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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

(Ag/AgCl, 11 mm outer diameter, 4 mm inner diameter contact surface) placed below the left eye. EMG activity of this muscle was amplified with a Biopac EMG amplifier and sampled (1000 Hz) by a Biopac MP150 workstation which stored four versions of the EMG input: raw unfiltered EMG, filtered EMG in a passband of 28–500 Hz (as advised by Blumenthal et al. (2005)), a rectification of the filtered EMG signal, and a rectified and smoothed (five sample boxcar filter) derivation of the filtered signal. The data reported in this paper are based on the smoothed EMG signal.

2.4. Self-report measures

To measure ASPD, BPD, and AD symptomatology, we employed the MCMI-III (Millon et al., 1996), which was designed to be in accord with the DSM-IV (APA, 1994), though it includes some scales for personality disorders found in the DSM-III-TR (APA, 1987; Strack, 1999). The MCMI-III consists of 175 true/false items, which compose 10 clinical syndrome scales and 14 personality disorder scales. These 24 scales have shown good reliability and validity (Davis, Wenger, & Guzman, 1997; Millon et al., 1996). In this study, we used the ASPD, BPD, and AD scales. We utilized the MCMI-III because of its comprehensiveness and specificity to DSM-IV constructs. The MCMI-III has been used widely in a variety of research and clinical settings, but is expressly not for diagnostic use in a normative population because its norms are based on a psychiatric sample (Millon et al., 1996; Strack, 2005). In the present study we were not concerned with diagnoses or clinically significant levels of disorders; rather, we were interested in variations on these scales in a normative sample, and in how scores on these scales vary with PPI of acoustic startle. The MCMI-III has been used in several studies with normative populations that also were primarily concerned with the covariance of MCMI-III scores with other constructs, rather than diagnoses (e.g., Franklin et al., 2009; McCann et al., 2001; Stredny, Archer, & Mason, 2006).

2.5. Procedure

In the first portion of the study, groups of 15–20 participants signed an informed consent form and were given 1 h to complete the MCMI-III. The MCMI-III contains a validity scale and no participants reached the cut-off for 'invalidity' as recommended by Millon et al. (1996). In the startle portion of the experiment, participants were seated individually in a sound-attenuated room, where they read and signed an additional informed consent form and filled out a brief medical history questionnaire. The skin on the left temple and below the left eye was cleaned with a cotton swab dipped in rubbing alcohol. Surface recording electrodes filled with Synapse conducting paste were then placed on the cleaned areas; one electrode was attached to the skin overlaying the orbicularis oculi muscle directly below the pupil, but below the lower eyelid, and another electrode was placed approximately 15 mm (center to center) lateral to and slightly higher than the other electrode. The ground electrode was placed on the skin overlaying the left temple. Headphones were then comfortably placed on the participant (see Franklin et al., 2007).

Background noise was then turned on (and remained on throughout the session) and participants were given five minutes to acclimate to it before any other stimuli were presented. Consistent with previous studies (e.g., Blumenthal et al., 2006; Franklin et al., 2007), three habituation trials of a 105 dB(A) startle stimulus were then presented (these trials are not included in the analyses). Then the session of 32 trials, each containing a startle stimulus, was presented. Each session was composed of eight blocks, with each block containing a random order of a control trial (no prepulse), and 75, 80, and 85 dB(A) prepulse trials. This resulted in eight trials for each of the four stimulus conditions. Block order was counterbalanced across participants. After the session, participants were debriefed, given credit, and allowed to leave.

2.6. Data analysis

Blink response amplitude was calculated for each stimulus condition (see Blumenthal et al., 2005). Response amplitude was the average of the difference between peak and onset voltage of the smoothed EMG, within a window of 20–150 ms after stimulus onset, for all trials on which a response was detected (3.7% of trials were deleted due to movement artifacts or unstable baselines). Because eight participants failed to respond on any trials in one of the stimulus conditions, the results are based on data from 45 participants.

PPI was calculated using the PDC (proportion of difference from control) method for response amplitude, as recommended by Blumenthal et al. (2004). The resulting data were multiplied by −1 so that negative correlations with self-report measures would signify a decrease in PPI. The effect of prepulse intensity was evaluated with repeated measures analysis of variance (ANOVA); Greenhouse–Geisser degrees of freedom were used to test for significance, but uncorrected degrees of freedom are reported.

