Violence inhibition mechanism functioning mediates the relationship between dietary eicosapentaenoic acid intake and physical aggression

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Abstract

Deficiency in long-chain omega-3 polyunsaturated fatty acids, in particular eicosapentaenoic acid (EPA), is implicated in aggression and callous-unemotional (CU) traits. A violence inhibition mechanism (VIM) has been proposed to regulate aggression through responding to expressions of distress. However, it remains unclear whether EPA intake is related to the VIM, and if so, whether this pathway can mediate relationships between EPA intake and deviant personality traits. The current investigation documents two, independently-sampled studies that tested relationships between EPA intake, personality (aggression, CU traits), and electrophysiological indices of the VIM (motor extinction cued by facial expressions of distress). In study one, 98 participants completed a food-frequency questionnaire, the inventory of callous-unemotional traits, and an aggression questionnaire. EPA intake was negatively correlated with physical aggression, even after controlling for age and sex. In study two, 47 participants completed the same measures in addition to having electroencephalography recorded during a novel paradigm assessing the distinct processing stages of the VIM. Stop-P300 (motor extinction) responses to facial expressions of distress mediated the relationship between EPA intake and physical aggression. For the first time, we have evidenced an association between EPA intake and indices of distress-induced motor extinction proficiency. Findings are in line with a proposed role of EPA in regulating aggression, possibly through associations with networks involved in distress-cued executive control over behaviour. Results are discussed in terms of the potential benefit of nutritional supplementation in clinical and forensic arenas. Data and a pre-print of this manuscript are available here:

https://osf.io/u3jdc/?view_only=b7f32cd1798344b5a6dffa2892253392

**Violence inhibition mechanism functioning mediates the relationship between dietary
eicosapentaenoic acid intake and physical aggression**

The violence inhibition mechanism (VIM) is a neurobiological network thought to regulate aggressive behaviour through the elicitation of an empathic response to the perception of distress in others (Blair, 1995; 2001). Deficiency of the VIM is thought to underpin psychopathy – a personality disorder characterised by atypical affect (e.g., callousness, low guilt, and poor empathy; Hare 2003) and behaviour (e.g., increased sensation seeking and aggression; Dawel, O’Kearney, McKone, & Palermo, 2012). In psychopathy, VIM deficiency is reflected through impaired processing of facial expressions of distress (e.g., fear and sadness; Dawel et al., 2012; Marsh & Blair, 2008; Wilson et al., 2011), coupled with poor motor extinction (Robinson & Bresin, 2014). While psychopathy occurs in fewer than 1% of the general population (Hare, 2003), associated personality traits, collectively referred to as callous-unemotional (CU) traits, are normally distributed and can be used to understand underpinning biological mechanisms, free from epiphenomena associated with a criminal lifestyle (Centifanti et al., 2016; Essau, Sasagawa, & Frick, 2006; Frick et al., 2000). This understanding is key to relieving the financial and personal burden associated with psychopathy (DeLisi et al., 2017).

Omega-3 within the context of psychopathy

Long-chain omega-3 polyunsaturated fatty acids (*n*-3 PUFAs) have been implicated in aggressive and antisocial personality traits, as well as in affective processes that purportedly underpin these traits (Gow, Vallee-Tourangeau et al., 2013; Raine et al., 2015). However, to date, *n*-3 PUFAs have not been directly investigated in relation to the VIM. Such work would

offer important insight into the neurobiological mechanisms underpinning the association between n -3 PUFAs and antisocial personality traits, and go some way to indicating a possible means of intervention.

Links between blood levels of n -3 and aggression, as well as other related cognitive and affective functions, are well established in animal models (*see* Fedorova & Salem Jr., 2006 *for review*). In humans, 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 negatively correlated with psychometric measures of aggression (Beier et al., 2014; Meyer et al., 2015; Zaalberg et al., 2016). Moreover, dietary supplementation of EPA has been associated with a reduction in physical aggression in borderline personality disorder patients (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 British and Dutch forensic populations (Gesch et al., 2002; Zaalberg et al., 2010). Where n -3 PUFA supplementation has not been shown to benefit aggression, such studies have used a supplement comprised entirely of DHA (Hirayama et al., 2004; Voigt et al., 2001). Regarding CU traits, blood levels of EPA have been found to negatively correlate with CU traits in children with attention deficit hyperactivity disorder (ADHD), with this association only observed at trend level for DHA (Gow, Vallee-Tourangeau et al., 2013). Thus, when considered together, EPA appears to play a key role in regulating psychopathy-related personality traits.

