



RESEARCH ARTICLE

Examining the cognitive function of individuals with antisocial traits using event-related potentials

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Abstract:

Background: While antisocial behavior, a significant contributor to undesirable and criminal behaviors, is well characterized and defined in some ways, this is not the case for the cognitive processes that might differ for this behavior, including those relating to behavioral control.

Objectives: Investigation of cognitive factors that contribute to antisocial behavior by examining inhibitory control to better characterize cognitive differences associated with such behavior and aid determination of methods for beneficial intervention.

Methods: Inhibitory control was measured using a stop signal task with concurrent recording of event-related potentials (ERPs) in individuals with high antisocial traits and low antisocial traits with trials preceded by either neutral or fearful facial cues. This allowed assessment of any differences in emotion processing, inhibitory control and error monitoring. Participants were a non-clinical adult sample recruited through a university online forum in Taiwan.

Results: Both higher- and lower-ASTS-20 score groups showed higher P2 ERP amplitudes for fearful-face trials compared to neutral-face trials, suggesting more perceptual processing of the former, but the higher-ASTS-20-score group had lower P2 amplitudes for these trials. No differences were seen for N2 and P3 amplitudes, often associated with inhibitory control. The higher-ASTS-20-score group showed weaker event-related negativities (ERNs) and stronger Pe responses than the lower-ASTS-20-score group as well as longer latencies for the ERN.

Conclusions: Higher antisocial personality traits were associated with reduced processing of fearful emotion information and differences relating to error processing rather than inhibitory control deficits. The associated processes might be good candidate processes for future mechanistic or intervention research, rather than general inhibitory control training.

Keywords: antisocial behavior, electroencephalography, inhibitory control, error processing

Ethical Approval: This study received ethical approval from the Human Research Ethics Committee of National Chung Cheng University (CCUREC111070801; Ref. #34071) and was conducted in accordance with the Declaration of Helsinki.

Informed Consent: All participants gave informed consent before taking part and were free to withdraw from the study at any time.

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Data Availability: The data supporting the findings of this study are available from the authors upon reasonable request.

Preregistration: The study design and analysis were not preregistered.

Introduction

Antisocial behavior is problematic in society, is associated with criminal acts and causes distress for the individuals affected by such acts (Foster & Jones, 2005). One of the strongest predictors of someone showing criminal behavior is their level of antisocial traits (Eddy and Reid, 2002). While there environmental and genetic elements associated with such traits (Niv et al., 2013), specific underlying contributions of cognitive and neural mechanisms associated with elevated levels of antisocial behavior remain unclear (Delfin et al., 2022). It is also unclear how individuals who show high levels of antisocial behavior can be effectively treated (Bateman et al., 2015; van den Bosch et al., 2018). Better characterization of cognitive differences in individuals with high levels of antisocial traits would clearly be beneficial for both of these questions.

There are two main points of issue when trying to better characterize antisocial behavior with the specific aim of guiding a beneficial reduction of such behavior. First is the fundamental issue of deciding who should be categorized as antisocial and, second, determining which cognitive processes are related to unacceptable levels of such behavior and so be potentially beneficial targets for intervention.

Identification of individuals with high levels of antisocial traits

Until relatively recently, scoring scales related to antisocial traits were primarily employed for clinical diagnosis or forensic investigation, employing measures such as those provided by the Psychopathy Checklist-Revised (PCL-R) (Hare, 2003). The subsets of the population in which these are used can often also show particularly high or low levels of antisocial traits. However, many individuals can have high levels of antisocial behavior/traits that are neither a clinical nor a criminal problem (despite them potentially being at risk of being so) so it is unclear whether they can be meaningfully indexed by scales that were not designed for use in non-clinical/forensic populations. In addition, there has been a broader question of a lack of agreement about the definition of antisocial traits (Rafiey et al., 2020), meaning defining a meaningful, generally applicable index could be

problematic. In light of this, Rafiey and colleagues (Rafiey et al., 2020) developed an antisocial traits scale with the specific aim of it being applicable to the general population, using an analytical approach to determine the factors that should be assessed by the associated questionnaire (the resulting Antisocial Traits Scale, ASTS-20). This was described as being potentially useful to identify individuals who may be at risk of showing criminal behavior.

This scale, therefore, allows for valid investigation of differences in cognitive processes in individuals with antisocial traits in the absence of clinical problems or criminal behavior. While there are, as mentioned above, several factors that *might* contribute to antisocial behavior, and several of these have been related to cognitive processes, such associations remain unclear (see, also, ‘Inhibitory Control’, below).

Cognitive processes potentially affected by antisocial behavior - inhibitory control

Ogloff (2006) reported that individuals with antisocial personality disorder, which has an occurrence rate of 1-3% of people in Western countries (Volkert et al., 2018), are more prone to showing impulsivity and poor behavioral control, so linking higher antisocial behavior (or traits) with lower inhibitory control function. Inhibitory control can be measured using cognitive tasks, such as the stop-signal task, which also allows measurement of impulsivity. This task typically requires a speeded response to be made to a presented ‘go’ stimulus on most trials. Occasionally, a stimulus is followed by a ‘stop signal’, indicating a response should be withheld and the time difference between the go stimulus and the stop signal can be varied to manipulate task difficulty. For example, if a stop signal is presented with a longer delay after the presentation of the ‘go’ signal, an individual is more likely to incorrectly make a response. Importantly, this task has been shown to show differences in various ‘non-normal’ populations, including clinical groups and drug users (Li et al., 2006) and can be neurochemically modulated (Chamberlain et al., 2006). The task can also be used in conjunction with electrophysiological recordings to supplement assessment of inhibitory control and impulsivity with evaluation of error processing. Despite reports such as that of Ogloff (2006), investigation of the nature of the link between *antisocial behavior* and inhibitory control remains somewhat limited and this is particularly the case for studies employing event-related potential data which can allow better understanding of the nature of any differences seen.

Event-related potentials (ERPs) and inhibitory control

Concurrent recording of electroencephalography (EEG) during an inhibitory control task allows observation of several electrophysiological components which relate to different processes required for successful task performance. Of these, the ERP N2 and P3 components are considered to be indicative of processes important for inhibitory control and, so, are of interest in the context of antisocial behavior. For example, Pasion et al. (2018, 2019) reported that a decrease in the amplitude of P3 can be generally regarded as a neurophysiological indicator of aggressive and antisocial behavior. This component is seen in trials in a Go/NoGo task, another task involving inhibitory control that requires a response to be made frequently but withheld occasionally. The P3 component is typically seen for trials where the response has to be withheld (Polich, 2007) and is thought to relate to monitoring processes and the evaluation of outcomes (Gajewski & Falkenstein, 2013; Huster et al., 2013), although there remains ambiguity of whether this is actually the case.

For the N2 component, and once again highlighting some of the uncertainty underlying characteristics of antisocial-related cognitive differences, a meta-analysis by Pasion et al. (2019) reported that there was a systematic decrease in N2 amplitude associated with antisocial behavior and related to inhibitory control, but only

when this component was recorded for stop signal tasks (SST) and not for Go/NoGo tasks. One possibility is that this is because of a higher inhibitory control requirement for SST tasks, with aggressive behavior and antisocial behavior potentially having a stronger effect on the control process required to stop making a response in these tasks.

