stopping reaction times or percentage of correct stopping trials for any emotion.

8.3.2 Correlations

8.3.2.1 N170

In the left, but not right hemisphere, the temporo-parietal N170 response to sad (r = -.56, p < .001, *padj* < .001) and neutral (r = -.47, p < .001, *padj* < .001) facial expressions exhibited statistically significant, negative, and moderately strong correlations with *uncaring* traits after controlling for age and sex. Similar, albeit weaker correlations were observed between *uncaring* traits and N170 responses to fearful (r = -.41, p < .05, *padj* = .07) and angry facial expressions (r = -.41, p < .05, *padj* = .06) at trend-level significance after correcting for multiple comparisons. These results suggest *uncaring* traits are inversely associated with the ability to identify facial structure regardless of the expression depicted. Correlations between *uncaring* traits and left N170 responses are depicted in subplots A to D of Figure 8.

8.3.2.2 P200

In the left, but not right hemisphere, the temporo-parietal P200 evoked to sad (r = .51, p < .001, *padj* = .01) and neutral (r = .44, p < .001, *padj* = .03) facial expressions exhibited statistically significant, positive, and moderately strong correlations with *uncaring* traits after controlling for age, sex, and stimulus-matched N170 amplitude. Although *uncaring* traits correlated in a similar, albeit weaker fashion to the left P200 evoked to angry (r = .41, p < .05, *padj* = .06) and fearful facial expressions (r = .37, p = .01, *padj* = .17), these did not survive multiple comparisons correction. Results suggest *uncaring* traits are positively associated with the neuronal response to task salient features depicted in sad and neutral facial expressions, as indexed by the P200. Correlations between

uncaring traits and left hemispheric P200 responses are depicted in subplots E to H of Figure 8.

No statistically significant correlations between either ERP evoked at bilateral temporo-parietal sites to sad, fearful, angry, or neutral facial expressions, and *physical aggression*, *callousness*, or *unemotional* traits survived multiple comparisons correction.

8.4 Discussion

This investigation sought to assess the distinct relationships between physical aggression and CU traits on early electrophysiological indices of face processing. After controlling for age, sex - and in the case of the P200 - N170 amplitude, physical aggression, callousness, and unemotional traits were not correlated with either N170 or P200 response to faces. In contrast, uncaring traits were negatively correlated with N170 responses uniformly across facial expressions, and positively correlated with P200 responses to neutral and sad facial expressions, specifically. Significant correlations were limited to the left hemisphere.

8.4.1 N170

CU traits were hypothesised to correlate with N170 attenuation. In this investigation, uncaring traits, but not callousness or unemotional traits, were inversely correlated with the N170 response to sad and neutral facial expressions, with similar trends observed in response to fearful and angry facial expressions. Though controlling stringently for multiple comparisons, this investigation used a large number of correlations and so the inverse relationship between uncaring traits and the N170 is likely pervasive across facial affect. This finding is supported, to some degree, by the meta-analysis reported in Dawel et al. (2012) that found individuals scoring high on the affective factor of psychopathy evidenced deficient face processing uniformly

across facial affect. However, the current results suggest a specific association with uncaring traits.

Previously, N170 attenuation has been associated with poor emotional sensitivity/expressivity (Meaux et al., 2014) and fearless dominance (Almeida et al., 2014) in cohorts derived from the community, as well as clinical samples characterised by blunted affect (Ibáñez et al., 2012). Whilst fearless dominance is thought to strongly relate to the uncaring subscale of the ICU (Kimonis et al., 2013), this is the first investigation to correlate distinct subscales of the ICU with N170 responses to facial affect. Therefore, although not measured in this investigation specifically, the current results partly support associations between fearless dominance and the N170 observed elsewhere (Almeida et al., 2014).

Neither callousness, the poor affective response to the feelings of others, nor unemotional traits, the lack of emotional expression (Frick, 2003) associated with N170 amplitude. Previously, only one investigation has measured the N170 in association with callous-related traits (Almeida et al., 2014). Here, Almeida et al. found positive associations between cold-heartedness scores and N170 responses to angry facial expressions in a community sample. Although a related measure to cold-heartedness (Patrick, 2010), callousness was not correlated with the N170 in this investigation when evoked to comparable stimuli. It is possible this disparity is attributed to task effects. Whilst the FAST orientates attention to facial affect, the paradigm used in Almeida et al. tasked participants with responding to non-face targets. Discussed in chapter three, section 3.2.1 (page 39), N170 amplitude might be associated with task-specific voluntary attention (Holmes et al., 2003).

8.4.2 P200

This is the first investigation to correlate psychometric measures of physical aggression with P200 responses to facial affect. One previous investigation observed P200 attenuation uniformly across the six core facial expressions (*see chapter one, section 1.3.1 [page 7]*) in relation to poor emotional control, an

index of the likelihood to aggress (Meaux et al., 2014). In contrast to expectations, physical aggression did not correlate with P200 amplitude in this investigation. This result supports previous findings using auditory stimuli, which concluded no correlation between auditory P200 amplitude and either antisocial behaviour or reactive aggression (Barratt et al., 1997; Lijffijt et al., 2012). In the wider context of distress-induced motor extinction, although physical aggression does not appear to associate with stages of face processing, its effect on later downstream mechanisms of cognitive and behavioural control remains unknown.

Prior to this investigation, scores on distinct subscales of the ICU have not been investigated in relation to the P200. In this investigation, uncaring, but not callousness or unemotional traits were found to positively correlate with P200 amplitude, measured over the left hemisphere, in response to sad and neutral facial expressions. Positive trends were also observed between uncaring traits and P200 responses to angry and fearful expressions. Somewhat counterintuitive, although these correlations cannot dictate causations, they indicate uncaring traits might benefit the processing of second-order facial features. As N170 amplitudes were included in this analysis as a confound regressor, temporally overlapping ERP responses cannot be used to explain these results. It is possible that the P200 augmentation observed here might reflect a compensatory measure for socially functioning individuals with uncaring traits during face processing. Whilst this explanation is not concurrent with the P200 augmentation observed in the psychopathic sample reported in Flor et al. (2002), as stated on page 149, the negative components in each study are not comparable.

8.4.3 Implications, Alternative Explanations, and Future Research

Efficient processing of facial affect is a core requirement of the VIM with the ability to accurately process facial distress considered key when triggering distress-induced behavioural inhibition (Blair, 2001). On an electrophysiological level, results of this investigation suggest uncaring traits in individuals drawn

from the community might contribute to a deficient ability to process both structural and configurable aspects of faces, as indexed by the N170 and P200, respectively. Ultimately, in the absence of sufficient executive control (Blair, 2001) dysfunctional processing of faces - particularly those depicting distress - might contribute to a failure to trigger the VIM, and in turn the manifestation of prolonged and uninhibited acts of aggression (Blair, 1995; Marsh & Ambady, 2007).

Dysfunction to the VIM has been evidenced in individuals with psychopathic traits (Blair, 1995, 2001; Blair et al., 2004) - characterised by affective, interpersonal, and behavioural malfunction (Hare, 2003). Recent meta-analytic work suggests an association between the affective factor of psychopathy (i.e. CU traits) and pervasive face processing impairment across a constellation of positive and negative emotions after controlling for aggressive traits (Dawel et al., 2012). This current investigation not only supports the view that distinguishing between CU and aggressive traits is important when investigating VIM malfunction but also extends this knowledge by showing specific associations between poor face processing efficacy and uncaring, above callousness, unemotional, and physically aggressive traits. Though limited to a community sample, such findings have implications for our understanding of how distinct psychopathy-related traits might moderate the face processing stage of the VIM and the methodological gain from distinguishing between such traits.

Results of this investigation should be discussed in light of alternative explanations and future avenues of investigation. First, it cannot be ruled out that the absence of correlations between ERP responses and physical aggression, callousness, and uncaring traits were an artefact of the duration of time that stimuli were presented for in the FAST. Previously, female psychopaths, characterised by a constellation of aggressive and CU traits, evidenced distress-processing deficits when presented with stimuli for a short, but not an *ad libitum* duration (Eisenbarth et al., 2008). As stimuli are presented

for 800 ms in the FAST, this duration might have masked trait-related facial affect processing deficits indexed on both electrophysiological and behavioural levels. Replication of this investigation using shorter stimulus durations might act to tease apart these trait associations in community samples thought to be proficient at facial affect recognition (Calder et al., 1996 Sawada et al., 2014).

Second, although nuisance regressors were controlled for where appropriate, conclusions in this investigation are drawn from correlations and so causation cannot be inferred. In order to verify these associations, it is necessary to replicate this investigation by comparing electrophysiological responses from groups of high and low trait responders. However, in order to investigate associations between ERP responses and all four psychometric traits observed in this investigation, large age and sex matched cohorts would be required to account for these groups.

8.4.4 Summary

Although wider associations might have been masked as a function of task design, this investigation concluded uncaring traits, but not physical aggression, callousness, and unemotional traits, were associated with both structural and second-order stages of face processing during a FAST. The ability to accurately process facial expressions is a key component of cognitive models of aggression inhibition, such as the VIM. Using EEG to investigate how aggressive/CU traits might moderate distinct stages of face processing is important for gaining a better understanding of how such traits might contribute to the manifestation, and disinhibition of an aggressive response. In addition to face processing, it is important to understand how physical aggression and CU traits might relate to stress-induced motor extinction.

Chapter Nine: Correlations between physical aggression/callous-unemotional traits and motor extinction ERPs

9.1 Introduction

The ability to extinguish a motor response as a function of social stimuli is key for adaptive behaviour (Huster et al., 2014; Sagaspe et al., 2011). As modelled by the VIM, facial distress is thought to halt aggressive/antisocial behaviour in typically developing humans - possibly reflecting the alleviation of a distress-induced empathic response (Blair, 1995, *see chapter one, section 1.3.4 [page 12]*). As such, the VIM is considered dysfunctional in individuals characterised by affective, interpersonal, and behavioural malfunction, such as those exhibiting CU and aggressive traits (Blair, 2001; Hare, 2003). In chapter eight, uncaring traits, but not physical aggression, callousness, or unemotional traits, were shown to correlate with electrophysiological indices of face processing (N170 and P200). The current investigation extends this knowledge by further delineating the relationships between physical aggression/CU traits and electrophysiological indices of distress-induced motor extinction (stop-N200 and stop-P300).

