
BOOK REVIEW ESSAYS ON LYKKEN'S *THE ANTISOCIAL PERSONALITIES*

Reconsidering the Low-Fear Explanation for Primary Psychopathy

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Lykken's book, *The Antisocial Personalities*, is an informative and entertaining book with much to commend it. Written by a senior scientist with established expertise in psychophysiology, behavior genetics, and psychopathy, Lykken's experience and expertise enable him to provide clear and concise tutorials on evolution, genetics, and the relevance of temperament-related traits to the socialization process. Especially valuable is Lykken's emphasis on the heterogeneity of criminal behavior which, it follows, requires diverse explanations. With this background, the reader is prepared to understand that antisocial behavior results from a complex and continuous interaction of innate characteristics and environment.

Lykken defines his subject matter by subdividing antisocial behavior into three broad categories: normal, psychopathic, and sociopathic. He goes on to outline alternative etiological explanations for these syndromes. One of the two major goals of his book, according to Lykken, is to review the experimental literature concerning a specific psychopathic subtype, the primary psychopath. The focus of this review includes: (a) differentiating primary psychopathy from the related syndromes of sociopathy and secondary psychopathy, (b) a reanalysis of the low-fear hypothesis that stems from his classic 1957 study on psychopathy, and (c) consideration of alternative explanations for this syndrome. Lykken concludes that primary psychopathy may result from "the rearing by typical parents of a child who is normal in all respects save for a below-average endowment of innate fearlessness" (p. 154). He argues that his proposal is superior to alternative models and states that "the time is ripe for the low-fear hypothesis of primary psychopathy to be reconsidered" (p. 155). Our goal is to evaluate Lykken's conclusions regarding the primary psychopath.

The Antisocial Personalities, David T. Lykken, 1995, New Jersey: Lawrence Erlbaum Associates, Inc.

Distinguishing Psychopathy and Sociopathy

Regarding the distinction between sociopathy and psychopathy, Lykken states:

For the purposes of this taxonomy, I am using sociopath to refer to persons whose lack of socialization is primarily attributable to neglectful or incompetent parenting, reserving psychopath for those whose antisocial behavior is due primarily to innate characteristics that either overmaster their socialization from time to time or which make them difficult to socialize within a traditional two-parent family structure. (p. 31)

Lykken describes his proposal as a "diathesis-developmental model" (p. 172) with difficult temperament corresponding to the diathesis and poor parenting corresponding to the developmental stress. The implication is that the continuum from psychopathy to sociopathy corresponds to the interaction of temperament and parenting such that the greater the diathesis (i.e., predisposing temperament) the less stress (i.e., poor parenting) is required to result in antisocial personality. Conversely, the greater the stress the less diathesis is required to manifest an antisocial personality. Thus, "the psychopath and the sociopath can be regarded as opposite endpoints on a common dimension with difficult temperament maximized at the psychopathic end and inadequate parenting maximized at the sociopathic end" (p. 7).

According to this model, the socialization of both groups is the product of the interaction between temperament and parenting style (i.e., the same etiological factors are at work in both groups). This implies that the difference between psychopathy and sociopathy boils down, not to a qualitative difference between groups, but to a quantitative distinction involving the degree of difficult temperament and inadequate parenting. Although Lykken's distinction between psycho-

pathy and sociopathy allows him to highlight two important factors contributing to the development of anti-social behavior, namely difficult temperament and poor parenting, we have concerns about his proposal. For the purposes of this article, we focus on three interrelated reservations.

First, Lykken's proposal appears to rob psychopathy of its distinctiveness. Psychopathy was originally proposed as a construct because the antisocial and self-destructive behavior of some seemingly normal individuals was so striking that traditional explanations of criminality were considered insufficient to describe the group. Such observations made early leaders in the field, like Cleckley, "convinced that psychopathy is a neuropsychiatric disorder, as qualitatively different from normality as the most profound psychosis" (Lykken, 1995, p. 172).