While ERP components are most often indexed in terms of the size (amplitude) of the component, ERP *latencies* can also be relevant. For example, Delfin et al. (2022) found a strong positive correlation between NoGo P3 latency and self-reported scores of aggression and antisocial tendency. Similarly, the NoGo N2 component latency and self-reported scores of aggression and antisocial tendency also had a strong positive correlation. These longer latencies may represent a decrease in neural effectiveness, with latency being an indicator of 'neural power' (van Dinteren et al., 2014). If this is the case, when cognitive resources are engaged by aggressive and/or antisocial information, the information processing speed relating to conflict monitoring and outcome evaluation may decrease. The study by Delfin and colleagues (Delfin et al., 2022) tested a group expected to score highly on these scales (violent mentally disordered offenders) and a group of healthy controls. Interestingly, while no significant effects were seen for ERP amplitudes, they did see effects on ERP component latencies. Also of interest is that they saw no significant effects on behavioral measures of performance on the Go/NoGo task.

Overall, this suggests that both the typically-investigated ERP amplitudes and the less frequently evaluated ERP latencies would be useful indices in ERP studies looking at inhibitory control and antisocial behavior, with the latency measures enabling assessment of a different facet of cognitive processing.

ERPs and error-related processing

In addition to inhibitory control, ERP measures can also be used to evaluate of the relationship between antisocial traits and cognition in terms of error monitoring mechanisms, another area in which problems have been suggested. There are studies that have controlled for variables such as anxiety, depression, and, relevantly here, impulsivity and have found that antisocial personality traits can predict the amplitude of two error-related ERP indicators, the error-related negativity (ERN) and error positivity (Pe) (Chang et al. 2010). Ruchow et al. (2005) also found that individuals with impulsive behavior tendencies have lower ERN and Pe amplitudes. Given the association between antisocial personality traits and these ERP components when *controlling for impulsivity*, it could be that some of the behavior seen in individuals with high antisocial behavior is a consequence of poor error monitoring capabilities, rather than a general inhibitory control problem.

The ERN ERP component is an indicator of performance monitoring related to cognitive control and with a peak approximately 50 to 100 milliseconds after an incorrect response. It reflects the cognitive conflict between incorrect responses and correct responses and could, in turn, be related to an error detection function. Alternatively, it could be a response to cognitive conflict, occurring when success is expected but an error is made. The Pe component follows an ERN, and typically reaches a peak about 200 to 400 milliseconds after the erroneous response. It is generally believed to reflect the individual's awareness of an error (Overbeek et al., 2005). Both of these components can be readily recorded in a stop-signal task, which typically generates numerous errors for the trials where response inhibition is required.

Face emotion processing

In addition to problems with inhibitory control, antisocial personality disorder has been associated with a general dysfunction in recognizing facial emotions, and particularly fearful emotions. For example, a meta-analysis by Marsh & Blair (2008) reported that antisocial groups showed dysfunction in recognizing facial emotions with antisocial behavior negatively correlated with the recognition of fearful, sad, and surprised face stimuli but not for faces showing happiness, anger or disgust. There was also low amygdala activity in such groups (Marsh & Blair, 2008). This can be indexed via an ERP component, with the deficiency associated with a decrease in the amplitude of the P2 event-related potential following presentation of a face-stimulus (Boutsen et al., 2006). This is of particular interest because the aforementioned study by Delfin et al (2022) didn't involve emotional face stimuli in the task but previous studies with a Go/NoGo task that have used emotional (happy/sad) faces as the Go/NoGo stimuli *did* report differences in performance for aggressive individuals in comparison to non-aggressive individuals (Madole et al., 2020). One possible explanation for this is that inhibitory control might be more affected by (or contingent on) emotional information being involved in the task.

Summary

While there have been several underlying cognitive causes suggested for the patterns of behavior associated with antisocial personality disorder, but there remains uncertainty regarding several of these. Additionally, and importantly, consideration of this in the broader context of individuals with *non-clinical* degrees of antisocial behavior is needed, given that studies tend to involve cases of more extreme antisocial behavior such as criminal aggression. Identifying cognitive processes with deficiencies in individuals with a higher likelihood of showing problematic behavior in the future might help consider how such behavioral differences could be beneficially addressed.

Here, the aim of the study was to investigate cognitive factors that might be related to antisocial behavior by looking at the performance of high and low antisocial individuals on a stop-signal task with trials preceded by an emotional (fearful) or neutral face cue in conjunction with electroencephalographic (EEG) recording. This would allow examination of inhibitory control, error monitoring and any effects of emotional information on these. The P2 ERP component can be looked at in relation to effects of fearful face stimuli and the stop N2 and P3 ERP components relate to inhibitory control. Additionally, the ERN and Pe ERP components can be examined in the context of error monitoring. All ERP components can be assessed in terms of amplitude (indicative of cognitive resource allocation) and latency (indicative of processing speed). The overall objective is to better characterize cognitive differences, if any, associated with antisocial behavior with this being potentially informative about processes which be suitable targets for beneficial intervention.

Methods

This study received ethical approval from the local Human Research Ethics Committee. Research was completed in accordance with the Helsinki Declaration.

Participants

Participants were recruited through advertisements posted on a university online forum. Screening of the 86 individuals who responded was then carried out using the Antisocial Traits Scale (ASTS). This is a 20-item, five-point scale (see below) and allowed selection of two groups, one with higher levels of antisocial traits and one with lower levels of antisocial traits. The mean and median scores of the 86 volunteers were both 48 points ($N = 86$, Mean score = 48.37, Median score = 48.5, $SD = 10.29$). The 16 lowest scoring participants (defined as the lower-ASTS-20-score group) and the 16 highest scoring participants (defined as the higher-ASTS-20-score group) were selected, making a total of 32 participants (16 males and 16 females) for the rest of the experiment. The two groups showed a significant difference in ASTS scores ($F_{(1, 30)} = 88.235$, $p < 0.001$, $\eta_p^2 = 0.746$), but no significant difference in age ($F_{(1, 30)} = 0.010$, $p = 0.920$, $\eta_p^2 < 0.001$) and no significant difference in Raven scores ($F_{(1, 30)} = 0.111$, $p = 0.741$, $\eta_p^2 = 0.004$) (from the Raven's Graphical Reasoning test, Raven, & Court, 1998). The Triarchic Psychopathy Measure (TriPM) (Patrick & Drislane, 2015) was also presented to all participants. More details of the participant characteristics and the ASTS, Raven and TriPM scores are shown in Table 1.

Table 1: Characteristics for the participants who took part in the study

Variable	Lower-ASTS-20-score Group (n = 16)	Higher-ASTS-20-score Group (n = 16)	<i>p</i>
Demographics			
Sex, <i>n</i> (%)			
Male	8 (50%)	8 (50%)	
Female	8 (50%)	8 (50%)	
Age (years)			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	22.63 ± 3.81 (0.95)	22.75 ± 3.13 (0.78)	0.920
Range	20–35	20–32	
Raven's Score			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	24.13 ± 4.84 (1.21)	24.69 ± 4.69 (1.17)	0.741
Range	15–32	16–31	
Antisocial & Personality Traits			
ASTS Total Score			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	38.94 ± 5.42 (1.36)	59.19 ± 6.71 (1.68)	<0.001**
Range	28–45	51–71	
TriPM Total Score			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	50.94 ± 10.45 (2.61)	70.50 ± 16.16 (4.04)	<0.001**
Range	25–67	34–98	
Boldness			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	24.44 ± 7.11 (1.78)	27.81 ± 9.91 (2.48)	0.277

Variable	Lower-ASTS-20-score Group	Higher-ASTS-20-score Group	<i>p</i>
	(<i>n</i> = 16)	(<i>n</i> = 16)	
Range	7–35	8–42	
Disinhibition			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	16.00 ± 6.58 (1.65)	21.25 ± 7.41 (1.85)	0.042*
Range	6–32	11–35	
Meanness			
<i>M</i> ± <i>SD</i> (<i>SEM</i>)	10.50 ± 4.29 (1.07)	21.44 ± 9.93 (2.48)	<0.001**
Range	4–17	8–48	
Note. <i>M</i> = Mean; <i>SD</i> = Standard Deviation; <i>SEM</i> = Standard Error of the Mean; ASTS = Antisocial Traits Scale; TriPM = Triarchic Psychopathy Measure; * <i>p</i> < .05. ** <i>p</i> < .001.			

