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# Psychopathic personality and racial/ethnic differences reconsidered: a reply to Lynn (2002)

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**Abstract**

Lynn (2002) argued that psychopathic personality is distributed disproportionately across racial and ethnic groups, and that these putative group differences were likely attributable to genetic factors. Lynn's analysis is problematic on three primary counts. First, he equates psychopathy with generalized antisocial behavior and social deviance and fails to distinguish longstanding personality-based from behavior-based conceptions of this syndrome. Second, Lynn presumes rather than demonstrates that genetic factors explain race differences in antisocial behavior and social deviance, neglecting such potential alternative explanations as socioeconomic status and measured verbal intelligence. Third, Lynn presents an evolutionary explanation for putative racial and ethnic group differences in psychopathy that fails to reflect current methods and practices of evolutionary biology and genetics. A lack of awareness that Lynn's conclusions are premature and unwarranted, given the present state of the data, may have far-reaching legal and public policy implications.


*Keywords:* Psychopathy; Antisocial personality; Race; Ethnicity; Crime; Violence

Psychopathic personality disorder (psychopathy) is a construct defined by a relatively distinct constellation of affective, interpersonal and behavioral characteristics (Cleckley, 1941; Hare, 1991). In a recent issue of this journal, Lynn (2002) argued that the prevalence of psychopathic personality is distributed disproportionately across racial groups, with Native Americans and Blacks evincing higher rates than Whites, and East Asians evincing lower rates than Whites.[1] Furthermore, he argued

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[1] Throughout this article, "Black" is used to refer to individuals of African descent and "White" is used to refer to Caucasian individuals.

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that these differences are probably attributable to genetic factors and are consistent with evolutionary theories of race differences. Lynn reviewed a wealth of data from multiple sources (e.g. group differences in self-report personality scales, crime rates, relationship stability) to support this argument.

Lynn's (2002) analysis of racial and ethnic differences in the prevalence of psychopathy is flawed in that he (a) equates psychopathy with generalized antisocial behavior and social deviance, (b) assumes that genetic factors explain putative differences in antisocial behavior and social deviance without addressing plausible alternative explanations, and (c) provides an evolutionary explanation for racial and ethnic group differences that is inconsistent with current methods and practices of evolutionary biology and genetics. In this article we review the primary limitations of Lynn's claims and the data upon which they are based. Notably, we limit our critique specifically to racial and ethnic differences in psychopathic personality per se. We do not address controversies about racial and ethnic differences in IQ estimates, nor do we examine directly the scientific basis of Rushton's (1988, 1991a, 1991b, 1994a, 1994b, 2000) theory of race differences (for reviews, see Anderson, 1991; Cernovsky & Litman, 1993; Cunningham & Barbee, 1991; Fairchild, 1991; Gorey & Cryns, 1995; Graves, 2002; Lieberman, 2001; Neapolitan, 1998; Vanderwolf & Cain, 1991), which underlies Lynn's basic arguments. However, our criticisms of Lynn's genetic/evolutionary explanations for race differences can be applied to Rushton's work as well.

## 1. Equating psychopathy with social deviance

Lynn begins his analysis by citing Herrnstein and Murray's (1994) observation that intelligence cannot explain fully racial and ethnic differences in such socially deviant phenomena as crime, poverty, long-term unemployment, and teenage pregnancy. He hypothesizes that psychopathy may further explicate these variations. To support this hypothesis, he presents effect size data on racial and ethnic group differences on the Psychopathic Deviate (Pd) subscale of the MMPI, followed by a mass of data on some of the group discrepancies that he proposes psychopathy explains. These include racial and ethnic differences in childhood disorders and developmental maturity (e.g., conduct disorder, ADHD, moral reasoning, delay of gratification), and adult social deviance (sexual, work/financial, child abuse/neglect, aggression/crime).

Lynn's argument is tautological in that it equates psychopathic personality disorder (the proposed explanatory variable) with social deviance (the proposed effect). His presentation of data on group differences in various indices of social deviance appears to be underpinned by an assumption that these indices are "expressions of psychopathy" (p. 306). However, his operationalization of psychopathic personality disorder (e.g. symptoms of antisocial personality disorder) overlaps heavily with some of its purported expressions. Although it is plausible that crime, unemployment, and other variables may lie causally downstream from psychopathy, Lynn presents no data on group differences in psychopathic personality disorder per se, far less on any link between psychopathy and these purported outcomes.