Although EPA is not highly concentrated in the brain (Chen, Ma, Kim, Mount, & Bazinet, 2008), possibly as a function of its rapid β -oxidation, relative to DHA (Chen, Liu, Ouellet, Calon & Bazinet, 2009), it may impact brain function and structure through its involvement in synaptic development (Cutuli et al., 2014; Robson, Dyall, Sidloff, & Michgael-

Titus, 2010), immunology, (Serhan, Chiang, & van Dyke, 2008), and gene expression (Allam-Ndoul, Guénard, Barbier, & Vohl, 2017). As such, EPA likely benefits the structure and function of networks implicated in regulating the distinct information processing stages of the VIM (i.e., face processing and motor extinction), which themselves have been associated with aggressive and CU traits (Fido et al., 2017).

Investigating aggressive and CU traits using electroencephalography

Electroencephalography (EEG) measures the gross change in extracellular, post-synaptic electrical potential, predominantly derived from pyramidal cells, and represents a useful biomarker of neural function associated with *n*-3. EEG has been used to investigate brain function in relation to *n*-3 intake in ADHD (Sumich et al., 2009), and CU traits and aggression in developmental (Sumich et al., 2012), adult forensic (Bernat, Hall, Stefan, & Patrick, 2007) and non-forensic (Fido et al., 2017) populations. The time-locked EEG response, referred to as event-related potentials (ERPs), can be evoked during a paradigm designed to index distinct processing stages of the VIM. The ERP in response to faces comprises several components, of which the N170 and P300 have been implicated in CU traits and reactive aggression (Fido et al., 2017).

N170 is a negative deflection in the EEG signal that peaks 150-200 ms post-stimulus at bilateral occipito-parietal sites and is responsive to expressions of threat and distress (Hinojosa et al., 2015). Whilst N170 amplitude in response to facial stimuli is negatively associated with uncaring and fearless dominance traits (Almeida et al., 2014; Fido et al., 2017; Meaux et al., 2014), positive correlations have been observed for other traits associated with psychopathy, such as cold-heartedness (Almeida et al., 2014). N170 responses to sad and neutral stimuli were

negatively associated with uncaring traits (a component of CU traits), but not with physical aggression in a non-forensic population (Fido et al., 2017).

P300 is a positive deflection in the ERP that peaks 300-500 ms post-stimulus with a widespread scalp distribution and parietal maxima (Hajcak, Weinberg, Macnamara, & Foti, 2013; Luck, 2005). Reduced P300 amplitude is associated with a range of externalising 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, likely reflecting the recruitment of frontal executive networks (Sumich et al., 2008). P300 amplitude during motor inhibition is reduced in delinquent men with ADHD (Meier, Perrig, & Koenig, 2012) and community samples evidencing psychopathic traits (Kim & Jung, 2014). However, in the general population, physical aggression, but not CU traits, was inversely associated with P300 amplitude during a task in which motor extinction was cued by facial expressions of distress (Fido et al., 2017). These findings support the idea that the N170 in response to facial affect reflects mechanisms underpinning the generation of empathy, whilst the Stop-P300 indexes the executive ability required to alter behaviour in response to empathic cues (e.g., facial expressions of distress). As such these indices are proposed to map onto VIM processes that facilitate an affective, aversive response to perceiving distress in others and inhibiting aggressive behavior towards them.

To date, only limited investigation exists into the relationship between *n*-3 and ERP responses to face processing and motor inhibition (Fontani et al., 2005; Gow et al., 2009). Blood levels of EPA have shown a negative trend with N170 amplitude to sad faces in adolescent boys with ADHD (Gow et al., 2009). Moreover, P300 responses to NoGo stimuli increased following 35 days of combined EPA and DHA supplementation in a sample derived from the general

population (Fontani et al., 2005). To our knowledge, no investigation has explored associations between EPA and motor extinction cued by facial affect.

