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# Differential relationships of impulsivity or antisocial symptoms on P50, N100, or P200 auditory sensory gating in controls and antisocial personality disorder

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## Abstract

Limited information is available on the relationship between antisocial personality disorder (ASPD) and early filtering, or gating, of information, even though this could contribute to the repeatedly reported impairment in ASPD of higher-order information processing. In order to investigate early filtering in ASPD, we compared electrophysiological measures of auditory sensory gating assessed by the paired-click paradigm in males with ASPD ($n$ = 37) to healthy controls ($n$ = 28). Stimulus encoding was measured by P50, N100, and P200 auditory evoked potentials; auditory sensory gating (ASG) was measured by a reduction in amplitude of evoked potentials following click repetition. Effects were studied of co-existing past alcohol or drug use disorders, ASPD symptom counts, and trait impulsivity. Controls and ASPD did not differ in P50, N100, or P200 amplitude or ASG. Past alcohol or drug use disorders had no effect. In controls, impulsivity related to improved P50 and P200 gating. In ASPD, P50 or N100 gating was impaired with more symptoms or increased impulsivity, respectively, suggesting impaired early filtering of irrelevant information. In controls the relationship between P50 and P200 gating and impulsivity was reversed, suggesting better gating with higher impulsivity scores. This could reflect different roles of ASG in behavioral regulation in controls versus ASPD.

### Keywords

Antisocial personality disorder; Impulsivity; BIS-11; Substance use disorder; Sensory gating; Information processing

## 1. Introduction

Antisocial Personality Disorder (ASPD) is a serious pathology associated with changes in evoked potential components reflecting impaired higher-order information processing (Bauer, 2001; Chang et al., 2010; Gao and Raine, 2009). Considerably less is known about relationships between ASPD and early stimulus encoding or filtering, although these processes may influence later information processing affected in ASPD (Boutros et al.,

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2004; Gjini et al., 2010). We studied pre- and early-attentional information processing in subjects with ASPD, and investigated relations with symptom severity and impulsivity, a key feature of ASPD (Swann et al., 2009).

Early information processing can be studied with the paired-click paradigm, a passive listening task in which two identical click stimuli are presented in rapid succession. The first click (S1) elicits P50, N100, and P200 auditory evoked potentials, reflecting stimulus encoding. The second click (S2) elicits corresponding, but attenuated, potentials (Fruhstorfer et al., 1970), reflecting filtering (auditory sensory gating, ASG) of information. ASG may reflect inhibitory mechanisms protecting higher-order functions from irrelevant information (Freedman et al., 1991). P50, N100, and P200 components reflect different underlying mechanisms and functions (Boutros et al., 2004; Brockhaus-Dumke et al., 2008; Crowley and Colrain, 2004; Jansen et al., 2004): P50 appears to be related to pre-attentional processes (Näätänen, 1992), N100 to early triggering of attention (Näätänen, 1992; Rinne et al., 2006), and P200 to early allocation of attention and initial conscious awareness (Näätänen, 1992).

Preliminary results showed later P50 peak latencies and reduced P50 ASG in subjects with ASPD, but not in subjects with adult-onset antisocial behavior, compared to controls (Lijffijt et al., 2009). There was also a trend for a more pronounced P50 ASG impairment in subjects endorsing more conduct disorder symptoms, similar to relationships between ASPD symptoms and changes in evoked potentials reflecting higher-order mechanisms (Bauer, 2001; Chang et al., 2010). These outcomes suggest delayed pre-attentional stimulus encoding and impaired pre-attentional filtering in ASPD.

However, study samples were small, and impaired filtering could have been moderated by co-occurring past alcohol or drug use disorders, which frequently coexist in subjects with ASPD (Goldstein et al., 2006, 2007; Krueger et al., 2002, 2005), and are also potentially related to impaired P50 gating (Boutros et al., 2006; Fein et al., 1996; Fuentemilla et al., 2009; Marco et al., 2005; Patrick et al., 1999; Rentzsch et al., 2007; Thoma et al., 2006), although not all studies showed this (Adler et al., 2001; Boutros et al., 2000a,b; Fein et al., 1996). N100 and P200 ASG have not been studied in ASPD, although subjects with substance use disorders had reduced N100 or P200 gating compared to healthy controls (Boutros et al., 2000a, 2006; Fuentemilla et al., 2009). These results suggest impaired P50, N100, or P200 gating in subjects with substance use disorders, potentially moderating gating deficits in ASPD. This would be consistent with relationships between ASPD or antisocial traits and enhanced early-attentional orienting to stimuli as measured by an increase in N100 or N100-like components, which has been interpreted as increased processing of potentially irrelevant information (Franken et al., 2005; Houston and Stanford, 2001; Liu et al., 2007). Automatic orientation to stimuli seems to be stronger in subjects who score higher on impulsivity (Franken et al., 2005; Hegerl et al., 1995), which is enhanced in subjects with ASPD (Swann et al., 2009). These results suggest ASPD could be related to increased automatic early-attentional triggering or orientation, potentially resulting in a smaller difference in N100 or P200 amplitude between S1 and S2, reflecting impaired sensory gating.

