Validating a Distinction Between Primary and Secondary Psychopathy With Measures of Gray’s BIS and BAS Constructs

Joseph P. Newman, Donal G. MacCoon, Leah J. Vaughn, and Naomi Sadeh
University of Wisconsin—Madison

Psychopathic individuals exhibit deficits in socialization that predispose them to poor school performance, irregular employment histories, inadequate marital adjustment, unreliable and irresponsible behavior, interpersonal conflict, and significant legal problems. In the prototypical, or primary, psychopath, this failure is not due to social disadvantage, low intelligence, neurotic anxiety, or other psychopathology (Cleckley, 1976). In contrast, such causes are likely to play a role in secondary psychopathy. That is, the inappropriate behavior of the primary psychopath is presumed to be a consequence of some intrinsic deficit that hampers self-regulation and normal adjustment, whereas secondary psychopathy is viewed as an indirect consequence of inadequate intelligence, psychotic thinking, excessive neurotic anxiety, unusual sex drive, or other attributes that increase a person’s vulnerability to chronic misbehavior (Lykken, 1995).

Despite efforts by Cleckley (1976) and others (e.g., Blackburn, 1975; Lykken, 1995) to distinguish primary psychopathy from other forms of antisocial behavior, the psychopathy construct remains heterogeneous, a problem that seriously hampers research on the etiology of primary psychopathy (Brinkley, Newman, Wider, & Lynam, 2004; Hicks, Markon, Patrick, Krueger, & Newman, 2004; Lilienfeld, 1998; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). Ultimately, successful evaluation of etiological hypotheses regarding primary psychopathy depends on the ability of researchers to distinguish between those syndromes that reflect the core underlying deficit and those that reflect alternative etiological processes (i.e., secondary psychopathy).

To achieve this distinction, investigators have historically used self-report measures of neurotic anxiety like the Welsh Anxiety Scale (WAS; Welsh, 1956) and the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953) to subdivide psychopathic individuals. This tradition of the use of anxiety to parse the etiological heterogeneity of psychopathy is supported by a substantial research literature showing that high- and low-anxious psychopathic individuals display a different constellation of performance deficits (e.g., Blackburn, 1975, 1979; Chesno & Kilmann, 1975; Fox & Lippert, 1963; House & Milligan, 1976; Newman, Widom, & Nathan, 1985; O’Brien & Frick, 1996; Painting, 1961; Schmauk, 1970; Widom, 1976a, 1976b, 1978; or see Newman & Brinkley, 1997 for a review). Furthermore, identifying primary psychopathy with low levels of neurotic anxiety is consistent with Cleckley’s (1976) view that primary psychopaths are “very sharply characterized by the lack of anxiety (remorse, uneasy anticipation, apprehensive scrupulousness, the sense of being under stress or strain) and, less than the average person, show what is widely regarded as basic in the neurotic” (p. 257).

Although there is good theoretical and empirical support for the use of anxiety to subdivide psychopathy (e.g., Brinkley et al., 2004; Newman & Brinkley, 1997; Schmitt & Newman, 1999), not all researchers agree that such measures should be used to distinguish between primary and secondary psychopathy. One issue relates to improvements in the assessment of psychopathy associated with Hare’s (2003) Psychopathy Checklist—Revised (PCL–R). In contrast to earlier measures of psychopathy that were positively correlated with anxiety, the PCL–R is relatively uncorrelated with trait anxiety and negative affect. According to some (e.g., Lykken, 1995), this fact obviates the necessity of controlling for anxiety when

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A second concern relates to the distinction between anxiety and fearfulness (e.g., Lilienfeld, 1994, 1998; Lykken, 1957, 1995). According to Lykken (1995), primary psychopathy is characterized by fearlessness, poor passive avoidance, weak electrodermal anticipation of punishment, and average levels of positive and negative emotionality. Secondary psychopathy, by contrast, is characterized by relatively high levels of positive and negative emotionality, impulsiveness, and sensation seeking but average levels of fearlessness, passive avoidance, and electrodermal activity in anticipation of punishment. In addition, Lykken has used Gray’s (1987) conceptual model of the nervous system to clarify his distinction between primary and secondary psychopathy. Gray’s model postulates two primary motivational systems: the behavioral activation system (BAS) and the behavioral inhibition system (BIS). The BAS is sensitive to reward cues and initiates behavioral approach, whereas the BIS is sensitive to punishment cues and initiates passive avoidance. In Gray’s model, both motivational systems serve to increase nonspecific (i.e., general) arousal and they are reciprocally related such that activation of one system inhibits activation of the other. According to Lykken, primary psychopathy is associated with a hyperreactive BIS and average BAS, whereas secondary psychopathy is associated with a hyperreactive BAS and average BIS.