The scores on the MCMI-III scales were calculated and intercorrelated. Scores on these scales were then correlated with PPI levels. All correlations were one-tailed Pearson correlations, and all alpha levels were .05.

3. Results

Correlational analyses revealed that the ASPD, BPD, and AD scales were all significantly positively intercorrelated ($p < .001$; see Table 1). Correlational analyses also showed that, whereas all three constructs were significantly negatively associated with PPI in the +15 dB(A) SnR condition ($p < .01$; see Table 2), only ASPD was associated with PPI in the +10 dB(A) SnR condition ($p < .05$; see Table 2), and no constructs were significantly associated with PPI in the +5 dB(A) SnR condition ($p > .05$; see Table 2). Indeed, in all cases the correlation between MCMI-III constructs and PPI increased in magnitude as SnR approached +15 dB(A) (see Table 2). Additionally, consistent with previous reports (Blumenthal et al., 2006; Franklin et al., 2007), a repeated measures analysis of variance (ANOVA) showed that there was a significant main effect of prepulse intensity on PPI of startle response amplitude, $F(2,88) = 35.52$, $p < .001$, epsilon = .765, with PPI increasing as prepulse intensity increased from +5 dB ($M = -.49$; SE = .04), to +10 dB ($M = -.68$; SE = .03), to +15 dB ($M = -.73$; SE = .03).

4. Discussion

In the present study, we examined the association between PPI of acoustic startle and symptoms of ASPD, BPD, and AD in a nonclinical sample of females. All hypotheses were supported, as each of these constructs was significantly negatively correlated with PPI and these associations became stronger at a SnR of +15 dB(A). Taken together, these results suggest that these three externalizing

Table 1
One-tailed intercorrelations between MCMI-III Scales.

	ASPD	BPD	Alcohol Dep.
ASPD	1	.688*	.860*
BPD		1	.634*
Alcohol Dep.			1

* $p < .001$.

Table 2
One-tailed correlations between MCMI-III Scales and PPI Variables.

	ASPD	BPD	Alcohol Dep.
PPI Amp 75 dB	−.192	−.141	−.187
PPI Amp 80 dB	−.283*	−.153	−.201
PPI Amp 85 dB	−.371**	−.364**	−.351**

* $p < .05$.
** $p < .01$.

spectrum constructs are associated with decreased PPI and that, to observe this association, optimal experimental parameters must be utilized.

The finding of a negative correlation between ASPD and PPI is consistent with the results of Kumari et al. (2005), who used similar parameters to the present study. The present study extends these findings by demonstrating decreased PPI in a relatively large nonclinical sample of college females with increasing ASPD symptomatology. Indeed, the addition of the results of the present study to those of Kumari et al. (2005) suggests that the ASPD–PPI association is robust, implying that the neurocognitive deficits indicative of PPI (see Braff et al., 2001; Campbell et al., 2007; Swerdlow et al., 2001) may underlie a tendency towards antisocial behaviors. However, as there may be two distinct groups of people with ASPD – those with impulsive symptomatology and those with psychopathic symptomatology (Blair et al., 2005; Patrick, Bradley, & Lang, 1993) – decreased PPI may only be associated with a subset of individuals with ASPD. Given that the neurological profile of the impulsive subset has a greater overlap with the neurological correlates of PPI than the psychopathic subset (see Blair et al., 2005; Swerdlow et al., 2001), it may be that decreased PPI can serve as a biological marker that distinguishes between the two subsets. In any case, the ASPD–PPI association deserves more study.