Impaired gating in ASPD might be moderated by impulsivity, a predisposition to action without planning or regard for consequences (Moeller et al., 2001) that is prominent in ASPD and anti-social behavior in general (Cale, 2006; Luengo et al., 1994; Swann et al., 2009), as well as in substance use disorders (Krueger et al., 2002; Moeller et al., 2001; Ruiz et al., 2008). Although substance use disorders and impulsivity overlap they could exert different effects on information processing in ASPD (Swann et al., 2009).

We investigated P50, N100, and P200 ASG in males with ASPD, expecting impaired P50, N100, and P200 ASG in ASPD compared to healthy controls. Deficits could be related to: 1) ASPD per se, 2) co-occurring substance use disorders, or 3) impulsivity. If impaired ASG relates to ASPD per se we would expect gating deficits irrespective of co-occurring substance use disorders, with a potential relationship between ASG and ASPD symptom count. If deficits are related to substance use disorders, we would expect impairments in subjects with combined ASPD and substance use disorders, but not ASPD only. Finally, if impaired gating is related to increased impulsivity we would expect more pronounced deficits in subjects with higher trait impulsivity. Effects of substance use disorders were investigated in subjects with ASPD with past substance use disorders because P50, N100, and P200 gating might improve during abstinence (Boutros et al., 2006), thus reducing possible confounding acute effects of substances.

## 2. Methods and materials

Study and study materials were approved by the Committee for the Protection of Human Subjects, IRB of the University of Texas Health Science Center at Houston (study number HSC-MS-05-0036), and complied with the Declaration of Helsinki. Before starting any research-related procedures subjects received in writing a thorough description of the study. After full opportunity for questions subjects provided written informed consent.

### 2.1. Participants

Subjects were recruited by advertisements in bulletins and newspapers freely available in the community. General inclusion criteria were age 18—55, and normal or corrected-to-normal vision. General exclusion criteria were history of head injury (HI) with loss of consciousness (LOC) for more than 30 min, or with reported lack of memory of the event or with lasting after-effects; history of epilepsy or migraine; current use of psychotropic medication; current alcohol or drug use disorder; history of delusions or hallucinations. Subjects with ASPD were excluded for HI occurring before onset of antisocial behavior, any axis-I disorder other than past substance use disorder, or schizoid, schizotypal or borderline personality disorder. Control subjects had to have never met criteria for any axis-I or -II disorder.

There were 28 controls and 37 subjects with ASPD. Analyses were limited to males because only 4 women with ASPD completed electrophysiological testing. Groups in this paper differed from our previous paper (Lijffijt et al., 2009): to increase sample size and generalizability controls were now allowed to have first-degree relatives with a psychiatric disorder (26 ASPD, 11 controls; no report: 4 ASPD, 6 controls), and to endorse ASPD symptoms without meeting full childhood or adulthood criteria. Six subjects with ASPD and 7 controls were in both studies.

Trained personnel administered the Structured Clinical Interview for DSM-IV axis-I Disorders (SCID-I) and axis-II Disorders (SCID-II) (First et al., 1996), complimented with updates downloaded from www.scid4.org. Diagnoses followed DSM-IV. ASPD required at least 2 symptoms for conduct disorder (CD) and 3 symptoms for adult antisocial behavior (AAB). Symptoms are not secondary to medication, substance use, or co-occurring axis-I or axis-II disorders. Additionally, subjects must have experienced dysfunction because of symptoms. The ASPD diagnosis has good internal consistency and convergence, and moderate divergence from other cluster B personality disorders (Blais and Norman, 1997). Diagnosis was confirmed by FGM or ACS. Counts of CD, AAB, or total ASPD symptoms varied between 2—14 (median 5), 3—6 (median 4), and 5—20 (median 9), respectively.

Subjects with ASPD had at least 1 conviction resulting in probation or incarceration. The most severe crime subjects were convicted for were violent (24 subjects), non-violent (9

subjects), or driving under influence (4 subjects). Lifetime history of aggression was defined as endorsing any SCID-II item addressing aggression against persons or animals in either child- or adulthood, or by conviction for an aggressive crime; 36 subjects with ASPD, and no controls, met these criteria. Co-occurring axis-I or -II diagnoses among subjects with ASPD were past alcohol use disorder (23 subjects); past drug use disorder (28 subjects: 27 marijuana, 18 cocaine); an extended period of simple bereavement (4 subjects); passive aggressive personality disorder (3 subjects); paranoid personality disorder (3 subjects); avoidant personality disorder (2 subjects); narcissistic personality disorder (2 subjects). Two subjects had attempted suicide. Subjects with past substance use disorder were in early partial (1 alcohol; 5 drug), early full (4 alcohol, 5 drug), or sustained full remission (18 alcohol, 18 drug).

