Don't slap the fish: The relationship between dietary Omega-3 intake and physical aggression is mediated by motor inhibition in response to distressed faces

Abstract

The innate violence inhibition mechanism (VIM) purportedly regulates maladaptive aggressive behavior through motor inhibition, in response to expressions of distress, and is implicated in psychopathy-related aggression. Deficiency in eicosapentaenoic acid (EPA; an omega-3 fatty acid) is implicated in aggression and callous-unemotional (CU) traits, however, its relationship to the VIM remains unknown. Two studies tested relationships between EPA intake, personality (aggression, CU traits), and electrophysiological indices of the VIM. In study one (N=98), participants completed omega-3 intake (FFQ), CU traits (ICU), and aggression (BPAQ) measures. Physical aggression correlated positively with callousness and negatively with EPA intake. CU traits were unrelated to EPA. In study two (N=47), participants completed the same measures and an electroencephalography assessment of VIM. Stop-P300 amplitude (motor inhibition success) in response to facial expressions of distress mediated the relationship between EPA intake and physical aggression. This is the first demonstration of an association between EPA intake and electroencephalographic indices of the VIM. Findings support a role of EPA in regulating aggression through networks involved in distress-cued executive control over behaviour; and provide supporting data to direct future trial designs for nutritional supplementation in non-clinical, clinical and forensic arenas.

Key words: *Omega-3 fatty acids; event-related potentials; eicosapentaenoic acid; callous-unemotional traits; aggression; FAST task*

1 Introduction

Blair (1995; 2001) proposed an innate violence inhibition mechanism (VIM) that regulates maladaptive aggressive behavior in psychpoathy. Various subtypes of anti-social behavior (e.g., detached vs disinhibited; McKinley et al., 2018) may be differentiated by deficits in distinct VIM processing stages, such as the initial empathic response to facial distress (Dawel et al., 2012; Marsh & Blair, 2008; Wilson et al., 2011) and/or subsequent motor inhibition (Robinson & Bresin, 2014). Study of these stages in relation to callous-unemotional (CU) and aggressive traits in the general population would offer insight into biological mechanisms underpinning psychopathy, in the absence of epiphenomena associated with a criminal lifestyle (Centifanti et al., 2016; Essau, Sasagawa, & Frick, 2006; Frick et al., 2000). Moreover, deficiency in eicosapentaenoic acid (EPA) is implicated in both, aggression (Fedorova & Salem Jr., 2006) and callous-unemotional (CU) traits (Gow, Vallee-Tourangeau et al., 2013) and may impact VIM functioning. Nevertheless, direct associations between EPA dietary intake and brain mechanisms underpinning the VIM remain uninvestigated. Therefore, this paper aims to understand the relationship between EPA dietary intake and neurocognitive mechanisms underpinning aggressive behavior from a VIM framework. To this end, specific objectives are to test: (i) associations between EPA dietary intake, self-report psychometric assessments aggression and CU traits, (ii) associations between EPA dietary intake and brain mechanisms implicated in VIM, and (iii) whether implicated brain mechanisms mediate the relationship between EPA dietary intake and maladaptive traits (aggressive behavior, ICU).

1.1 Omega-3 within the context of the VIM

Insufficient intake of long-chain omega-3 polyunsaturated fatty acids (*n*-3 PUFAs) is implicated in violence and maladaptive aggressive behavior (Fedorova & Salem Jr., 2006). For example, blood levels of *n*-3 PUFA eicosapentaenoic acid (EPA, 20:5*n*-3) either alone, or alongside docosahexaenoic acid (DHA, 22:6*n*-3), have been inversely associated with self-report measures of aggression in adults (Beier et al., 2014; Meyer et al., 2015; Zaalberg et al., 2016) and CU traits in children with attention deficit hyperactivity disorder (ADHD; Gow, Vallee-Tourangeau et al., 2013). Dietary supplementation with EPA reduces physical aggression in people with borderline personality disorder (Zanarini & Frankenburg, 2003), anger in substance abusers (Buydens-Branchey & Branchey, 2008), reactive and proactive aggression in children (Raine et al., 2015), and violent, rule-breaking behavior in forensic populations (Gesch et al., 2002; Zaalberg et al., 2010). Although some inconsistent findings are reported in studies of DHA supplementation alone (Hirayama et al., 2004; Voigt et al., 2001). Taken together, findings suggest a role for EPA in regulating brain mechanisms implicated in psychopathy-related personality traits and behaviours.

