Gender Differences in Autonomic Indicators of Antisocial Personality Disorder Features

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We investigated the autonomic indicators of antisocial personality disorder (APD) features in a mixed gender student sample. One hundred college students (50 men, 50 women) were administered an interview of APD and self-report measures of aggression and psychopathy. Participants completed a passive coping task and viewed emotionally valenced slideshows while their electrodermal activity (EDA), pre-ejection period (PEP), and respiratory sinus arrhythmia (RSA) were measured. Associations between APD features and autonomic reactivity were examined, controlling for aggression and psychopathy. APD features were associated with EDA hyporeactivity in men, but not women, during passive coping. While viewing threatening slides, APD features were associated with RSA hyperreactivity in women and with PEP hyperreactivity in men. APD features were associated with RSA hyperreactivity in women, but not men, while viewing slides of others in distress. These findings suggest that APD features are characterized by parasympathetic nervous system dysfunction in women but sympathetic nervous system dysfunction in men.

Keywords: antisocial personality disorder, psychophysiology, gender

Antisocial personality disorder (APD) is characterized by persistent deceitfulness, recklessness, failure to conform to social norms, and irresponsibility (American Psychiatric Association [APA], 2000). The prevalence of APD in the population is estimated at 3% for men and 1% for women. Although a few taxometric studies using measures of APD have found a taxon (Ayers, 2000; Skilling, Harris, Rice, & Quinsey, 2002), recent studies using the Structured Clinical Interview for the DSM–IV, Axis II (SCID–II; First, Spitzer, Benjamin, Gibbon, & Williams, 1997) have suggested that APD is a dimensional rather than categorical construct (Marcus, Lilienfeld, Edens, & Poythress, 2006; Marcus, Ruscio, Lilienfeld, & Hughes, 2008). These recent findings provide justification for examining the dimensional correlates of APD, such as autonomic functioning, in nonclinical samples. Several studies have investigated the autonomic correlates of antisocial behavior (see Lorber, 2004, for a review), but few have examined APD specifically. Moreover, no published studies have examined the autonomic correlates of APD in women. Given that many researchers have pointed out the advantages of investigating the autonomic characteristics of mental disorders (e.g., Beauchaine, 2001; Iacono, 1991), studies investigating associations between autonomic reactivity and APD features are warranted, as are comparisons of these associations across genders.

Gender Differences and APD

Historically, the literature on APD has focused largely on men; however, several studies have investigated the role of gender in the etiology and manifestation of APD (Cale & Lilienfeld, 2002). Some authors have posited that APD features in women result from an impaired ability to tolerate negative emotions (Bell, Foster, & Mash, 2005; Litt, Hien, & Levin, 2003), which suggests inhibitory system dysfunctions, whereas others have posited that APD features in men result from insufficient arousal to the threat of punishment (e.g., Brennan & Raine,
1997; Coren, 1999), which suggests excitatory system dysfunctions. Consistent with studies suggesting impaired emotional coping in women with APD, epidemiological studies have found stronger associations between APD and alcohol and drug use disorders in women than men (Grant et al., 2004). Moreover, researchers have found that females with APD describe themselves in more negative terms than males with APD (Sutker, DeSanto, & Allain, 1983). Nevertheless, this latter finding may reflect base rate differences between men and women, as substance use disorders are more prevalent in males than females, and high levels of negative emotionality are more prevalent in females than males. Despite the growing literature investigating gender and APD features, there are no published studies, to our knowledge, comparing autonomic indicators of these features across gender.

**Autonomic Indicators of APD**

With regard to autonomic functioning in individuals exhibiting antisocial behavior, one consistent finding is that men with psychopathic personality traits exhibit reduced electrodermal arousal to the anticipation of an aversive stimulus (Lorber, 2004). However, the construct of psychopathy differs from APD in its emphasis on interpersonal and affective features, such as grandiosity and lack of empathy (Lilienfeld, 1994). Although many researchers have examined autonomic functioning in male inmates with elevated psychopathy and aggression (e.g., Benning, Patrick, & Iacono, 2005; Fung et al., 2005; House & Milligan, 1976), only two published studies have investigated APD per se (Dinn & Harris, 2000; Raine, Lencz, Bihrl, LaCasse, & Colletti, 2000).