The Current Research

This manuscript documents two, independently-sampled studies conducted within the general population. Study one investigated self-reported 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 facial affect processing and distress-cued motor extinction mediated the relationship between self-reported EPA intake and both physical aggression and CU traits. Increases in Stop-P300 and N170 responses were hypothesised to mediate negative associations between EPA intake and aggressive and CU traits, respectively.

Study One

Methods

Participants

To determine our target sample size, we conducted an a priori power analysis using G*Power (version 3.1.9.2). Assuming an anticipated medium effect size ($R^2 = .40$), and a standard alpha level of .05, 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. Participants were required to be fluent in English, aged between 18-55 years, 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.

Materials

Buss-Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992). The BPAQ comprises 34 items that measure the propensity to aggress using a 5-point scale. In addition to the physical aggression subscale reported here (9 items; e.g., “If somebody hits me, I hit back”), subscales include verbal aggression (5 items), anger (7 items), and hostility (8 items). 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.

Inventory of Callous–Unemotional Traits (ICU; Frick, 2003). The ICU comprises 24 items that assess 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 (Kimonis, Branch, Hagman, Graham, & Miller, 2013).

Food Frequency Questionnaire (FFQ; Sublette et al., 2011). The FFQ comprises 21 items and was developed as a means of rapidly assessing *n*-3 PUFA consumption. Self-reported EPA scores are significantly correlated with blood measures ($r = .47$; 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.

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. On average, the study took less than 10 minutes to complete. This study was approved by an institutional ethical review panel prior to data collection.

Statistical Analysis

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). Hochberg correction accounted for multiple comparisons.

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 as described in Beier et al. (2014).

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	-

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.

221

222 Callousness was positively correlated with both physical aggression ($r [96] = .249, p =$
 223 $.014, padj = .034$) and uncaring traits ($r [96] = .434, p < .001, padj < .001$). Uncaring traits were
 224 positively correlated with unemotional traits ($r [96] = .276, p = .006, padj = .020$). There was a
 225 negative correlation between physical aggression and EPA intake ($r [96] = -.408, p < .001, padj$
 226 $< .001$), and this was confirmed by a partial correlation ($r = -.423, p < .001$) controlling for
 227 covariates age and sex (see Figure 1). EPA did not correlate with any subscale of the ICU.

228

229 **Figure 1:**

230 Scatter plot of Physical Aggression trait score (t -transformed) against EPA intake ($\ln$ -
 231 transformed). Pearson correlation.

Study Two

Methods

Participants

Forty-seven participants (aged 18.96 ± 1.22 years, 60% female) were subsampled from the cohort of 54 reported in Fido et al. (2017). 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. Participants were right-handed, aged 18-55 years, and did not report any diagnosed psychiatric or neurological disorders, or use of medication that might impact electrophysiology. Participants provided written informed consent in accordance with approved central university research protocols and national guidelines.

Materials

Before completing a Stop-Go paradigm under EEG conditions, 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.

The Barratt Simplified Measure of Social Status (BSMSS; Barratt, 2006). The

BSMSS is a two-section proxy measure of socioeconomic status, which combines data on educational attainment and occupation of one's own family. 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.

The Alcohol Use Disorder Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1992). The AUDIT 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.

The Cannabis Use Disorder Identification Test (CUDIT; Adamson & Sellman, 2003). The CUDIT 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.

The Facial Affect Stop-Go Task (FAST; Fido et al., 2017). The FAST is an experimental paradigm designed to simultaneously investigate the cognitive mechanisms underpinning the face processing and motor extinction processes, which form the VIM. Participants were asked to 'react' to facial stimuli (e.g., fearful, sad, neutral, or angry faces; 800 $\pm$ 100 ms) by moving their right index finger from a red button box key to an adjacent green key

after the extinction (i.e., face replaced with a black screen; 160 ± 40 ms) of angry faces (Go stimulus). Further, participants were tasked with interrupting this response (i.e., returning their finger to the red key) if a fearful or sad face (Stop stimulus) appeared before this response was completed. This task measures one's capacity to extinguish a motor response that is causing distress to another.

The paradigm was presented in two blocks using OpenSesame (version 3.0). Each block began with a 4000 ms lead-in, followed by 136 trials. A red fixation cross separated each trial (1800 ± 200 ms). Stimuli consisted of open-mouthed expressions so as to increase valence (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).

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. This study was approved by an institutional ethical review panel prior to data collection.