It is difficult to reconcile how unique the psychopath seems in relation to other criminals with Lykken's notion that psychopathy and sociopathy are distinguished only by their relative standing on a temperament-parenting continuum. This is particularly true given that Lykken does not even require an individual to have an extreme temperament to meet the criteria for psychopathy. He states, "the psychopath begins life as a normal child with less than the average endowment of harm avoidance ... which is not pathological in itself but which does make him more difficult to socialize" (p. 171). Given that such statements are also used to describe sociopaths (p. 21) and normal criminals (p. 18), Lykken's conceptualization of the psychopath hardly seems to distinguish it from other antisocial groups.

Furthermore, this view seems incompatible with some important aspects of psychopathy. If psychopaths do have a relatively normal temperament as Lykken suggests, one might expect them to be fairly common in the population as a whole. We know, however, that psychopaths are rare in both prisons (Hare, 1991) and noninstitutionalized settings (Babiak, 1995; Levenson, Kiehl, & Fitzpatrick, 1995). If psychopaths have a relatively normal temperament, why is it that they are so difficult to socialize even in good homes? Even when extraordinary circumstances enable them to succeed socially, Lykken believes "there remains lurking in the background an antisocial disposition ..." (p. 22). Why would someone with a relatively normal temperament have these problems? Such difficulties suggest to us a temperamental predisposition that is rarer and more difficult than would be found in the normal spectrum.

Our second concern with Lykken's proposal is that his distinction between psychopathy and sociopathy seems difficult or impossible to implement. Although Lykken's proposal may distinguish individuals who

exist at the far sociopathic end of the continuum from those who are at the extreme psychopathic end, the majority of individuals will not fall into either of these categories. Because difficult temperament and poor parenting are correlated (Lykken, 1995, p. 11), the majority of cases will be characterized by both. In addition, classifying individuals who fall between these extremes will present a formidable challenge. For example, what differentiates sociopaths who "are aggressive or fearless, stimulus seekers or Machiavellian manipulators, people who, as children, posed too great a problem for their well-intending but overmatched parents to cope with" (p. 21) from psychopaths? How does one decide when a person's "difficult temperament" has sufficient etiological significance to regard him or her as psychopathic as opposed to sociopathic?

To be fair, Lykken acknowledges that his distinction between psychopathy and sociopathy is "fuzzy" and allows for substantial overlap between the sociopathic and psychopathic categories (e.g., p. 42). He constructed his taxonomy "for the purpose of emphasizing the probable complexity of psychological motives, temperament peculiarities, and personality configurations that can contribute to criminal behavior" (p. 41). Whereas we agree that such understanding is important, we find Lykken's armchair taxonomy to be unwieldy. The usefulness of a taxonomic system concerns its ability to classify subjects so that etiological processes underlying the phenomena may be identified.

Our third concern relates to the potential impact of Lykken's proposal on the field of psychopathy. After decades of research hampered by fuzzy distinctions and assessment strategies with dubious reliability and validity, there is an emerging consensus regarding the concept of psychopathy and the optimal strategy for assessing the concept. Based on decades of research, Professor Robert Hare has clarified the concept of the Cleckley psychopath and developed an instrument for assessing it reliably. In turn, this contribution has generated a wealth of research that supports its validity (Hare, 1991). Why, just as the field is exploding with renewed interest and quality research, should we consider an alternative taxonomy?

Distinguishing Primary and Secondary Psychopathy

Investigators have traditionally distinguished between primary psychopaths, who coincide with Cleckley's characterization of the psychopath, and secondary psychopaths whose antisocial behavior may resemble primary psychopaths' but whose conduct problems appear to be the result of (i.e., secondary to) neurotic

conflicts and/or pervasive negative affect (Hare, 1970; Karpman, 1948). In his 1957 study, Lykken drew a similar distinction between primary and neurotic "sociopaths" (now psychopaths). The former included inmates who "best fitted the Cleckley prototype ..." whereas the latter, according to Lykken, "did not meet the criteria in important respects" (p. 6).