Dinn and Harris (2000), using self-report questionnaire measures to diagnose APD, compared electrodermal activity (EDA) in 12 men with APD and 10 healthy community men who viewed 30 emotionally valenced words (positive, neutral, and negative). Participants with APD exhibited hyporeactive EDA to negatively valenced words relative to controls. One limitation of their study, acknowledged by the authors, was the heterogeneity of their sample, which displayed elevated levels of psychopathy in addition to APD. Given the extensive literature associating psychopathy with EDA hypo-reactivity (Lorber, 2004), the relation between APD and EDA is difficult to interpret in this sample.

Raine et al. (2000) investigated heart rate (HR) and electrodermal activity in 21 men with APD (measured using the SCID–II), 34 healthy men, 26 men with substance dependence, and 21 male psychiatric controls. Participants gave videotaped speeches describing their faults while their physiology was monitored. The APD group exhibited reduced HR and EDA during the task compared with other groups. Although the APD group scored more than 2 standard deviations higher than the control group on a measure of psychopathy, Raine et al., like Dinn and Harris (2000), did not control for psychopathy scores.

In a review of the literature investigating the autonomic indicators of antisocial behavior, Lorber (2004) highlighted two important conceptual and methodological limitations. First, studies investigating autonomic responding in APD have not controlled for scores on measures of aggression or psychopathy. Although aggression is correlated with APD, it is not necessary or sufficient for a diagnosis of APD (APA, 2000). Moreover, the failure to conform to social norms, irresponsibility, and illegal behaviors of APD are not necessarily characteristic of the core features of psychopathy. As aggression and psychopathy are separable from APD, it is unclear whether autonomic reactivity in these studies was characteristic of APD per se or attributable to aggression, psychopathy, or both.

Second, studies often examined HR as an autonomic indicator. As HR is influenced by both the parasympathetic (PNS; inhibitory and reduces HR) and sympathetic nervous systems (SNS; excitatory and increases HR), which function independently, it is unclear which autonomic nervous system branch primarily drove resting HR or HR reactivity in these studies. Modern psychophysiological methodology allows researchers to differentiate the influence of the two systems. Specifically, cardiac pre-ejection period (PEP; Sherwood et al., 1990) indexes SNS influence on HR, whereas high frequency respiratory sinus arrhythmia (RSA; Berntson et al., 1997; Porges, 1995) indexes PNS influence (Grossman & Kollai, 1993).

PEP refers to the time interval between the onset of the q-wave and cardiac ejection, and is inversely associated with cardiac SNS activity.
RSA refers to beat-to-beat changes in heart rate (measured within the .18 to .40 Hz band) controlling for respiration rate and is positively associated with cardiac PNS activity. In healthy adults, RSA increases in response to sudden threat (Jonsson & Hansson-Sandsten, 2008). Several authors have posited that RSA is a sensitive indicator of emotion coping skills, whereby abnormal RSA reactivity may characterize a host of psychopathological processes (Appelhans & Luecken, 2006).

The separation of parasympathetic and sympathetic influences on HR is especially important when considering gender differences. A number of studies have suggested that men and women do not differ in their HR responses to stress (Glynn, Christenfeld, & Gerin, 1999; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004), but that these changes in HR are predominately sympathetically mediated (activated) in men and parasympathetically mediated (withdrawal) in women (Evans et al., 2001; Kuo et al., 1999). These findings suggest that healthy men and women physiologically tolerate stress differently. Therefore, the autonomic dysfunction associated with APD may be parasympathetically and sympathetically mediated in women and men, respectively.

The Present Study

The overarching goal of our study was to investigate the role of gender in the association between APD and autonomic responses to threat. Our study adds to the literature by examining the role of parasympathetic and sympathetic influences on the relationship between autonomic reactivity and APD features. Moreover, it addresses the potentially confounding influence of psychopathic personality features and aggression by controlling for measures of these constructs. We advanced two primary hypotheses:

H1: We predict EDA hyporeactivity (decreased responsivenes) and PEP hyporeactivity (lengthened time required for PEP) in men, but not women, while anticipating an aversive stimulus are associated with APD features. We based this hypothesis on Dinn and Harris (2000) and Raine et al.'s (2000) psychophysiological findings in men and Bell et al.'s (2005) psychological conceptualization of women with APD (i.e., inhibitory system dysfunction).