1.2 Brain mechanisms in aggressive and CU traits

Electroencephalography (EEG) and event-related potentials (ERPs) have been used to investigate brain function in relation to *n*-3 intake in ADHD (Sumich et al., 2009), as well as CU traits and aggression in developmental (adolescents; Sumich et al., 2012), forensic (adult; Bernat, Hall, Stefan, & Patrick, 2007) and non-forensic (adult; Fido et al., 2017) populations. For example, the N170 amplitude (a negative EEG deflection that peaks 150-200ms post-stimulus at bilateral occipito-parietal sites) has been shown to be responsive to facial expression of threat and distress (Hinojosa et al., 2015), and is negatively associated with uncaring and fearless

dominance traits (Almeida et al., 2014; Meaux et al., 2014); though, positive correlations have been observed for other traits associated with psychopathy, such as cold-heartedness (Almeida et al., 2014). Reduced P300 amplitude (a positive deflection in the ERP that peaks 300-500ms post-stimulus with a widespread scalp distribution and parietal maxima; Hajcak, Weinberg, Macnamara, & Foti, 2013; Luck, 2005) has been associated with a range of externalizing disorders, including substance abuse and reactive aggression (Bernat et al., 2007; Hicks et al., 2007). During Stop and NoGo tasks, P300 maxima shows an anterior shift, reflecting the recruitment of frontal executive networks (Sumich et al., 2008). Reduced P300 amplitude during motor inhibition is seen in delinquent men with ADHD (Meier, Perrig, & Koenig, 2012) and in relation to psychopathic traits (Kim & Jung, 2014).

Fido et al., (2017) investigated distinct processing stages of the VIM using a Facial Affect Stop-Go Task (FAST). Adults recruited from the community respond to angry faces (Go stimuli) and extinguish their responses to STOP stimuli, expressions of distress (fear, sadness). As such, it can be mapped onto the distinct VIM processing stages (initial empathic response to facial distress, subsequent motor inhibition). The N170 response to sad and neutral stimuli were negatively associated with uncaring CU traits (measured by the Inventory of Callous-Unemotional Traits (Frick, 2003)) but not with physical aggression. The STOP-P300 amplitude was inversely associated with physical aggression but not CU traits (Fido et al., 2017). These findings support the idea that the N170 in response to facial affect reflects mechanisms underpinning an affective response (e.g., initial empathic response to facial distress), whilst the Stop-P300 indexes the executive ability required to alter behavior (e.g., subsequent motor inhibition) - mirroring processes underpinning the VIM.

To date, limited investigation exists into the relationship between *n*-3 and ERPs to face processing and motor inhibition (Fontani et al., 2005; Gow et al., 2009). Blood levels of EPA were inversely associated with N170 amplitude to sad faces (Gow et al., 2009), whilst P300 in responses to NoGo stimuli increased following *n*-3 PUFA supplementation (Fontani et al., 2005). To our knowledge, no investigation has explored associations between EPA and motor inhibition cued by facial affect.

1.3 The Current Research

This manuscript documents two studies using independent samples from the general population. Study one investigated self-reported EPA intake (henceforth referred to solely as EPA intake) in relation to aggression and CU traits. Here, EPA intake was hypothesised to negatively correlate with both aggressive and CU traits. Study two investigated whether electrophysiological indices of VIM processing stages mediated relationships between EPA intake and i) physical aggression and/or ii) CU traits. All study protocols were approved by an institutional ethics committee.

2 Study One

2.1 Methods

2.1.1 Participants

To determine our target sample size, we conducted an a priori power analysis using G*Power (version 3.1.9.2). Due to an absence of existing cross-sectional research using our target measures, we assumed a conservative medium effect size (R^2=.40) and a standard alpha level of .05, which indicated that a minimum of 63 participants were required for 95% power.

Ninety-eight participants (aged 21.47 ± 3.07 years, 89% female) responded to an online advertisement distributed across social, professional, and institutional networks. Inclusion criteria required participants to be fluent in English, aged 18 years or over, not currently taking any dietary supplements, and without any diagnosed psychiatric or neurological disorder. Participants provided written informed consent in accordance with approved central university research protocols and national guidelines.

2.1.2 Materials

Reactive physical aggression was measured using the subscale of the Buss-Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992). The BPAQ comprises 34 items using a 5-point scale. The physical aggression subscale comprises 9 items (e.g., "If somebody hits me, I hit back"). Each item is rated using a scale anchored from "*uncharacteristic of me*" to "*very characteristic of me*" with higher scores indicating greater aggression. As recommended by Buss and Perry (1992), aggression scores were *t*-transformed as a function of age and sex.