All participants gave informed consent prior to taking part in the experiment and they were free to withdraw from the experiment at any time for any reason. All participants had normal or corrected to normal visual acuity as well as normal color discrimination ability. Additionally, none had a history of neural damage or other physical or mental diseases or disorders.

Following recruitment of the participants and description of the experimental procedures, the questionnaires were presented to the participants and these were followed by the behavioral testing/ERP collection.

Questionnaires

Antisocial Traits Scale (ASTS) (Rafiey et al., 2020): This is a 5-point self-report scale, from 1 (‘strongly disagree’) to 5 (‘strongly agree’) containing 20 items and it measures six aspects of antisocial traits: maleficence, distraction, law-breaking, risk-taking, lack of planning, and impulsive decision-making. Scores are totaled, such that an individual can obtain a minimum of 20 points and a maximum of 100 points.

Triarchic Psychopathy Measure (TriPM): This is a 4-point self-report scale with 58 items. There are three subscales relating to the different phenotypic constructs used to measure psychopathy: disinhibition (20 questions), meanness (19 questions), and boldness (19 questions). Participants have to report whether a question description is consistent with the themselves, on a range from 0 (not at all) to 3 (completely match). Scores are summed for each phenotype construct and the sum of these is an overall psychopathy score.

Behavioral task and electrophysiological recording

Participants performed a version of the stop signal task (SST) presented on a PC. The task was presented via a 33.28 cm monitor (28.78 cm horizontal width, 32.11 degrees of visual angle) with a refresh rate of 60 Hz, a 1600×900-pixel resolution and viewed from a distance of 50 cm. A 500 ms fixation (the + character, using a font with a 60-pixel width, 1.24° of visual angle) was followed by either a neutral expression face or a fearful expression face (400×300 pixels, 8.23×6.18°) for 500 ms in the formal trials (no picture was presented in the practice trials). For ‘Go’ trials, an arrow (using a 400×262 pixels, 8.23×5.40° image), indicating whether a ‘left’ or ‘right’ response should be made (via a finger press of the left or right keyboard button) was presented for up to 500 ms. The left and right directions occurred equally frequently but in a random order. For ‘Stop’ trials, a red

circle (60-pixel width font, 1.24° of visual angle) was presented centrally following arrow presentation and indicated that no response should be made. Details regarding the timing of the arrow-stop signal offset are described in the sections below and the task is illustrated in Figure 1.

Experimental stimuli

Practice trials

40 practice trials of the task were presented prior to the main testing trials. No face images were used for these trials. The stop signal delay (SSD) was initially 200 milliseconds, and was dynamically adjusted so that the probability of participants failing to inhibit their response on stop trials was approximately 50%. Stop signals were presented on 25% of trials. Whenever the participant successfully inhibited a response following a stop signal, the SSD for the next stop trial was increased by 34 ms (i.e., two screen refreshes of the 60 Hz refresh-rate monitor used to present the task). When the participant failed to inhibit their response on such a trial, the SSD for the next stop trial was decreased by 34 ms.

Formal trials

For these trials fearful and neutral emotional stimuli were added to the trials, using face pictures from the Asian Emotional Face Picture Database (<https://www.paulekman.com/product/pictures-of-facial-affect-pofa/>). 320 trials were presented, composed of 4 blocks of 80 trials. As before, 25% trials had a stop signal appear and the SSD was altered in the same manner as before. The average SSD and average reaction time for Go trials was calculated for each of the 80-trial blocks and then these four SSDs and average reaction times were used to calculate an overall average SSD and average reaction time for each participant. In each case, the difference between the overall average reaction time and the average SSD was used to calculate the stop signal reaction time (SSRT), a measure indicative of the time taken for the inhibitory processes needed to withhold a response. Post error slowing was calculated as the mean response times for go-trials immediately following a stop signal presentation where a response had been made (i.e., when an error was on a stop trial) minus the mean response times for go-trials immediately following a stop trial where no response had been made (i.e., a successful response inhibition).