H2: Consistent with Raine et al.'s (2000) findings regarding reduced task HR, we predict that APD features will be positively associated with changes in PEP (less SNS activation) and RSA (more PNS activation) in men and women, respectively, in response to visual stimuli of threatening or distressing situations.

Method

Participants

Participants were 100 undergraduates, 50 women and 50 men, from a private Southern university. Full participation in the study consisted of two phases, the first comprising interview and questionnaire measures and the second laboratory and psychophysiological measures. Partial physiological data from 10 participants were dropped due to equipment failure or excessive movement artifact, and one participant elected not to participate in the countdown task (see Laboratory Tasks). The ethnic composition of the sample was 81 (81%) White, 11 (11%) Asian/Pacific Islander, 7 (7%) African American, and 1 (1%) Hispanic/Latino. Students received partial course credit and $10 for completing both phases of the study.

Interview Measure

SCID–II—APD scale. The SCID–II is a well-validated semistructured interview measure of personality disorders. Studies investigating the interrater reliability of the SCID–II show high levels of agreement for the APD (κ = .95) module (Maffei et al., 1997). The interview consists of a screening protocol, which is a list of questions assessing DSM–IV Axis II criteria. The questions representing these criteria are low-threshold and intended to capture many false endorsements. Any items endorsed are followed up with more specific questions about the criteria. Items are scored on a 1 to 3 ordinal scale. A 1 indicates that the criterion in question is not present; a 2 indicates that it is present at subthreshold levels; and a 3 indicates that it is clearly present. Clinical psychology graduate students, trained using the SCID–II manual,
training DVD, and practice interviews, administered SCID–II interviews. SCID–II APD scores reflect a count of criteria endorsed as clearly present (i.e., scores of 3 were dummy coded as 1, whereas scores of 1 and 2 were dummy coded as 0).

**Questionnaire Measures**

**AQ.** The Aggression Questionnaire (AQ; Buss & Perry, 1992) is a 34-item Likert-type self-report measure of overt aggression, anger, and hostility. Studies have found adequate support for the convergent validity of the AQ (e.g., Harris, 1997; Williams, Boyd, Cascardti, & Poythress, 1996). Cronbach’s alpha for the AQ in this sample was .86. Normative scores for the AQ in the original college sample (Buss & Perry, 1992) for men was $M = 77.8$, $SD = 16.5$ and for women was $M = 68.2$, $SD = 17.0$. In a large forensic sample (Williams et al., 1996), normative scores for the AQ in males was $M = 72.8$, $SD = 19.7$ and for females was $M = 68.4$, $SD = 21.5$.

**PPI–SF.** Derived from the lengthier PPI (Lilienfeld & Andrews, 1996), the Psychopathic Personality Inventory—Short Form (PPI–SF; Lilienfeld & Andrews, 1996) is a self-report measure of psychopathic personality traits consisting of 56 items in a Likert-type format. Like its parent measure, the PPI–SF was designed primarily to detect relatively mild manifestations of psychopathic personality traits in nonclinical samples. The PPI–SF correlates $r > .95$ with its parent measure (Lilienfeld & Hess, 2001). The PPI correlates moderately to highly with other self-report and interview-based measures of psychopathy, and negligibly with measures of depression, schizotypy, and social desirability (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Chapman, Gremonre, & Farmer, 2003; see Lilienfeld & Fowler, 2006, for a review). The PPI–SF is made up of two primary underlying factors: Factor 1 (fearless dominance), which reflects the core personality features of psychopathy and Factor 2 (antisocial deviance), which reflects the impulsivity and irresponsibility consistent with APD. The PPI–SF is particularly advantageous for the purposes of this study because it assesses the core personality features of psychopathy, with minimal emphasis on overt antisocial and criminal behavior. Scores for the PPI–SF in a college sample (Lilienfeld & Hess, 2001) were $M = 130.9$, $SD = 13.9$ for men and $M = 115.4$, $SD = 15.3$ for women.