Callous–Unemotional Traits were measured using Frick (2003)'s inventory (ICU) which comprises 24 items, assessing the occurrence and intensity of callous (11 items; e.g., "I do not care who I hurt to get what I want"), uncaring (8 items; e.g., "I try not to hurt others' feelings"), and unemotional (5 items; e.g., "I hide my feelings from others") traits. Each item is rated on a 4-point scale anchored from "*not at all true*" to "*definitely true*" with higher scores indicative of greater levels of CU traits. Although developed for use within adolescents, the ICU has been validated for use in adult samples (Byrd, Kahn, & Pardini, 2013; Kimonis, Branch, Hagman, Graham, & Miller, 2013).

EPA intake was measured using Sublette et al., (2011)'s Food Frequency Questionnaire (FFQ) that comprises 21 items. Self-reported EPA scores are significantly correlated with blood plasma measures of EPA (r=.47; Sublette, 2011). EPA intake was measured in milligrams (mg) per day, and calculated as a function of the sex of the responder, as well as fish type (e.g., salmon, sardines, tuna), portion sizes (i.e., < 2 ounces, 2-7 ounces, > 7 ounces), and frequency (e.g., 1 time each month, 2 times each week, 1 time each day) consumed over the previous 6-month period. The FFQ also documents consumption of nuts, seeds, and oils that contain EPA.

2.1.3 Procedure

Participants were presented with the BPAQ, ICU, and FFQ in a randomised order through online survey software to reduce the likelihood of order effects influencing the data.

2.1.4 Statistical Analysis

One data point (one item for one participant) was missing, and so was replaced with the sample mean. Moreover, there was no indication of the presence of response biases. Pearson correlations were computed between psychometrics (i.e., physical aggression, callousness, uncaring, and unemotional traits) and daily intake of EPA. For correlations of interest (i.e., EPA intake-related), Pearson's partial correlations were computed; controlling for age and sex (demeaned). A Benjamini-Hochberg correction was used to adjust for multiple comparisons (Benjamini & Hochberg, 1995).

2.2 Results

Means, standard deviations, Cronbach's alpha coefficients, and bivariate correlations for psychometric measures and EPA intake are displayed in Table 1. Due to low self-reported EPA consumption in this sample, this data was positively skewed (z-skew=14.28, z-kurtosis=27.29), and so underwent *ln*-transformation (Beier et al., 2014).

[Please place table 1 about here]

Callousness was positively correlated with both physical aggression (p_{adj}=.034) and uncaring traits (p_{adj} < .001). Uncaring traits were positively correlated with unemotional traits (p_{adj} =.020). There was a negative correlation between physical aggression and EPA intake (p_{adj} < .001), and this was confirmed by a partial correlation (r=-.423, p < .001) controlling for covariates age and sex (*see* Figure 1). EPA did not correlate with any subscale of the ICU.

[Please place figure 1 about here]

3. Study Two

3.1 Methods

3.1.1 Participants

Forty-seven participants (aged 18.96 ± 1.22 years, 60% female) were subsampled from the cohort of 54 reported in [REMOVED FOR REVIEW]. Five participants were excluded due to self-reported *n*-3 supplementation within the previous six-months, and a further two were excluded due to incomplete datasets. Inclusion criteria required participants to be right-handed,

aged 18 years and over, and without any diagnosed psychiatric or neurological disorders, or use of medication that might impact electrophysiology.

3.1.2 Materials

Before completing the FAST, participants completed measures of EPA intake (FFQ; Sublette et al., 2011), physical aggression (BPAQ; Buss & Perry, 1992), and CU traits (ICU; Frick, 2003) as described in Study 1. In addition, socioeconomic status and intake of alcohol and cannabis were also assessed as control variables.

Socioeconomic status was measured using the Barratt Simplified Measure of Social Status (BSMSS; Barratt, 2006), a two-section proxy measure of socioeconomic status, which combines data on educational attainment and family occupation. Participants are required to specify the current occupation of, and level of education completed by, their mother, father, spouse, and self, respectively. Each choice is weighted accordingly, with higher scores (range of 8 to 66) indicative of higher socioeconomic status. Scoring for this scale is adjusted as a function of growing up in a single parent family, living alone, and/or being a student.

Alcohol use was measured using the Alcohol Use Disorder Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1992), which comprises 10 items that assess one's quantity (e.g., "How many drinks containing alcohol do you have on a typical day when you are drinking?") and frequency (e.g., "How often do you have a drink containing alcohol?") of alcohol consumption, as well as problems caused by alcohol (e.g., "Have you or someone else been injured because of your drinking?"). Each item is rated on a 5-point scale with high scores indicative of greater use of, and problems associated with alcohol.