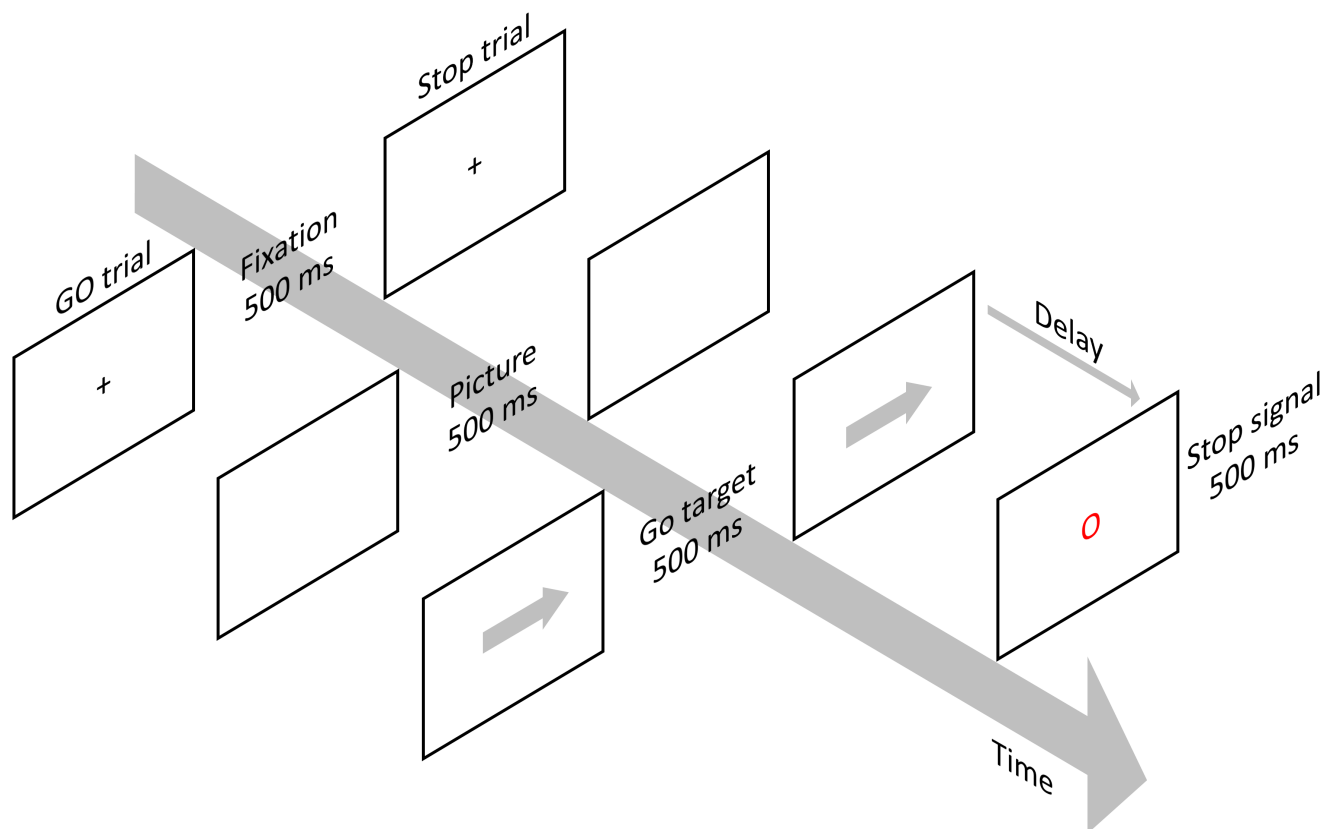


Figure 1. The time-line and procedure of the emotional stop-signal task. The stop signal, if presented, remained on the screen until the end of the trial or until the participant made a button press.

Electrophysiological recording and analysis

Electrophysiological recording during task performance was carried out using a Neuroscan Grael amplifier and Curry 8 acquisition software (Compumedics Neuroscan, Charlotte, NC, USA) with a sampling rate of 1024 Hz and via a 32-electrode cap with electrodes placed according to the 10-20 system.

Prior to analysis of the recordings, ocular artifact reduction, filtering, epoching, baseline correction, and artifact rejection were all carried out. Filtering used a 24dB band pass filter, with high and low pass frequencies of 0.01 Hz and 30 Hz, respectively. Segmentation (epoching) used a period of 100 milliseconds before stimulus presentation or response execution (the baseline period for stimulus-related and response-related components), followed by a sufficient time period for the ERP components of interest, with amplitude correction according to the mean amplitude of the baseline period. Subsequently, the VEOG channel was used for removal of trials with vertical eye movements with a standard threshold criterion of $\pm 70 \mu\text{V}$, and the HEOG channel was used to exclude trials with horizontal eye movements with a $\pm 100 \mu\text{V}$ criterion. Finally, to obtain the specific information for analysis, segments from the same participant, for the same category, and responses were averaged.

ERP amplitudes were determined as the mean amplitude during the relevant time-window for the component of interest (see below).

Brainwave latency analysis used the relative criterion method. This method first selects the peak or trough with the largest amplitude in the relevant time segment of data for each participant, category and for correct responses. The voltage (in μV) of the peak or trough can be used to calculate the value of 50% of the amplitude voltage, and

the time point at which the 50% amplitude voltage value is reached for the first time is used as the brain wave latency time. In addition, if the time point is earlier than the time segment of interest, the earliest time point in its time segment was used as the latency time.

Transparency and Openness

The data upon which the findings of the present study are based can be obtained from the authors by request. The study design and analysis were not preregistered.

Results

Questionnaire: Triarchic Psychopathy Measure (TriPM)

Using a one-way independent samples ANOVA, no significant difference was seen in the Boldness subscale of the TriPM ($F_{(1, 30)} = 1.225$, $p = 0.277$, $\eta_p^2 = 0.039$). However, significant differences were found between the two groups on the Disinhibition subscale ($F_{(1, 30)} = 4.491$, $p = 0.042$, $\eta_p^2 = 0.13$), the Meanness subscale ($F_{(1, 30)} = 16.369$, $p < 0.001$, $\eta_p^2 = 0.353$), and the total score ($F_{(1, 30)} = 16.534$, $p < 0.001$, $\eta_p^2 = 0.355$), with the higher-ASTS-20-score group scoring significantly higher than the lower-ASTS-20-score group.

Behavioral Performance

The behavioral performance data is shown in Table 2.

Table 2: Performance data for the stop signal task

		Lower-ASTS-20-score Group	Higher-ASTS-20-score Group
		<i>M ± SD</i>	<i>M ± SD</i>
Go accuracy	Neutral	0.92 ± 0.09	0.94 ± 0.05
	Fear	0.92 ± 0.09	0.94 ± 0.08
Stop accuracy	Neutral	0.45 ± 0.06	0.46 ± 0.04
	Fear	0.46 ± 0.05	0.46 ± 0.04
GORT^a	Neutral	392.49 ± 37.32	397.49 ± 29.36
	Fear	391.01 ± 35.02	399.67 ± 31.81
SSRT^b	Neutral	232.03 ± 29.33	237.29 ± 18.31
	Fear	228.78 ± 19.15	236.57 ± 14.27
SSD^c	Neutral	160.47 ± 43.58	160.2 ± 35.53
	Fear	162.22 ± 34.4	163.1 ± 29.09
PES^d	Neutral	8.26 ± 17.7	12.87 ± 16.85
	Fear	10.5 ± 17.4	11.85 ± 15.23

^aGORT: GO Reaction Time; ^bSSRT: Stop Signal Reaction Time; ^cSSD: Stop Signal Delay;

^dPES: Post-Error Slowing

Go reaction times and accuracies

A two-way ANOVA (Group x Emotion) for Go reaction time showed no significant interaction ($F_{(1, 30)} = 0.639$, $p = 0.430$, $\eta_G^2=0.001$), nor significant main effects of emotion ($F_{(1, 30)} = 0.023$, $p = 0.881$, $\eta_G^2<0.001$) or group ($F_{(1, 30)} = 0.345$, $p = 0.561$, $\eta_G^2=0.011$). Similarly, the two-way ANOVA (Group x Emotion) for Go accuracy showed no significant interaction ($F_{(1, 30)} = 0.943$, $p = 0.339$, $\eta_G^2=0.003$), nor a significant main effect of emotion ($F_{(1, 30)} = 0.006$, $p = 0.937$, $\eta_G^2<0.001$) or group ($F_{(1, 30)} = 0.524$, $p = 0.475$, $\eta_G^2=0.021$).

Stop-related performance

The two-way ANOVA (Group x Emotion) for SSD showed no significant interaction ($F_{(1, 30)} = 0.027$, $p = 0.871$, $\eta_G^2<0.001$), nor significant main effects of emotion ($F_{(1, 30)} = 0.451$, $p = 0.507$, $\eta_G^2=0.001$) or group ($F_{(1, 30)} = 0.001$, $p = 0.980$, $\eta_G^2=0.001$). Similarly, the two-way ANOVA (Group x Emotion) for SSRT revealed no significant interaction ($F_{(1, 30)} = 0.179$, $p = 0.675$, $\eta_G^2=0.001$), nor significant main effects of emotion ($F_{(1, 30)} = 0.438$, $p = 0.513$, $\eta_G^2=0.002$) or group ($F_{(1, 30)} = 0.920$, $p = 0.345$, $\eta_G^2=0.025$). Finally, the two-way ANOVA (Group x Emotion) for Stop Response Accuracy showed no significant interaction ($F_{(1, 30)} = 0.657$, $p = 0.424$, $\eta_G^2=0.003$), nor significant main effects of emotion ($F_{(1, 30)} = 0.233$, $p = 0.632$, $\eta_G^2=0.001$) or group ($F_{(1, 30)} = 0.253$, $p = 0.619$, $\eta_G^2=0.007$).

