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Antisocial and borderline personality disorder symptomatologies are associated with decreased prepulse inhibition: The importance of optimal experimental parameters

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ABSTRACT

Although antisocial personality disorder (ASPD) and borderline personality disorders (BPD) have been hypothesized to be associated with decreased prepulse inhibition (PPI) of the acoustic startle response, empirical support for this contention has been inconsistent. Accordingly, we measured symptoms of ASPD, BPD, and a common feature of both disorders – alcohol dependence symptomatology – in a sample of 53 nonclinical college females using the MCMI-III, and then correlated their scores with their PPI levels. Results indicated that all constructs were intercorrelated ($p < .001$), and that all constructs were negatively correlated with PPI of amplitude ($p < .05$), but only at a signal-to-noise ratio (SnR) of +15 dB. These findings suggest that, even in a nonclinical sample, ASPD, BPD, and alcohol dependence symptomatology are associated with decreased PPI, and further specify that a SnR of +15 dB and other optimal experimental parameters should be employed when investigating these associations.

1. Introduction

The acoustic startle reflex is a protective response that occurs in reaction to a sufficiently sudden and intense sound. In humans, startle reactivity is usually quantified as the electromyographic (EMG) activity of the orbicularis oculi, the muscle that causes the eye to blink (Blumenthal & Franklin, 2009). Prepulse inhibition (PPI) of the startle reflex occurs when a stimulus (i.e., the prepulse) precedes the startle-eliciting stimulus by 30–500 ms and, subsequently, inhibits the startle response (Blumenthal, 1999). Over the past 30 years, PPI has proved to be a valuable index of neurocognitive dysfunction, such that decreased PPI is associated with decreased frontal activity, abnormal striato-limbic activity, and poorer information processing (Campbell et al., 2007; Kumari, Antonova, & Geyer, 2008; Swerdlow, Geyer, & Braff, 2001). Consistent with evidence of such neurocognitive dysfunction in patients diagnosed with schizophrenia, decreased PPI has been found across much of the schizophrenia spectrum (e.g., Cadenhead, Swerdlow, Shafer, Diaz, & Braff, 2000; Duncan et al., 2006), and is considered by some to represent an endophenotype of schizophrenia (Braff & Freedman, 2002).

Recently, however, studies have indicated that decreased PPI may be associated with a greater range of psychopathology than was initially suspected. In the last 15 years, decreased PPI has been found in several internalizing disorders (e.g., Franklin, Bowker, & Blumenthal, 2009; Grillon, Morgan, Southwick, Davis, & Charney, 1996; Hoenig, Hochrein, Quednow, Maier, & Wagner, 2005; Ludewig et al., 2005), with initial reports also suggesting that decreased PPI is indicative of externalizing disorders (Castellanos et al., 1996; Grillon, Sinha, Ameli, & O'Malley, 2000; Kumari et al., 2005). In light of these associations between decreased PPI and both internalizing and externalizing constructs, it is apparent that: (1) decreased PPI should be considered a *sensitive*, rather than *specific*, correlate of psychotic disorders; and (2) in addition to psychosis, decreased PPI also may be sensitive to emotion dysregulation, which is an underlying feature of both internalizing and externalizing pathology (Clark, 2007; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Linehan, 1993). However, further tests of this latter hypothesis, particularly within the externalizing spectrum, are needed. Given that antisocial personality disorder (ASPD), borderline personality disorder (BPD), and alcohol dependence (AD) are strongly associated with externalizing symptomatology (Krueger

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et al., 2007) and may be associated with decreased frontal and abnormal striato-limbic activity (Blair, Mitchell, & Blair, 2005; Johnson, Hurley, Benkelfat, Herpertz, & Taber, 2003; Swerdlow, Braff, & Geyer, 2000), these three interrelated constructs may be associated with decreased PPI. Consistent with this hypothesis, Kumari et al. (2005) found that nine male inpatients diagnosed with ASPD had decreased PPI relative to a nonclinical control group and, moreover, that PPI in the ASPD group was negatively correlated with violence ratings. However, this study is the only published report of an ASPD–PPI association, with no replications of this finding in larger, nonclinical, or female samples.

Only two studies have investigated the association between BPD and PPI, with both concluding that there is no BPD–PPI association (Grootens et al., 2008; Herpertz & Koetting, 2005); nevertheless, both of these studies employed nonoptimal experimental parameters that might have made it difficult to detect a BPD–PPI association. In methodological guidelines papers for startle research (Blumenthal, Elden, & Flaten, 2004; Blumenthal et al., 2005; Braff, Geyer, & Swerdlow, 2001; Franklin, Moretti, & Blumenthal, 2007; Franklin et al., 2009), researchers have made several recommendations that were not followed in these two studies. First, Blumenthal et al. (2005) advised that EMG activity be filtered with a 28–500 Hz band-pass filter to avoid 'aliasing', a process by which EMG signals above 500 Hz are folded back into the EMG frequency range, thereby contaminating the signal. As Herpertz and Koetting (2005) used a 100–1000 Hz band-pass filter, their data may have been contaminated with EMG folding artifacts. Second, Braff et al. (2001) suggested that group differences are best observed at stimulus onset asynchronies (SOA; elapsed time from prepulse onset to startle stimulus onset) of 120 ms. Given that Herpertz and Koetting (2005) employed a SOA of 70 ms and Grootens et al. (2008) utilized a SOA of 100 ms, these studies may have been limited in their ability to detect group differences in PPI. Third, Blumenthal et al. (2004) found that the proportion of difference from control (PDC; [prepulse magnitude − startle-alone magnitude]/startle-alone magnitude) PPI quantification method provided the most protection from contamination by variations in baseline startle reactivity (and thus, the most pure index of PPI). Neither Herpertz and Koetting (2005) nor Grootens et al. (2008) used the PDC quantification method and, consequently, may not have adequately indexed PPI.