Cannabis use was measured using the Cannabis Use Disorder Identification Test (CUDIT; Adamson & Sellman, 2003), which comprises 10 items that assess one's quantity and frequency of cannabis use, as well as problems caused by cannabis over the last six months. Each item is modified from the AUDIT, with references to 'drinks containing alcohol' replaced with 'cannabis' (e.g., How often do you use cannabis?"). Items are rated on a 5-point scale with high scores indicative of greater use of, and problems associated with cannabis.

3.1.3. Event-related potentials

The Facial Affect Stop-Go Task (FAST; Fido et al., 2017) is an experimental paradigm designed to simultaneously investigate cognitive mechanisms underpinning VIM stages of face processing and motor inhibition. Participants were presented with faces that varied in expression (e.g., fearful, sad, neutral, or angry faces; duration=800 ± 100ms), followed by a black screen (duration 160 ± 40ms). They were asked to move their right index finger from a red button box key to an adjacent green key as soon as the black screen appeared, if it was preceded by an angry face (Go stimulus). However, participants were asked to interrupt this response (i.e., by returning their finger to the red key) if a fearful or sad face (Stop stimulus) appeared before the GO response was completed. No behavioural response was required to neutral faces.

The paradigm was presented in two blocks using OpenSesame (version 3.0). Each block began with a 4000ms lead-in, followed by 136 trials. A red fixation cross separated each trial (1800 ± 200ms). Stimuli consisted of open-mouthed expressions to increase the intensity and clarity of the emotion presented (IDs 01, 03, 05, 06, 07, 08, 09, 10, 20, 21, 23, 25, 26, 32, 34, 35, 36; MacBrain NimStim Face Stimulus Set; Tottenham et al., 2009). Further information regarding the FAST, as well as an example trial can be found in Fido et al. (2017).

3.1.3 Procedure

Participants completed the BPAQ, ICU, FFQ, BSMSS, AUDIT, and CUDIT in a randomised order to reduce the likelihood of order effects influencing the data. On average, the survey measures took less than 15 minutes to complete. Afterwards, participants were fitted with the EEG cap before completing the FAST which, on average, took 20 minutes to complete.

3.1.4 EEG recording and signal processing

EEG was recorded using an active-electrode, 64-channel Active-Two acquisition system and ActiView v.6.05 software (BioSemi, Amsterdam, Netherlands), sampled at 2048 Hz and digitised at 24-bits.

The Matlab toolbox EEGLAB (v.13.6.5b) was used to correct electrooculography artefacts (Jung et al., 2000) and to apply a band-pass filter of 0.01–0.35 Hz. Trials were baseline corrected before averaging (-200ms). The N170 was average-referenced to avoid ERP attenuation at temporal-parietal sites (Joyce & Rossion, 2005) and the P300 was re-referenced to linked mastoids to minimize spatial distortion (Luck, 2005). Averaged ERP amplitudes were calculated across posterior (P7, PO7, O1, PO3, P8, PO8, O2, PO4) sites for N170 (130-200ms post-stimulus) evoked to fearful, sad, neutral, and angry facial expressions, and at anterior midline (Fz) for Stop-P300 (300-450ms post-stimulus, successful trials only) to fearful and sad facial expressions. To facilitate comprehension when discussing findings, additive inverse values were used for the N170 (e.g., more negative N170 values are discussed as being ‘higher’).

3.1.5 Statistical Analysis

Mean ERP amplitudes for each trial type were calculated. Pearson correlations were computed between EPA intake, VIM indices (i.e., N170 responses to fearful, sad, angry, and neutral facial expressions; Stop-P300 responses to fearful and sad facial expressions), and personality (i.e., physical aggression and callous, uncaring, and unemotional traits). Further, Pearson correlations were computed between EPA intake and behavioral responses (i.e., reaction time in successful trials, accuracy). Benjamini-Hochberg correction accounted for multiple comparisons (Benjamini & Hochberg, 1995). To determine any indirect effects of EPA intake on aggressive and CU traits, through ERP responses, the PROCESS procedure was used to test mediation (Hayes, 2018, model type 4). Socioeconomic status, as well as use of alcohol and cannabis were modelled as covariates. All *Beta* values reported are unstandardised as per Hayes' (2018) recommendations.

3.2 Results

As with study one, mean EPA consumption data (.01 ± .02 g) was positively skewed (*z*-skew=5.91, *z*-kurtosis=6.81) and so underwent *ln*-transformation prior to analysis (-5.98 ± 2.54 g).