Post error slowing

The two-way ANOVA (Group x Emotion) for post-error slowing showed no significant interaction ($F_{(1, 30)} = 0.002$, $p = 0.965$, $\eta_G^2<0.001$), nor significant main effects of emotion ($F_{(1, 30)} = 0.044$, $p = 0.836$, $\eta_G^2=0.044$) or group ($F_{(1, 30)} = 0.474$, $p = 0.496$, $\eta_G^2=0.474$).

ERP analysis

The following indicates the electrodes and time-windows for the ERP components of interest. The P2 (200~250 ms) component was recorded from the Cz electrode (Ashley et al., 2004), the N2 (180~250 ms) component was recorded from the Fz, Cz and Pz electrodes (Čeponien et al., 2002; Mahajan & McArthur, 2011), the P3 (270-370 ms) component was recorded from the Fz, Cz and Pz electrodes (270~370 ms) (Walhovd et al., 2002). These were relative to the image onset for the P2 component (320 trials) and relative to the onset of the stop-signal, of which there were 80 trials per participant, for the N2 and P3 components. The ERN (135-235 ms) was recorded from the Cz electrode (135~235 ms) (Grisetto et al., 2019; Simó et al., 2018) and the Pe (235~335 milliseconds) component (Overbeek et al., 2005) was recorded from the Cz electrode. These components were recorded relative to responses made following presentation of a stop-signal (i.e., errors on stop trials). This gave a mean of 43.4 (S.D. = 3.8) trials per participant. The amplitudes and latencies of were all dependent variables with factors of group (lower-ASTS-20-score group, higher-ASTS-20-score group) and face emotional stimulus (neutral, fearful).

ERP component amplitudes

P2 component

A two-way ANOVA (Group x Emotion) analysis of the P2 amplitude revealed a significant interaction between groups ($F_{(1, 30)} = 4.660$, $p = 0.039$, $\eta_G^2 = 0.004$). Further simple main effect analysis within each condition indicated that for the lower-ASTS-20-score group, there was a significant simple main effect of emotion ($t_{(15)} = -7.223$, $p < 0.001$, Cohen's $d = 0.559$). Post-hoc comparisons showed that the P2 amplitude in response to fearful stimuli was significantly greater than for neutral stimuli in the lower-ASTS-20-score group. For the higher-ASTS-20-score group, emotion also had a significant simple main effect ($t_{(15)} = -6.70$, $p < 0.01$, Cohen's $d = 0.534$). Post-hoc analysis revealed that the P2 amplitude was significantly greater for fearful stimuli than for neutral stimuli. Further simple main effects analysis by group showed no significant difference between groups in response to neutral stimuli ($F_{(1, 31)} = 3.388$, $p = 0.076$, $\eta_p^2 = 0.101$), but a significant difference was found for fearful stimuli ($F_{(1, 31)} = 5.799$, $p = 0.022$, $\eta_p^2 = 0.162$). Post-hoc analysis indicated that, under the fearful condition, the P2 amplitude in the higher-ASTS-20-score group was significantly lower than that in the lower-ASTS-20-score group.

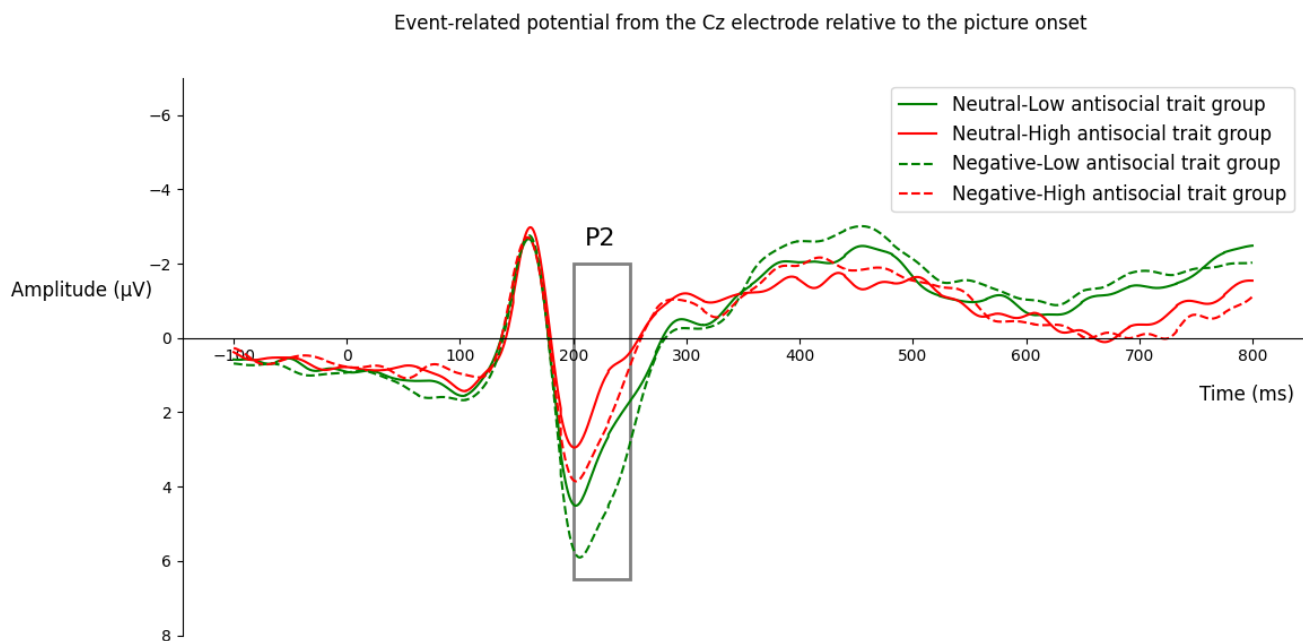


Figure 2: The P2 amplitude recorded from Cz locked to picture onset for both groups and depending on the face-stimulus category (fearful or neutral face) presented at the start of the trials.

N2 and P3 components

Three-way ANOVA (Group x Emotion x Electrode Site) was conducted to examine N2. The three-way interaction ($F_{(2, 47.05)} = 0.255$, $p = 0.722$, $\eta_G^2 < 0.001$), as well as the two-way interactions between Emotion and Group ($F_{(1.57, 47.05)} = 0.004$, $p = 0.952$, $\eta_G^2 < 0.001$), Emotion and Electrode Site ($F_{(1.57, 47.05)} = 1.914$, $p = 0.166$, $\eta_G^2 = 0.001$), and Group and Electrode Site ($F_{(1.63, 48.90)} = 2.407$, $p = 0.110$, $\eta_G^2 = 0.012$), were not significant. Similarly, no significant main effects were found for Emotion ($F_{(1, 30)} = 0.024$, $p = 0.877$, $\eta_G^2 < 0.001$) or Group ($F_{(1, 30)} = 0.349$, $p = 0.559$, $\eta_G^2 = 0.009$). However, there was a significant main effect of Electrode Site ($F_{(1.63,$

48.90) = 22.087, $p < 0.001$, $\eta_G^2=0.096$). Post hoc comparisons indicated that the N2 amplitude at the Cz site was significantly greater than at the Fz ($p < 0.01$) and Pz sites ($p < 0.01$), with the Fz amplitude also significantly greater than the Pz amplitude ($p < 0.01$).