Table 1 shows demographics. Completion of high school or GED was considered equivalent to 12 years of education. Subsequent completed courses for specialization in a field (eg, mechanics) lasting 6 months to a year were counted as 1 additional year of completed education. Controls had significantly more education than subjects with ASPD.

Among subjects with ASPD, 67.6% had 12 years of education. Behavioral consequences of ASPD may shorten or disrupt educational career independent of intellectual capacity (Block, 1995; Glahn et al., 2006) Therefore, intellectual ability was measured by the Shipley Institute of Living Scale (Shipley, 1940; Zachary, 1986), generating age-corrected *T*-scores for verbal (40 items), abstraction (20 items), and combined scales, and an estimate of Wechsler Adult Intelligence Scale (WAIS) IQ scores. Twenty-two controls and 34 subjects with ASPD completed the Shipley. Table 1 shows that groups did not differ on Shipley scores.

Shipley scores may not have differed due to bias in levels of education among subjects completing the Shipley. However, subjects who did not complete the Shipley had levels of education similar to those who had completed it, suggesting that 1) obtained Shipley scores are representative of the full sample, 2) level of education is most likely not a valid reflection of intellectual ability in ASPD, 3) intellectual ability is not different between groups. Finally, smoking was more prevalent in ASPD (24 subjects) than controls (7 subjects) ($\chi^2 = 10.15$, $p = .001$), and subjects with ASPD had a higher incidence of head injury without LOC over 30 min and lack of lasting after-effects (14 ASPD, 3 controls) ($\chi^2 = 6.04$, $p = .014$). During interviews subjects mentioned that head injury occurred after onset of ASPD during rough play or wild behavior or during fights. This suggests head injury may be a characteristic of course-of-illness of ASPD (Felde et al., 2006).

## 2.2. Measures

**2.2.1. Paired-click paradigm—**Eighty to 120 paired clicks were presented across 3 blocks. Clicks (40 ms, 80 dB SPL, 1000 Hz, 4 ms raise-fall) were presented binaurally through headphones using STIM software (Neuroscan, Inc., El Paso, USA). The inter-click interval was 500 ms; the inter-pair interval was 8—10 s. Test—retest reliability was moderate to high for P50, N100, and P200 latencies, amplitudes, and difference scores, low to moderate for N100 and P200 gating ratios, but low for P50 ratio (Rentzsch et al., 2008; Fuerst et al., 2007).

**2.2.2. BIS-11—**The Barratt Impulsiveness Scale, 11th revision (BIS-11) (Patton et al., 1995) is a 30-item self-report questionnaire measuring dysfunctional impulsivity (Mobini et al., 2007). The BIS-11 measures 3 interrelated subfactors: non-planning (no future sense), motor (acting without thinking), and attentional (inability to focus attention or to concentrate). Internal consistency for the total score is good across control and pathological samples ($\alpha \geq 0.79$) (Patton et al., 1995), and is somewhat lower for the subscales ($\alpha$ 0.59—

0.74) (Stanford et al., 2009). One-month test—retest reliability was good for the total score (rho 0.83), and somewhat lower for the subscales (rho 0.61—0.72) (Stanford et al., 2009).

### 2.3. Procedure

A full description of all procedures is provided as Supplemental material to this paper. In short, participation required abstention from drugs, alcohol, caffeinated products and smoking at testing. EEG was recorded from 30 electrodes (impedance below 5 kΩ; sample rate 1000 Hz, gain 1000, band-pass filter 0.1—100 Hz) referenced to linked mastoid electrodes. The ground was attached in the cap anteriorly to F3—F4. Signals were filtered off-line (1—50 Hz, 48 dB/oct roll-off) and corrected for blinks (Semlitsch et al., 1986). Artifacts were detected manually and rejected as S1 – S2 stimulus pairs. Retained pairs differed between groups (mean ± SD, range: NC = 89.9 ± 19.2, 69—118; ASPD = 104.9 ± 15.1, 70—119) ($Z = -2.35$, $p = .019$). Repeating all analyses with number of retained pairs as a covariate revealed no changes in results, suggesting no effect of group difference of retained pairs on current outcomes. Before averaging, signals were filtered with a 10 Hz high-pass filter for the P50 or with a 20 Hz low-pass filter for the N100 and P200 (Jerger et al., 1992).

Peaks were scored semi-automatically at Cz relative to preceding troughs (Boutros et al., 2004). Peaks were scored blind for group status. N100 occurred 79—149 ms, P50 occurred 35—86 ms, and P200 occurred 154—278 ms. S2 peaks were detected within 13, 48, and 98 ms of S1 P50, N100, and P200 peaks, respectively. ASG was measured as S2/S1 ratio, and S1 – S2 difference score.