[Please place figure 2 about here]

3.2.1 Behavioral Response

There was a positive association between motor inhibition success in response to fearful facial expressions (M=65.71 ± 15.92%) and dietary intake of EPA (r [45]=.39, p=.007, p_{adj}

=.028) (*see* Figure 2). This association was confirmed with a partial correlation (*r*=.36, *p*=.015) controlling for potential covariates age and sex (demeaned). Intake of EPA did not significantly correlate with motor inhibition success to sad facial expressions (77.97 ± 13.34%; *r* [45]=.06, *p*=.674) nor motor inhibition reaction times to sad (733.61 ± 189.44ms; *r* [45]=-.11, *p*=.460) or fearful (788.48 ± 225.86ms; *r* [45]=-.10, *p*=.499) facial expressions.

3.2.2 ERPs

Preliminary analysis in the form of bivariate correlations revealed no statistically significant correlations between EPA intake and the N170 response to fearful (*r* [45]= -.10, *p*=.490), sad (*r* [45]= -.17, *p*=.262), angry (*r* [45]=-.10, *p*=.512), or neutral (*r* [45]= -.08, *p*=.576) facial stimuli. EPA intake showed a negative relationship with physical aggression (*r* [45]= -.35, *p*=.015, p_{adj} =.050), but there was no association with callous (*r* [45]= -.12, *p*=.905), uncaring (*r* [45]= -.15, *p*=.313), or unemotional traits (*r* [45]= -.20, *p*=.176). As such, mediation analysis was limited to the association between EPA intake and physical aggression using only the Stop-P300 responses to fearful and sad facial expressions as mediator variables.

[Please place figure 3 about here]

EPA intake was negatively associated with physical aggression (*B*= -1.66, *SE*=.41, *t*(42)= -4.08, $p < .001$, 95%CI [-2.49, -.84]) and positively associated with the Stop-P300 response to both fearful (*B*=.47, *SE*=.15, *t*(42)=3.14, *p*=.003, 95% CI [.17, .77]) and sad (*B*=.53, *SE*=.13, *t*(42)=4.16, $p < .001$, 95% CI [.27, .78]) facial expressions (*see* Figure 3). Moreover, Stop-P300 responses to fear (*B*= -.69, *SE*=.34, *t*(40)= -2.04, *p*=.049, 95% CI [-1.38, -.00]) and sadness (*B*= -

.92, *SE*=.45, *t*(40)= -2.06, *p*=.046, 95% CI [-1.83, -.02]) were inversely associated with physical aggression, with the direct inverse relationship between EPA intake and physical aggression rendered non-significant (*B*= -.86, *SE*=.54, *t*(40)= -1.59, *p*=.119, 95% CI [-1.94, .23]). What this indicates is that variation in the Stop-300 response to distress may mediate the relationship between EPA intake and physical aggression (*see* Figure 4). We could not, however, separate out the independent contributions of the indirect effects of the Stop-P300 response to fear and sadness (*B*= -.16, *SE*=.42, 95%CI [-.91, .78], 5,000 bootstrap resamples). Although the inclusion of socioeconomic status as a covariate was associated with a statistically significant decrease in physical aggression in this model (*B*= -.18, *SE*=.08, *t*(40)= -2.26, *p*=.030, 95%CI [-.35, -.02]), the overall effect remained significant (R^2=.39, *F*(6, 40)=11.70, $p < .001$). The covariates of alcohol and cannabis use were not statistically significant. Grand averaged Stop-P300 ERPs to distress, as a function of EPA intake can be seen in Figure 5.

[Please place figure 4 and 5 about here]

4. Discussion

Using both cross-sectional online sampling and laboratory-based designs, self-reported dietary intake of EPA was investigated in relation to physical trait aggression and CU traits, and to electrophysiological indices of VIM stages: face processing and motor inhibition. EPA intake was consistently found to correlate with reduced physical aggression, with this association possibly mediated by a positive association between EPA-intake and Stop-P300 amplitude

315 (indicative of motor inhibition proficiency to distress). EPA intake was neither associated with
316 CU traits, nor N170 responses to facial affect.

317 Relationships between aggression and CU traits are well established (Frick et al., 2003;
318 Frick & White, 2008), yet their independent associations with EPA remained unclear. As
319 expected, findings of study one revealed an inverse association between intake of EPA and
320 physical aggression. Previously, negative associations between physical aggression and *n*-3
321 intake have been observed in male offenders (Meyer et al., 2015), and physical aggression has
322 been shown to decrease following a two-month intervention of EPA supplementation in females
323 with borderline personality disorder (Zanarini & Frankenburg, 2003). As such, our findings add
324 to an emerging literature, which defines a role of EPA in behavioural and trait aggression.