For P3, a three-way ANOVA (Group x Emotion x Electrode Site) also showed no significant three-way interaction ($F_{(1.53, 45.74)} = 0.005$, $p = 0.985$, $\eta_G^2<0.001$) or two-way interactions between Emotion and Group ($F_{(1, 30)} = 2.033$, $p = 0.164$, $\eta_G^2=0.003$), Emotion and Electrode Site ($F_{(1.53, 45.74)} = 1.195$, $p = 0.302$, $\eta_G^2<0.001$), or Group and Electrode Site ($F_{(2, 60)} = 1.328$, $p = 0.273$, $\eta_G^2=0.006$). Additionally, the main effects for Emotion ($F_{(1, 30)} = 0.778$, $p = 0.385$, $\eta_G^2=0.001$) and Group ($F_{(1, 30)} = 1.563$, $p = 0.221$, $\eta_G^2=0.041$) were non-significant. There was, however, a significant main effect of Electrode Site ($F_{(2, 60)} = 28.122$, $p < 0.001$, $\eta_G^2=0.106$), with post hoc comparisons showing that P3 amplitude at Cz was significantly greater than at Fz ($p < 0.01$) and Pz ($p < 0.01$), and that Pz amplitude was significantly greater than Fz ($p < 0.01$).

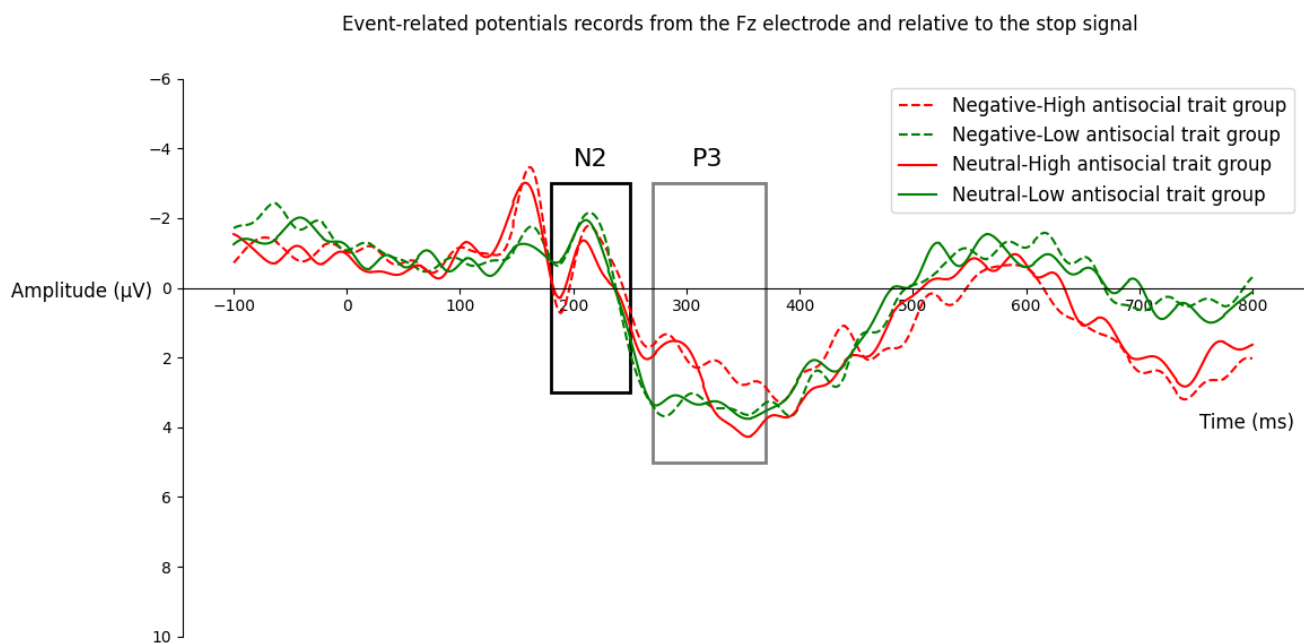


Figure 3: Comparison of N2 and P3 amplitudes recorded from the Fz electrode locked to stop signal onset for the two groups and two situational stimuli.

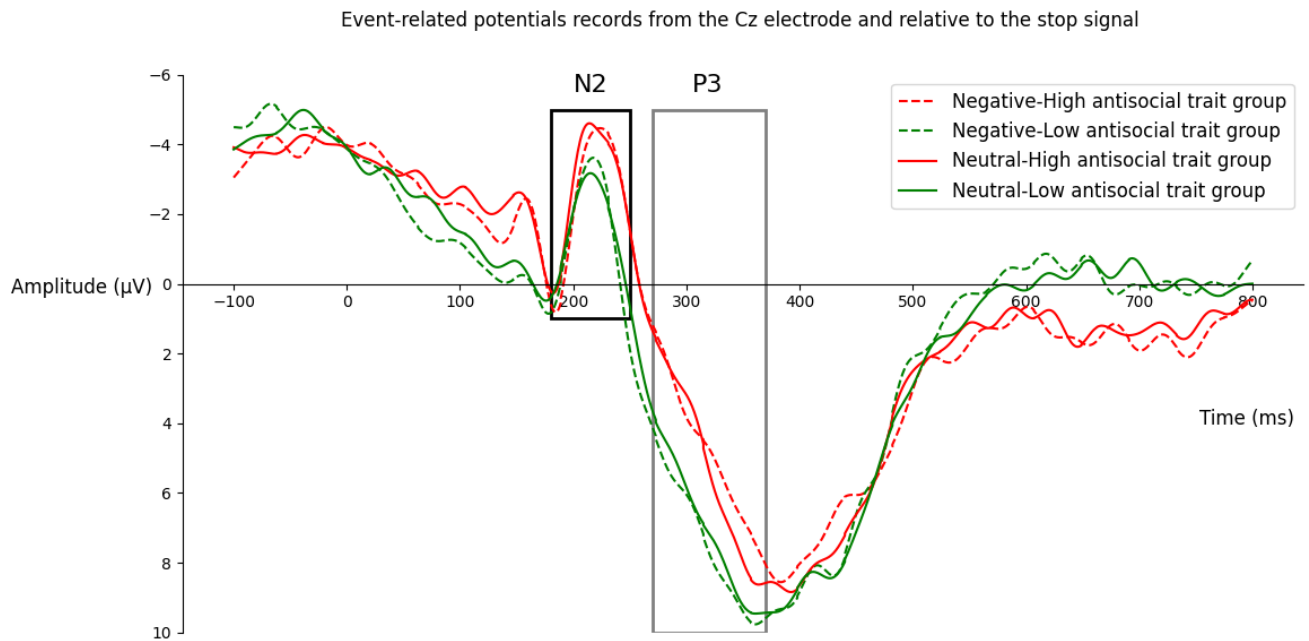


Figure 4: N2 and P3 amplitudes recorded from the Cz electrode locked to stop signal onset for the two groups and situational stimuli.