In response to stimuli denoting the need to inhibit or extinguish a motor response, the N200 and P300 are thought to index the recognition of inhibitory need and inhibitory control efficiency, respectively (Bruin & Wijers, 2002; Hughes et al., 2012; Kok et al., 2004). To date, whilst not previously investigated in response to stop stimuli, physical aggression and CU traits have been shown to moderate N200 and P300 responses evoked to paradigms requiring the passive viewing of stimuli and/or premotor inhibitory control (*see chapter three, sections 3.4.1 [page 50] and 3.4.2 [page 52] for review*).

There is disparity as to how physical aggression and CU traits might moderate the N200 response. In both adolescents with CD (Sumich et al., 2012) and

adults with psychopathy (Kiehl et al., 2006), N200 augmentation has been reported in response to background (visual letters) and nogo (auditory tones) stimuli, respectively. This positive association is thought to reflect the emergence of CU traits alongside disrupted cortical remodelling (de Brito et al., 2009; Sumich et al., 2012). Elsewhere, N200 *attenuation* has been reported in offenders with psychopathy, relative to offenders with and without schizophrenia (Kiehl et al., 2000). However, this result might be an artefact of either methodological variation or co-varying aggressive traits (*see chapter three, section 3.6.3 [page 57]*). As anterior midline nogo-N200 attenuation has been observed in male offenders characterised by impulsive and violent behaviour, relative to age-matched controls (Chen et al., 2005, 2008), aggressive traits might be associated with poor mobilisation of neuronal resources to inhibitory stimuli and a deficit for the recognition of inhibitory need.

Similarly, auditory and visual P300 responses, across both target and nogo stimuli, have shown attenuation in aggressive samples derived from forensic and community settings (s*ee chapter three, section 3.6.4 [page 58] for review*). In contrast, P300 amplitude is thought to remain stable (Patrick, 2008; Raine & Venables, 1987, 1988) or even augment (Carlson & Thái, 2010; Flor et al., 2002; Raine, 1989) in individuals characterised by CU traits. This interaction is further supported by findings of P300 attenuation in individuals with psychopathy during, but not outside of, interpersonal-affective encounters (Hicks et al., 2007). However, as P300 attenuation has been observed in individuals with psychopathy during inhibitory, but not target detection trials (Kim & Jung, 2014), the relationship between CU traits and the P300 might be dependent on task – possibly reflecting specific moderation of inhibitory networks. This finding warrants further investigation.

Taken together, although N200 and P300 responses to background, target, and nogo stimuli have been investigated in samples characterised by aggressive and CU traits, such ERP/trait associations have not been investigated during stop tasks. This investigation delineates these associations in the context of the

VIM, using a FAST, whereby stop ERPs were evoked to fearful and sad facial expressions. It was hypothesised that [1] physical aggression would be inversely correlated with N200 and P300 responses, to both background and stop stimuli. CU traits were hypothesised to [2] positively correlate with N200 responses to both background and stop stimuli, but [3] inversely correlate with P300 responses to stop stimuli only.

9.2 Methods

Core methodology has been documented in chapter seven, section 7.3.2 (page 117). For this investigation, only N200 and P300 responses to fearful/sad background and stop stimuli were reported.

9.2.1 Measures

Participants completed the AQ and ICU (*see chapter five, sections 5.3.1 [page 83] and 5.3.2 [page 84], respectively).* For this investigation, only the physical aggression subscale of the AQ was used.

9.2.2 Statistical Analysis

Questionnaire responses and mean ERP amplitudes for each component (i.e. N200, P300, stop-N200, stop-P300) across each facial expression (i.e. fearful, sad) were calculated and entered into an R Statistics v.2.15 dataset (R Core Team, 2012). Averaged N200 and P300 responses to background and stop stimuli were measured at anterior- (Fz) and parietal- (Pz) midline sites. Pearson's partial-correlations were calculated for *trait* (i.e. callousness, uncaring, unemotional, physical aggression)*, trial type* (i.e. background, stop), *ERP* (i.e. N200, P300), *emotion* (i.e. fearful, sad), and *site* (i.e. anterior, parietal). Further associations were explored between *trait* (i.e. callousness, uncaring, unemotional, physical aggression) and *behavioural measures* (i.e. stopping time, accuracy) for each *emotion* (i.e. fearful, sad). Correlations

controlled for age and sex (demeaned), and were corrected for multiple comparisons (*see Benjamini & Hochberg, 1995*).

9.3 Results

9.3.1 Psychometrics

Means and standard deviations for psychometric measures were as follows: *physical aggression* (M = 52.98 [*t*-transformed], SD = 8.61), *callousness* (M = 5.98, SD = 2.97), *uncaring* (M = 8.00, SD = 3.33), and *unemotional* traits (M = 8.16, SD = 3.06). Mean amplitudes for N200 and P300 responses are reported in Tables 6 (page 123) and 7 (page 133), respectively.

9.3.2 Behavioural data

Correlations between *trait* (i.e. callousness, uncaring, unemotional, physical aggression) and *behaviour* (i.e. stopping time, accuracy) across *emotion* (i.e. fearful, sad) are displayed in Table 9. Callousness shared a moderate, positive correlation with reaction times to both fearful (r = .27, p = .05, *padj* = .62) and sad facial stimuli (r = .32, p = .02, *padj* = .27), as did uncaring traits (fearful [r = .26, p = .05, *padj* = .62], sad [r = .31, p = .02, *padj* = .29]). However, these correlations did not survive multiple comparison correction.

Table 9.

Correlations between psychometric measures and behavioural data

	Fearful		Sad	
	Stopping Accuracy	RT	Stopping Accuracy	RT
P. Aggression	-.03	-.22	-.02	-.16
Callousness	-.08	.27	-.09	.32
Uncaring	.08	.26	.02	.31
Unemotional	.04	.04	.01	.02

Note.* * p < .05, ** p < .005, *** p < .001, *P.Aggression = Physical Aggression

9.3.3 Correlations

9.3.3.1 N200

At anterior midline sites, physical aggression was strongly and positively correlated with N200 responses to facial distress during stop conditions (fear [r = .51, p < .001, $padj$ < .001], sad [r = .42, p < .001, $padj$ = .03]). Physical aggression did not correlate with N200 responses evoked to background stimuli. At parietal midline sites, uncaring traits were positively correlated with N200 responses to background facial distress (fear [r = .29, p = .03, $padj$ = .19], sad [r = .32, p = .02, $padj$ = .16]), but did not survive multiple comparison correction. No other CU traits correlated with N200 responses evoked to either background or stop stimuli. Significant correlations are displayed in subplots A and B of Figure 9.

9.3.3.2 P300

At anterior midline sites, physical aggression shared a moderately strong, negative correlation with P300 responses to facial distress during stop conditions (fear [r = -.40, p < .05, $padj$ = .04], sad [r = -.32, p = .02, $padj$ = .16]). However, the latter did not survive multiple comparison correction. Physical aggression did not correlate with P300 responses evoked to background stimuli. Uncaring and unemotional traits were inversely correlated with stop-P300 responses to sad facial expressions at anterior and parietal midline sites, respectively (uncaring [r = -.30, p = .02, $padj$ = .18], unemotional [r = -.25, p = .01, $padj$ = .12]). However, neither correlation survived multiple comparison correction. No other CU traits correlated with P300 responses to either background or stop stimuli. Significant correlations are displayed in subplots C and D of Figure 9.

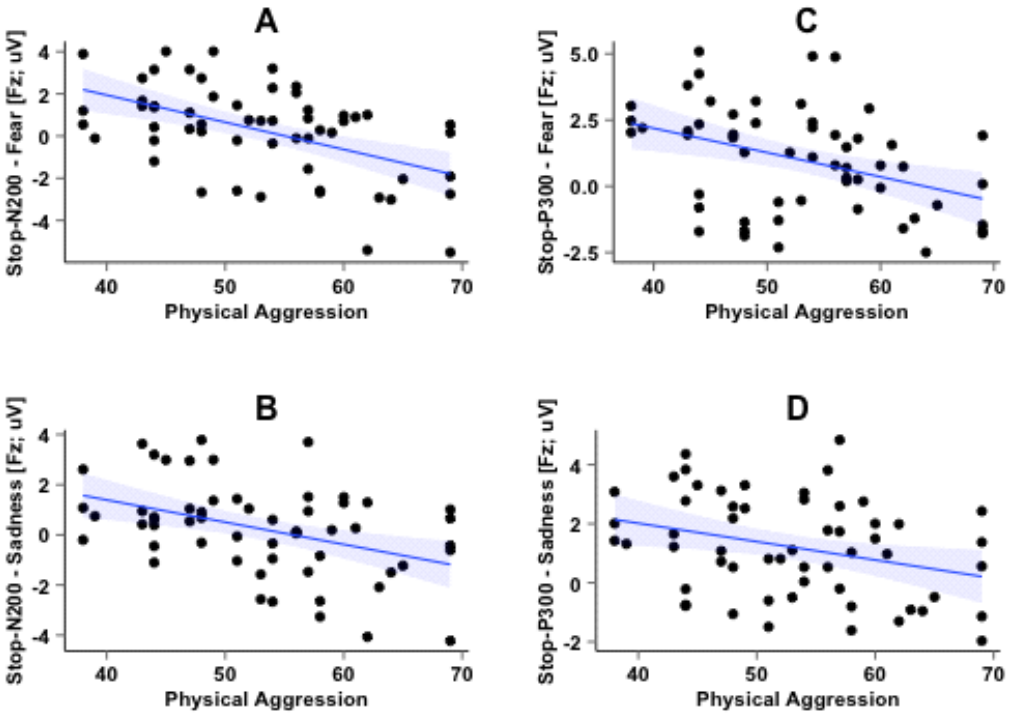

Figure 9. Correlations between physical aggression and stop-N200 (subplots A [*r* = .51] and B [*r* = .42]) and stop-P300 amplitudes (subplots C [*r* = -.40] and D [*r* = -.32]) evoked to fearful and sad facial expressions, measured at Fz. Shaded areas denote 95% confidence interval areas along linear associations.