The present study is the first to demonstrate that BPD symptomatology is associated with decreased PPI. Although this finding converges with reports of neurocognitive deficits in BPD (e.g., Johnson et al., 2003) that overlap with the neurocognitive substrates of decreased PPI (Braff et al., 2001; Campbell et al., 2007; Kumari et al., 2008; Swerdlow et al., 2001), they contradict earlier reports of normal PPI in individuals diagnosed with BPD (Grootens et al., 2008; Herpertz & Koetting, 2005). As discussed earlier, this discrepancy is likely due to methodological differences between earlier reports and the present study. This contention is supported by the fact that, across the three studies, as experimental parameters approach optimal levels, the hypothesized BPD–PPI association becomes stronger. Although both earlier studies included several nonoptimal parameters, relative to Herpertz and Koetting (2005), Grootens et al. (2008) used a more appropriate SOA, band-pass filter, and PPI quantification method, and included a +15 dB(A) SnR condition. Accordingly, whereas Herpertz and Koetting (2005) found no indication of decreased PPI in their BPD group ($F[1,50] = .005$, $p = .82$), Grootens et al. (2008) found a nonsignificant trend for decreased PPI in their BPD group in the +15 dB(A) condition ($F[1,61] = 2.71$, $p = .11$), but not in the +25 dB condition ($p > .2$). Further optimizing experimental parameters, the present study demonstrated significantly decreased PPI in association with BPD symptomatology, but only in the +15 dB(A) condition ($p < .01$; see Table 2). As such, the present study suggests that BPD symptomatology is associated with decreased PPI, but specifies that optimal parameters must be employed to observe this association.

The present study also found that AD symptomatology was associated with decreased PPI. This result is consistent with reports of decreased PPI in the children of individuals with AD symptomatology (Grillon et al., 2000), in alcohol-dependent participants in withdrawal (Keedwell et al., 2001), after the administration of alcohol (Hutchinson et al., 2003), and in a group of veterans diagnosed with PTSD, half of whom had a history of AD (Grillon et al., 1996). This finding represents the first direct demonstration that AD symptomatology is associated with decreased PPI and, as with ASPD and BPD symptomatology, indicates that this association is best observed under specific experimental parameters. Converging with studies on the neural substrates of addiction propensity, these findings suggest that the neurocognitive deficits associated with PPI may underlie AD symptomatology (Swerdlow et al., 2000).

The results of the present study should be interpreted in accord with its limitations, namely the use of a nonclinical sample of college females, correlational design, and self-report measures. First, we employed a normative female sample because we wanted to index BPD symptomatology and avoid the drug and medication confounds that are more likely in a clinical sample, and because previous studies had included only clinical samples (and Kumari et al. (2005) only included males). Additionally, we sought to rigorously test the hypothesis that ASPD, BPD, and AD symptomatology are associated with decreased PPI, and the use of a nonclinical sample best satisfied this aim. Nevertheless, future studies should examine PPI in both male and female participants with clinically significant levels of ASPD, BPD, and AD. Second, because this study was largely exploratory, we used a simple correlational design that prevented us from inferring the directionality of the PPI – externalizing spectrum relationships. However, studies that separate groups based on clinical diagnoses use essentially the same correlational design, with the only difference being that such studies have categorical independent variables (i.e., group membership), whereas the present study had continuous independent variables (i.e., scores on constructs). Consequently, one might argue that the design of the present study is as strong as, if not stronger than, the design of other studies in this area (see MacCallum, Zhang, Preacher, & Rucker, 2002). Nonetheless, longitudinal studies examining the co-development of symptomatology and decreased PPI are needed to further examine the nature of PPI-symptomatology associations. Third and finally, in order to collect information on a large number of constructs within a short amount of time, the present study utilized a self-report measure, which is subject to self-report bias. Although we obtained promising results using this method with the MCMI-III, future studies may benefit from using other more specific questionnaires (e.g., a more comprehensive BPD measure), structured clinical interviews, and observational methods to index these constructs.

To summarize, the present study indicates that ASPD, BPD, and AD symptomatology are associated with decreased PPI, even in a nonclinical sample. As these constructs are strongly interrelated and have been proposed to represent externalizing psychopathology (Clark, 2007; Krueger et al., 2007), the present findings imply that decreased PPI may be a feature of externalizing psychopathology in general. Moreover, these findings support the hypothesis that decreased PPI is generally associated with emotion dysregulation, regardless of its specific clinical presentation (i.e., internalizing or externalizing). Importantly, the findings of the present study emphasize the need to utilize information about the optimal experimental parameters for PPI studies. Future studies are needed both to replicate and extend the present findings and, additionally, to further specify the optimal parameters of PPI studies.

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References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: American Psychiatric Association.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Blair, J., Mitchell, D., & Blair, K. (2005). *The psychopath: Emotion and the brain*. Malden, MA: Blackwell Publishing.