### 2.4. Statistical analysis

Distributions were tested for normality using Shapiro—Wilk tests. Non-normal distributions were transformed to normalize the data. Age, Shipley, and P200 S1 and S2 latency were normally distributed; P50, N100, and P200 S1 and S2 amplitudes, P50, N100, and P200 ratios, P50 difference score, and BIS-11 scores were normalized with logarithmic transformations; education, P50 and N100 S1 and S2 latencies, and N100 and P200 difference scores could not be normalized. Normalized variables were analyzed with univariate general linear models (GLM), repeated measures GLM with Group (ASPD—control) as between-subject factor and Stimulus (S1 – S2) as within-subjects factor, or Pearson correlations, where appropriate. Non-normally distributed variables were analyzed with non-parametric tests for independent or dependent samples or Kendall's correlation, where appropriate. Effects of substance use disorders were tested with separate GLM analyses; control subjects and subjects with ASPD without histories of alcohol and drug use disorders were combined into 1 group. Potential outliers were not removed because we tried to use as many subjects of the small samples as possible. Analyses conducted with or without potential outliers revealed similar outcomes. Effect size (ES) and 95% confidence interval (95%-CI) was calculated as difference in mean for the control compared to ASPD group, divided by the standard deviation pooled across both groups, and weighted by sample size.

## 3. Results

### 3.1. Group differences

Fig. 1 shows P50, N100, and P200 evoked potentials. Table 2 shows outcomes for electrophysiology and statistical tests. P50, N100, and P200 amplitudes were smaller, and N100 and P200 peaked earlier, for S2 than for S1. Group effects were not significant (ESs < 0.5, 95%-CIs including 0). There were no effects of smoking. GLM analyses with group and history of HI revealed no differences in evoked potential or gating measures between

subjects with ($n$ = 17) or without a history of head injury ($n$ = 47) ($F$ < 3.70, $p$ > .06). This suggests history of HI with no residual after-effects did not relate to changes in sensory processing or gating.

### 3.2. Effects of past substance use disorder

Subjects had no substance use disorder (28 controls, 7 ASPD), past alcohol use disorder (2 subjects), past drug use disorder (7 subjects), and past alcohol + drug use disorder (21 subjects). Subjects with past substance use disorder were in early partial (6 subjects), early full (6 subjects), or sustained full remission (18 subjects). GLM revealed no significant effects of past substance use disorder. Dunnett tests, comparing controls with ASPD without substance use disorder or with combined alcohol + drug use disorder, revealed outcomes comparable to those obtained with GLM analysis. Duration of abstinence was not tested further due to small samples for early partial and early full remission.

### 3.3. Effects of symptom count

Table 3 shows correlations between ASPD symptom counts and P50 variables for ASPD. Symptom counts correlated significantly with P50 ratio and difference score. Fig. 2 depicts the relationship between ASPD total symptoms and P50 ratio. No significant correlations were found for N100 or P200 variables.

### 3.4. Effects of impulsivity

**3.4.1. BIS-11 and P50—**Table 3 shows group differences for BIS-11, and correlations between BIS-11 and P50 variables. Subjects with ASPD had significantly higher BIS-11 scores than controls. BIS-11 total score correlated positively with P50 ratio in subjects with ASPD, but negatively with P50 ratio in controls (Table 3). BIS-11 total score also correlated with P50 difference score and P50 S2 amplitude in controls only. These correlations differed significantly between groups ($r$-to-$z$ tests, $z$ > |2.55|, $p$ < .011).

Fig. 3 suggests a non-linear relationship between impulsivity and P50 gating across all subjects. Non-linear regression across all subjects revealed a significant quadratic relationship between BIS-11 total score and P50 ratio ($F$(1,61) = 3.70, $p$ = .031), and a marginally significant quadratic relationship with P50 difference score ($F$(1,61) = 3.09, $p$ = 3.09, $p$ = .053). Non-linear regression analyses for each group separately revealed a significant quadratic relationship between BIS-11 total score and P50 ratio only in controls, which seemed less pronounced compared to the linear relationship ($F$ = 11.46 and 5.81 for linear and quadratic relationships, respectively). These outcomes cannot distinguish between a linear, but opposite, relationship between impulsivity and P50 gating in controls and subjects with ASPD, or a quadratic relationship with controls and subjects with ASPD falling differently along the curve.