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. An average reference was calculated online. Impedance was maintained below 5 k Ω .

Electrooculography artefacts were corrected (Jung et al., 2000) and a band-pass filter of 0.01–0.35 Hz (notch filter of 50 Hz) was applied. Trials were baseline corrected before averaging (-200ms). The N170 was average-referenced and the P300 was re-referenced to linked mastoids (Fido et al., 2017). ERPs consisted of averaged (P7, PO7, O1, PO3, P8, PO8, O2, PO4) temporo-parietal N170 responses evoked to fearful, sad, neutral, and angry facial expressions, and anterior midline Stop-P300 (Fz) responses to fearful and sad facial expressions. Additive inverse values were used for the N170.

Statistical Analysis

EPA intake and 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, accuracy). Hochberg correction accounted for multiple comparisons. 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, 2013, 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' (2017) recommendations.

Results

As with study one, mean EPA consumption data ($.01 \pm .02$ 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).

Behavioral Analysis

There was a positive association between motor extinction success in response to fearful facial expressions ($M = 65.71 \pm 15.92$ %) 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 correlated with neither measure of motor extinction success to sad facial expressions (77.97 ± 13.34 %; $r [45] = .06, p = .674$) nor motor extinction reaction times to sad (733.61 ± 189.44 ms; $r [45] = -.11, p = .460$) or fearful (788.48 ± 225.86 ms; $r [45] = -.10, p = .499$) facial expressions.

Figure 2:

Scatter plot of motor extinction success (%) in response to fearful facial stimuli against EPA intake ($\ln$ -transformed). Pearson correlation.

Mediation Analyses

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.

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.

Figure 3:

Scatter plot of EPA Intake ($\ln$ -transformed) against anterior midline stop-P300 amplitude (μV) to fearful (a) and sad (b) facial stimuli. Pearson correlations.

Figure 4:

Mediation model showing the association between EPA intake and physical aggression through the Stop-P300 response to fearful and sad facial expressions ($n = 47$; 5,000 resamples); Covariates of socioeconomic status, alcohol use, and cannabis use are modelled but not shown for the purpose of clarity; $p < .05^*$, $p < .01^{**}$, $p < .001^{***}$.

Figure 5:

Grand average ERPs to fearful and sad (stop) facial stimuli measured from the anterior midline electrode (Fz) as a function of EPA intake (median split). ERPs are representative of EEG signal referenced to averaged mastoids.

Discussion

Using both cross-sectional online sampling and laboratory-based designs, self-reported dietary intake of EPA was investigated in relation to physical aggression and CU traits, and to electrophysiological indices of face processing and motor extinction (the VIM). 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 (indicative of distress-induced motor extinction proficiency). EPA intake was neither associated with CU traits, nor N170 responses to facial affect.

Relationships between aggression and CU traits are well established (Frick et al., 2003; Frick & White, 2008), yet their independent associations with EPA remain unclear. As expected, findings of study one revealed an association between self-reported intake of EPA and lower scores on a psychometric measure of physical aggression. Previously, negative associations between physical aggression and *n*-3 intake have been observed in male offenders (Meyer et al., 2015). Further, physical aggression has been shown to decrease following a two-month intervention of EPA supplementation in females with borderline personality disorder (Zanarini & Frankenburg, 2003). As such, our findings add to a growing body of literature defining a key role of EPA in reducing behavioural and psychometric measures of aggression, as well as associated affective responses (e.g., anger; Buydens-Branchey & Branchey, 2008).

There was no association between self-reported EPA intake and CU traits in either of our non-clinical, adult samples. This finding conflicts with previous reports of negative associations between EPA blood concentrations and CU traits in boys with ADHD (Gow, Vallee-Tourangeau et al., 2013). It does, however, support intervention studies which evidence no impact of *n*-3 supplementation on either self- (Raine et al., 2016) or parent-reported (Raine et al., 2015) CU

traits in children with conduct disorder. Disparity of results could be explained by differences in quantifying EPA intake and sample characteristics (e.g., age, comorbidity of ADHD, and/or conduct disorder symptomology), and so further investigation is warranted to accurately delineate this association across heterogeneous community, clinical, and forensic samples.