9.4 Discussion

This investigation delineated the relationships between physical aggression/CU traits and electrophysiological indices of distress-induced motor extinction. Although no associations were observed between psychometric measures and behavioural responses, physical aggression positively correlated with N200 amplitude, and negatively correlated with P300 amplitude to stop, but not background stimuli. CU traits did not exhibit statistically significant correlations with electrophysiological responses to either stop or background stimuli. Results are discussed in the context of the VIM, in light of limitations and future investigation.

9.4.1 N200

Irrespective of stimulus type, N200 amplitude was hypothesised to inversely correlate with physical aggression and positively correlate with CU traits. Unexpectedly, the current investigation suggested that physical aggression was both [1] statistically unrelated to N200 responses to *background* stimuli, and [2] positively, not negatively, correlated with N200 responses to fearful and sad *stop* stimuli.

The N200 response to stop stimuli is thought to index the preparatory response preceding motor extinction (Kok et al., 2004). Previously, N200 responses to nogo stimuli have shown attenuation over anterior midline sites in samples of male offenders characterised by impulsive and violent behaviour, relative to age-matched controls (Chen et al., 2005, 2008). The observation of a positive correlation between the anterior N200 response to stop stimuli and physical aggression in this investigation might be explained in two ways. First, N200 augmentation in community-based individuals with high trait scores for physical aggression might reflect the need for increased recruitment of neuronal resources when preparing to extinguish a motor response. Second, stop-N200 augmentation might be an artefact of co-varying CU traits previously shown to associate with N200 augmentation (Kiehl et al., 2006; Sumich et al., 2012). However, as no CU traits evidenced statistically significant correlations with N200 responses in this investigation (*see page 164*), this explanation is unlikely.

The relationship between the N200 response to facial expressions and physical aggression was limited to stop stimuli. This finding echoes previous investigation identifying an absence of auditory N200 amplitude variation between male offenders, characterised by behavioural control problems, and non-offender controls (Barratt et al., 1997; Munro et al., 2007). However, as this is the first investigation to associate self-reported physical aggression with N200 amplitude in a community sample, further replication of this null finding is required to verify this association.

In contrast to expectations, no subscale of the ICU was significantly correlated with N200 responses to either fearful or sad facial expressions, across background or stop stimuli. Previously, N200 augmentation, indicative of disrupted cortical remodelling (de Brito et al., 2009; Sumich et al., 2012), has been evidenced in adults with psychopathy and adolescents with CD (Kiehl et al., 2006; Sumich et al., 2012). The null effect reported in this investigation suggests callousness, uncaring, and unemotional traits do not correlate with electrophysiological indices of automatic alerting/stimulus classification (background) or the preparation of motor extinction (stop). However, as this community sample is unlikely to display CU traits to the extent of those in forensic or clinical populations, a group comparison between low- and high-extreme ICU scorers might better answer this question. Here, larger N200 responses to background and stop stimuli would be expected in samples reporting high, relative to low, CU traits.

9.4.2 P300

As P300 attenuation has been repeatedly observed in forensic and community cohorts characterised by aggressive traits (s*ee chapter three, section 3.6.4 [page 50] for review*), physical aggression scores were hypothesised to negatively correlate with FAST-evoked P300 responses to both background and stop stimuli. However, in this investigation, whilst physical aggression scores negatively correlated with the P300 response to stop stimuli, comparable results were not observed to background stimuli. Previously, aggression-related P300 attenuation has been observed in community samples in response to both auditory and visual stimuli (Bartholow et al., 2006; Gerstle et al., 1998). Results of this investigation might be explained by methodology. First, P300 responses were measured in response to background, but not target stimuli, and second, said stimuli consisted of facial expressions.

In this investigation, whilst not elicited in response to background stimuli, the observation of an inverse correlation between physical aggression and P300 responses to stop stimuli is novel. However, as this relationship is limited to [1]

self-reported physical aggression, in [2] a community sample, and [3] in response to facial expressions depicting distress, further replication is required. Results of this investigation suggest individuals reporting higher trait physical aggression might be less efficient at engaging motor extinction mechanisms. Although not supported by behavioural data, it remains to be seen whether variation in behavioural responses would emerge from sampling individuals with higher degrees of trait aggression. Previously, increased reaction times have been positively associated with AQ-measured aggressive traits during an affect categorisation paradigm (Pawliczek et al., 2013). However, Pawliczek et al. defined high and low aggression groups based on non-transformed, total AQ scores, and not the physical aggression subscale, specifically.

In light of previous investigations documenting [1] comparable P300 responses to target/background stimuli between individuals with and without CU traits (Patrick, 2008; Raine & Venables, 1987, 1988), but [2] P300 attenuation during inhibitory tasks (Kim & Jung, 2014), CU traits were hypothesised to negatively correlate with P300 responses to stop, but not background facial stimuli. After correcting for multiple comparisons, no CU trait was associated with P300 responses to either stop or background stimuli in this investigation. Disparity between results reported here and those in Kim & Jung (2014) could be explained by variation in task (i.e. nogo vs. stop), stimuli (i.e. geometric shapes vs. faces), or assay of CU traits (i.e. PPI-R vs. ICU), and so warrants further investigation.

9.4.3 Chapter Summary

Findings of this investigation have implications for better understanding how CU traits and physical aggression associate with distress-induced motor extinction on an electrophysiological level. In contrast to associations reported in chapter eight (*see page 147*), whereby uncaring traits, but not physical aggression, were correlated with electrophysiological indices of face processing, this investigation suggests that individuals with high trait scores of physical aggression might require greater recruitment of neuronal resources when

preparing a motor extinction response (N200). Moreover, such individuals appear less able to mobilise neuronal resources during actual response extinction as a function of facial distress (P300). Although findings in this investigation are novel and contribute to theoretical knowledge relating to the distinct stages of the VIM, it remains to be seen whether such findings are comparable to those observed in populations characterised by extreme physical aggression/CU traits.

Chapter Ten: Relationships between omega-3 intake, callous-unemotional traits, and physical aggression

10.1 Introduction

Malnourishment and dietary deficiency contribute to the development of psychopathologies characterised by poor cognition, impulsivity, and aggression (Freeman et al., 2006; Grantham-McGregor et al., 2007; Pollitt, 2000, *but see chapter four for review [from page 63]*). With a growing interest in using diet, specifically omega-3, as a moderator of aggressive behaviour (Buydens-Branchey & Branchey, 2008) and recent investigation into the effects of omega-3 on CU traits (Gow, Vallee-Tourangeau et al., 2013; Raine et al., 2015), there is reason to expect omega-3 might moderate the VIM response. Focusing solely on omega-3s EPA and DHA, this investigation explores relationships between omega-3 dietary intake, physical aggression, and CU traits. Results are discussed in light of methodological and practical utility, limitations, and future avenues of investigation.

Low plasma levels of composite omega-3 and sub optimal ratios of omega-3:-6 have been associated with increased aggressive behaviour in rodent (DeMar et al., 2006; Raygada et al., 1998) and canine (Re et al., 2008) samples. In humans, an inverse association between fatty fish consumption and homicide rates has been observed in a large cross national analysis (Hibbeln, 2001), with dietary deficiency of both EPA and DHA being associated with increased historical incidents of violence (Buydens-Branchey et al., 2003; Umhau et al., 2006). Elsewhere, whilst DHA has been inversely correlated with psychometric measures of aggression (Zaalberg et al., 2015), similar associations with EPA have only been observed in the absence of comorbid substance abuse (Beier et al., 2014). Alcohol and drug use is thought to moderate the structural composition of fatty acids (Pawlosky et al., 2001), and so substance use might be a possible confound when associating fatty acids and aggression.

Further support for the relationship between omega-3 and aggression comes from supplementation research. Although not conclusive (*see chapter four, section 4.3.2.3 [page 71]*), DHA and EPA supplementation has been found to reduce incidents of violence and rule breaking behaviour in forensic settings (Gesch et al., 2002 [44 mg DHA; 80 mg EPA]; Zaalberg et al., 2010 [400 mg DHA; 400 mg EPA]). Moreover DHA-rich supplementation (510 mg DHA; 120 mg EPA) has benefited parent- and teacher-rated aggression in adolescents with ADHD (Hamazaki & Hirayama, 2004; Stevens et al., 2003). However, as such studies vary in terms of target population, qualification of aggression, and supplementation type/dose, direct comparisons are difficult to draw.

Throughout this thesis, focus has been placed on trait measures of physical aggression. Composite levels of omega-3 intake have been inversely correlated with self-reported physical aggression in a large cohort of male offenders, but only at trend-level significance (Meyer et al., 2015). Decreased physical aggression has also been observed in a small sample of female patients with BPD following two months of daily EPA, relative to placebo, supplementation (Zanarini & Frankenburg, 2003 [1000 mg EPA]). However, whilst a three-month period of daily DHA supplementation (670 mg DHA) has been shown to reduce aggressive dialogue in adults – as measured by a picture-frustration paradigm - no effect on physical aggression was observed (Long & Benton, 2013). It is possibly that whilst omega-3 might benefit aggression as a whole, there might be variation in the effect of DHA/EPA on physical aggression, specifically.

In contrast to trait aggression, very few studies have investigated the relationship between omega-3 intake and CU traits, with none to date controlling for the co-variation of aggressive traits. Because of the intricate relationships between aggressive and CU traits explored in chapter six (*see from page 90*), incorporating these relationships into a statistical model is key. Although limited to an adolescent sample with ADHD symptomology, total blood measures of omega-3, EPA, and DHA have been shown to inversely relate to psychometric measures of callousness, uncaring, and unemotional traits (Gow,

Vallee-Tourangeau et al., 2013). However, these results only reached trend-level significance, and in contrast to results reported above, omega-3 did not correlate with trait aggression in either ADHD or control sample. Furthermore, supplementation of 300 mg DHA and 200 mg EPA resulted in reduced parent-reported cold-heartedness, fearlessness, and CU traits, which persisted over a six-month period (Raine et al., 2015).