**3.4.2. BIS-11 and N100, P200—**In ASPD, BIS-11 total, motor, and attentional scores correlated significantly with N100 difference score (tau = 0.26, 0.23, 0.24, $p$ < .05), although these correlations may not differ significantly from those found in controls (tau = 0.13, 0.05, 0.17). As N100 amplitudes and difference scores were expressed as negative values, positive correlations suggest worse N100 gating with increased impulsivity. In controls, BIS-11 attentional score correlated with P200 difference score ($r$ = 0.40, $p$ = .034; ASPD $r$ = −0.08), suggesting better P200 gating with increased impulsivity. No significant relationships were found with S1 or S2 amplitudes.

**3.4.3. BIS-11 and latencies—**In controls, P50 S2 latency correlated with attentional score (tau = 0.35, $p$ = .027; ASPD tau = 0.10), N100 S2 correlated with non-planning score (tau = −0.33, $p$ = .018; ASPD tau = 0.13), and P200 S2 correlated with motor score (tau =

−0.30, $p$ =.029; ASPD tau = −0.03). As correlations may differ between ASPD and controls for the relationship between BIS-11 and P50, N100, and P200 S2 latency, outcomes suggest increased impulsivity could relate to later P50, but earlier N100 and P200 S2 peaking in controls.

### 3.5. Gating in ASPD: symptoms versus impulsivity

In ASPD, outcomes for P50 ASG showed significant relationships with symptom count and impulsivity. ASPD total symptoms correlated significantly with BIS-11 total score ($r$ = 0.42, $p$ = .009). To disentangle effects of symptoms and impulsivity, we conducted GLM analyses with P50 ratio and difference score as dependent variables, BIS-11 total score and ASPD total symptom count as continuous variables, and past substance use disorders as dichotomous variables. Total symptom count, but not BIS-11 total score, related to P50 difference score ($F$(1,29) = 7.25, $p$ = .012) and, less pronounced, to P50 ratio ($F$(1,29) = 3.36, $p$ = .077). This suggests that in ASPD P50 gating is impaired in subjects endorsing more symptoms.

## 4. Discussion

This study confirmed previously reported relationships between P50 gating and ASPD symptoms, but did not replicate P50 gating deficits in subjects with ASPD compared to controls (Lijffijt et al., 2009). BIS-11 motor score correlated with N100 difference score in ASPD, suggesting impaired N100 gating with higher impulsivity, but correlated positively with P200 gating in controls, suggesting better P200 gating with higher impulsivity. ASG measures were not affected by past alcohol or drug use disorders. These results suggest impaired early filtering of information in subjects with more severe ASPD, potentially indicating limited protection against processing irrelevant information.

### 4.1. P50 gating in ASPD

Subjects with ASPD and controls did not differ in P50 ASG or amplitudes, suggesting similar pre-attentional encoding and filtering of information across groups. However subjects with ASPD with higher BIS-11 impulsivity and more ASPD symptoms had higher P50 ratio and reduced P50 difference score, suggesting impaired pre-attentional filtering of information in subjects with more severe ASPD. GLM analyses suggested that impaired P50 gating was related to symptom count rather than impulsivity, suggesting the relationship between impulsivity and P50 gating in ASPD was moderated by ASPD symptoms. Although exact relationships need to be established, these outcomes appear similar to relationships between more ASPD symptoms and reduced sensitivity to salient information (Bauer, 2001) or errors (Chang et al., 2010).

Impaired P50 gating in subjects with more severe ASPD is also consistent with the few available studies on pre-attentional filtering using pre-pulse inhibition of the startle response, possibly reflecting pre-attentional mechanisms different from P50 ASG (Oranje et al., 2006). Pre-pulse inhibition was reduced in ASPD or antisocial behavior (Kumari et al., 2005), and correlated with increased antisocial symptoms in healthy females (Franklin et al., 2009). Taken together, this suggests that more severe ASPD may relate to impaired pre-attentional filter mechanisms, potentially resulting in less inhibition of irrelevant information that might eventually affect higher-order mechanisms.

### 4.2. Impulsivity and P50 gating

Controls and subjects with ASPD had opposite correlations between BIS-11 impulsivity and P50 ratio, suggesting that increased impulsivity is related to worse ASG in ASPD, but better ASG in controls. However, in ASPD, P50 gating appears not to be directly related to trait

impulsivity but to ASPD symptoms. In contrast, in controls, the association between impulsivity and P50 gating seems to be more direct as evidenced by the additional significant correlation between impulsivity and P50 S2 amplitude and S2 latency. For controls this might indicate increased impulsivity related to better P50 gating partially due to slower processing of S2, which might have contributed to better inhibition of the stimulus. These outcomes suggest two different pathways: one for controls between higher impulsivity and better inhibition of irrelevant information, and one for subjects with ASPD between a more severe course-of-illness and deficient inhibition of irrelevant information.