Lykken's (1995) recommendation for distinguishing primary and secondary psychopathy is clear and specific. After describing Hare's (1991) Psychopathy Checklist-Revised (PCL-R), a diagnostic instrument based on the original Cleckley criteria with demonstrated reliability and validity, Lykken notes it "may not be the optimum instrument for distinguishing the primary psychopath from other varieties of unsocialized offenders" (p. 127). As an alternative, Lykken (1995) recommends using a combination of factor 1 items from the PCL-R (PCL-1) and his Activities Preference Questionnaire (APQ) or Tellegen's (1982) Harm Avoidance (HA) Scale. "If the low-fear hypothesis is valid, then the Cleckley or PCL-1-denominated psychopaths who are low in harmavoidance should constitute the purest group of primary psychopaths, whereas the prison inmates who are not psychopathic according to the PCL-1 and who are also high on harmavoidance should provide the best contrast" (p. 127).

We have three concerns with his approach: First, Lykken's justification for preferring the APQ or HA scale appears related to an a priori assumption that primary psychopathy is distinguished by low fear. To the extent that the low fear hypothesis is incorrect or incomplete, however, his approach may not be optimal for identifying primary (i.e., Cleckley) psychopaths. This issue is addressed in the next section of this article. Second, Lykken asserts that the APQ and HA scales provide superior indices of anxiety, but the basis for this conclusion is unclear. Because Lykken provided no evidence for this assertion, we will not debate the issue, but rather request that Lykken identify the body of evidence supporting the superiority of these measures as indices of anxiety/fearlessness. Third, Lykken fails to discuss the evidence regarding the ability of these measures to distinguish primary and secondary psychopaths. The rest of this section is devoted to this issue.

Although Lykken's (1957) original application of the APQ successfully differentiated primary psychopaths from the noninstitutionalized control group, it failed to distinguish primary and secondary psychopaths. Subsequent investigations employing the measure were no more successful. For instance, Schmauk (1970) reported average APQ scores of 30.3, 32, and 34.4 for primary psychopaths, secondary psychopaths, and nonpsychopathic controls, respectively. None of the differences approached statistical significance.

Widom (1976) administered the APQ to primary, secondary, and nonpsychopathic offenders and concluded that "although primary psychopaths scored lowest on the APQ, with controls next lowest and secondary psychopaths the highest, none of the differences was significant" (p. 616).

We recently began administering Tellegen's (1982) personality questionnaire to investigate Lykken's claims regarding the HA scale. Thus far, we have acquired data on 71 inmates who were also assessed using Hare's (1991) PCL-R. The correlation between HA and PCL-R total scores was $-.147$. Lykken also predicted that HA would be more associated with PCL-1 than PCL-2 scores because PCL-1 items relate more closely to the Cleckley-like features of psychopathy (p. 128). However, the correlations for PCL-1 and PCL-2 scores were $-.110$ and $-.206$, respectively. None of these correlations was significant. We also computed means for three groups using our usual cutting scores of 22 and 30 on the PCL-R to form low, middle, and high psychopathy groups. The mean HA scores for these groups were 18.5, 16.1, and 16.5, respectively. As in earlier studies, these data are directional but do not approach significance, $F(2, 64) < 1.0$.

In addition to promoting his own strategy, Lykken actively rejected a more common strategy that subdivides psychopathic and nonpsychopathic offenders using traditional measures of anxiety, such as the Taylor Manifest Anxiety Scale (Taylor, 1953), the Welsh Anxiety Scale (WAS; Welsh, 1956), or Spielberger's Trait Anxiety Scale (Spielberger, Gorsuch, & Lushene, 1970). According to Lykken, these measures "do not measure the fearfulness or harmavoidant trait but, instead, are loaded strongly on the superfactor of neuroticism" (p. 128). Referring to research employing this alternative strategy, Lykken states, "there is no clear theoretical justification for this practice" (p. 128).