Blumenthal, T. D. (1999). Short lead interval modification. In M. E. Dawson, A. M. Schell, & A. H. Boehmelt (Eds.), *Startle modification: Implications for neuroscience, cognitive science, and clinical science* (pp. 51–71). Cambridge, UK: Cambridge University Press.

Blumenthal, T. D., Cuthbert, B. N., Filion, D. L., Hackley, S., Lipp, O. V., & van Boxtel, A. (2005). Committee report: Guidelines for human startle eyeblink electromyographic studies. *Psychophysiology, 42*, 1–15.

Blumenthal, T. D., Elden, A., & Flaten, M. A. (2004). A comparison of several methods used to quantify prepulse inhibition of eyeblink responding. *Psychophysiology, 41*, 326–332.

Blumenthal, T. D., & Franklin, J. C. (2009). The startle eyeblink response. In E. Harmon-Jones & J. S. Beer (Eds.), *Methods in social neuroscience* (pp. 92–117). New York, NY: Guilford Press.

Blumenthal, T. D., Noto, J. V., Fox, M. A., & Franklin, J. C. (2006). Background noise decreased both prepulse elicitation and inhibition of acoustic startle blink responding. *Biological Psychology, 72*, 173–179.

Braff, D. L., & Freedman, R. (2002). The importance of endophenotypes in studies of the genetics of schizophrenia. In K. L. Davis, D. Charney, J. T. Coyle, & C. Nemeroff (Eds.), *Neuropsychopharmacology: The fifth generation of progress* (pp. 703–716). Baltimore, MD: Lippincott, Williams & Wilkins.

Braff, D. L., Geyer, M. A., & Swerdlow, N. A. (2001). Human studies of prepulse inhibition of startle: Normal subjects, patient groups, and pharmacological studies. *Psychopharmacology, 156*, 234–258.

Cadenhead, K. S., Swerdlow, N. R., Shafer, K. M., Diaz, M., & Braff, D. L. (2000). Modulation of the startle response and startle laterality in relatives of schizophrenia patients and in subjects with schizotypal personality disorder: Evidence of inhibitory deficits. *American Journal of Psychiatry, 157*, 1660–1668.

Campbell, L. E., Hughes, M., Budd, T. W., Cooper, G., Fulham, W. R., Karayanidis, F., et al. (2007). *European Journal of Neuroscience, 26*, 2327–2333.

Castellanos, F. X., Fine, E. J., Kaysen, D., Marsh, W. L., Rapoport, J. L., & Hallett, M. (1996). Sensorimotor gating in boys with Tourette's syndrome and ADHD: Preliminary results. *Biological Psychiatry, 39*, 33–41.

Clark, L. A. (2007). Assessment and diagnosis of personality disorder: Perennial issues and emerging conceptualization. *Annual Review of Psychology, 58*, 227–258.

Davis, R. D., Wenger, A., & Guzman, A. (1997). Validation of the MCMI-III. In T. Millon (Ed.), *The Millon inventories: Clinical and personality assessment* (pp. 327–359). New York, NY, US: Guilford Press.

Duncan, E. J., Bollini, A. M., Lewison, B., Keyes, M., Jovanovic, T., Gaytan, O., et al. (2006). Medication status affects the relationship of symptoms to prepulse inhibition of acoustic startle in schizophrenia. *Psychiatry Research, 145*, 137–145.

Franklin, J. C., Bowker, K. B., & Blumenthal, T. D. (2009). Anxiety and prepulse inhibition of acoustic startle in a normative sample: The importance of signal-to-noise ratio. *Personality and Individual Differences, 46*, 369–373.

Franklin, J. C., Moretti, N. A., & Blumenthal, T. D. (2007). Impact of stimulus signal-to-noise ratio on prepulse inhibition of acoustic startle. *Psychophysiology, 44*, 339–342.

Grillon, C., Morgan, C. A., Southwick, S. M., Davis, M., & Charney, D. S. (1996). Baseline startle amplitude and prepulse inhibition in Vietnam veterans with posttraumatic stress disorder. *Psychiatry Research, 64*, 169–178.