This investigation delineates associations between EPA and DHA intake on self-reported physical aggression and CU traits in a community sample. Driven by the limited literature documenting the specific associations between omega-3 intake and physical aggression/CU traits, it was hypothesised that [1] physical aggression would be negatively correlated with EPA, but not DHA intake, and [2] CU traits would be negatively correlated with both EPA and DHA intake.

10.2 Methods

10.2.1 Participants

A total of 98 participants (88 female; M age = 21.47; SD = 3.07) were recruited via online, snowball sampling. Participants consented to take part in a brief online questionnaire after responding to an advertisement disseminated via social media.

10.2.2 Measures

Participants completed a battery of questionnaires consisting of demographics and online versions of the AQ, ICU, and FFQ (*see chapter five, sections 5.3.1 [page 83], 5.3.2 [page 84], and 5.3.3 [page 87]*). For this investigation, only the physical aggression subscale of the AQ, and EPA and DHA subscales of the FFQ were used.

10.2.3 Procedure

Participants were briefed and provided informed consent. On average, the questionnaire battery took less that 20 minutes to complete, and respondents were provided with means of redistributing the survey through social media, if they so wished. Respondents were debriefed and provided a means of contacting the lead researcher (DF).

10.2.4 Statistical Analysis

Scores for the physical aggression subscale of the AQ and the callous, uncaring, and unemotional subscales of the ICU were reverse scored and calculated into an R Statistics v.2.15 dataset (R Core Team, 2012). Physical aggression scores were *t*-transformed as a function of age and sex. Guided by Sublette et al. (2011), EPA and DHA intake were calculated as a function of fish type, portion size, consumption frequency, and sex of responder. Due to low self-reported omega-3 consumption, EPA and DHA consumption data were not normally distributed (skew [EPA (3.04, SE = .13), DHA (3.61, SE = .13)], kurtosis [EPA (11.13, SE = .25), DHA (15.87, SE = .25)]), and so underwent *ln*-transformation. Precedent for *ln*-transformation of FFQ data is set in Beier et al. (2014).

A total of eight Pearson's partial correlations were used to delineate relationships between psychometric traits (i.e. physical aggression, callousness, uncaring, unemotional) and daily intake of EPA and DHA. Correlations controlled for demeaned age, sex, and scores on psychometric measures *not* included in the specific analysis (*see chapter six, section 6.2.4 [page 93] for further explanation*). Partial correlations were corrected for multiple comparisons (*see Benjamini & Hochberg, 1995*).

10.3 Results

Means and standard deviations for each self-report measure were as follows: *physical aggression* (M = 51.16, SD = 7.48), *callousness* (M = 4.14, SD = 3.34),

uncaring (M = 7.45, SD = 3.73), *unemotional* (M = 6.86, SD = 3.10). Mean daily EPA consumption was .01 g (SD = .02) and mean daily DHA consumption was .13 g (SD = .28). Following *ln*-transformation the mean EPA score was -2.70 (SD = 1.02) and the mean DHA score was -1.78 (SD = 1.17).

Table 10.

Partial correlations between fatty acid intake (EPA, DHA) and psychometric scores (physical aggression, callousness, uncaring, unemotional)

	P. Aggression	Callousness	Uncaring	Unemotional
EPA	-.39***	-.13	.07	-.14
DHA	-.24	-.14	.16	.09

***Note.* * $p < .05$, ** $p < .005$, *** $p < .001$, P. Aggression = Physical Aggression**

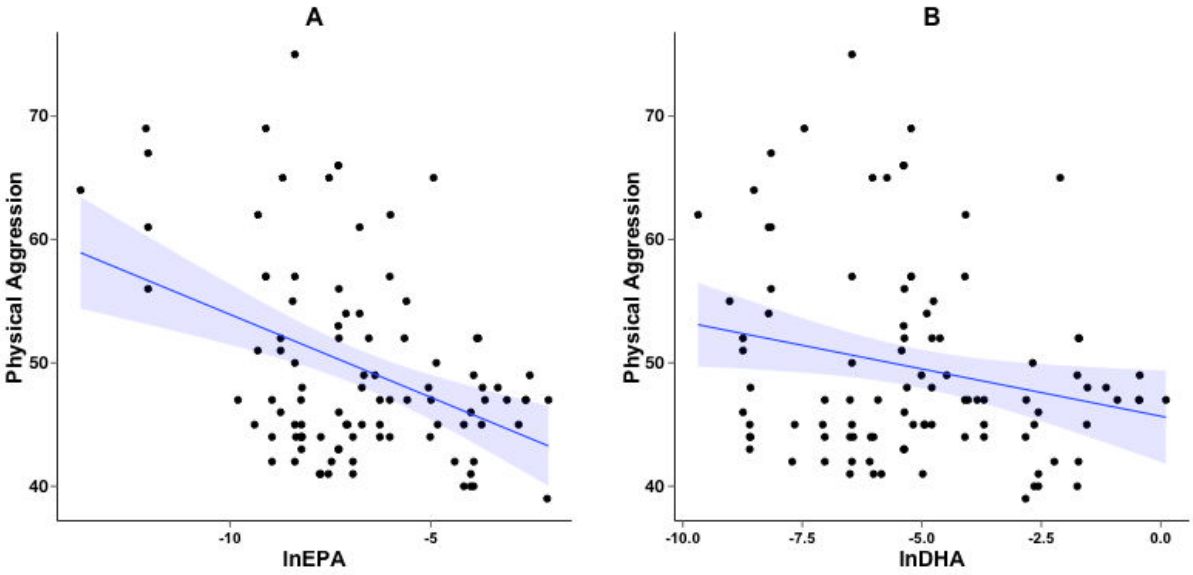

Fig 10. Correlations between physical aggression and *ln*-transformed EPA ($r = -.39$, subplot A) and DHA intake ($r = -.24$, subplot B). Prior to *ln*-transformation, EPA and DHA intake were measured in g/day. Shaded areas denote 95% confidence interval areas along linear associations.

Partial correlations between *ln*-transformed EPA and DHA intake and psychometric measures are reported in Table 10. Neither EPA nor DHA intake correlated with any subscale of the ICU. EPA shared a moderately strong, inverse correlation with self-reported physical aggression (r = -.39, p < .001, *padj* < .001, *see subplot A of Figure 10*), and contributed to 27.11% of explained variance. Although a similar association was observed between DHA and physical aggression (r = -.24, p = .02, *padj* = .16, *see subplot B of Figure 10*), this correlation did not survive multiple comparison testing.

10.4 Discussion

Whilst relationships between aggression and CU traits are well established (Frick et al., 2003; Frick & White, 2008, *but see chapter one, section 1.3.4 [page 12]*), their independent associations with omega-3 are unclear. This investigation explored correlations between self-reported EPA and DHA intake with psychometric measures of physical aggression, callousness, and uncaring and unemotional traits. Results suggested a single inverse association between physical aggression and intake of EPA, but not DHA. Neither DHA nor EPA correlated with scores on any subscale of the ICU.

To date, investigation between omega-3 and physical aggression has suggested an inverse association between composite dietary omega-3 intake and scores on self-reported measures of physical aggression, in male offenders (Meyer et al., 2015). Although restricted to EPA, and not DHA, this investigation extends these findings into a community sample, using the FFQ to measure omega-3 intake. Results reported here are further supported by investigations using supplementation. Whilst Zanarini & Frankenburg (2003) observed decreased self-reported physical aggression in female patients with BPD following EPA supplementation, no effect on physical aggression was observed in the sample of non-forensic adults reported in Long & Benton (2013) following DHA supplementation.

Thought present prior to multiple comparison correction, self-reported intake of DHA and physical aggression were not statistically associated in this investigation. However, as blood assays of DHA have previously shown negative associations with the antecedence to aggress (Fontani et al., 2005; Légaré et al., 2007), and observed aggression in both canine (Re et al., 2008) and human samples (Buydens-Branchey et al., 2003; Hamazaki et al., 2004; Hibbeln et al., 1998; Stevens et al., 2003; Virkkunen, et al., 1987), this result warrants further replication.

This is the first investigation to document associations between self-reported omega-3 intake and scores on subscales of the ICU. Opposing the study hypothesis - bore out of, albeit, a limited literature - this investigation indicated that neither EPA nor DHA intake was significantly correlated with any subscale of the ICU. Previously, low EPA and total omega-3 blood measures have been associated with increased CU traits in male children with ADHD (Gow, Vallee-Tourangeau et al., 2013). Although worth noting that results in Gow, Vallee-Tourangeau et al. only reached trend-level significance, disparity of results could be explained by differences in blood-/self-assessed omega-3 intake and sample populations.

Findings reported in this investigation should be considered in light of three limitations. First, the FFQ has only been validated in patients with major depressive disorder (Sublette et al., 2011) and so its validity across cognitive function, race, and culture remains unknown. However, as noted in chapter five, section 5.3.3 [page 87], the use of the FFQ is justified in this investigation. Nevertheless, results here would benefit from a blood assay of omega-3. Blood measurements would allow for both the accurate quantification of omega-3, and provide a means of assessing omega-6 without the need for additional questionnaire measures. Omega-6 intake has been positively associated with aggressive behaviour (Hibbeln, 2001; Virkkunen et al., 1987; Zaalberg et al., 2015), and would likely moderate the relationship between physical aggression and omega-3 observed here.

Second, this investigation did not control for variation in the intake of substance use (e.g. alcohol, drug). Alcohol consumption and substance abuse have been shown to compromise omega-3 structural composition (Pawlosky et al., 2001), and contribute to the relationship between EPA deficiency and trait aggression (Beier et al., 2014). Beier et al. observed an inverse association between EPA blood measures and aggression in patients with major depressive disorder, but only in the presence of comorbid substance abuse. Although not ethically viable in this online cohort, future investigation would be expected to show a greater effect between low omega-3 intake and increased physical aggression in individuals with high substance consumption.