### *1.1. Personality- and behavior-based theories of psychopathy*

This element of Lynn's argument neglects a longstanding debate about "the primacy of and relationship between two constructs that are consistently distinguished in the [psychopathy] literature"

(Pilkonis & Klein, 1997, p. 109). The first construct is a constellation of core personality traits that generally include callousness, egocentricity, superficial charm, failure to form close emotional bonds, deceitfulness, and externalization of blame (Lilienfeld, 1998). The second construct reflects a long history of observable socially deviant and antisocial behaviors, including many of those reviewed by Lynn. "Personality-based" models of psychopathy, exemplified by Cleckley's seminal work (1941; see also Karpman, 1961; McCord & McCord, 1964) reflect the first construct, whereas "behavior-based" models, typified by recent versions of the DSM (APA, 1980, 1987, 1994; see also Robins, 1966), reflect the second (Lilienfeld, 1994; Widiger et al., 1996).

According to the personality-based model, socially deviant behavior is neither necessary nor sufficient for a diagnosis of psychopathy (Lilienfeld, Purcell, & Jones-Alexander, 1997). First, in sharp contrast with Lynn's apparent assumptions, one may possess core traits of psychopathy without manifesting behavior that those in power would define as socially deviant, such as criminality. As observed by Hare (1993), "subcriminal" psychopaths

> . . .never go to prison or any other facility. They appear to function reasonably well—as lawyers, doctors, psychiatrists, academics, mercenaries. . .and so forth—without breaking the law, or at least without being caught and convicted. These individuals are every bit as egocentric, callous, and manipulative as the average criminal psychopath; however, their intelligence, family background, social skills, and circumstances permit them to construct a façade of normalcy and to get what they want with relative impunity (p. 113; see also Cleckley, 1941).

Second, general criminality may be caused by a host of factors other than psychopathy (Blackburn, 1998; Harpur, Hakistan, & Hare, 1988). As observed by Hare, Hart, and Harpur (1991), "[a]ll of those who fulfill the APD criteria may be antisocial, but they may differ greatly in their motivations for being so and in significant interpersonal, affective, and psychopathological features, such as the capacity for empathy, remorse, guilt, anxiety or loyalty" (p. 393). Because they have multiple determinants (e.g. poverty, substance abuse, genetic factors), deviant and antisocial behavior are insufficient grounds for a diagnosis of psychopathy and belong in a different conceptual domain (Blackburn, 1998).

Lynn dismisses this debate with an assertion that, "[d]espite these fine distinctions, for practical purposes psychopathic personality and anti-social personality can be regarded as largely synonymous descriptions of the same condition" (p. 274). He applies the term "psychopathic personality" to both the current, behavior-based diagnostic criteria for antisocial personality disorder (APD, APA, 1994) and the diagnostic criteria for psychopathic personality summarized in the most widely accepted measure of psychopathy, the Psychopathy Checklist-Revised (PCL-R, Hare, 1991).

### *1.2. Empirical support for distinguishing personality- and behavior-based constructs*

A substantial body of research on the PCL-R supports the distinction between psychopathy and APD. The PCL-R is underpinned by a two-factor model of psychopathy that is undoubtedly the most influential recent conceptualization of this construct. This instrument contains two

moderately correlated ($r \approx 0.50$) scales identified via factor analysis. Using Harpur et al.'s (1988) descriptive labels, Factor 1 items reflect the interpersonal and affective core of psychopathy, or the "selfish, callous and remorseless use of others," whereas Factor 2 items describe a collection of socially deviant behaviors, or a "chronically unstable and antisocial lifestyle" (see also Hare et al., 1990; Hart, Cox, & Hare, 1995).[2] Factors 1 and 2 grossly represent the personality-based and behavior-based models, respectively. As shown in Table 1, most of the overlap between the content