**Laboratory Tasks**

**Countdown task.** The countdown task, designed to assess passive coping, has yielded well-replicated results in the criminal literature, with inmates displaying EDA hyporesponsivity while anticipating aversive stimuli (Lorber, 2004). In this paradigm, participants watch the screen “count” to 12, one count per second. On reaching the twelfth second, they hear a 105 dB, 1s blast of white noise with minimum rise time. This blast causes momentary discomfort in some participants, but not pain. There were five trials; between each trial, participants rested for 2.5 min, with the last 12 s measured as a baseline for the next trial.

**IAPS.** The International Affective Picture System (IAPS; Bradley & Lang, 1999) is a collection of well-validated emotionally valenced photographs. Slides depicted people in various situations and were presented in three trials: one threat (e.g., a masked person holding a knife), one other person’s distress (e.g., a woman mourning a male companion), and one neutral (e.g., a factory worker looking at a machine). Slides in the threat and other people’s distress conditions were rated high in intensity and negative in valence, whereas slides in the neutral conditions were rated low in intensity and neutral in valence (Bradley & Lang, 1999). Each trial lasted 1 min and consisted of 10 slides, displayed for 6 s each. Between each trial, participants rested for 2.5 min, with the last 60 s measured as a baseline for the next trial.

**Psychophysiological Data**

**Electrodermal activity level.** EDA was measured using the Biopac (Santa Barbara, CA) GSR 100C amplifier. Prior to affixing the electrodes, participants washed their hands using soap and warm water. Ag/AgCl electrodes were filled with .05 molar NaCl electrode paste and affixed to the medial phalanges of the first and second fingers of the participant’s nondominant hand with Velcro straps. The Biopac GSR 100C...
outputs a constant 0.5V current between the two electrodes. EDA reactivity was quantified as task EDA—baseline EDA, in μS/mm, during passive coping.

**PEP.** PEP was measured using the Biopac Niko 100C amplifier and the Biopac ECG 100C amplifier. The research assistant cleaned the contact area using rubbing alcohol and a cotton swab prior to affixing the electrodes. Electrodes were configured according to previous research (Sherwood et al., 1990). The QRS and dZ/dt B waveforms were ensemble-averaged using Biopac Acquisition v3.9 software, and PEP was measured as the time elapsed, in milliseconds, between the onset of the Q-wave and the impedance dZ/dt B-point. PEP is an inverse measure of sympathetic β-adrenergic activity, such that reductions in PEP are indicative of increased reactivity. PEP reactivity was quantified as task PEP—baseline PEP, in milliseconds, during passive coping and the IAPS task.

**RSA.** The ECG and respiratory data used to calculate RSA were measured using the Biopac ECG 100C electrocardiogram amplifier and the Biopac RESP 100C respiratory transducer. RSA was scored for each epoch using Mindware HRV v. 2.33 software (Mindware Technologies LTD, 2008). RSA reactivity was quantified as task RSA—baseline RSA during the IAPS task.

**Psychophysiological data reduction.** For the countdown to aversive stimuli (passive coping) task, EDA and PEP were measured during 10, 12-s epochs: five baseline epochs and five countdown epochs. PEP waveforms were ensemble-averaged for each of the 10 epochs. The mean of the baseline and countdown epochs were calculated for both EDA and PEP. For the IAPS task, RSA and PEP were measured during 1-min baseline periods preceding each slide presentation and during each 1-min slide presentation. Each epoch was scored separately.

**Procedure.**

Participants first completed a battery of questionnaires and were then administered the SCID–II. The laboratory tasks were administered during a subsequent visit to the laboratory. During the second visit, participants seated in a darkened, sound-proofed room completed the countdown and IAPS tasks while their HR, cardiac output, skin conductance, and respiration were monitored by a noninvasive physiological system (Biopac MP 100, Santa Barbara, CA). Participants sat in a cushioned, leather chair approximately 6 feet from the stimulus screen, a 27” TV. Prior to the first task, they sat quietly for a 5-min baseline.