325 Given the absence of any significant correlation between EPA intake and CU traits,
326 current findings are not in line with previous reports of inverse associations between EPA blood
327 concentrations and CU traits in boys with ADHD (Gow, Vallee-Tourangeau et al., 2013).
328 However, they do concur with intervention studies, which suggest no effect of *n*-3
329 supplementation on CU traits in children with conduct disorder (Raine et al., 2016; Raine et al.,
330 2015). Disparity of results might be explained by differences in quantifying EPA intake and
331 sample characteristics (e.g., age, comorbidity of ADHD, and/or conduct disorder
332 symptomology). Thus, further investigation is warranted to explore this association across
333 heterogeneous community, clinical, and forensic samples.

334 In study two, electrophysiological indices of VIM were investigated as mediators of the
335 relationships between self-reported EPA intake and both physical aggression and CU traits. As
336 intake of EPA was neither associated with callous, uncaring, or unemotional traits, nor N170
337 responses (irrespective of facial expression), mediation analysis was constrained to the

association between EPA intake and physical aggression through Stop-P300 amplitude in response to distress. As expected, Stop-P300 amplitude in response to fearful and sad facial expressions mediated the negative association between EPA intake and physical aggression. Although the use of mediation analysis has been used in similar-sized samples, larger sample sizes would help validate some of the smaller (in)direct effects observed in this report (*see* Fritz & MacKinnon, 2007). Moreover, this data is correlational and so cannot rule out the possibility that individuals with higher trait aggression (and associated variation in ERPs) may simply consume less EPA. Although causation cannot be directly inferred, our results build on the work of Fontani et al. (2005), which found increases in the P300 amplitude to NoGo geometric shapes following EPA-rich supplementation. Together, these findings support a potential role of EPA in motor inhibition, but future studies should confirm these through larger cohort and intervention studies using the FAST.

EPA did not correlate with face-evoked N170 responses. Thus, EPA may not to be involved in the initial face processing stage of the VIM as modelled here. However, the findings contrast a report on EEG evoked to facial stimuli in adolescents with ADHD, in which Gow et al. (2009) found a negative trend between blood levels of EPA and N170 amplitude to sad faces. Further research should investigate whether the disparity between this and the current findings is a function of psychopathology (e.g., general population vs. ADHD), age (e.g., adults vs. children), and/or task (e.g., responding to target vs. passive viewing of facial stimuli).

Although the FFQ is an indirect self-report measure of EPA intake through primarily oily fish consumption, it has been shown to correlate with plasma levels of EPA (Sublette et al., 2011). Nevertheless, blood measurements would enable a more accurate quantification of current EPA levels in light of physical characteristics, such as height and weight. On the other hand, a

measure of EPA consumption over a six-month period (as reported in the FFQ) may be less sensitive to acute fluctuations and provide a better average estimate of EPA intake over time. Moreover, an historical index of EPA consumption or longitudinal assessment may be beneficial given that synaptic reorganisation occurs particularly during child development (Crawford et al., 2003).

An index of DHA consumption was not reported in this investigation due to [i] previous findings indicating a predominant role of EPA in the manifestation of psychopathy-related traits, and [ii] high multicollinearity between DHA and EPA intake preventing precise delineation of their independent contribution. In addition, it should be noted that oily fish (consumption of which measured by the FFQ) contains several micronutrients implicated in modulation of brain function and aggressive behavior (e.g., Tryptophan, Magnesium, vitamin D; e.g., Zaalberg et al., 2016). In future research, relative contribution of these micronutrient would be better delineated using a combination of current and historical intake indices, and blood measures.

4.1 Conclusion

The current study is the first to identify an association between EPA intake and EEG-measured motor inhibition proficiency to facial stimuli. Results suggest EPA intake is associated with lower physical aggression and higher Stop-P300 amplitude (thought to map onto the motor inhibition VIM stage), but not callous, unemotional, or uncaring traits or N170. As such, the findings suggest a role of EPA in executive control over behavior, as cued by affective stimuli, rather than the initial encoding of the emotional content of the face. It might therefore be expected that EPA supplementation would be more effective for disinhibited, as compared to

383 detached, subtypes of antisocial personality disorder, and this should be investigated further in
384 future research.

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Tables

Table 1. Intercorrelations, means, and standard deviations for psychometric measures and EPA consumption

	α	M	SD	1	2	3	4	5
1 Physical Aggression	.81	48.7	9.53	-				
2 Callousness	.72	4.14	3.34	.25*	-			
3 Uncaring	.75	7.45	3.73	.19	.43***	-		
4 Unemotional	.65	6.86	3.10	.09	.15	.28*	-	
5 EPA intake	-	0.01	0.02	-.41***	-.19	-.08	-.15	-

Note: N=98; Benjamini-Hochberg corrections: *$p < .05$, **$p < .01$, ***$p < .001$.