Third, though not detrimental to statistical evaluation, self-reported measures of EPA and DHA required logarithmic transformation due to a large proportion of respondents reporting only minimal, if any, omega-3 consumption. This data distribution might reflect the sample comprising mostly of students, who might consume fewer, more expensive omega-3-containing foods. Future investigation should seek to delineate direct group comparisons between high and low omega-3 consuming samples, in regards to the presence of aggressive/CU traits. If such results were true to those reported here, high, relative to low, omega-3 consumers would be expected to evidence increased trait physical aggression.

In summary, whilst not associated with CU traits, self-assessed EPA, but not DHA intake was shown to inversely correlate with psychometric-based physical aggression. Such findings are novel and contribute to a growing literature delineating the role of omega-3 in aggressive/CU traits. Future investigation should seek to extend these results using biological assays and demographic-matched group comparisons.

Chapter Eleven: Correlations between omega-3 intake and the electrophysiological indices of the Violence Inhibition Mechanism

11.1 Introduction

The FAST is a useful tool for investigating the VIM as it allows for the simultaneous measurement of electrophysiological responses to facial affect and distress-induced motor extinction (*see chapter seven [from page 102]*). When investigated alongside personality traits associated with VIM dysfunction, distinct correlations have been observed between uncaring traits/physical aggression, and electrophysiological indices of [1] face processing (*see chapter eight [from page 149]*) and [2] distress-induced motor extinction (*see chapter nine [from page 159]*), respectively. From a preventative/treatment perspective, there is a growing interest into how omega-3 might serve to moderate aggressive and CU traits (Raine et al., 2015). Specifically, omega-3 has been shown to negatively correlate with both physical aggression (Meyer et al., 2015; Zanarini & Frankenburg, 2003, *but see chapter 10 [from page 169*) and ICU-measured CU traits (Gow, Vallee-Tourangeau et al., 2013). However, little is known about how omega-3 intake might relate to the VIM, and how this might reflect on an electrophysiological level.

In response to faces, the N170 and P200 are thought to reflect activation of medial temporal networks comprising the fusiform gyrus and STS (Halgren et al., 1994; Nummenmaa et al., 2010; Sadeh et al., 2010). Although an abundance of omega-3 has been identified in the medial temporal lobe (McNamara, 2010), omega-3 localised to the fusiform gyrus/STS has not been used to predict face processing efficacy or electrophysiological indices there of. To date, the single investigation to measure both ERP responses to faces and omega-3 blood concentrations did not report correlations between omega-3 and ERPs prior to 250 ms (Gow, Sumich et al., 2013). However, had this data been presented, conclusions would have been limited due to ERPs being recorded

over midline, and not optimum temporo-parietal sites (Luck & Kappenman, 2013, *see chapter three, section 3.2.1 [page 41]*). In this investigation, there were no statistically significant correlations between omega-3 fatty acid levels and the face-evoked P300.

Motor extinction can be indexed on an electrophysiological level by stop-N200 and stop-P300 ERPs, representative of the recognition of inhibitory need and motor extinction efficacy, respectively (Hughes et al., 2012; Kok et al., 2004). In this thesis, both N200 and P300 responses to stop stimuli have been correlated with trait physical aggression (*see chapter nine [from page 159]*), which in turn, has shown an inverse relationship with self-reported EPA intake (*see chapter ten*). However, previously, no investigation has delineated the relationship between omega-3 intake and electrophysiological indices of motor extinction.

Although a function of alcohol and drug use (Pawlosky et al., 2001), omega-3 might benefit neuronal networks associated with motor extinction through its role in synaptic development (Carlson, 2001; Serfaty & de Velasco, 2014; Suzuki et al., 1997). For example, efficiency of synaptic pruning, a process key for cell specialisation and inter-cellular communication (Bazan, 2005; Kitajka et al., 2004; Litman et al., 2001; Michael-Titus & Priestley, 2014; Mitchell et al., 2001; Uauy & Dangour, 2006), has been associated with greater cell concentrations of DHA (Carlson, 2001). On an electrophysiological level, though not previously investigated alongside omega-3 intake, dysfunctional synaptic pruning has been associated with delayed N200 attenuation, as a function of age, in males with CD (Sumich et al., 2012).

This investigation aimed to delineate associations between EPA and DHA intake on electrophysiological indices of face processing and distress-induced motor extinction. Previously, physical aggression and CU traits have been shown to moderate face processing and motor extinction (*see chapters seven and eight*), as well as being correlated with omega-3 intake (*see chapter ten*). In this investigation, these covariates were held constant. EPA and DHA intake

were hypothesised to positively correlate with [1] N170 and P200 responses, with [2] no prior literature to suggest discriminate effects of emotion. Furthermore, [3] DHA intake was hypothesised to inversely correlate with stop-N200 amplitude and [4] EPA intake was predicted to positively correlate with stop-P300 amplitude.

11.2 Methods

Core methodology has been documented in chapter seven, section 7.3.2 (page 117). In the interest of working in a parsimonious manner, only ERPs of direct interest, such as those evidencing statistically significantly associations with physical aggression or CU traits in chapters eight (*see from page 149*) and nine (*see from page 159*) of this thesis, were co-opted. For this investigation, N170 and P200 responses measured over the left hemisphere, and stop-N200 and stop-P300 responses measured over anterior midline sites were reported.

11.2.1 Measures

Participants completed the FFQ (*see section 5.3.3 [page 87]*), AUDIT (*see section 5.3.4 [page 88]*), and CUDIT (*see section 5.3.5 [page 88]*).

11.2.2 Statistical Analysis

FFQ, AUDIT, and CUDIT responses, as well as mean ERP amplitudes for each trial type were calculated and entered into an R Statistics v.2.15 dataset (R Core Team, 2012). ERPs consisted of averaged left (P7, PO7, O1, PO3), but not right, temporo-parietal N170 and P200 responses evoked to fearful, sad, neutral, and angry facial expressions, as well as anterior midline stop-N200 and stop-P300 responses evoked to fearful and sad facial expressions. Correlations between omega-3 intake and ERPs were investigated using two sets of Pearson's partial correlations. This first associated *omega-3* (i.e. DHA, EPA) and *ERPs* (i.e. N170, P200, stop-N200, stop-P300) across *emotion* (i.e. anger, fear, sadness, neutral). The second associated *omega-3* (i.e. DHA, EPA) and *behavioural responses* (i.e. reaction time, percentage correct). For reasons

documented in chapter ten, section 10.4 (page 174), partial correlations controlled for the effects of demeaned alcohol and cannabis use, age, and sex. All correlations were corrected for multiple comparisons (*see Benjamini & Hochberg, 1995*).

11.3 Results

Mean daily EPA consumption was .01 g (SD = .02) and mean daily DHA consumption was .15 g (SD = .23). Both EPA and DHA data violated assumptions of skew (EPA [2.21, SE = .32], DHA [1.59, SE = .32]) and kurtosis (EPA [5.54, SE = .62], DHA [1.18, SE = .62]) and so underwent *ln*-transformation prior to analysis (*ln*-EPA [M = -6.29, SD = 2.63], *ln*-DHA [M = -3.96, SD = 2.87]).

11.3.1 Behavioural Data

Neither EPA nor DHA correlated with stopping times or accuracy.

11.3.2 N170 and P200

Neither EPA nor DHA were correlated with left temporo-parietal N170 or P200 responses to fearful, sad, neutral, or angry facial expressions. Although EPA initially correlated with the N170 response to angry facial expressions ($r = .32$, $p = .03$, $padj = .51$), such that N170 amplitude increased alongside EPA intake, this finding did not survive multiple comparison testing.

11.3.3 Stop-N200 and Stop-P300

EPA intake shared moderately strong, positive correlations with both stop-N200 and stop-P300 responses to fearful (stop-N200 [$r = .43$, $p < .05$, $padj = .05$], stop-P300 [$r = .49$, $p < .001$, $padj = .01$]) and sad (stop-N200 [$r = .32$, $p = .03$, $padj = .50$], stop-P300 [$r = .34$, $p = .02$, $padj = .38$]) facial expressions. However only responses to fearful expression survived multiple comparison correction.