On the basis of this characterization of primary and secondary psychopathy, Lykken (1957, 1995) has argued that investigators should use measures of fearlessness (e.g., harm avoidance/constraint; Tellegen, 1982) to distinguish between primary and secondary psychopathy. Moreover, because fearlessness is essentially uncorrelated with measures of anxiety–negative emotionality such as the WAS and TMAS (Watson & Clark, 1984), Lykken has expressed strong reservations about the use of anxiety scales to distinguish between primary and secondary psychopathy.

Given the methodological and theoretical importance of this question and the lack of empirical evidence documenting the postulated associations, there is a strong need for empirical research to address the validity of alternative methods for identifying primary and secondary psychopathy. Toward this end, we examine the extent to which primary and secondary psychopathy identified with the PCL–R and WAS satisfy Lykken’s (1995) criteria with respect to Gray’s (1987) BIS and BAS constructs. Our focus on Gray’s (1987) BIS/BAS systems reflects their prominence in models of primary and secondary psychopathy (e.g., Fowles, 1980; Lykken, 1995) and the absence of empirical evidence involving well-validated measures of these constructs. We hypothesize that (a) primary psychopathy identified through the use of a combination of high PCL–R and low-WAS scores will be characterized by a weak BIS and a normal (i.e., average) BAS and (b) secondary psychopathy identified through the use of a combination of high PCL–R and high-WAS scores will be characterized by a strong BAS and normal (i.e., average) BIS. In other words, we predict that the BIS/BAS profiles that Lykken associates with primary and secondary psychopathy will be satisfied by using the PCL–R and WAS to identify these groups.
worrying about making mistakes), whereas the BAS—Reward Responsiveness, Drive, and Fun Seeking subscales measure sensitivity to anticipated/acquired rewards, motivation to achieve desired goals, and willingness to approach new appetitive stimuli, respectively. Because all three BAS subscales load strongly on a second-order (BAS) factor, we tested our hypotheses with the total BAS score.

Classification

Participants were divided into groups on the basis of PCL–R scores and a median split on the WAS. Primary psychopathy was defined as having a PCL–R score of 30 or greater and a WAS score of 11 or less. Secondary psychopathy was defined as having a PCL–R score of 30 or greater and a WAS score of 12 or more. In order to make the comparison groups for these two groups as comparable as possible, we performed statistical analyses to compare (a) individuals meeting criteria for primary psychopathy with all participants not meeting these criteria and (b) individuals meeting criteria for secondary psychopathy with all participants not meeting these criteria. Table 1 provides cell sizes, means, and standard deviations for these groups on the two assessments of Gray’s (1987) motivational systems. To facilitate comparison across measures, we report z scores for SR/SP and BAS/BIS measures. Table 2 shows the nonstandardized means and standard deviations for these measures along with relevant correlations.

Results

Preliminary analyses examined the effects of race in moderating the association between group and the dependent measures used in the following analyses. In no case, did the Group × Race interaction approach statistical significance (all ps > .20).

To test the hypothesis that primary psychopathy is associated with a weak BIS and a normal BAS relative to control participants, we conducted a mixed-model analysis of variance (ANOVA) with group (primary psychopathy, control) as the between-participants variable and motivational system (SP, SR) as the repeated measure. Consistent with our hypothesis, the ANOVA revealed a significant Group × Motivational System interaction, F(1, 249) = 14.06, p < .001, η² = .05. Consistent with predictions, the BIS scores of the primary psychopathy group were significantly lower than those of controls, F(1, 249) = 28.45, p < .001, η² = .10, whereas the BAS scores of the two groups did not approach statistical significance, F(1, 249) < 1.0.