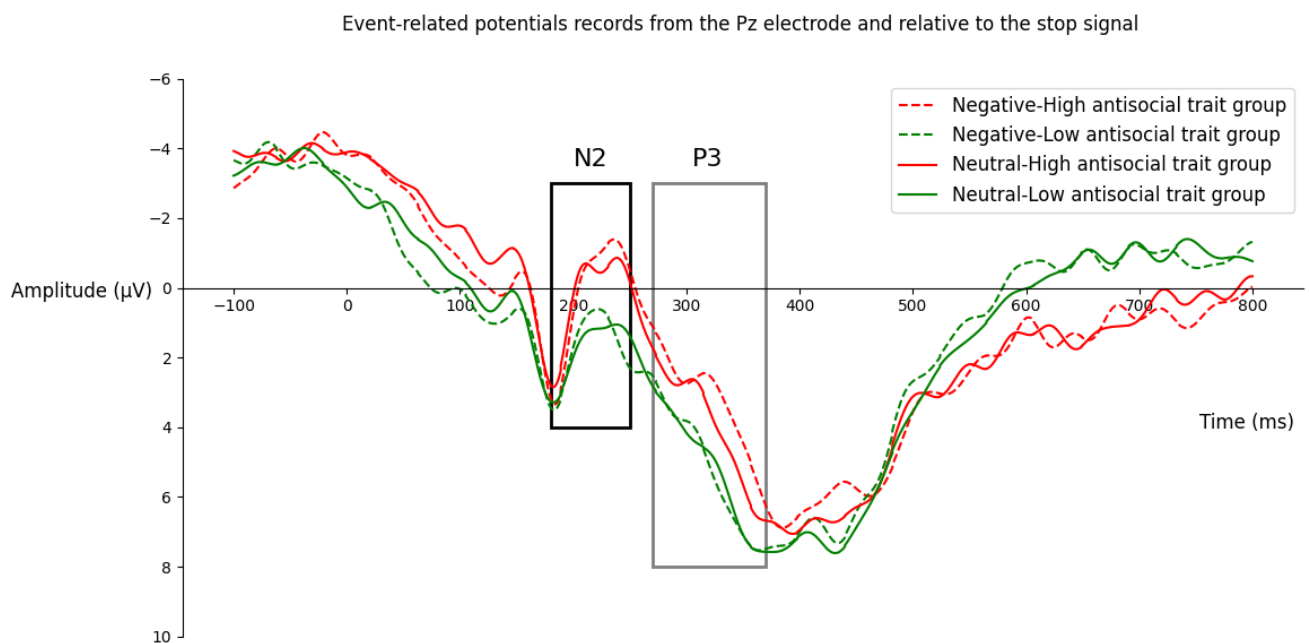


Figure 5: N2 and P3 amplitudes recorded from the Pz electrode locked to stop signal onset for both groups and both situational stimuli.

ERN and Pe components

A two-way ANOVA (Group x Emotion) was conducted to examine ERN. The analysis revealed no significant interaction between Group and Emotion ($F_{(1, 30)} = 0.900$, $p = 0.350$, $\eta_G^2 = 0.002$), and Emotion did not have a significant main effect ($F_{(1, 30)} = 0.722$, $p = 0.402$, $\eta_G^2 = 0.002$). However, there was a significant main effect of Group ($F_{(1, 30)} = 4.275$, $p = .047$, $\eta_G^2 = 0.118$), indicating that the ERN amplitude in the higher-ASTS-20-score group was significantly smaller than in the lower-ASTS-20-score group.

There was no significant interaction between Group and Emotion for Pe ($F_{(1, 30)} = 0.292$, $p = 0.593$, $\eta_G^2=0.002$), and Emotion did not have a significant main effect ($F_{(1, 30)} = 0.092$, $p = 0.763$, $\eta_G^2=0.001$). A significant main effect of Group was observed ($F_{(1, 30)} = 6.560$, $p = 0.016$, $\eta_G^2=0.154$), showing that the Pe amplitude in the higher-ASTS-20-score group was significantly greater than in the lower-ASTS-20-score group.

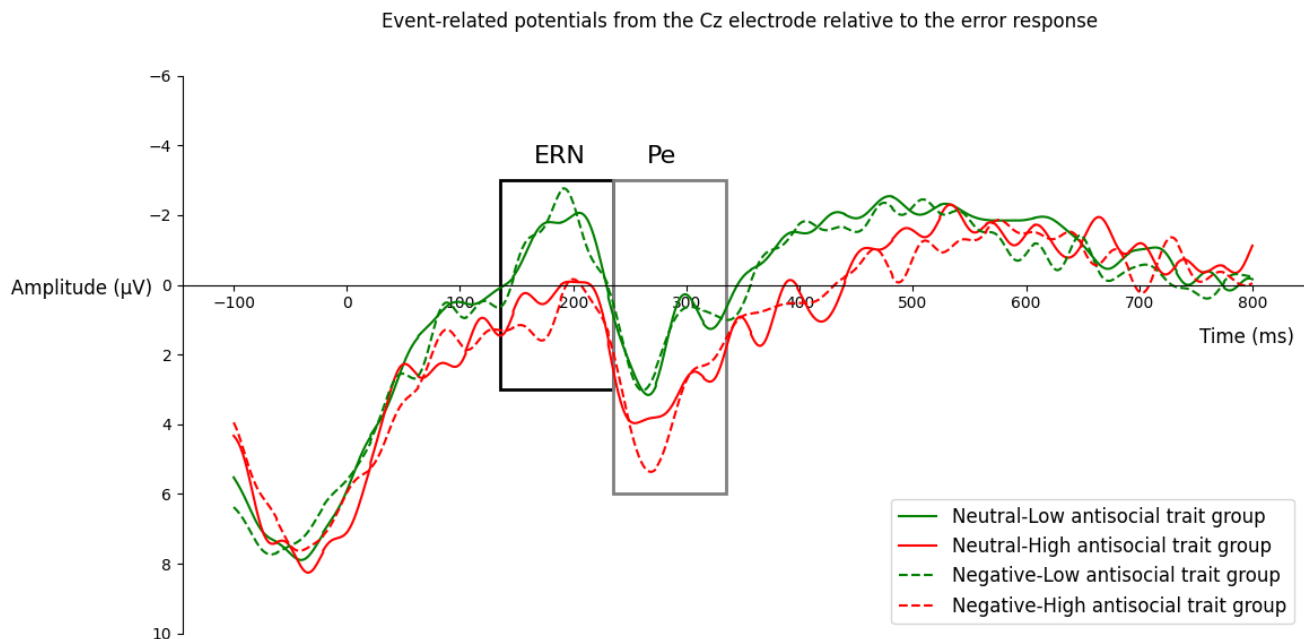


Figure 6: ERN and Pe amplitudes of participants recorded from the Cz electrode locked to the error responses and for the two types of situational stimulation (fearful and neutral faces).

ERP latency analysis

Latency data is shown in Table 3 for each of the components analyzed as described as follows. For the N2 latency at the Fz electrode, a two-way ANOVA (Group x Emotion) showed no significant interaction effect ($F_{(1, 30)} = 0.011$, $p = 0.918$, $\eta_G^2<0.001$) and no significant Group effect ($F_{(1, 30)} = 0.025$, $p = 0.876$, $\eta_G^2=0.001$). Emotion had a significant main effect ($F_{(1, 30)} = 4.285$, $p = 0.047$, $\eta_G^2=0.012$). *Post-hoc* analysis showed that the N2 latency in the fear condition was significantly longer than that in the neutral condition. The two-way ANOVA (Group x Emotion) for P3 latency at the Fz electrode showed no significant interaction ($F_{(1, 30)} = 0.507$, $p = 0.482$, $\eta_G^2=0.002$), and there were no significant main effects of emotion ($F_{(1, 30)} = 1.863$, $p = 0.182$, $\eta_G^2=0.006$) or group ($F_{(1, 30)} = 1.087$, $p = 0.306$, $\eta_G^2=0.031$).