Fourth and finally, Franklin et al. (2007, 2009) suggested that a signal-to-noise ratio (SnR; prepulse intensity − background noise intensity) of +15 dB is optimal for detecting PPI group differences. As Herpertz and Koetting (2005) utilized a +18 dB SnR (70 dB prepulse over 52 dB background), their ability to detect group differences may have been compromised. Interestingly, although Grootens et al. (2008) included a +25 SnR (75 dB prepulse/50 dB background) in which the BPD group displayed similar PPI relative to a control group, they also included a +15 dB SnR (65 dB prepulse/50 dB background) condition in which they found a nonsignificant trend for decreased PPI in the BPD group. Moreover, although studies indicate that there may not be large differences in absolute PPI levels between background noise intensities of 50 and 70 dB (Blumenthal, Noto, Fox, & Franklin, 2006; Franklin et al., 2007), it may be that these differences (and the resultant differences in prepulse intensity) affect the ability to detect group differences in PPI (Braff et al., 2001). Taken together, an analysis of these two studies indicates that the BPD–PPI association has heretofore been inadequately tested.

Consistent with the fact that there is substantial overlap between the neural circuits that modulate PPI and the reinforcement of most drugs (Swerdlow et al., 2000), decreased PPI has been found in the offspring of individuals with AD symptomatology (Grillon, Sinha, Ameli, & O'Malley, 2000), as a result of the acute administration of alcohol (Hutchinson, MGeary, Wooden, Blumenthal, & Ito, 2003) and in alcohol-dependent participants in withdrawal (Keedwell, Kumari, Poon, Marshall, & Checkley, 2001). In addition, Grillon et al. (1996) found that a group of 21 combat veterans diagnosed with PTSD demonstrated significantly decreased PPI relative to a noncombat control group, and reported that 10 members of this patient group had a history of AD, although none had used alcohol during the three months prior to the study. Despite these promising findings, there has yet to be a direct examination of the association between AD symptomatology and PPI.

The purpose of the present study was to: (1) replicate findings of decreased PPI in ASPD in a nonclinical female sample; (2) test the hypothesis that BPD symptomatology is associated with decreased PPI when optimal experimental parameters are utilized, even in a nonclinical sample; and (3) directly test the possibility that AD symptomatology is associated with decreased PPI. Accordingly, this study not only has the potential to strongly establish that decreased PPI is associated with externalizing symptomatology and to accordingly provide new avenues of research in this area, but it also has the potential to demonstrate that certain experimental parameters may be necessary to detect these associations.

2. Methods

2.1. Participants

Female participants ($N = 53$) ranging from 18–22 years of age were randomly selected from a group of introductory psychology students earning credit for a research participation option. Because BPD is primarily diagnosed in females (APA, 1994) and we sought to replicate the findings of Kumari et al. (2005) in a female sample, the sample of the present study only included females. Participants signed an informed consent form and all procedures were approved by the Institutional Review Board of Wake Forest University. In the first portion of the experiment, participants completed the third version of the Millon Multiaxial Clinical Inventory (MCMI-III; Millon, Davis, & Millon, 1996). One to six weeks later, they completed the startle portion of the experiment. None of the participants in the startle portion indicated that they had any hearing-related illnesses or psychiatric diagnoses, used any psychoactive medication, or used any alcohol, tobacco, or caffeine within 4 h prior to the experiment.

2.2. Stimuli

Startle stimuli were 105 dB(A) broadband noises (20 Hz–20KHz), with a 50 ms duration and a rise/fall time of <1 ms. Prepulses were 75, 80, and 85 dB(A) broadband noises, each with a 40 ms duration and a rise/fall time of 5 ms. As recommended by Braff et al. (2001), the SOA for each trial was 120 ms. Consistent with convention (Braff et al., 2001) and empirical studies (Blumenthal et al., 2006; Franklin et al., 2007), background noise was a continuous 70 dB(A) broadband noise present during the entire testing session. The respective prepulse and background noise intensities resulted in three SnR conditions: +5, +10, and +15 dB(A). Intertrial intervals varied randomly from 14 to 23 s. All stimuli were generated by Coulbourn S-series noise generators, gated through Coulbourn rise/fall gates, amplified by Coulbourn audio mixer amplifiers, and presented to the participants through Telephonics TDH-39 headphones. Stimulus intensities were calibrated with steady-state signals presented through the headphones and measured with a Quest sound level meter with a fitted earpiece.

2.3. Response measures

Eyeblink EMG responses were measured from the orbicularis oculi muscle with In Vivo Metric surface recording electrodes