Lykken's vehement rejection of the Welsh and Taylor anxiety scales is difficult to understand in light of the theoretical and empirical support for this strategy. It is not clear to us why a high factor loading on neuroticism should disqualify a measure of "anxiety" as suggested by Lykken (pp. 145-146). Although we found no definition of anxiety in his book, Lykken uses *anxiety* and *fearlessness* interchangeably and he clearly identifies his anxiety construct with that of Jeffrey Gray (1987). For Gray (1987), however, trait anxiety reflects a combination of neuroticism and introversion with a greater loading on neuroticism (about 0.7) than introversion (about 0.40; pp. 350-351). Indeed, Gray (1987) explicitly associated his anxiety dimension to the Taylor anxiety scale (p. 350). Thus, an association with neuroticism would hardly compromise a measure's relevance for assessing Gray's anxiety construct.

Moreover, Lykken appears to embrace the WAS while discussing Blackburn's distinction between primary and secondary psychopathy. Referring to the secondary psychopaths in his own 1957 study, Lykken claims they "resembled those who have been subsequently referred to as secondary psychopaths by Blackburn (1988) and others" (p. 145). According to Lykken, Blackburn's primary psychopaths are relatively free of anxiety and resemble the Cleckley psychopath in many respects. In contrast, secondary psychopaths are "anxious, depressed, and emotional but also hostile, aggressive, impulsive, and undersocialized" (p. 130). Given Blackburn's reliance on the WAS to distinguish primary and secondary psychopaths, Lykken's own comments suggest that the WAS may be an important diagnostic tool.

Empirically, there is good evidence that the WAS has been more successful than the APQ in differentiating primary and neurotic psychopaths. Beginning with Lykken's own study, the Welsh and Taylor measures revealed an average difference of 85 standard units between primary and secondary groups ($p < .01$) whereas the APQ difference was a nonsignificant 41 units. In a related study, Schmauk (1970) observed significant differences between primary and secondary psychopaths using the Welsh and Taylor scales (both $ps < .01$) but not using the APQ. Moreover, the Welsh and Taylor scales were also more successful than the APQ in differentiating Schmauk's primary psychopaths from his normal control group.

Numerous other investigations have employed the Welsh, Taylor, or Spielberger measures to subdivide psychopaths and nonpsychopaths into high- and low-"anxious" groups. Such studies provide additional justification for distinguishing primary and secondary psychopathic groups using these measures (e.g., Arnett, Howland, Smith, & Newman, 1992; Chesno & Kilmann, 1975; Fagan & Lira, 1980; Newman, Kosson, & Patterson, 1992; Newman, Patterson, Howland, & Nichols, 1990; Smith, Arnett, & Newman, 1992; Widom, 1976).

A final point concerns the potential role of anxiety as a confounding variable. The WAS and other measures of anxiety are correlated with a wide variety of personality scales tapping negative affect and psychopathological attributes. They are also correlated with behavioral inhibition, passive avoidance, and electrodermal activity. If an investigator compares groups of psychopaths and controls that are unmatched on the WAS (or a similar measure), it is quite possible that observed differences in one's dependent variable will reflect anxiety/neuroticism as opposed to psychopathy (see Arnett, Smith, & Newman, 1996). By contrast, when hypotheses are tested using low-anxious psycho-

paths and controls (i.e., comparing primary psychopaths with a group of anxiety-matched controls), the results are likely to reflect processes specific to psychopathy as opposed to anxiety. Lykken's suggestion to compare a high PCL-1, low HA group with a low PCL-1, high HA group seems especially problematic. Although it would not be surprising if this approach yielded significant differences in electrodermal responding, self-reported anxiety and other fear-related indices as predicted by his theory, it would be impossible to conclude that such differences related to psychopathy as opposed to harm avoidance. Addressing this potential confound provides further justification for employing the WAS.

In short, Lykken's advocating of the APQ and HA measures seems incomprehensible, given the lack of evidence regarding their ability to distinguish primary and secondary psychopaths, the lack of clear theoretical rationale or evidence supporting their superiority over other measures of anxiety, and the generally impressive body of evidence showing that the WAS and similar measures predict outcomes consistent with the primary-secondary psychopathy distinction and are strongly related to other indices of anxiety.

Lykken's Low-Fear Hypothesis: State of the Art?