To test the hypothesis that secondary psychopathy is associated with a strong BAS and a normal BIS relative to control participants, we conducted a mixed-model ANOVA with group (secondary psychopathy, control) as the between-participants variable and motivational system (SP, SR) as the repeated measure. Consistent with our hypothesis, the ANOVA revealed a significant Group × Motivational System interaction, F(1, 505) = 8.94, p = .003, η² = .02. As predicted, SR scores for the secondary psychopathy group were significantly greater than those for control participants, F(1, 505) = 49.68, p < .001, η² = .09. Contrary to prediction, SP scores were also significantly greater in the secondary psychopathy group, F(1, 505) = 9.15, p = .003, η² = .02.

The ANOVA that used the BIS/BAS scales also yielded a significant Group × Motivational System interaction, F(1, 249) =

### Table 1

<table>
<thead>
<tr>
<th>Sample and statistic</th>
<th>Primary psychopathy</th>
<th>Primary psychopathy control participants</th>
<th>Secondary psychopathy</th>
<th>Secondary psychopathy control participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>107</td>
<td>400</td>
<td>110</td>
<td>397</td>
</tr>
<tr>
<td>M</td>
<td>-0.67a</td>
<td>-0.01</td>
<td>0.18a</td>
<td>0.00</td>
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<tr>
<td>SD</td>
<td>0.58</td>
<td>0.97</td>
<td>1.00</td>
<td>1.00</td>
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### Table 2

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. SPa</td>
<td>—</td>
<td>.21*</td>
<td>.49*</td>
<td>.04</td>
<td>.67*</td>
<td>8.90</td>
<td>5.40</td>
</tr>
<tr>
<td>2. SRa</td>
<td>—</td>
<td>—</td>
<td>.09</td>
<td>.54*</td>
<td>.32*</td>
<td>13.00</td>
<td>4.90</td>
</tr>
<tr>
<td>3. BISb</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>.16</td>
<td>.35*</td>
<td>18.60</td>
<td>3.70</td>
</tr>
<tr>
<td>4. BASb</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>.18*</td>
<td>38.60</td>
<td>6.00</td>
</tr>
<tr>
<td>5. WAS</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>12.20</td>
<td>8.60</td>
</tr>
</tbody>
</table>

Note. SP = Sensitivity to Punishment subscale; SR = Sensitivity to Reward subscale; BIS = behavioral inhibition system; BAS = behavioral activation system.

Paralleling results for the SR/SP analysis, an ANOVA that used the BIS/BAS scales yielded a significant Group × Motivational System interaction, F(1, 249) = 14.06, p < .001, η² = .05. Consistent with predictions, the BIS scores of the primary psychopathy group were significantly lower than those of controls, F(1, 249) = 28.45, p < .001, η² = .10, whereas the BAS scores of the two groups did not approach statistical significance, F(1, 249) < 1.0.
Discussion

Our results provide strong support for the hypothesis that primary psychopathy, as identified by high-PCL–R and low-WAS scores, is characterized by a weak BIS and a normal BAS. Across two different measures of the BIS and BAS constructs, the primary psychopathy group was associated with significantly lower BIS scores (mean $z$ score $= -6.60$) and normal BAS scores (mean $z$-score $= -.025$). This finding serves simultaneously to bolster Lykken’s (1995) characterization of primary psychopathy and the utility of using traditional measures of anxiety such as the WAS to distinguish primary psychopathy from other antisocial syndromes.

Results for the secondary psychopathy group are also consistent with Lykken’s (1995) predictions and provide additional support for the use of the WAS to differentiate primary and secondary psychopathy. Across two measures of the construct, secondary psychopathy was associated with significantly higher BAS scores (mean $z$ score $= .58$) than control participants. On the basis of Lykken’s characterization of secondary psychopathy, we also predicted that secondary psychopathy would be associated with average BIS scores, but this prediction received only partial support (mean $z$ score $= .205$), with one of the comparisons yielding a significant group difference. Despite this limitation, both analyses yielded significant Group $\times$ Motivational Construct interactions with the secondary psychopathy group distinguished primarily by their scores on the BAS measures. Moreover, in light of Lykken’s claim that secondary psychopathy involves “high scores on negative emotionality or neuroticism” (p. 122) and Gray’s (1987) claim that BIS activation increases with increasing levels of neuroticism, the modest correlation between secondary psychopathy and the BIS construct is not very surprising.