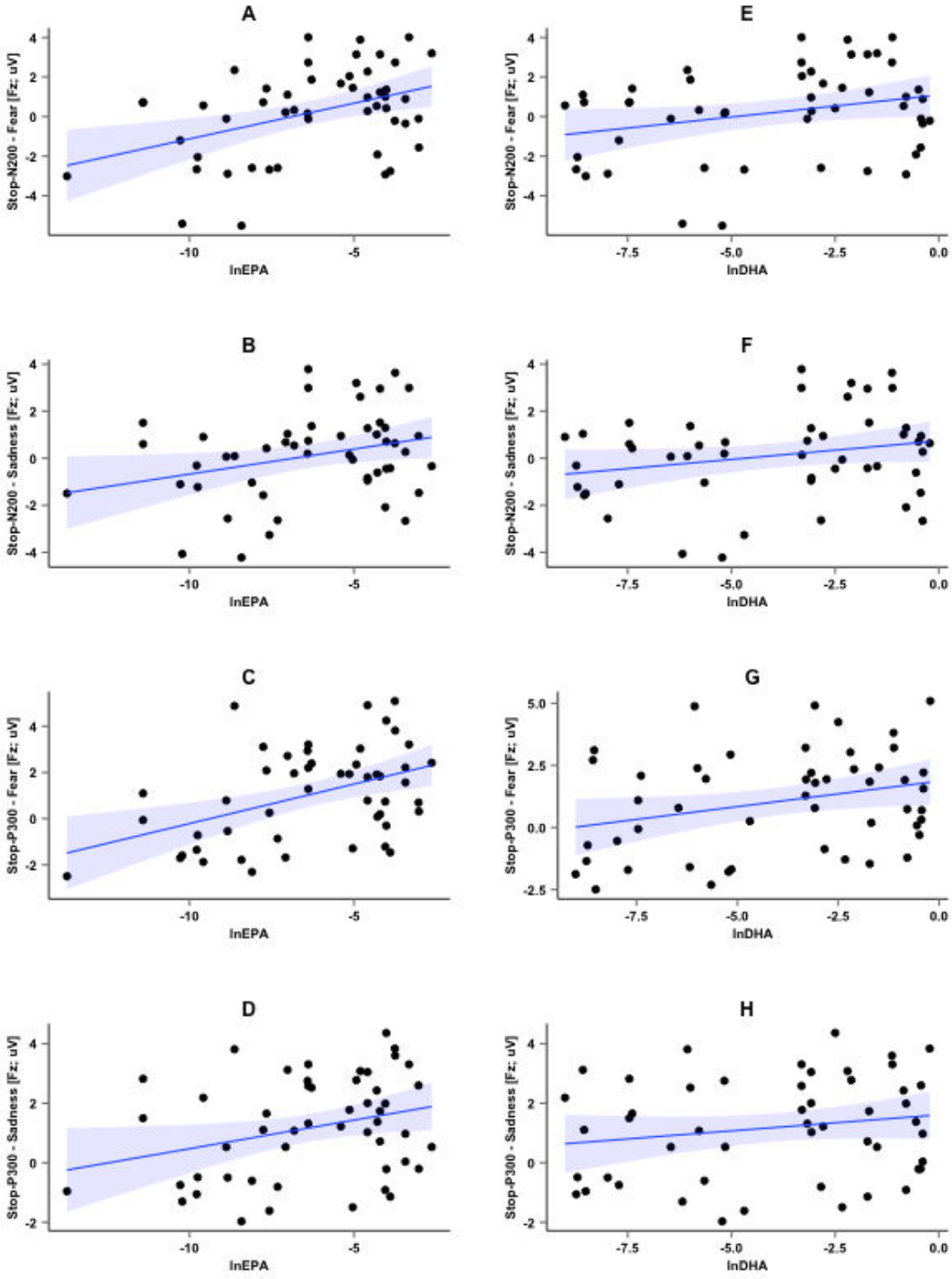

Figure 11. Partial correlations between central-midline stop-N200 and stop-P300 responses to sad and fearful facial expressions, and *ln*-EPA (subplots A to D) and *ln*-DHA intake (subplots E to H). Shaded areas denote 95% confidence interval areas along linear associations.

DHA intake was positively and moderately correlated with stop-N200 and stop-P300 responses to fearful (stop-N200 [r = .30, p = .04, $padj$ = .72), stop-P300 [r = .33, p = .02, $padj$ = .51)], but not sad (stop-N200 [r = .26, p = .07, $padj$ = .87], stop-P300 [r = .21, p = .15, $padj$ = .87]) facial expressions. However no correlations survived multiple comparison correction. Partial correlations describing EPA and DHA intake are evidenced in subplots A to D and E to H of Figure 11, respectively.

11.4 Discussion

Omega-3s EPA and DHA are considered important for healthy development and brain function (Carlson, 2001; Chen & Su, 2012; Conklin et al., 2007, de Velasco et al., 2012). The primary aim of this investigation was to delineate the associations between EPA/DHA consumption and the electrophysiological indices of face processing and distress-induced motor extinction. Results suggest that whilst omega-3 intake did not correlate with either N170 or P200 responses to faces, DHA and EPA were correlated with electrophysiological indices of the preparatory response preceding motor extinction and motor extinction efficiency, respectively. In light of limitations, results are discussed in the context of implications for cognitive and neurobiological models of distress-induced motor extinction, and future investigation.

The primary aim of this investigation was achieved through exploring four key hypotheses. First, because of its role in visual acuity and attention (Birch et al., 2010; Fedorova & Salem, 2006; Uauy et al., 2000), self-reported intake of omega-3 was hypothesised to correlate positively with electrophysiological indices of face processing. However, in this investigation neither N170 nor P200 responses to faces correlated with omega-3 intake. Whilst a single correlation suggested a relationship between EPA intake and the N170 response to angry faces, this did not survive correction for multiple comparisons. Although the second hypothesis proposed omega-3 intake would interact with face processing efficiency equally across facial expressions, the overall absence of

associations between omega-3 intake and N170/P200 responses was unexpected.

Findings presented here suggest that self-reported omega-3 intake over a six-month period does not correlate with ability to process structural and featural aspects of faces. In the absence of previous literature, it remains to be seen whether this effect is restricted to FAST-evoked ERPs, or whether similar results are elicited using other paradigms. As noted in chapter seven, section 7.3.3 (page 118), FAST-evoked N170/P200 responses might be atypical due to their susceptibility to instruction-led voluntary attention (Holmes et al., 2003).

In regards to the electrophysiological indices of motor extinction, DHA was hypothesised to inversely correlate with stop-N200 amplitude, indicative of disrupted synaptic reorganisation (Carlson, 2001; Sumich et al., 2012). The stop-N200 is thought to represent a negatively distributed, preparatory response preceding motor extinction (Hughes et al., 2012). As DHA did not associate with either stop-N200 or stop-P300 amplitudes, results did not support this hypothesis. Unexpectedly, *increased* EPA intake was associated with stop-N200 augmentation. Although this finding suggests EPA intake might be associated with poorer recognition of the need to extinguish a motor response, there is no supporting evidence to reason this association.

Whilst further replication of these results is required in order to assess their validity, it is possible these finding might represent an artefact of the FFQ. Disrupted synaptic reorganisation is thought to occur during child development (Crawford et al., 2003), yet the FFQ only assesses omega-3 intake for the six-months prior to questionnaire completion. As diet can differ throughout life, it is possible that the relationship between omega-3 consumption and stop-N200 amplitude might be better answered through an historical index of omega-3 consumption, or via a longitudinal assay.

As hypothesised, there was a positive correlation between EPA intake and stop-P300 responses to both fearful and sad facial stimuli. Similar associations were observed in regards to DHA, but these did not survive multiple comparison testing. Building on evidence derived from patients with ADHD indicating stop-P300 attenuation (Overtoom et al., 2002) and baseline EPA deficiency (Bekoroglu et al., 1996; Colquhoun & Bunday, 1981; Young et al., 2004, 2005), this is the first investigation to directly correlate omega-3 intake with the stop-P300. Supported by recent investigations suggesting a role of omega-3 in the fluidity of molecular information transmission (Michael-Titus & Priestley, 2014; Serfaty & de Velasco, 2014), the current results suggest that omega-3s might facilitate the mobilisation of neuronal responses resulting in motor extinction.

A clear limitation of this investigation is the lack of analysis between omega-3 intake and [1] the N170/P200 over the right hemisphere, [2] the stop-N200/stop-P300 over parietal sites, and [3] the background N200/P300 over both parietal and anterior sites. These ERPs were omitted from the analysis in order to focus statistical power on ERPs associated with physical aggression/CU traits within this thesis. However, with omega-3 found to associate with P300 responses (at trend-level significance) elsewhere (Gow, Sumich et al., 2013), there is scope for future investigation to fully delineate associations between omega-3 intake and face-evoked ERPs.

In summary, whilst neither self-reported EPA nor DHA related with electrophysiological indices of face processing, EPA, but not DHA, was found to differentially moderate electrophysiological indices of the recognition of inhibitory need and motor extinction efficiency. Discussed in the absence of supporting literature and methodological (i.e. omega-3 assay) limitations, these findings ground future investigation into the role of omega-3 as a treatment intervention for individuals with VIM deficits. However, current results prove this knowledge to be fragmentary, warranting further investigation.

Chapter Twelve: Conclusion

12.1 Introduction

This thesis aimed to [1] explore associations between physical aggression and CU traits in an adult community sample, [2] develop a sensitive experimental paradigm for characterising electrophysiological indices of different processing components of the VIM, [3] explore associations between physical aggression/CU traits and electrophysiological responses to face processing (i.e. N170, P200), and distress-induced motor extinction (i.e. stop-N200, stop–P300), [4] explore associations between omega-3 intake, physical aggression, and CU traits, and [5] explore associations between omega-3 intake and electrophysiological responses to face processing (i.e. N170, P200), and distress-induced motor extinction (i.e. stop-N200, stop–P300).

ERPs have been used to index face processing (N170 [Bentin et al., 1996; Rossion, 2014; Rossion & Jacques, 2011]; P200 [Itier & Taylor, 2004; Latinus & Taylor, 2006]) and motor extinction (stop-N200 [Albrecht et al., 2005; Huster et al., 2013]; stop-P300 [Bae et al., 2011; Bekker et al., 2005; Liotti et al., 2005]). However, to date, this knowledge has not been used to better the understanding of the VIM on an electrophysiological level. Whilst used in some motor extinction paradigms (Meier et al., 2012; Pawliczek et al., 2013), facial affect has not been used as a stopping stimulus, nor has its neural response been indexed via EEG. Prior to this thesis, both the electrophysiological response to distress-induced motor extinction and its relationships with personality traits associated with poor face processing and motor extinction were unknown. Furthermore, although omega-3 has been shown to benefit aggressive behaviour in forensic (Gesch et al., 2002; Zaalberg, 2010), clinical (Hamazaki & Hirayama, 2004; Stevens et al., 2003), and community (Long & Benton, 2013; Raine et al., 2015) samples, this knowledge was previously unexplored in relation to the VIM. This thesis serves as a necessary progression in resolving these gaps in knowledge.

The following sections draw together the key empirical findings of this thesis and show how they systematically combine in order to answer the research questions outlined above. Theoretical implications and the place of these findings within the current literature are discussed, before outlining how these results might advise future policy. Finally, future avenues of research are outlined in order to overcome wider limitations and extend knowledge presented within this thesis.

12.2 Empirical Findings and Theoretical Implication

Empirical findings are discussed within their respective chapters: "*Chapter Six: Correlations between physical aggression & callous-unemotional traits*", "*Chapter Seven: Baseline assessment of the Facial Affect Stop-go Task*", "*Chapter Eight: Correlations between physical aggression/callousness-unemotional traits and face-evoked ERPs*", "*Chapter Nine: Correlations between physical aggression/callous-unemotional traits and motor extinction ERPs*", "*Chapter Ten: Relationships between omega-3 intake, callous-unemotional traits, and physical aggression*", and "*Chapter Eleven: Correlations between omega-3 intake and the electrophysiological indices of the Violence Inhibition Mechanism*". This section draws together key empirical findings and discusses [1] their relevance to answering the main research questions, and [2] their theoretical contributions.