### 4.3. Early information processing: N100 and P200

In ASPD, N100 difference score correlated with impulsivity, suggesting impaired early-attentional filtering (i.e., a positive relationship with N100 amplitude or difference score indicates a lower N100 amplitude) of information with higher impulsivity and less protection against irrelevant information. Although this relationship appears not to differ from that found in controls, it seems to complement findings in subjects with higher self-reported impulsive aggression who had enhanced N100 amplitudes in a passive visual task (Houston and Stanford, 2001), in subjects with recent alcohol use disorder with co-occurring antisocial tendencies who had increased strength of dipolar sources related to the visual N100 and P200 (Hegerl et al., 1995), and with increase mismatch negativity, assessing automatic orientation to a stimulus, with increased impulsivity (Franken et al., 2005). The N100 component has been related to triggering of attention toward a stimulus (Näätänen, 1992; Rinne et al., 2006) and involuntary attention switches (Rinne et al., 2006). Thus, increased N100 component amplitude and reduced filtering could both be related to involuntary switches to irrelevant information.

In contrast, P200 difference score correlated positively with impulsivity in controls, but not in ASPD, suggesting improved early-attentional gating in control subjects with higher impulsivity (i.e., a positive relationship with P200 amplitude or difference score indicates a more pronounced P200 amplitude). As for P50 ASG, this may indicate better early-attentional filtering of irrelevant information in controls with higher impulsivity, potentially reflecting more efficient information processing suggested by earlier P200 peaking times for S2 in subjects with increased impulsivity. Additional research is needed to investigate relationships in controls between impulsivity, sensory gating, and faster early-attentional orienting (P200) or slower pre-attentional processing (P50) to S2.

### 4.4. Alcohol and drug use disorders and ASPD

Impaired P50 gating in ASPD (Lijffijt et al., 2009) might have been influenced by co-existing substance use disorders (Boutros et al., 2006; Fein et al., 1996; Fuentemilla et al., 2009; Marco et al., 2005; Patrick et al., 1999; Rentzsch et al., 2007; Thoma et al., 2006) which frequently co-occur with ASPD (Goldstein et al., 2006, 2007; Krueger et al., 2002, 2005). We selected subjects with past rather than current substance use disorders because gating deficits could attenuate with abstinence (Boutros et al., 2006), suggesting that part of gating deficits in substance use disorders are due to substance use instead of inherent to the disorder. We found no effects of past substance use disorders on any measure, suggesting that impaired gating in ASPD in association with symptom count (P50) or impulsivity (N100) was not due to potential deficits in substance use disorders.

### 4.5. Limitations

This study did not replicate impaired P50 gating in subjects with ASPD reported in our pilot study (Lijffijt et al., 2009). One possible cause is differences in control samples. To increase power and generalizability, we included males having first-degree relatives with any psychiatric history, or who had endorsed any SCID-II ASPD items without meeting ASPD

criteria. Secondly, we did not measure psychopathy which overlaps with ASPD (Basoglu et al., 2011), and with BIS-11 impulsivity (Snowden and Gray, 2011). Subjects with ASPD with higher symptom counts may be more likely to have psychopathic traits than those with fewer ASPD symptoms (Coid and Ullrich, 2010), suggesting psychopathic traits may mediate the relationship between P50 gating deficits and ASPD symptom count in ASPD. Third, almost all of the subjects with ASPD had a history of aggression, not necessarily reflected by their criminal record. Thus, effects reported in this paper might be related in part to aggression (Fresán et al., 2007) rather than ASPD or impulsivity. Finally, the reliability of the P50 ratio, the dependent variable in the majority of study on P50 gating, is low (Rentzsch et al., 2008).

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Appendix

## Appendix. Supplementary material:

Supplementary data associated with this article can be found, in the online version, at doi: 10.1016/j.jpsychires.2012.03.001.

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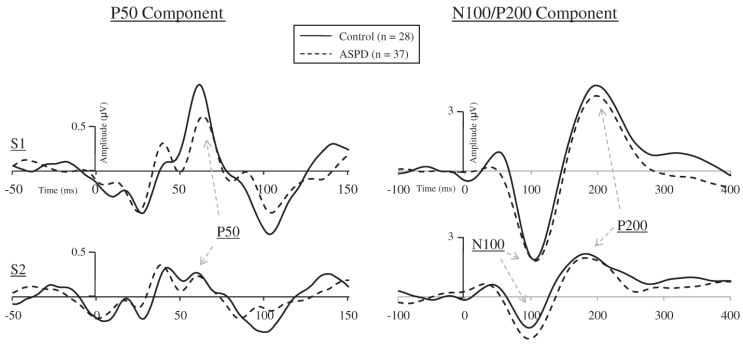


**Fig. 1.**

Averages for controls and subjects with ASPD for P50 potentials (left panel, filter 10—50 Hz), and N100 and P200 potentials (right panel, filter 1—20 Hz).