One limitation of the current findings is that they are restricted to self-report indices of BIS and BAS functioning and, thus, do not address the behavioral (e.g., passive avoidance) and physiological indices (electrodermal hyporeactivity) of BIS functioning identified by Lykken (1995). However, there is already published evidence demonstrating that poor passive avoidance is more strongly related to primary psychopathy than to secondary psychopathy as defined in this study (e.g., Newman & Schmitt, 1998). Results for the electrodynamical hyporeactivity are more equivocal. Evaluating predictions from the Folkles (1980) model of psychopathy, Arnett (1997) found that group differences in electrodynamical responses to punishment cues were specific to comparisons involving high-anxious (i.e., secondary) psychopaths and control participants. Although this result appears counterintuitive, Folkles (2000) has noted that psychopathic individuals’ electrodynamical hyporeactivity in anticipation of aversive stimuli “may relate to an impulsivity dimension among psychopaths rather than to the core personality features of psychopathy” (p. 177; see also, Gatzke-Kopp, Raine, Loebel, Stouthamer-Loeber, & Steinhauser, 2002). Indeed, there is growing awareness that many correlates of psychopathy initially attributed to weak BIS functioning, and low levels of neurotic anxiety may actually reflect a low constraint/impulsivity dimension that is more associated with the antisocial lifestyle aspect of psychopathy (i.e., PCL–R, Factor 2) and secondary (i.e., high-anxious) psychopathy than with primary psychopathy (Hicks et al., 2004; Newman, 1997; Schmitt & Newman, 1999).

Another potential limitation is that because Gray (1982, 1987) characterized his BIS as a hypothetical anxiety system and, in large part, based his model on the effects of antianxiety drugs (e.g., benzodiazepines), its association with a definition of primary psychopathy that includes low anxiety could be construed as circular and as undermining the significance of the current study. In this regard, it is worth noting that (a) the BIS and the WAS measures were only moderately correlated ($r = .35$); (b) although the correlation between the SP and the WAS was much higher ($r = .67$), results for the BAS and the SP scales were virtually identical, indicating that our results were not simply a reflection of the magnitude of correlation between the WAS and measures of the BIS construct; (c) partialing the effects of anxiety from the group analyses does not eliminate the significant effects reported; and (d) despite the correlation between the WAS and the BAS measures, secondary psychopathy was more strongly related to the BAS than to the BIS constructs; and (e) others (e.g., Fowles & Missel, 1994; Lykken, 1995) have proposed that the BIS deficiencies associated with psychopathy are attributable to fearlessness and low constraint rather than to low anxiety. Finally, regardless of the processes responsible for the association, our results provide good empirical support for the use of the WAS to distinguish primary and secondary psychopathy.

Despite these potential limitations, the current investigation provides an important addition to the field of psychopathy. To our knowledge, the current investigation is the first to provide evidence that primary psychopathy is differentially associated with the BIS construct (i.e., relatively uncorrelated with BAS) and that secondary psychopathy is significantly and differentially associated with the BAS construct.

This demonstration has potentially important implications for models that relate psychopathy to Gray’s (1987) motivational systems (e.g., Arnett, 1997; Folkles, 1980; Quay, 1988). As proposed by Folkles (1980), many findings in psychopathy appear to reflect weak BIS functioning, but this characterization may not apply to high-anxious psychopathic individuals. Similarly, Arnett (1997) and others (e.g., Quay, 1988) have characterized psychopath as BAS- or reward-dominant, but this description may apply specifically to secondary psychopathy as opposed to primary psychopathy. More generally, without assessing anxiety and distinguishing between primary and secondary psychopathy, a significant difference between psychopathic and nonpsychopathic participants may reflect weak BIS functioning, strong BAS functioning, or BAS versus BIS dominance. This problem was also highlighted by Lykken (1995) when he noted that secondary psychopathy may mimic primary psychopathy because a strong BAS may overwhelm a person’s normal BIS and mimic the fear deficit that he associates with primary psychopathy.

These and other sources of confusion can be avoided through the clarification of the diverse etiological processes associated with primary and secondary psychopathy. The current study suggests that measures of anxiety can help make this distinction, and, when combined with measures of Gray’s (1987) motivational constructs, can play an important role in the clarification of these processes.
References