**Data Analysis.**

For all analyses, we controlled for PPI–SF Factor 1 and AQ scores. However, analyses conducted without controlling for these constructs (not reported here) yielded the same pattern of findings. To test whether the laboratory tasks elicited physiological responses, we conducted eight paired-sample $t$ tests (baseline value–task value). As expected, we found increased EDA and decreased PEP (both indicative of sympathetic activation) during passive coping. Partially supporting the validity of the threatening slides, significant increases in RSA, but no significant changes in PEP, occurred. Across the whole sample, in contrast, no significant physiological changes occurred during the others’ distress or neutral slides. Therefore, findings regarding psychophysiological reactivity to distress slides should be interpreted with caution.

**Results.**

**Descriptive Statistics.**

Men ($M = 1.04, SD = 1.13$) scored significantly higher on the SCID–II APD scale, $t(98) = 3.51, p < .01, d = .71$ than women ($M = .47, SD = .94$). Men ($M = 78.69, SD = 14.50$) also scored significantly higher on the AQ, $t(98) = 4.01, p < .01, d = .81$ than women ($M = 68.54, SD = 13.16$). Similarly, men ($M = 128.94, SD = 14.51$) scored significantly higher on the PPI–SF, $t(98) = 5.5, p < .001, d = 1.11$ than women ($M = 114.58, SD = 13.73$). Zero-order correlations among the major scales and measures of physiological reactivity are presented in Table 1.

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1 Neutral Slides 2025, 2190, 2191, 2214, 2215, 2270, 2372, 2383, 2393, and 2394; Others People’s Distress Slides 2095, 2141, 2276, 2455, 2800, 2900, 9041, 9415, 9421, and 9530; Threat 6230, 6243, 6250.1, 6260, 6313, 6315, 6350, 6510, 6540, and 6560.
Table 1
Zero-Order Correlations Between Interview, Self-Report, and Physiology Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. AQ</td>
<td></td>
<td>.45</td>
<td>.25</td>
<td>-.24</td>
<td>.07</td>
<td>-.05</td>
<td>-.03</td>
<td>.05</td>
<td>-.21</td>
</tr>
<tr>
<td>2. PPI–SF</td>
<td></td>
<td></td>
<td>.32</td>
<td>-.24</td>
<td>-.07</td>
<td>.07</td>
<td>-.07</td>
<td>.27</td>
<td>-.22</td>
</tr>
<tr>
<td>3. SCID–II APD</td>
<td>.35</td>
<td>.43</td>
<td></td>
<td>-.42</td>
<td>.01</td>
<td>-.09</td>
<td>-.36</td>
<td>-.07</td>
<td>-.10</td>
</tr>
<tr>
<td>4. PC EDA</td>
<td>-.04</td>
<td>-.04</td>
<td>-.12</td>
<td></td>
<td>-.07</td>
<td>.05</td>
<td>.19</td>
<td>-.34</td>
<td>.00</td>
</tr>
<tr>
<td>5. PC PEP</td>
<td>-.19</td>
<td>-.14</td>
<td>-.19</td>
<td>.28</td>
<td></td>
<td>-.07</td>
<td>.23</td>
<td>-.03</td>
<td>-.17</td>
</tr>
<tr>
<td>6. Threat RSA</td>
<td>-.04</td>
<td>.29</td>
<td>.39</td>
<td>-.07</td>
<td>-.12</td>
<td></td>
<td>.16</td>
<td>-.34</td>
<td>-.07</td>
</tr>
<tr>
<td>7. Threat PEP</td>
<td>-.04</td>
<td>-.10</td>
<td>-.14</td>
<td>.04</td>
<td>.15</td>
<td>-.17</td>
<td></td>
<td>.10</td>
<td>.01</td>
</tr>
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<td>8. OD RSA</td>
<td>.12</td>
<td>.30</td>
<td>.38</td>
<td>.04</td>
<td>.16</td>
<td>.29</td>
<td>-.12</td>
<td></td>
<td>-.22</td>
</tr>
<tr>
<td>9. OD PEP</td>
<td>-.01</td>
<td>.44</td>
<td>.11</td>
<td>.05</td>
<td>.30</td>
<td>.00</td>
<td>.09</td>
<td>.25</td>
<td></td>
</tr>
</tbody>
</table>

Note. Men: n = 43–50; women: n = 47–50. Correlations for men are above the diagonal, women are below the diagonal. AQ = Aggression Questionnaire; PPI–SF = Psychopathic Personality Inventory–Short Form; SCID–II APD = Structured Clinical Interview for the DSM–IV, Axis II Personality Disorders, Antisocial Personality; PC EDA = electrodermal reactivity during passive coping; PC PEP = pre-ejection period reactivity during passive coping; Threat RSA = respiratory sinus arrhythmia reactivity during threat slides; OD = other people’s distress slides; RSA = respiratory sinus arrhythmia reactivity.