Grillon, C., Sinha, R., Ameli, R., & O'Malley, S. S. (2000). Effects of alcohol on baseline startle and prepulse inhibition in young men at risk for alcoholism and/or anxiety disorders. *Journal of Studies on Alcohol, 61*, 46–54.

Grootens, K. P., van Luijtelaar, G., Miller, C., Smits, T., Hummelen, J. W., Buitelaar, J. K., et al. (2008). Increased P50 gating but intact prepulse inhibition in borderline personality disorder. *The Journal of Neuropsychiatry and Clinical Neurosciences, 20*, 348–356.

Herpertz, S. C., & Koetting, K. (2005). Startle response in inpatients with borderline personality disorder vs. healthy control. *Journal of Neural Transmission, 112*, 1097–1106.

Hoenig, K., Hochrein, A., Quednow, B. B., Maier, W., & Wagner, M. (2005). Impaired prepulse inhibition of acoustic startle in obsessive–compulsive disorder. *Biological Psychiatry, 57*, 1153–1158.

Hutchinson, K. E., MGeary, J., Wooden, A., Blumenthal, T. D., & Ito, T. (2003). Startle magnitude and prepulse inhibition: Effects of alcohol and attention. *Psychopharmacology, 167*, 235–241.

Johnson, P. A., Hurley, R. A., Benkelfat, C., Herpertz, S. C., & Taber, K. H. (2003). Understanding emotion regulation in borderline personality disorder: Contributions of neuroimaging. *Journal of Neuropsychiatry and Clinical Neurosciences, 15*, 397–402.

Keedwell, P. A., Kumari, V., Poon, L., Marshall, E. J., & Checkley, S. A. (2001). Information processing deficits in withdrawing alcoholics. *Addiction Biology, 6*, 239.

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S. D., & Kramer, M. D. (2007). Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology, 116*, 645–666.

Kumari, V., Antonova, E., & Geyer, M. A. (2008). Prepulse inhibition and "psychosis-proneness" in healthy individuals: An fMRI study. *European Psychiatry, 23*, 274–280.

Kumari, V., Das, M., Hodgins, S., Zachariah, E., Barkataki, I., Howlett, M., et al. (2005). Association between violent behavior and impaired prepulse inhibition of the startle response in antisocial personality disorder and schizophrenia. *Behavioural Brain Research, 158*, 159–166.

Linehan, M. M. (1993). *Cognitive behavioral treatment of borderline personality disorder*. New York: Guilford Press.

Ludewig, S., Geyer, M. A., Ramseier, M., Vollenweider, F. X., Rechsteiner, E., & Cattapan-Ludewig, K. (2005). Information-processing deficits and cognitive dysfunction in panic disorder. *Journal of Psychiatry and Neuroscience, 30*, 37–43.

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods, 7*, 19–40.

McCann, J. T., Flens, J. R., Campagna, V., Collman, P., Lazzaro, T., & Connor, E. (2001). The MCMI-III in child custody evaluations: A normative study. *Journal of Forensic Psychology Practice, 1*, 27–44.

Millon, T., Davis, R. D., & Millon, C. (1996). *The Millon clinical multiaxial inventory-III manual*. Minnetonka, MN: National Computer System.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology, 102*, 82–92.

Strack, S. (1999). *Essentials of Millon inventories assessment*. Hobken, NJ, US: John Wiley & Sons, Inc..

Strack, S. (2005). Combined use of the PACL and MCMI-III to assess normal range personality styles. In R. J. Craig (Ed.), *New directions in interpreting the Millon Clinical Multiaxial Inventory-III* (pp. 94–128). Hoboken, NJ, US: John Wiley & Sons, Inc..

Stredny, R. V., Archer, R. P., & Mason, J. A. (2006). MMPI-2 and MCMI-III characteristics of parental competency examinees. *Journal of Personality Assessment, 87*, 113–115.

Swerdlow, N. R., Braff, D. L., & Geyer, M. A. (2000). Animal models of deficient sensorimotor gating: What we know, what we think we know, and what we hope to know soon. *Behavioural Pharmacology, 11*, 185–204.

Swerdlow, N. R., Geyer, M. A., & Braff, D. L. (2001). Neural circuit regulation of prepulse inhibition of startle in the rat: Current knowledge and future challenges. *Psychopharmacology, 156*, 194–215.