Intensity Sensitivity and Impulsivity in Antisocial Personality Disorder

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1. Introduction

Antisocial behaviors and antisocial personality disorder (ASPD) have been related to (a) increased trait impulsivity, impulsive action (poor response inhibition), and impulsive choice (more delay discounting) (Swann et al., 2009); and (b) enhanced early-attentional processing of irrelevant stimuli (Houston and Stanford, 2001), and diminished pre-attentional sensory gating (Lijffijt et al., 2012). These outcomes suggest impaired early orienting to irrelevant information in people with antisocial behaviors or ASPD which may relate to increased impulsivity.

Stimulus orienting can be measured as changes in P1, N1, or P2 evoked potential amplitudes to increased stimulus intensity (Buchsbaum and Silverman, 1986). P1 (or P50) could reflect pre-attentional stimulus processing (Näätänen, 1992), N1 could reflect early triggering of attention (Näätänen, 1992; Rinne et al., 2006), and P2 could reflect early allocation of attention and initial conscious awareness (Näätänen, 1992). More pronounced increases in amplitudes with more intense stimuli may reflect increased stimulus orientation.

In healthy controls amplitude increases are less pronounced or even blunted at the highest intensities, whereas people with impulsive aggression (Houston and Stanford, 2001) or with high trait impulsivity or hypomania symptoms (Barratt et al., 1987; Carrillo-de-la-Peña and Barratt, 1993; Heinisch et al., 2007; Lijffijt et al., submitted; Norra et al., 2003) showed a continued increase in amplitudes. This suggests increased orienting to more intense stimuli with increased trait impulsivity which might trigger impulsive actions.

We explored stimulus orientation in ASPD. We expect ASPD to have steeper intensity-amplitude slopes than controls, and people with ASPD with high trait impulsivity or more symptoms to have steeper intensity-amplitude slopes than those with low impulsivity or fewer symptoms.

2. Methods

Study and study materials were approved by the local IRB. Thirty-eight men with ASPD (First et al., 1996, 1997) (age 32.4 years ± 12.2 [SD], 23 smoking) and 18 healthy control men (age 31.5 years [SD], 12.9 years [IQR 1.1 or 1.2]; b) trait impulsivity. Barratt Impulsiveness Scale, BIS-11, that assesses total impulsivity (12). Groups differ in BIS scores (ASPD: T = 0.07, P = 0.02; NC: T = 0.07, P = 0.02). S smoking (χ² = 1.9, P = 0.06) were recruited from the general population.

For ASPD, most severe crime was non-aggressive (n = 9), violent (n = 27) or DWI (n = 2). Subjects could have histories of aggression (n = 39), suicide attempt (n = 21), of head injury with loss or consciousness (LOC) fewer than 30 minutes but without loss of memory or other adverse effects (n = 14). Subjects with substance use disorder history were at least in partial remission. Subjects used no psychoactive medication. Controls met no criteria for lifetime alcohol or drug dependence and had no history of using psychoactive medication, although 1 had a history of aggression in childhood or adolescence, and 2 had a head injury without LOC.

Assessed were (a) P1, N1, and P2 evoked potentials from Cz (0.1–30 Hz band-pass filter; linked-mastoid reference) while subjects passively listened to randomly presented 50–90 dB (100 presentations each; 1000 Hz, 40 ms; ISI 1 or 1.2 s); (b) trait impulsivity. Barratt Impulsiveness Scale, BIS-11, that assesses total impulsivity. We assessed P1, N1, and P2 amplitudes and compared them across groups.

3. Results

Table 1: Impulsivity and Intensity-Amplitude Slopes

<table>
<thead>
<tr>
<th>Trait</th>
<th>NC</th>
<th>ASPD</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIS-11 Total</td>
<td>54.2 (7.6)</td>
<td>65.8 (10.2)</td>
<td>t = 4.3</td>
</tr>
<tr>
<td>IMT Hits</td>
<td>82.3 (11.3)</td>
<td>83.2 (9.5)</td>
<td>t = 0.3</td>
</tr>
<tr>
<td>CE/Hits</td>
<td>0.33 (0.27)</td>
<td>0.39 (0.17)</td>
<td>t = 1.2</td>
</tr>
<tr>
<td>A’</td>
<td>0.36 (0.05)</td>
<td>0.84 (0.06)</td>
<td>t = -1.0</td>
</tr>
<tr>
<td>Beta</td>
<td>-0.20 (0.57)</td>
<td>-0.35 (0.44)</td>
<td>t = 0.7</td>
</tr>
<tr>
<td>TCIP Delayed</td>
<td>50</td>
<td>76</td>
<td>Z = 0.7</td>
</tr>
<tr>
<td>Cons-Delay</td>
<td>7</td>
<td>25</td>
<td>Z = 1.1</td>
</tr>
<tr>
<td>SKIP Shortest RT</td>
<td>3.3</td>
<td>2</td>
<td>Z = -1.5</td>
</tr>
<tr>
<td>Longest RT</td>
<td>157.8</td>
<td>195.9</td>
<td>Z = -0.1</td>
</tr>
</tbody>
</table>

Fig. 1: Intensity-Amplitudes across Groups

Fig. 2: P1, N1, and P2 Slopes for ASPD and NC

4. Discussion

In contrast to our expectations, intensity sensitivity for auditory information did not differ between men with ASPD and healthy controls. Intensity sensitivity did not also relate to trait impulsivity or number of ASPD symptoms.

Relationships between P2 intensity sensitivity and measures of impulsive action (IMT CE/hits) in men with ASPD suggests increased sensitivity to intense stimuli of evoked potentials reflecting orienting of attention and early conscious awareness of information related to more commission errors and diminished signal-noise discriminability. This implies increased attention to intense stimuli that subsequently enter awareness could result in impulsive actions in men with ASPD.

Although there was also a relationship between P2 slope and measures of impulsive choice (SKIP), Fig. 3 (bottom panel) suggests this may only be for people with ASPD who are able to delay responding, which seems to be uncommon in this group.

Although not significant, in controls it appears increased sensitivity to more intense stimuli of evoked potentials reflecting triggering of attention towards a stimulus (P1N1 slope) may relate to less commission errors and better signal-noise discrimination. This implies less impulsive actions with better triggering of attention to more intense stimuli in controls.

Outcomes:

(a) Main effects of Intensity for N1, P2, P1N1, and N1P2 (F(4,216) = 45.89, p < 0.001) indicates more pronounced amplitudes with higher intensities (Fig. 1). No Effects of Group (F(1,54) < 3.1, P > 0.08) or Group x Intensity (F(4,216) = 2.3, P > 0.09) were found.

(b) This latter finding is also reflected by a lack of significant group differences in slopes (Fig. 2).

(c) BIS-11, but not the other impulsivity measures, differed between groups (Table 1).

(d) In ASPD, IMT CE/Hits, A’, and SKIP shortest delay correlated significantly with P2 slopes (Fig. 3).

(e) In controls, IMT CE/Hits and A’ correlated with P1N1 slope (r = 0.38 and -0.42, ns), although these relationships were not significant (ASPD: r = 0.07 and -0.02, respectively).

(f) ASPD symptoms did not correlate with intensity-amplitude slopes (r < 0.12).

Outcomes:

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Acknowledgements

References

11. Lijffijt M et al. (submitted). Trait impulsivity and intensity sensitivity of N1 and P2 auditory evoked potentials in bipolar disorder and controls.