Hypothesis 1: Passive Coping and Autonomic Reactivity

Partially supporting Hypothesis 1, our findings (see Table 2) indicated that APD features in men, but not women, significantly predict EDA hyporeactivity during passive coping (countdown task). Moderated multiple-regression analysis (MMRA), with a dichotomous gender term and a centered APD features term in Step 1 and a Gender × Centered APD features term in Step 2 revealed that gender moderated the association between APD features and EDA reactivity, ΔF(1, 90) = 6.82, p = .01, ΔR = .06. Inconsistent with Hypothesis 1, PEP reactivity was not related to APD features in men or women.

Hypothesis 2: IAPS Tasks and Autonomic Reactivity

Table 2 displays the regression coefficients for APD features regressed on RSA and PEP reactivity during threat and others’ distress slide presentations. MMRA indicated that gender moderated the association between APD features and RSA reactivity during threat, ΔF(1, 90) = 6.54, ΔR = .07, p = .01. Partially supporting Hypothesis 2, APD features significantly predicted RSA hyperreactivity in women, but not men, during threat slides. Contrary to Hypothesis 2, APD features significantly predicted increased PEP reactivity (shortened PEP) in men, but not women, during threat slides. MMRA, however, indicated that

Table 2
APD Symptoms Regressed on Autonomic Reactivity Indicators, Controlling for Psychopathy Factor 1 and Aggression

<table>
<thead>
<tr>
<th>Measure</th>
<th>β</th>
<th>ΔR²</th>
<th>ΔF</th>
<th>p</th>
<th>β</th>
<th>ΔR²</th>
<th>ΔF</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC EDA</td>
<td>-.44</td>
<td>.15</td>
<td>7.19</td>
<td>.01</td>
<td>-.07</td>
<td>.00</td>
<td>0.17</td>
<td>.68</td>
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<tr>
<td>PC PEP</td>
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<td>.00</td>
<td>0.07</td>
<td>.80</td>
<td>.20</td>
<td>.03</td>
<td>1.45</td>
<td>.24</td>
</tr>
<tr>
<td>Threat RSA</td>
<td>-.20</td>
<td>.03</td>
<td>1.24</td>
<td>.27</td>
<td>.53</td>
<td>.21</td>
<td>12.18</td>
<td>.00</td>
</tr>
<tr>
<td>Threat PEP</td>
<td>-.35</td>
<td>.11</td>
<td>3.80</td>
<td>.04</td>
<td>-.23</td>
<td>.04</td>
<td>1.32</td>
<td>.26</td>
</tr>
<tr>
<td>OD RSA</td>
<td>-.09</td>
<td>.01</td>
<td>1.01</td>
<td>.64</td>
<td>.34</td>
<td>.09</td>
<td>4.30</td>
<td>.04</td>
</tr>
<tr>
<td>OD PEP</td>
<td>.02</td>
<td>.00</td>
<td>0.01</td>
<td>.91</td>
<td>.27</td>
<td>.05</td>
<td>2.21</td>
<td>.15</td>
</tr>
</tbody>
</table>

Note. Men: n = 43–50; women: n = 47–50. APD = antisocial personality disorder; PC EDA = electrodermal reactivity during passive coping; PC PEP = pre-ejection period reactivity during passive coping; Threat RSA = respiratory sinus arrhythmia reactivity during threat slides; Threat PEP = pre-ejection period reactivity during threat slides; OD RSA = respiratory sinus arrhythmia reactivity during others’ people’s distress slides; OD PEP = pre-ejection period reactivity during others people’s distress slides.
gender did not moderate this association, $\Delta F(1, 90) = .14, \Delta R = .002, p = .71$. Underscoring benefits of measuring PEP and RSA rather than HR, APD was not related to HR reactivity during threat slides, $\Delta F(1, 90) = 3.11, p = .08$. Partially supporting Hypothesis 2, APD features predicted RSA hyperreactivity to others’ distress in women, but not men. MMRA indicated that the gender interaction was significant, $\Delta F(1, 90) = 4.52, \Delta R = .05, p = .03$. PEP reactivity during other people’s distress was not associated with APD symptoms in men or women (see Figure 1).