Lykken (1995) regards his 40-year-old theory as state of the art because it has survived testing in a variety of studies, provides a more plausible account of the psychopathic syndrome than other theories, and "unlike any of the competing hypotheses ... is compatible with the fact that about half of the MZ cotwins of psychopathic probands ... " are not themselves psychopaths (p. 154).

In our view, this conclusion depends on one's expectation for a state-of-the-art theory. We believe that such a theory should be consistent with past evidence, be able to accommodate new evidence, yield relatively unique predictions that provide differential support for the theory, and stimulate investigators to ask more refined questions. Ultimately, such a theory would also give rise to meaningful interventions or applications. Although Lykken's theory is consistent with much of the past evidence, we do not believe that it has performed well in accommodating new findings, generating differential support, or guiding the field in recent years.

The low-fear hypothesis is consistent with evidence that primary psychopaths frequently display poor passive avoidance learning and weaker electrodermal activity (EDA) in response to fear- or punishment-related stimuli (Lykken, 1995). However, this is not sufficient

to justify Lykken's assertion that the low-fear hypothesis is superior to alternative explanations. Although consistent with the low-fear hypothesis, the poor avoidance learning and EDA evidence are open to alternative interpretations and may be explained by other hypotheses. For instance, our response modulation hypothesis would attribute these findings to an information processing deficit that interferes with psychopaths' ability to reflect on their behavior or circumstances (see Newman & Wallace, 1993; Patterson & Newman, 1993, for a more detailed explanation). As noted by Lykken (1995, p. 179), Damasio's (1994) research with brain-damaged patients demonstrates that attenuated EDA may reflect a person's automatic (i.e., passive, involuntary) tendency to appreciate the significance of motivationally significant stimuli as opposed to innate differences in fearlessness.

Does the low-fear hypothesis explain new findings as well or better than other theories? We believe that there is reason to be skeptical. One of the early problems for the low-fear hypothesis concerns the fact that although fear stimuli typically do not generate as much EDA in psychopaths as in controls, psychopaths' heart rate response to such stimuli is at least as great as controls' (Hare, 1978). Of course, one interpretation of these heart rate data is that, like controls, psychopaths are fearful of punishment stimuli, although their sensitivity is manifested in a qualitatively different fashion. Lykken (1995), however, interprets this problematic finding as a "defensive" or inhibitory response that contributes to psychopaths' fearlessness. Unfortunately, because Lykken does not discuss the relation of the defensive response to innate fearlessness, it is unclear how the low-fear hypothesis accommodates this finding. Is the defensive response a manifestation of psychopaths' fearlessness or does the innate fearlessness of the psychopath derive from the ability to block out aversive stimuli?

Another consistent finding that has emerged since 1957 is that psychopaths exhibit inhibitory/regulatory deficits in nonthreatening situations. A common design, for instance, involves the use of monetary rewards and punishments to examine passive avoidance learning in psychopathic and nonpsychopathic participants. In a typical experiment, psychopaths can win money for making appropriate responses and lose money for inappropriate responses. Under such circumstances, psychopaths display poor self-regulation and lose more money than controls, despite the absence of fear-eliciting stimuli (e.g., Newman & Kosson, 1986; Newman, Patterson, & Kosson, 1987; Siegel, 1978; Thornquist & Zuckerman, 1995).

A low-fear theorist might argue that learning to avoid monetary punishments, like physical punishment

(e.g., shocks), in some way relies on fear or the capacity for fear conditioning. Of course, doing so would require them to reinterpret Schmauk's (1970) study in which psychopaths demonstrated poor passive avoidance in the physical punishment condition but good passive avoidance in the monetary punishment condition. To examine this issue more specifically, Newman and Kosson (1986) assessed passive avoidance learning using monetary punishments without a competing reward contingency. Psychopaths performed this task as well as controls, suggesting that they are no less motivated or capable of avoiding monetary punishments than controls when this is their primary task (i.e., their focus of their attention).¹

Following up on this early work, we recently examined sensitivity to "motivationally neutral" contextual cues in psychopaths and controls while they performed a speeded reaction time task. Despite instructions to ignore the contextual (i.e., peripheral) cues, the cues elicited associations that "automatically" interfered with performance of the primary task in normal participants (Gernsbacher & Faust, 1991). Although there are no punishment cues or punishment contingencies in this task, psychopaths were significantly less sensitive to (i.e., impaired by) the contextual cues (Newman, 1995; Newman, Schmitt, & Voss, 1996). Such findings are not easily accommodated by the low-fear hypothesis.