Table 1
Comparison of APD and PCL-R diagnostic criteria

| PCL-R Factor 1 | APD Criteria | PCL-R Factor 2 | APD criteria | PCL-R unclassified | APD criteria |
|---|---|---|---|---|---|
| Glibness/superficial charm | | Need for stimulation /proneness to boredom | Reckless behavior | Promiscuous sexual behavior | Failure to sustain a monogamous relationship for >1 year |
| Grandiose | | Parasitic lifestyle | Inability to sustain consistent work | Many short-term marital relationships | |
| Pathological lying | No regard for truth (lying/conning) | | Failure to honor financial obligations | Criminal versatility | Unlawful behavior |
| Conning/ manipulative | | | | | Inability to function as a responsible parent |
| Lack of remorse | Lacking remorse | Poor behavior controls | Frequent fights/ assaults | | |
| Shallow affect | | Early behavior problems | Childhood CD | | |
| Callous/lack of empathy | | Lack of realistic long-term goals | | | |
| Failure to accept responsibility | | Impulsivity | Failure to plan ahead/ impulsivity | | |
| | | Irresponsibility | | | |
| | | Juvenile delinquency | | | |
| | | Revocation of conditional release | | | |

[2] Notably, the PCL-R's inclusion of this factor is "not particularly satisfying if psychopathy is to be understood as a constellation of personality traits" (Widiger & Lynam, 1998, p. 180). In fact, Cooke and Michie (2001) recently argued that a three-factor model of psychopathy was superior to the traditional two-factor model. This three-factor model dropped several Factor 2 items that were found to be poor indicators of psychopathy, including promiscuous sexual behavior, many short-term marital relationships, poor behavioral controls, early behavior problems, juvenile delinquency, and criminal versatility. Notably, these items tap many of the variables on which Lynn relies to support his claim for racial and ethnic differences in psychopathy.

of the PCL-R and the current APD criteria is explained by Factor 2 items rather than the interpersonal and affective traits of Factor 1 (see Hare et al., 1991).

The results of research conducted over the past two decades with the PCL-R and other measures also support the distinction between APD and psychopathy. First, psychopathy is less prevalent and more powerful in predicting a range of negative outcomes than APD. In correctional settings where these disorders have been most thoroughly studied, 50–80% of offenders typically meet diagnostic criteria for APD, whereas only about 15–30% of offenders are classified as psychopathic, based on cutting scores established for the PCL-R (Hart & Hare, 1997; see also Widiger & Corbitt, 1997). Moreover, most studies of correctional, forensic, and psychiatric populations have shown that the predictive efficiency of psychopathic personality disorder exceeds that of APD for such outcomes as community violence, violent and general criminal recidivism, treatment termination and failure, and disciplinary infractions (see Edens, Skeem, Cruise, & Cauffman, 2001; Hare, Clark, Grann, & Thornton, 2000; Hare et al., 1991; Hart & Hare, 1997; Hemphill, Templeman, Wong, & Hare, 1998; Monahan et al., 2001; Rice, 1997; Salekin, Rogers, & Sewell, 1996).

Second, the two factors of the PCL have different correlates, supporting the discriminant validity of personality- and behavior-based constructs (Hare, 1991; Harpur, Hare, & Hakistan, 1989). Across correctional, forensic, psychiatric, and college samples, Factor 2 scores have been found to be much more strongly related to clinical diagnoses and prototypicality ratings of APD than Factor 1 scores (for reviews, see Hare, 1991; Hart et al., 1995). For example, based on a sample of 48 psychiatric patients, Hart et al. (1995) found that interviewers' Personality Disorder Examination (PDE; Loranger, 1988) ratings of APD correlated highly with PCL:SV Factor 2 scores ($r = 0.83$), but only moderately with Factor 1 scores ($r = 0.48$). In contrast, the interpersonal and affective traits captured by Factor 1 scores consistently correlated more highly with other "Cluster B" personality disorders (e.g. narcissistic; Hare, 1991; Hart et al., 1995).