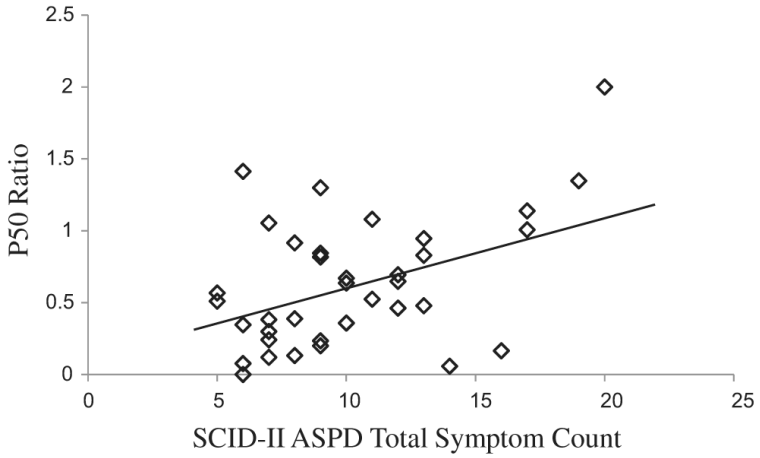


**Fig. 2.**
Relationship between ASPD total symptom count and P50 ratio ($r = 0.41$) across subjects with ASPD.

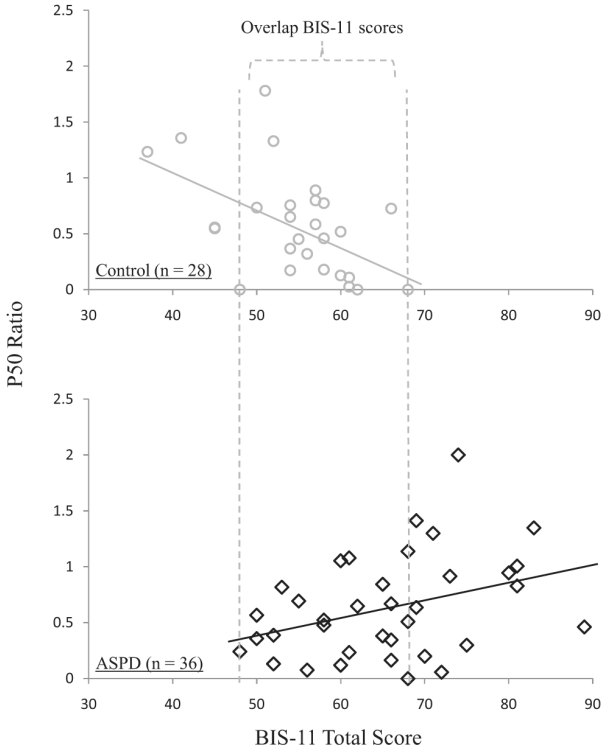


**Fig. 3.**

Relationship between BIS-11 total score and P50 ratio across controls (upper panel, $r = -0.55$) and subjects with ASPD (lower panel, $r = 0.35$).

## Table 1

Demographics for controls (NC, $n = 28$) and subjects with antisocial personality disorder (ASPD, $n = 37$)

| | NC | ASPD | Statistics |
|---|---|---|---|
| Age[a] | 32.18 (8.39) | 32.76 (8.80) | $F(1,63) = 0.07$ |
| Education (median [range]) | 13.5 (11–20) | 12 (9–14) | **$Z = -3.81$** |
| Shipley-estimated IQ[a,b] | 109.0 (11.95) | 106.74 (6.97) | $F(1,54) = 0.80$ |
| Verbal *T*-score | 49.14 (11.72) | 45.12 (7.84) | $F(1,54) = 2.37$ |
| Abstraction *T*-score | 53.91 (9.0) | 54.21 (6.23) | $F(1,54) = 0.02$ |
| Smoking ($n$ [%]) | 7 (25.0) | 24 (64.9) | **$\chi^2 = 10.15$** (df = 1) |
| Head injury[w/o] LOC ($n$ [%]) | 3 (11.1) | 14 (38.9) | **$\chi^2 = 6.04$** (df = 1) |

Bold: $p < .05$. Age, Shipley-estimated IQ, and verbal and abstraction *T*-scores are expressed as mean (SD).

[a] Statistical outcomes based on logarithmically transformed data.

[b] NC, $n = 22$; ASPD, $n = 34$.

