Journal of Psychiatric Research 46 (2012) 743–750

Contents lists available at SciVerse ScienceDirect

Journal of Psychiatric Research

journal homepage: www.elsevier.com/locate/psychires

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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ARTICLE INFO

Article history:
Received 21 November 2011
Received in revised form
28 February 2012
Accepted 1 March 2012

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

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.

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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., 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

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0022-3956/$ – see front matter Published by Elsevier Ltd.
doi:10.1016/j.jpsychires.2012.03.001

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 antisocial 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.

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)	$\mathbf{Z = -3.81}$
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)	$\mathbf{\chi^2 = 10.15}$ (df = 1)
Head injury$^{w/o}$ LOC (n [%])	3 (11.1)	14 (38.9)	$\mathbf{\chi^2 = 6.04}$ (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, $n = 34$.