**Discussion**

The overarching goal of this study was to investigate the role of gender in the relationship between autonomic functioning and APD features. This study also sought to clarify whether the autonomic indicators associated with APD in men are characteristic of APD per se or result

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**Figure 1.** Scatterplots: Psychophysiological reactivity by antisocial personality disorder (APD) symptoms. EDA = electrodermal activity; SCID–II = Structured Clinical Interview for the DSM–IV, Axis II; PEP = pre-ejection period; RSA = respiratory sinus arrhythmia.
from its overlap with aggression and the core personality features of psychopathy. Consistent with studies investigating the autonomic correlates of APD diagnoses in men (Dinn & Harris, 2000; Raine et al., 2000), subclinical APD was associated with EDA hyporeactivity while anticipating threat. Moreover, the relationship between APD and EDA hyporeactivity was characteristic of APD in men even when controlling for the core features of psychopathy or aggression. However, EDA hyporeactivity was not characteristic of APD features in women. These findings suggest that sympathetic hypovascularism to imminent threat may be characteristic of APD features in men but not women (see also Raine, 2002).

APD features in women, but not men, predicted RSA hyperreactivity to visually threatening images and images of others’ distress. Conversely, APD features predicted PEP hyperreactivity in men, but not women, to threatening images. These findings suggest that APD features in women are associated with cardiac PNS abnormalities during threatening situations, whereas APD features in men are associated with SNS abnormalities. In addition to their relation to APD features, these findings are consistent with studies investigating gender differences in cardiac autonomic reactivity to stress more broadly (Evans et al., 2001; Kuo et al., 1999). Taken together, the results from this study provide suggestive physiological evidence that APD features are differentially motivated in men and women. Moreover, they support the contention that an impaired ability to tolerate negative emotions (Bell et al., 2005; Litt et al., 2003) characterizes APD features in women, whereas an insufficient arousal to the threat of punishment (e.g., Brennan & Raine, 1997; Coren, 1999) characterizes APD features in men.

There were several limitations to our study, most notably the small sample size, which limited our statistical power to detect moderation effects. A second limitation was the ethnic homogeneity of the sample and our substantial reliance on self-report rather than multi-informant measures of antisocial behavior and personality pathology. Personality pathology, especially in Cluster B (the “dramatic, emotional” cluster), is largely ego-syntonic (Grove & Tellegen, 1991), suggesting that the addition of peer-reported personality pathology in future research may reduce the error associated with individuals’ lack of insight (Oltmanns & Turkheimer, 2009). In addition, the mean levels of APD were low, so our results may be limited in their generalizability to samples with higher levels of APD. Future studies should also consider the potential influence of substance abuse and dependence, as these disorders are highly comorbid with APD and associated with autonomic abnormalities (e.g., Taylor, Carlson, Iacono, Lykken, & McGue, 1999). A third limitation of this study was the other people’s distress paradigm, which did not result in any measured baseline to task physiological changes during manipulation checks. A fourth limitation was the use of the PPI–SF as a measure of psychopathy, as the psychophysiological correlates of this measure are not well researched.

This study had several major strengths, including the interview-based assessment of DSM–IV APD, the use of PEP and RSA as autonomic indexes, the inclusion of women, and the statistical control of psychopathy and aggression. The continuation of this line of research may ultimately lead to a better understanding of the autonomic correlates of APD, as well as potential sex differences. Future lines of research may seek to investigate the autonomic correlates of APD in female inmates, investigate potential structural and functional brain abnormalities in women with APD, and continue to investigate differential etiological factors contributing to the development of APD across genders.

References


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