12.2.1 Research Question One

The first research question asked how personality traits associated with poor face processing and motor extinction related to one another within an adult cohort. CU traits were broken down into callousness, uncaring, and unemotional traits, with physical aggression chosen as the key index of aggression due to its greater relevance for reactive aggression and physical harm (Buss & Perry, 1992). Other motivations for and manifestations of aggression were deferred for parsimonious value.

In contrast to previous investigations evidencing greater scores on both physical aggression (Bernstein & Gesn, 1997; Buss & Perry, 1992; Morales-Vives & Vigil-Colet, 2010; Reyna et al., 2011; Williams et al., 1996), and CU traits for males than females, findings of this investigation suggested that males only out-scored females on callous traits. As this finding does not compare to other samples comprising predominantly of students (García-León et al., 2002), it was explained by the low yield of male respondents and so requires further investigation. Furthermore, partial correlations, controlling for age and sex suggested that the relationship between aggression and CU traits might be driven by callousness in adults. This finding differs from those in adolescent samples, where aggression was observed to correlate with all three subscales of the ICU (Fanti et al., 2009; Feilhauer et al., 2012; Guelker et al., 2014; Kimonis et al., 2008). Whilst warranting future age-specific, group-contrasts, this finding has theoretical implication as to how uncaring and unemotional traits might manifest in adulthood and how they relate to aggressive traits and behaviours. It is possible that the ICU is not ideal for measuring these traits in an adult population.

12.2.2 Research Question Two

The second research question asked whether a FAST could be used to accurately index the VIM on an electrophysiological level, and whether associated ERP responses would be comparable to those evoked by typical face presentation and motor inhibition/extinction paradigms. Following three pilot investigations (*see chapter seven, sections 7.2.2 [page 105] and 7.2.3 [page 108] and 7.2.4 [page 112]*), a FAST was developed that succeeded in teasing apart the distinct stages of the VIM. Specifically, indices of structural face encoding (N170) and second-order face processing (P200), as well as motor preparation (stop-N200) and extinction efficiency (stop-P300). For the first time, FAST-evoked ERP responses were delineated.

In line with previous characterisations, electrophysiological indices of face processing were maximal at temporo-parietal sites (N170 [Hinojosa, et al.,

2015; Luck & Kappenman, 2013]; P200 [Farah, 1990; Watanabe, Kakigi, Koyama, & Kirino, 1999]), likely reflecting their neuronal origins in the OFA, STS, and fusiform gyrus (Adolphs, 2002; Itier & Taylor, 2004; Nummenmaa, Hyönä, et al., 2010; Rossion et al., 2003; Sadeh et al., 2010). However, in contrast to expectations, the N170 was augmented to angry facial expressions, and the P200 evidenced augmentation to neutral facial stimuli, but not those depicting facial affect. Previously, both the P200 (Ashley et al., 2004; Eimer et al., 2003; Schulz, 2012), and the N170 in some (Ibáñez et al., 2012; Hinojosa et al., 2015; Smith et al., 2003), but not all (Ashley et al., 2004; Eimer, 2000; Eimer & Holmes, 2002) investigations have evidenced augmentation in response to facial affect.

Findings of this investigation were explained by task instructions orientating the attention of participants to facial affect. Specifically, participants were tasked with evoking a motor response to angry facial expressions (go stimuli) and extinguishing said response to fearful or sad facial expressions (stop stimuli). The observation of anger-specific N170 augmentation is likely an artefact of angry, as opposed to neutral, fearful, or sad facial expressions being target stimuli, and so might reflect a readiness to respond to anger. Previously, affect-driven N170 responses have been observed in tasks where participants were required to attend to the affective elements of facial stimuli (Holmes et al., 2003; Ibánez et al., 2012). It remains to be seen if comparative findings would be observed using other facial expressions as go stimuli. Similarly, the neutral-specific P200 augmentation might reflect participants recruiting greater neuronal resources in an attempt to resolve the ambiguity of neutrality when tasked with attending/responding to facial affect. P200 augmentation to neutral facial expressions has been observed once before in participants tasked with assigning reward, based on facial affect (Tortosa et al., 2013).

In regards to N200 and P300 responses, in line with previous nogo and stop investigations using non-affect stimuli, both the N200 (Enriquez-Geppert et al., 2010; Huster et al., 2013; Liu et al., 2015; Patel & Azzam, 2005) and P300 (De

Jong et al., 1990; Dimoska et al., 2006; Kok et al., 2004) were larger over anterior than parietal sites in response to stop facial-stimuli. Furthermore, whilst neither stop-N200 nor stop-P300 responses varied as a function of emotion, the composite P300 (recorded over anterior-midline sites) was larger in response to fearful than sad facial distress. In accordance with the VIM (Blair, 1995, 2000), this finding likely reflects a predominant effect of the fear due to its biological and social importance (Blair, 2000; Blair et al., 2004; Dadds et al., 2008; Fairchild et al., 2009).

Taken together, the FAST appears a useful tool for indexing the VIM on an electrophysiological level and provides research utility that is likely to remain current as it forms a benchmark for future experimentation and manipulation. Previously, simple face presentation paradigms have facilitated investigation of the N170 and P200 to neutral and affective facial expressions. However, facial expressions, particularly those depicting distress have been overlooked in motor inhibition and motor extinction paradigms. Development of the FAST contributes experimental knowledge to the motor extinction literature and provides a means of measuring electrophysiological responses during distress-induced stopping.

12.2.3 Research Question Three

The third research question asked how physical aggression and CU traits moderated electrophysiological indices of face processing (N170, P200) and distress-induced motor extinction (stop-N200, stop-P300). Building on knowledge obtained in chapters six (from page 90) and seven (from page 102), results suggested that whilst uncaring, but not physical aggression, callousness, or unemotional traits, was associated with left hemispheric N170 (attenuation) and P200 (augmentation) responses during face processing, only physical aggression was associated with stop-N200 (augmentation) and stop-P300 (attenuation) responses during motor extinction.

The inverse association between scores on the uncaring scale of the ICU and the N170 is supported by previous investigation in community samples. Here, structural face processing, as indexed by the N170, was associated with poor emotional expression (Meaux et al., 2014) and fearless dominance (Almeida et al., 2014). In particular, fearless dominance is strongly related to the uncaring subscale of the ICU (Kimonis et al., 2013). Furthermore, in contrast to expectations, after controlling for N170 amplitude, positive correlations were observed between uncaring traits and the P200 response. This finding was explained as reflecting a compensatory mechanism for socially functioning individuals presenting uncaring traits. However, it remains to be seen whether P200 augmentation is abolished in samples presenting higher levels of uncaring traits. The absence of an association between N170/P200 amplitude and physical aggression is also supported by the meta-analysis reported in Dawel et al. (2012) where face processing was associated with affective, but not antisocial components of psychopathy. This is the first investigation to associate electrophysiological indices of face processing with subscales of the ICU and physical aggression.

In regards to electrophysiological indices of motor extinction, the positive correlation between physical aggression and the stop-N200 was thought to reflect increased neuronal activity during motor extinction preparation. In a similar manner to the P200, noted above, as nogo-N200 attenuation has been observed in forensic samples characterised by extreme aggression, it remains to be seen whether comparative stop-N200 attenuation, in response to facial distress, is elicited in samples presenting higher levels of aggressive traits. Moreover, the inverse association between physical aggression and the stop-P300 both supports prior investigations reporting P300 attenuation as a function of aggression (Chen et al., 2005; Costa et al., 2000; Vilà-Balló et al., 2014) and extends these findings into the context of facial-distress stop stimuli.

12.2.4 Research Questions Four and Five

Using the findings from research questions one to three as a benchmark, research questions four and five asked how physical aggression, CU traits, and FAST-evoked ERPs might relate to dietary intake of omega-3. With a growing interest into the benefit of omega-3 for aggressive behaviour (Buydens-Branchey & Branchey, 2008), and recent support for the reduction of aggressive/CU traits as a function of omega-3 supplementation (Raine et al., 2015), answering these research questions has important theoretical and practical utility.

Neither self-reported EPA nor DHA intake correlated with any subscale of the ICU. Previously, CU traits have been shown to inversely correlate with EPA blood content in children with ADHD (Gow, Vallee-Tourangeau et al., 2013). Whilst this disparity might reflect choice of omega-3 assay (i.e. blood content vs. self-report), it is possible that the null finding in the current investigation is explained by statistical analysis controlling for physical aggression. Additionally, physical aggression was negatively correlated with EPA, but not DHA intake over a six-month period. Previously, EPA deficiency has been related to increased aggression in animal (DeMar et al., 2006; Raygada et al., 1998; Re et al., 2008) and human samples (Buydens-Branchey et al., 2006; Umhau et al., 2006), with supplementation of EPA shown to benefit physical/verbal aggression (Zanarini & Frankenburg, 2003), anger (Buydens-Branchey & Branchey, 2008), and violent incidents (Gesch et al., 2002). Although not supplemented by biological measures, these findings have theoretical implications for [1] the use of the FFQ as a quick and inexpensive measurement of omega-3 intake and [2] the importance of disentangling CU and aggressive traits.

On an electrophysiological level, although not correlated with N170 or P200 components, self-reported EPA and DHA intake was positively correlated with both stop-N200 and stop-P300 responses. Combined with evidence presented in chapters ten (from page 169) and nine (from page 159), which delineated

inverse associations between physical aggression and omega-3 intake and electrophysiological indices of motor extinction efficiency, respectively, these latter findings were expected. Understanding how omega-3 might moderate personality traits associated with dysfunctional VIM responses and associated ERPs at baseline provides a theoretical foothold, and so warrants further investigation into the practical utility of omega-3 supplementation on these indices.