## Table 2

Electrophysiology for controls (NC, $n = 28$) and subjects with antisocial personality disorder (ASPD, $n = 37$)

| | | Group | | Statistics | | |
|---|---|---|---|---|---|---|
| | | NC | ASPD | Stimulus (S)[a] | Group (G) | S*G |
| P50 component[b] | | | | | | |
| Latency | S1 | 60.96 ± 12.24 | 60.33 ± 14.86 | $Z = -0.71$ | N/A | $Z = -0.10$ |
| | S2 | 57.75 ± 10.71 | 59.54 ± 15.65 | | | $Z = -0.39$ |
| Amplitude | S1 | 2.51 ± 1.39 | 2.06 ± 1.16 | **F (1,62) = 57.71** | $F(1,62) = 0.29$ | $F(1,62) = 1.53$ |
| | S2 | 1.24 ± 1.07 | 1.24 ± 1.0 | | | |
| Ratio | | 55.22 ± 46.50 | 63.53 ± 45.53 | N/A | $F(1,62) = 0.68$ | N/A |
| Difference score | | 1.27 ± 1.38 | 0.90 ± 1.08 | N/A | $F(1,62) = 1.41$ | N/A |
| N100 component | | | | | | |
| Latency | S1 | 104.82 ± 11.74 | 109.95 ± 15.15 | **Z = −4.71** | N/A | $Z = -1.35$ |
| | S2 | 93.43 ± 11.07 | 98.39 ± 14.92 | | | $Z = -1.18$ |
| Amplitude | S1 | −6.54 ± 3.72 | −5.82 ± 3.38 | **F (1,63) = 112.27** | $F(1,63) = 0.01$ | $F(1,63) = 3.08$ |
| | S2 | −3.05 ± 1.53 | −3.55 ± 2.08 | | | |
| Ratio | | 53.32 ± 27.89 | 64.23 ± 27.0 | N/A | $F(1,63) = 2.69$ | N/A |
| Difference score | | −3.50 ± 2.94 | −2.27 ± 2.21 | N/A | $Z = -1.82$ | N/A |
| P200 component | | | | | | |
| Latency | S1 | 205.18 ± 27.02 | 199.95 ± 27.16 | **F (1,63) = 22.37** | $F(1,63) = 0.12$ | $F(1,63) = 0.82$ |
| | S2 | 184.89 ± 27.86 | 186.16 ± 26.47 | | | |
| Amplitude | S1 | 10.75 ± 5.41 | 9.62 ± 5.53 | **F (1,63) = 196.14** | $F(1,63) = 0.31$ | $F(1,63) = 2.27$ |
| | S2 | 4.86 ± 2.09 | 4.97 ± 2.30 | | | |
| Ratio | | 51.84 ± 25.84 | 56.16 ± 17.04 | N/A | $F(1,63) = 1.12$ | N/A |
| Difference score | | 5.89 ± 4.22 | 4.66 ± 3.93 | N/A | $Z = -1.43$ | N/A |

Bold: $p < .05$.

[a] Difference testing between S1 and S2 across groups.

[b] ASPD: $n = 36$ for P50 S2, ratio, and difference score due to $n = 1$ with P50 S1 = 0 uV.

## Table 3

Antisocial personality disorder (ASPD) symptom counts, BIS-11 impulsivity scores, and correlations with P50 S1 and S2 amplitudes, P50 S2/S1 ratio, and S1 – S2 difference score (P50 Δ)

| | Group | | Statistics | ES | Correlations (*r*) | | | |
|---|---|---|---|---|---|---|---|---|
| | **NC** | **ASPD** | | | **P50 ratio** | **P50 Δ** | **P50 S1** | **P50 S2** |
| Symptom count (median [range]) | | | | | **ASPD** [b] | | | |
| CD | 0 (0–1) | 5 (2–14) | | | 0.33 | −0.42 | −0.24 | 0.19 |
| *AAB* | 0 (0–1) | 4 (3–6) | | | *0.24* | *−0.24* | *−0.12* | *0.21* |
| ASPD | 0 (0–2) | 9 (5–20) | | | 0.41 | −0.48 | −0.24 | 0.24 |
| BIS-11 (mean [SD])[a] | | | | | **NC/ASPD** [b] | | | |
| Non-planning | 21.3 (4.5) | 24.9 (4.7) | $F(1,63) = 9.8$† | −0.79 | −0.32/0.29† | 0.23/−0.10 | −0.03/0.08 | −0.36/0.31† |
| Motor | 20.6 (3.6) | 24.2 (4.0) | $F(1,63) = 14.5$† | −0.95 | −0.37/0.24† | 0.23/−0.24† | 0.11/−0.16 | −0.21/0.09 |
| Attentional | 13.4 (3.3) | 16.2 (3.9) | $F(1,63) = 10.0$† | −0.79 | −0.55/0.31† | 0.46/−0.23† | 0.12/0.01 | −0.44/0.26† |
| Total | 55.3 (7.4) | 65.3 (10.0) | $F(1,63) = 19.5$† | −1.11 | −0.55/0.35† | 0.41/−0.23† | 0.09/−0.02 | −0.46/0.28† |

Italics: Kendall's tau instead of Pearson *r*; Underlined: $p < .05$.

† Between-group difference $p < .05$.

[a] Statistical outcomes based on logarithmically transformed data.

[b] ASPD: $n = 36$ for P50 S2 amplitude, ratio, and difference score.