Physical aggression data was *t*-transformed as a function of age and sex; EPA intake mean and standard deviation (SD) data is presented untransformed for clarity (mg) and correlations are presented using *ln*-transformed data.

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Figures

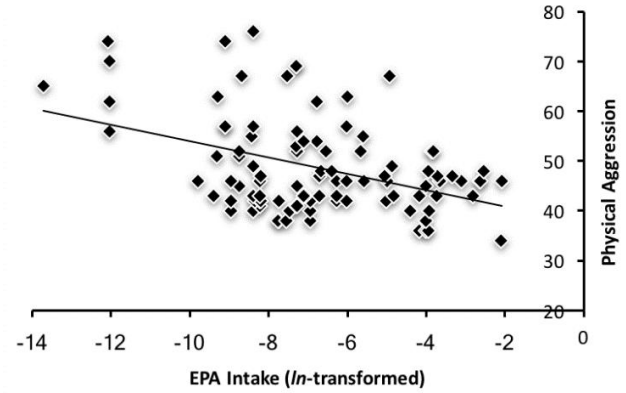


Figure 1: **Scatter plot of Physical Aggression trait score (*t*-transformed) against daily EPA intake (*ln*-transformed). Pearson correlation.**

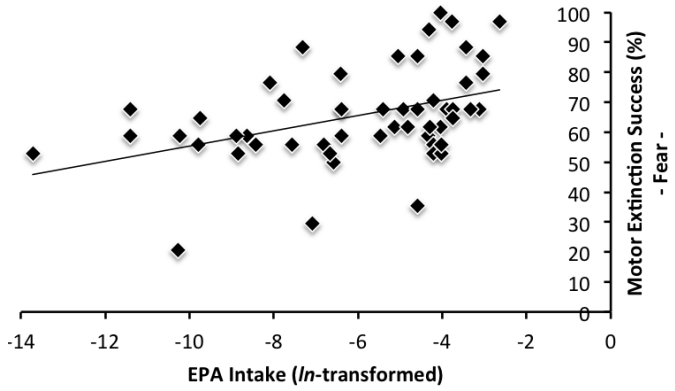
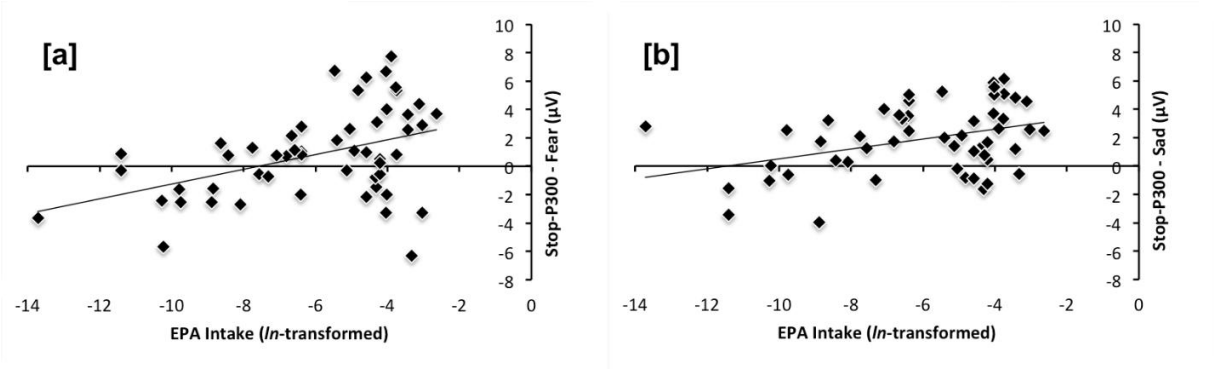


Figure 2: **Scatter plot of motor inhibition success (%) in response to fearful facial stimuli against daily EPA intake (*ln*-transformed). Pearson correlation.**



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568 ***Figure 3:*** **Scatter plot of EPA Intake (*ln*-transformed) against anterior midline stop-P300**

569 **amplitude (µV) to fearful (a) and sad (b) facial stimuli. Pearson correlations.**