In study two, electrophysiological indices of the emotional face processing (N170) and motor extinction (P300) stages of the VIM were investigated as possible mediators of the relationships between self-reported EPA intake and both physical aggression and CU traits. As intake of EPA was neither associated with callous, uncaring, or unemotional traits, nor N170 responses (irrelevant of facial expression), mediation analysis was constrained to the association between EPA intake and physical aggression through distress-induced Stop-P300 responses. As expected, Stop-P300 responses to fearful and sad facial expressions mediated the negative association between EPA intake and physical aggression.

This is the first investigation to identify a correlation between EPA intake (over a 6-month period, at least) and motor extinction proficiency, as measured by EEG. Although causation cannot be inferred from the current findings, the P300 response to NoGo stimuli (geometric shapes) has previously shown to increase following EPA-rich supplementation (Fontani et al., 2005). Together, these findings support a potential role of EPA in motor inhibition/extinction, however we endorse future studies to integrate molecular analysis with neurocognitive measures in order to clarify whether, and by what mechanism, EPA actually benefits fear-cued executive control over behaviour (e.g., motor extinction in response to distress). It is possible that EPA might directly facilitate mobilisation within neural networks underpinning motor extinction by affecting fluidity of molecular information transmission

(Michael-Titus & Priestley, 2014) or indirectly through regulation of systemic inflammation and/or the hypothalamic-pituitary-adrenal response (Simpoulos, 2002).

In line with psychometric findings from study one, EPA did not correlate with face-evoked N170 responses. Again, this contrasts 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).

Limitations and Future Direction

Although the FFQ has been shown to correlate with plasma levels of EPA (Sublette et al., 2011), blood measurements would allow for a more accurate quantification of EPA. Moreover, this static measure of EPA intake reflects consumption over a six-month period and so may not be robust enough to fully explain the role of EPA in variation in personality traits or EEG. Specifically, as synaptic reorganisation is thought to occur during child development (Crawford et al., 2003), such questions might be better answered through a historical index of EPA consumption, or via a longitudinal assessment. Further, consumption of DHA was not reported in this investigation due to [a] previous findings indicating a predominant role of EPA in the manifestation of psychopathy-related traits, and [b] high multicollinearity between DHA and EPA intake preventing precise delineation of their independent contribution to the variation in data. As such, this further supports the use of blood measures of *n*-3 in the future. Additionally, due to study-related constraints, variation in substance use (e.g., alcohol, drug, nicotine) and

socio-economic status was only controlled for in the latter, laboratory-based study, and not in the cross-sectional cohort. Substance use has been shown to compromise n -3 structural composition and absorption (Pawlosky, Bacher, & Salem, 2001; Pawlosky, Hibbeln, & Salem, 2007), and to contribute to the relationship between EPA deficiency and trait aggression (Beier et al., 2014). Socioeconomic status has been associated with increased habitual fish intake (Rice et al., 2016) and so may account for some of the observed variation in the data. Finally, although the use of mediation analysis has previously been used in samples of around $n = 50$, larger sample sizes would be preferred to ascertain the validity of some of the smaller (in)direct effects observed in this report (*see* Fritz & MacKinnon, 2007). Future studies should control for these limitations by investigating blood measures, historical indices, and controlling for other lifestyle factors. Specifically, these findings would benefit from future studies within larger forensic populations in order to ascertain the replicability of results in samples characterised by more extreme manifestations of psychopathy-related traits.

Conclusion

The current study found that higher levels of self-reported EPA intake were associated with lower traits scores of physical aggression, but not measures of callous, unemotional, or uncaring traits in two adult samples derived from the community. Furthermore, whilst EPA intake did not associate with electrophysiological indices of face processing (N170), higher levels of EPA intake were associated with larger Stop-P300 responses to facial distress, an electrophysiological index of motor extinction proficiency, with this relationship thought to mediate the negative association between EPA intake and physical aggression. These findings are in line with a role of EPA in executive control over behavior, as cued by affective stimuli; rather than the initial

482 encoding of the emotional content of the face. There appears to be great logistical and financial
483 value in pursuing the use of *n*-3 supplementation as a potential, internationally-robust, tool for
484 reducing physical aggression. As such, due to their notable implications for clinical and forensic
485 populations, as well as community-based cohorts more generally, results reported in this
486 investigation should be of interest to forensic psychologists and behavioral scientists alike, and
487 provide a strong basis for future replication using more statistically-robust, biological methods.

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