Table 3: Latencies for each of the ERP components

Latencies in ms		Lower-ASTS-20-score Group	Higher-ASTS-20-score Group
		<i>M ± SD</i>	<i>M ± SD</i>
N2	Neutral	193.88 ± 17.58	194.63 ± 18.38
	Fear	197.44 ± 15.01	198.56 ± 19.42
P3	Neutral	289.13 ± 26.04	301.13 ± 31.78
	Fear	287.06 ± 20.96	294.56 ± 31.49
ERN	Neutral	158.63 ± 17.15	173.13 ± 22.80
	Fear	162.60 ± 22.82	184.50 ± 27.50
Pe	Neutral	254.00 ± 24.21	245.75 ± 20.51
	Fear	258.25 ± 30.54	242.88 ± 9.01

The two-way ANOVA (Group x Emotion) for ERN latency showed no significant interaction ($F_{(1, 30)} = 0.634$, $p = 0.432$, $\eta_G^2=0.008$), and no significant main effect of emotion ($F_{(1, 30)} = 2.209$, $p = 0.148$, $\eta_G^2=0.027$). However, a significant main effect was found for group ($F_{(1, 30)} = 8.420$, $p = 0.007$, $\eta_G^2=0.148$), indicating that the higher-ASTS-20-score group had a longer latency compared to the lower-ASTS-20-score group.

Finally, a box covariate equality test showed that the variance of Pe latency times between the two groups of participants was heterogeneous ($F_{(3, 162000)} = 7.497$, $p < 0.001$), so this dependent variable was not further analyzed.

Discussion

High- and low-antisocial individuals selected from a normal adult population based on Antisocial Traits Scale (ASTS) scores were tested to investigate any differences in inhibitory control and ERPs were obtained to better characterize any underlying cognitive differences associated with the different levels of antisocial traits. The task also employed cues of either emotionally fearful or neutral faces to look at differences in modulation of inhibitory control by emotional information.

Behavioral performance

No differences were seen between the two groups for performance of the inhibitory control task, nor were there any group differences due to the fearful/neutral face cues presented before trials. This may be due to the participants in this study being from a community sample group that essentially would be viewed as having normal (or, at least, not atypical) cognitive functioning with there being no other factors such as criminal behavior or a history of physical or mental illness. However, this lack of difference is consistent with some previous studies reporting no differences in behavior but there being meaningful differences in other measures, such as electrophysiology. For example, Delfin et al. (2022) saw no differences in performance of a Go/NoGo task when comparing mentally disordered offenders and normal individuals but did see differences in ERP-related measures.

ERPs – the P2 component

The P2 component was collected in relation to presentation of neutral or fearful faces that preceded the stop signal trials. In line with previous work (e.g., Carretié et al., 2001), this component was found to be of larger amplitude across participants for fearful faces than for neutral faces. Additionally, this component was of larger amplitude for the low antisocial group than the high antisocial group. This is consistent with previous suggestions that groups with higher antisocial traits may have recognition deficits for fearful face stimuli (Marsh & Blair, 2008) so it is possible that this may be the case here, although further investigation would be needed to test this. It could also mean that greater bottom-up attention processes are engaged when facing fearful face stimuli, but this is less the case for individuals with higher levels of antisocial traits.

ERPs – N2 and P3 components

Neither the N2 component nor the P3 component showed group-related or face-emotion-related differences. This seems to contrast with previous work which has associated antisocial behavior with decreased N2 and P3 amplitudes (Pasion et al., 2019). It is possible that such differences are not seen in otherwise normal individuals or that the size of any such effect would require more participants for it to be seen.

ERPs – ERN and Pe

The ERN can be viewed as an indicator of error detection and Pe can reflect the level of an individual's awareness of an error. Here, the ERN amplitude of the higher-ASTS-20-score group was significantly smaller than that of the lower-ASTS-20-score group as well as showing a significantly longer latency. This could mean that when people with higher antisocial traits make mistakes they have weaker/less effective neural responses to such mistakes, a possibility that would be useful to test.

The Pe amplitude was seen to be significantly greater for the higher-ASTS-20-score group than in the lower-ASTS-20-score group. These results may be explained by the error-awareness hypothesis, in which Pe is regarded as indicative of the degree to which an individual is aware of the error and is positively related to high alertness to misguidance information (Leuthold & Sommer, 1999), whereas smaller Pe amplitude means that individuals may underestimate the importance of errors (Overbeek et al., 2005). Again, the relationship between the pattern of the data and the potential *meaning* of the data would benefit from further investigation and, overall, the pattern of data may be indicative that higher antisocial traits are associated with less activity indicating detection of an error but assign more importance to them (but this interpretation remains to be investigated).

Questionnaire data

The TriPM questionnaire (that measures the degree of psychopathy) showed higher sub-scores related to disinhibition and meanness, in higher overall scores for the suprathreshold group, i.e., the group with high antisocial traits, were all significantly higher than those of the threshold group. No significant difference between the two groups was seen in the boldness subscale. This measure in the triadic model of psychopathy (Patrick et al., 2009) relates to an individual's exposure to situations involving stress or threats, with being able to remain calm representing a resilient ability to recover from such situations. The results of this study are consistent with those of Venables et al. (2014) and it may be that boldness is a phenotypic variable that can distinguish psychopathy from antisocial personality disorders.

Limitations

The work in this paper aims to investigate typical adult populations and, specifically, to characterize effects of the presence of high- and low-antisocial traits on the effects of emotional information and behavior on an inhibitory control task in such individuals. It is expected that the pattern of effects will be generalizable but that, for different populations, it would be necessary to ensure appropriate images are used for the emotional face stimuli. Here, these came from the Asian Emotional Face Picture Database but it would be important to ensure suitable images were employed if different populations were tested. While it is not expected that different patterns of behavior or electrophysiological results would be seen for different populations, it would certainly be interesting to see whether any patterns of differences did occur.

It is also important to note that testing in more individuals would be beneficial. While significant differences were seen in some ERP measures, this was not the case for all measures nor for behavioral measures. While it is the case that such a dissociation between behavioral effects and electrophysiology has been previously reported, more studies would be needed to increase the confidence in such a dissociation. Similarly, larger group sizes could help determine the likelihood of absence of effects being likely to be a real absence of effect versus a smaller, harder to detect difference (both for behavior and electrophysiological measures).

Conclusion

The present study examined inhibitory control and brain electrophysiology in individuals with high and low levels of antisocial traits selected from a general population sample. While no significant differences were seen in behavior, there were differences in several ERP measures, particularly including those relating to error processing, between the groups. Some of these differences seem to be inconsistent with previous studies, potentially because of the different nature of the individuals investigated with participants having neither clinical levels of the traits nor having shown criminal behavior. Future research could beneficially extend the results from this study and, in particular, to examine overlap and differences between clinical groups, criminal groups and antisocial traits. In particular, there may be additional differences that may depend on either particularly extreme levels of antisocial traits or based on additional, clinical differences. Additionally, the current study highlights specific processes which could be candidate processes for future mechanistic or intervention research. Further work to clarify the extent to which the antisocial traits of general adults in the community can be extrapolated to clinical samples would be beneficial in this context.

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