12.3 Scientific Implications

Results documented within this thesis pose several scientific implications. First, though contradictory to evidence derived from adolescent samples (Fanti et al., 2009; Feilhauer et al., 2012; Guelker et al., 2014; Kimonis et al., 2008), physical aggressive traits in adults were solely associated with callous, but not uncaring or unemotional traits using comparable measures. Though potentially indicating poor utility of the ICU as a tool for distinguishing CU traits in adult samples, it cannot be ruled out that uncaring and unemotional traits manifest differently in adulthood. With such knowledge seemingly overlooked in the current literature, results reported here warrant further investigation.

Second, although individuals with psychopathic traits exhibit notable dysfunction in activating the VIM (Blair, 1995, 2001; Blair et al., 2004), there is importance, at least on an electrophysiological level in adults drawn from the community, to distinguish between CU and aggressive traits when investigating the different processing stages of the VIM. Such findings build on previous suggestions of trait specificity when investigating face processing proficiency (Dawel et al., 2012). Specifically, results documented within this thesis evidence associations between uncaring traits and a deficient ability to process both structural and configurable aspects of faces, and physically aggressive traits and deficient facial distress-induced motor extinction. In the context of the VIM, whilst CU traits might relate to vulnerability for poor responses to distress-related social cues, and a possible failure to trigger the VIM (Blair, 2005),

aggressive traits might ultimately associate with deficits in distress-induced motor extinction.

Third, this thesis implicates the use of the FFQ as both a time- and cost-efficient means of assessing omega-3 intake. Though limited to an historical assessment of omega-3 intake over a six-month period, results of this thesis suggest specific associations between omega-3 intake and both decreased self-reported physical aggression and increased efficiency of distress-induced motor extinction – a trait/ERP association identified earlier in this thesis. Relationships between omega-3 intake and CU traits/face processing efficacy were not observed. Such findings have importance for understanding the mechanisms relating omega-3 intake to psychopathy-related traits and distinct processing stages of the VIM. Furthermore, findings supplement the growing interest into how omega-3 might be used to benefit and/or alleviate psychopathy-related traits.

12.4 Recommendation for Future Research and Limitations

Although specific experiment-by-experiment limitations have been discussed throughout this thesis, it is important to consider the experimental results in light of wider limitations and future avenues of research. This section discusses '*sample*', '*diet*', and '*statistical*' limitations.

12.4.1 Sample

Experiments reported in this thesis predominantly sampled from the student population. Whilst important to understand the effect of physical aggression and CU traits on distress-induced motor extinction, student-biased samples might limit the spectrum of psychometric responses. As documented in chapters six (from page 90) and ten (from page 169), although psychometric measures were normally distributed, they were centred on a low mean, and so would benefit from an increased number of data points across the response spectrum. Such sampling methods would have additional value for facilitating the ability to

contrast high- and low- scoring subgroups in order to negate the limitations of correlational analysis (i.e. the inability to infer causation). In this thesis, data predominantly underwent partial correlational analysis. Moreover, collapsing individuals into subgroups would reduce between-subject variation - further benefiting group comparisons (Luck, 2005).

Furthermore, sex was not equally distributed within the samples and so, in the most part, sex-based comparisons could not reliably be observed. Although bivariate correlations as a function of sex were observed in chapter six (from page 90), this decision was made because [1] it was an initial baseline investigation, [2] the overall sample size was sufficiently large enough to warrant this comparison, and [3] unlike other investigations in this thesis, this particular investigation was not saturated in terms of multiple comparisons. However, in acknowledgment of the importance of sex differences in the manifestation of aggression and CU traits, where possible, investigations within this thesis sought to control for the variation in sex. Clearly, it is important that future investigations into this area acknowledge sex differences.

12.4.2 Reported Diet

This thesis is also limited to the assumptions formed around diet. First, associations between omega-3 intake and psychometric/electrophysiological measures are formed from self-reports that index omega-3 over a six-month period. Whereas blood assays of omega-3 have been associated with aggressive (Meyer et al., 2015) and CU (Gow, Vallee-Tourangeau et al., 2013) traits, which might vary over time (Barratt, 1991; Raine, 2013), it is difficult to accurately compare current levels of omega-3 using questionnaire methods. Furthermore, as reported in chapter four (from page 63), omega-3 plays a vital role in brain maturation during embryonic growth and early years of life (Carlson, 2001; Crawford et al., 2003; Riemer et al., 2010). It is possible that associations between omega-3 intake and electrophysiological indices of face processing, not observed in chapter eleven (from page 177), might be better indexed through historical/longitudinal assessments.

Second, the FFQ has not previously been validated in non-major depressive populations (Sublette et al., 2011), or as a function of cognitive ability and other social/developmental variables, and so might not be stable in samples of young adults derived from the community. In light of this limitation, although a quick and cost-effective assay of omega-3 intake, which has been justified in chapter five, section 5.3.3 (page 87), blood assays would likely provide a more reliable and robust measure of omega-3. However, it is important to note that the collection of blood is time consuming and expensive to analyse, especially in the context of investigations requiring large sample sizes.

12.4.3 Statistical Considerations

Finally, it is important to contextualise the results of this thesis in light of statistical considerations. First, as documented in chapters ten (from page 169) and eleven (from page 177), EPA and DHA consumption were not normally distributed. Although mediated through logarithmic transformation, skewed distributions suggest homogeneous fish consumption. Whilst homogeneity might ultimately prevent the accurate delineation of subtle relationships, the survival of an observed inverse association between EPA intake and physical aggression suggests enough variation within the data was present to capture this association. However, in order to more accurately explore associations between omega-3 intake and psychometric traits/ERP responses, future investigation would benefit from sampling individuals with a wider variety of fish consumption.

Second, as mentioned in section 12.4.1 (page 193), conclusions of this thesis are predominantly drawn form correlational analyses, which prevent the inference of causation. In relation to dietary intake of omega-3, specifically, group analysis has benefit for understanding differences in VIM-related personality traits and electrophysiological responses between high and low omega-3 consumers. Furthermore, experimental manipulation in the form of omega-3 supplementation would provide an effective measure of the direct

impact of omega-3 on both psychometric measures of physical aggression/CU traits and FAST-evoked ERPs. With omega-3 supplementation previously shown to reduce parent-, teacher-, and self-reported aggression, as well as incidents of aggressive behaviour (Gesch et al., 2002; Hamazaki & Hirayama, 2004; Stevens et al., 2003; Zaalberg et al., 2010), omega-3/ERP associations observed in this thesis suggest that supplementation of omega-3 would likely augment stop-N200 and stop-P300 responses to facial distress.

Third, although this thesis has focused on trait scores of physical aggression as a function of its relationship to reactive aggression and physical harm (Buss & Perry, 1992), this decision partly reflects consideration of statistical power. Future investigations should seek to investigate other manifestations of aggression (e.g. verbal and indirect aggression) and associated traits (e.g. anger and hostility) in a similar manner, or in contrast to one another using a larger sample. As aggression can be characterised in a multitude of ways (Barratt et al., 1997, *but see chapter one for review*), it is important to understand how such characterisations might differentially relate to CU traits, omega-3 intake, and electrophysiological indices of the VIM.

12.5 Policy Implication

Whilst expanding theoretical and scientific knowledge, this thesis has additional relevance for policy consideration. Currently, in England and Wales, each of the forty-two criminal justice areas are required to establish a Multi-Agency Public Protection Arrangement (MAPPA) in order to better protect the public from violent offenders (Criminal Justice Act 2003, s. 325). Specifically, whilst violent behaviour is managed, and so reduced, as a function of social, financial, and educational teams working alongside criminal justice agencies, distinct contributions from food agencies are absent (MAPPA Guidelines 2012, s. 3.7). Though correlational in nature, findings reported in this thesis, alongside others reviewed in chapter four, section 4.3.2 [*from page 68*]) suggest a potential benefit of omega-3 for scores on psychometric measures of physical aggression and aggressive-related behaviours.

Within forensic settings, whilst diets are required to be healthy, balanced, low in saturated fat, sugar, and salt, and of sufficient fruit and fibre (Catering: Meals for Prisoners [England] Prison Service Instruction 2010 No. 44) they place only little importance on omega-3 consumption. Currently, the Catering: Meals for Prisoners instruction only requires two portions of fish, with only one comprising omega-3-rich oily fish, to be served per week. Disregarding other dietary sources of omega-3 (e.g. seeds, grains, or leafy vegetables, *see chapter four, section 4.3.1 [page 64]*) or menu fluctuations across institutions and seasons, this level of omega-3 consumption is far lower than the daily recommended 0.25 to 3 g (Food and Agriculture Organisation, 2010).

Taken together, these results suggest possible practical implication for the use of omega-3 as a preventative or treatment measure for physical aggression, but perhaps not CU traits. However, such utility requires more rigorous testing, over additional assays of aggression, and biologically robust measures of omega-3. Although omega-3 intake has recently been explored as having important public health ramifications (Beier et al., 2014), since this and other investigations only support a reduction, and not an abolition of aggression, it is important that future investigation of the benefit of omega-3 considers it alongside wider treatment strategies.

12.6 Thesis Conclusion

In spite of the wealth of investigation outlining traits associated with atypical activation of the VIM - and their benefit from an omega-3-rich diet - empirical research has overlooked the investigation of these associations on an electrophysiological level. The VIM comprises distinct stages of face processing and distress-induced motor extinction. For the first time, this thesis provides a means to tease apart the various stages of the VIM using EEG. Key findings suggest that whilst uncaring traits associate with electrophysiological indices of structural/featural face processing, physical aggression associates with electrophysiological indices of distress-induced motor extinction. Moreover, in light of a growing interest in using omega-3 as a moderator of aggressive and

CU traits, self-reported omega-3 intake was found to correlate with physical aggression/distress-induced motor extinction, but not CU traits/face processing.

Together, these results signify the importance for not only distinguishing between aggressive and CU traits, but for discussing VIM dysfunction in terms of distinct processing stages. Discussed in the context of its limitations and wider application to both theoretical knowledge and policy, this thesis provides a strong foothold for future experimentation in order to better establish its position within the psychological and forensic literature.

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