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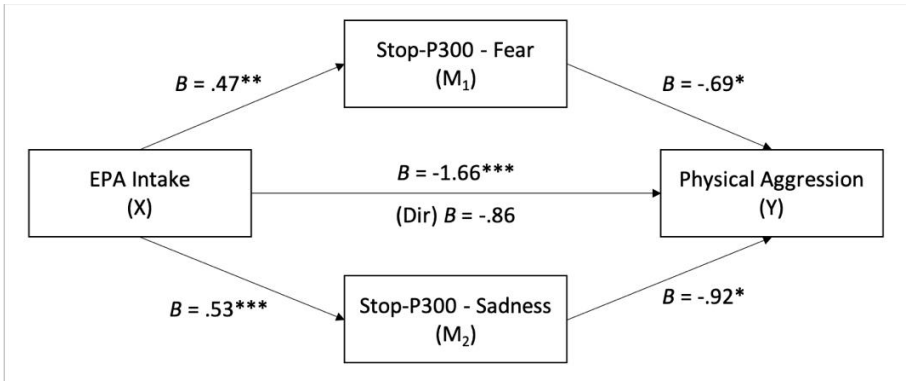

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577 ***Figure 4:*** **Mediation model showing the association between daily EPA intake and physical**

578 **aggression through the Stop-P300 response to fearful and sad facial expressions (*n*=47;**

579 **5,000 resamples); Covariates of socioeconomic status, alcohol use, and cannabis use are**

580 **modelled but not shown for the purpose of clarity; $p < .05$*, $p < .01$**, $p < .001$*****.**

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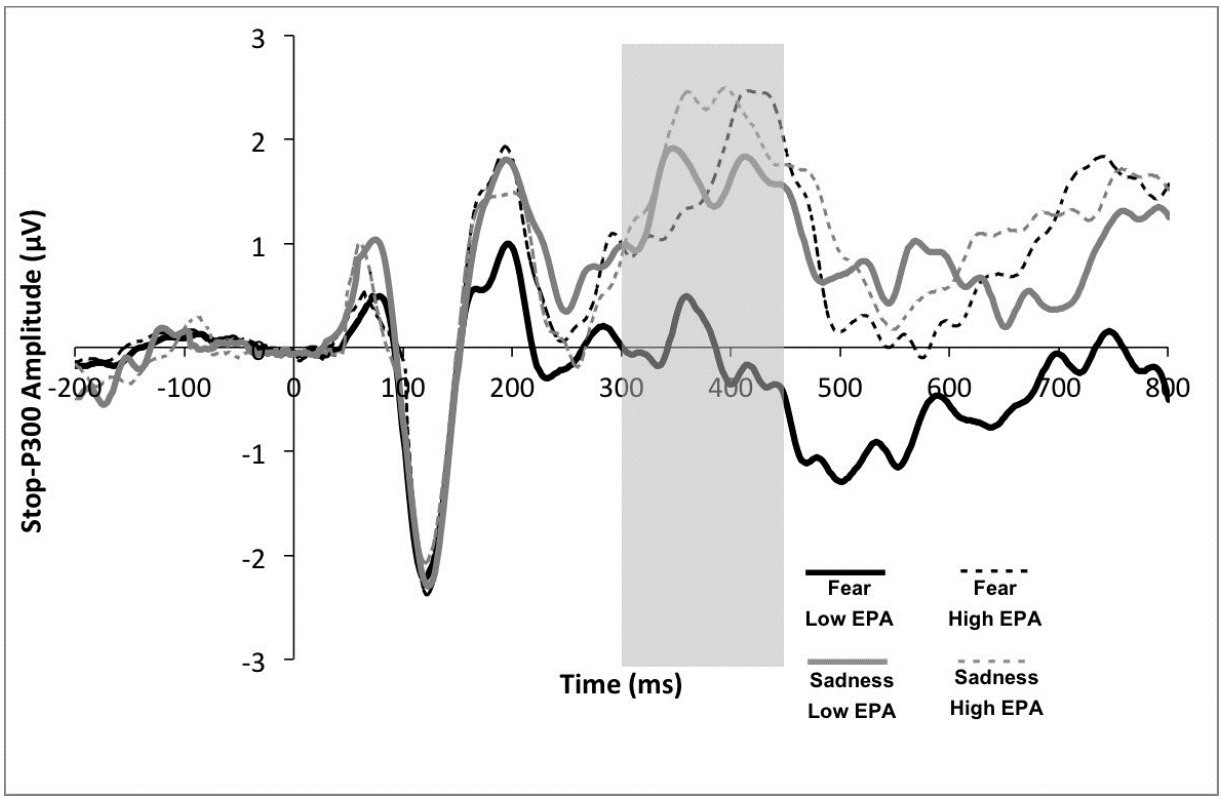

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584 ***Figure 5:*** **Grand average event-related potentials to fearful and sad (stop) facial stimuli as a**

585 **function of EPA intake (median split).** Waveforms indicate activity at the anterior midline

586 electrode (Fz) referenced to averaged mastoids**.** P300 time-window shaded in grey.

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