How would Lykken reconcile these findings with the low-fear hypothesis? It is unclear, because instead of dealing with the data directly, he attempts to dismiss their significance. Although most of these studies employed PCL-defined psychopaths and controls who were well matched on age, gender, and intelligence, Lykken implies that the experiments may be invalid because psychopaths and nonpsychopaths were subdivided using the WAS. As already discussed, we disagree with Lykken's assertions concerning the WAS and note, further, that much of the evidence documenting inhibitory/regulatory deficits in psychopaths in the

¹ Lykken suggests that this finding is flawed because psychopaths did not commit more errors in the reward-punishment condition than in the punishment-only condition, but as noted in the report, the punishment condition was more difficult overall. Lykken takes issue with this interpretation because the tasks were similar in every way with the exception of the instructions and payoffs employed. However, the results were unambiguous and the greater difficulty of the punishment-only task is not difficult to understand. In the reward-punishment stimuli, participants were instructed to respond to "good numbers" for reward and withhold responses to "bad numbers" to avoid punishment. This task is arguably more straightforward than responding to good numbers to avoid punishment and inhibiting responses to bad numbers to avoid punishment. Indeed, most participants in the first condition adopt the strategy "respond unless a bad number is present," whereas those in the latter try to learn both sets of stimuli (see Arnett et al., 1993).

absence of fear-related stimuli does not involve the WAS.² Thus, it seems important for a state-of-the-art theory to accommodate such findings.

Another body of evidence that has emerged since 1957 concerns the psychopath's language processing anomalies (Hare, Williamson, & Harpur, 1988). Comparisons between high and low scorers on the PCL reveal that the language of psychopaths is less fluent and more poorly structured (Williamson, 1991), is associated with distinctive nonverbal gestures (Gillstrom & Hare, 1988), and results in performance differences in experimental tasks involving language-related stimuli (Hare et al., 1988; Williamson, Harpur, & Hare, 1991). Moreover, their response to verbal stimuli suggests that their language processing is unusual in its lateralization (Hare, 1979; Hare & Jutai, 1988; Hare & McPherson, 1984). It is not immediately obvious how the low-fear hypothesis can account for these findings from Hare's lab. With regard to Williamson et al.'s (1990, 1991) results involving emotional language, Lykken states "the low-fear hypothesis also predicts weaker reaction to disturbing emotional stimuli, including the implications or connotations of emotional words or pictures" (p. 172). Whereas the low-fear hypothesis could certainly be stretched to accommodate such findings, Lykken's present attempt seems to contradict his previous assertion that "the psychopath has an attenuated experience, not of all emotional states, but specifically of anxiety and fear" (p. 118).

The results concerning unusual lateralization, distinctive gestures, and poor structure of psychopathic language are more difficult for Lykken to explain from a low-fear perspective. Indeed, Lykken is conspicuously silent about these data. His only response to this rigorous, systematic, and creative work with potential for addressing Cleckley's hypothesis in a meaningful and direct fashion is "it is too early to be sure what we can make of Hare's studies ... " (p. 173). We, too, recognize that independent replication of these findings is necessary, and so we have begun examining some of these effects in our own lab. Preliminary results provide striking support for the existence of language processing anomalies in psychopaths (Brinkley, 1995). In addition, Raine and his colleagues have independently replicated Hare's lateralization findings in a sample of

adolescent psychopaths (Raine, O'Brien, Smiley, Scerbo, & Chan, 1990). Although Lykken points out that the findings concerning "semantic aphasia" may be difficult to explain, continued independent replication of Hare's findings will make it necessary for any state-of-the-art theory to do just that.

Does the low-fear hypothesis yield relatively unique predictions that provide differential support for the theory? Lykken seems to think so, although we found his argument more confusing than convincing:

Unlike any of the competing hypotheses, most of which assume some qualitative innate defect of the central nervous system rather than a simple parametric difference, the low-fear hypothesis is compatible with the fact that about half of the MZ cotwins of psychopathic probands do not meet criteria for psychopathy themselves. (p. 154)

This statement is baffling for two reasons: First, the referent of Lykken's remark is unclear. What competing hypotheses assume an innate defect of the central nervous system? Throughout his book, Lykken refers repeatedly to the "septal-defect" psychopaths (p. 163) in discussing the Gorenstein and Newman model. Lykken refers to Newman's research investigating cognitive and learning deficits in psychopaths as "attempts to confirm his brain defect theory of psychopathy" (p. 176) and grossly distorts our hypothesis testing by stating that we "expected that psychopaths, due to their brain defect, would ... " (p. 180). We have not proposed that psychopaths are characterized by a septal or other brain defect, and the effects of experience on the development of psychopathy are not less apparent in our theorizing than in Lykken's own work (see Gorenstein & Newman, 1980; Patterson & Newman, 1993).

Perhaps Lykken (1995) is referring to Hare's theory. He notes, "Hare has long been partial to the view that something must be wrong with the psychopath's brain" (p. 171), but compare this with Hare's writing: "Some comments are needed on our studious avoidance of brain damage interpretations of psychopathy. We do so because our findings are more suggestive of group differences in function or structure than they are of neurological deficit, damage or dysfunction. ... Psychopaths may be 'wired up' differently without being neurologically damaged or impaired" (Hare et al., 1988). Although the theories that guide research in Newman's and Hare's laboratory are informed by physiology, Lykken's characterization of these perspectives as "brain defect" theories (p. 176) is inaccurate.

A second reason for our bewilderment regarding Lykken's characterization of the genetic evidence is that Lykken is knowledgeable in the area of behavior genetics and, as such, knows that concordance rates for

² Lykken asserts that our laboratory only employs the WAS when it is necessary to obtain significant results. This is not true. Our very first publication in the area employed the WAS (Newman, Widom, & Nathan, 1985). When we first began using the PCL, we did not administer the WAS because Hare and Harpur (1986) argued that it was not necessary to divide PCL-identified psychopaths into primary and secondary groups. However, with experience we came to a different conclusion and resumed using the WAS as soon as we could.

schizophrenia do not exceed 50% either. Does he deny the existence of cognitive deficits or brain involvement in this disorder, too? More to the point, none of the competing theories cited by Lykken rejects the significant role played by developmental or other psychobiological processes in moderating socialization and the expression of psychopathy. Perhaps Lykken could clarify the significance of this evidence.

Finally, a state-of-the-art theory of psychopathy should drive interest and research in the field. Lykken's (1995) own assessment regarding the low-fear hypothesis is that it "has not been taken very seriously, even by researchers whose own findings have contributed to its empirical support." Even Lykken appears to have been unmoved by the theory: "I have to confess to having been a neglectful parent of my (adopted) brainchild, having sent it forth on its own without further support after that initial study" (p. 155).

Why hasn't the low-fear hypothesis generated more research or been taken more seriously? One possibility is that Lykken's theory or presentation is too compelling. It is as if all of the important questions have been answered and there is nothing left to do but retell the story every decade or so. Indeed, Lykken's book provides few leads regarding research questions in need of investigation.

A second possibility concerns the nonspecific nature of Lykken's proposal. What particular processes are responsible for the psychopath's failure to avoid punishment or respond emotionally to punishment stimuli? Lykken's theory does not clearly identify specific mechanisms that may be tested or compared to the proposals of others. Although such proposals entail a risk of refutation, such proposals motivate investigation to evaluate them and provide a basis for comparing theories.

A third possibility is that the active investigators in the field no longer agree with the premise of Lykken's theory, which places psychopathy on a continuum of "normal" behavior. In this regard, it is worth highlighting the fact that Lykken (1957) set out to investigate a different concept of psychopathy than that currently embraced by the field. Whereas Cleckley (1976) believed that psychopathy "constituted a grave personality disorder that distances the psychopath from the reality of human experience to a degree comparable to the effects of psychosis" (Lykken, 1995, p. 134), Lykken (1957) argued that assessing the "normality of the affective ... experience" was destined to be "subjective and unreliable" (p. 6). He proposed "that a simpler hypothesis was both adequate and testable" (p. 134): "My theory of primary psychopathy was that all the components of the syndrome outlined by Cleckley's 16 criteria might be expected to be found in a normal but

relatively fearless child who had been subjected to the typical parenting methods ... " (p. 135). Thus, instead of investigating the pervasive, cognitive-emotional deficit postulated by Cleckley, Lykken focused on low fear/anxiety.

Lykken (1995) works hard to establish the relevance of his proposal for the primary (i.e., Cleckley) psychopath but his task is not easy (pp. 135-144). Whereas many experts regard "pathological egocentricity and incapacity for love" and "general poverty in major affective reactions" as core features of psychopathy, Lykken regards them as "derivative" symptoms (p. 140), meaning that they are secondary to inadequate fear. This reinterpretation of the psychopath's core features simplifies one's theorizing, but does the simplified theory elucidate the original syndrome? Indeed, as already discussed, we have serious misgivings about the applicability of the low-fear hypothesis to primary psychopathy. We believe that the term "primary psychopathy" should be reserved for individuals whose cognitive-emotional deficits interfere with their social adjustment regardless of their opportunities for socialization.

Despite our reservations concerning the applicability of Lykken's low-fear hypothesis to primary psychopathy, we recognize that Lykken has provided a valuable framework for conceptualizing the relation between temperament and socialization. As such, the model appears relevant for the large majority of antisocial personalities described in his book. Although the model may not be optimum for primary psychopathy, it may well be a state-of-the-art theory of sociopathy and the other psychopathies depicted in his book.

Lykken's conceptualization of sociopathy emphasizes poor parenting, but it is clear that such environmentally mediated influences interact with a child's temperament. Given a disadvantageous family environment, children with an above average endowment of impulsivity, sensation seeking, or fearlessness will be at greater risk for developing sociopathy than a highly introverted child exposed to the same parenting conditions. Lykken's diathesis-developmental model is readily applied to such conditions: Whereas shy, risk-averse individuals will be unlikely to develop an antisocial personality despite extremely poor parenting, the most fearless sensation seekers will be likely to display antisocial behavior, despite the most earnest parenting. Moreover, Lykken's proposals regarding a continuum of predisposing, but otherwise, normal temperaments are well-suited to the task of predicting which children, exposed to ineffective parenting, are at greatest risk. By referring to the predisposing temperament, it is even possible for Lykken to predict the specific manner in which a child's risk will be expressed. This is no small accomplishment.

Summary and Conclusions

The Antisocial Personalities provides a broad and useful perspective on the range of factors influencing antisocial behavior. We have, however, expressed concerns about Lykken's treatment of the primary psychopath. Specifically, we have reservations about: (a) the distinctiveness of Lykken's psychopathy construct, (b) Lykken's idiosyncratic approach to differentiating primary and secondary psychopaths, and (c) the apparent inability of the low-fear hypothesis to organize and drive research on primary psychopaths.

Although we applaud Lykken's general strategy of advancing a theory and contrasting it with competing hypotheses, we cite problems with the specifics of his arguments. In particular, Lykken's low-fear hypothesis is an encompassing theory capable of "explaining" past research, but he neglects to demonstrate how it can accommodate more recent evidence involving language processing anomalies and deficient self-regulation in nonthreatening situations. Moreover, by inaccurately characterizing the etiological foundation of other theoretical perspectives, Lykken attacks "straw men" instead of providing a rigorous comparison of the evidence concerning their diverse predictions. We hope that the foundation provided by Lykken's book will stimulate other researchers to examine the competing theories, identify their competing hypotheses, and subject them to rigorous investigation.

Notes

Preparation of this article was supported by a research grant from the National Institute of Mental Health. We thank Tim Baker and David Kosson for their valuable comments on an earlier version of this article.

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