The type of variables that are associated most strongly with the socially deviant and antisocial behavior of Factor 2 is particularly relevant to Lynn's analysis. Unlike Factor 1 scores, Factor 2 scores correlate moderately to strongly with trait anxiety, substance abuse, socioeconomic status (SES), educational attainment, and (despite Lynn's claim to the contrary) intelligence test scores (for reviews, see Hare, 1991; Hart et al., 1995). For example, based on a sample of 313 correctional inmates, Harpur et al. (1989) found that low social index and occupational class (as operationalized by Hollingshead & Redlich, 1958) correlated negligibly with Factor 1 scores ($r = 0.02$, 0.08, respectively), but moderately with Factor 2 scores ($r = 0.49$, 0.50, respectively). Moreover, inmates' educational attainment upon entry to prison was weakly associated with Factor 1 ($r = -0.12$), but moderately associated with Factor 2 ($r = -0.42$). Factor 2 is also disproportionately associated with various intelligence test scores, particularly those that tap individuals' experience, acculturation, and education (see Hare, 1991; Ridenour, Marchant, & Dean, 2001).

In summary, Lynn's argument is underpinned by an equation of psychopathy with APD that contradicts both a longstanding theoretical distinction between these constructs and a substantial body of research conducted over the past two decades. This research indicates that psychopathy is less prevalent, possesses greater predictive efficiency for a range of negative outcomes, and manifests a different pattern of correlates than APD. APD is neither unique nor specific to psychopathic personality deviation, as suggested by its association with individuals' SES, educational attainment, and intelligence test scores.

### 1.3. MMPI Pd scale is a poor proxy for psychopathic personality

Lynn's equation of psychopathy with APD and social deviance is expressed in his reliance on racial and ethnic group differences on the "Psychopathic Deviate" (Pd) scale of the MMPI and MMPI-II. Lynn argues that the average effect size on Pd (i.e. White/Black $d = 0.40$) "should be adopted provisionally as the best available estimates of the racial and ethnic differences in psychopathic personality" (p. 277). There are strong reasons for questioning this argument.

First, Pd correlates only modestly (and often nonsignificantly) with psychopathy as defined by total scores on the PCL/PCL-R, with correlations ranging from 0.19 to 0.29 (Cooney, Kadden, & Litt, 1990; Hare, 1991). For example, a study of 274 prison inmates, Hare (1985) found that the Pd *and* Hypomania (Ma) scales of the MMPI were only moderately correlated with the PCL, both singly (Pd, $r = 0.26$, Ma, $r = 0.27$), and in combination (Pd + Ma, $r = 0.35$). When the latter scale combination (Pd + Ma) was factor analyzed with the PCL, it loaded much more strongly (0.71) on a factor comprised of various self-report scales than on the factor including the PCL and other rating scales of psychopathy (0.20). Second, the modest relationship between Pd and the PCL is largely explained by the association between Pd and Factor 2 ($r = 0.28$–$0.31$) rather than Factor 1 ($r = 0.05$–$0.11$; see Cooney et al., 1990; Hare, 1991). Thus, the Pd scale is described more accurately as a measure of "chronic antisocial behavior" than a specific constellation of the callous and unemotional traits of psychopathy (Lilienfeld, 1996).

Third, the Pd Scale, like antisocial behavior in general, is multifactorial and heterogeneous. For this reason, Harris and Lingoes (1955) rationally derived five subscales to assist clinicians in interpreting Pd. In a study of forensic patients (Meloy & Gacono, 2000), only one subscale ("Authority Problems"), which contains many items that merely assess nonspecific antisocial behaviors, was significantly related to PCL-R psychopathy ($r = 0.31$). Thus, even when attempts are made to reduce the heterogeneity of the Pd scale, its overlap with the core interpersonal and affective traits of psychopathy is limited.

### 1.4. Childhood behavior disorders are poor proxies for psychopathic personality

To support his hypothesis regarding racial and ethnic group differences in psychopathy, Lynn (2002) provides data regarding group differences in conduct disorder (CD), which he asserts is "...an analogue of psychopathic personality in older adolescents and adults" (p. 277). This assertion overreaches what is currently known about the relationship between antisocial conduct among children and adolescents and adult psychopathy. First, like APD among adults, CD is a heterogeneous behavioral disorder that may be caused by a host of factors other than psychopathic personality deviation. Only a subset of children and adolescents with CD ($\sim$30%) manifest the personality characteristics associated with psychopathy (see Forth & Burke, 1998; Forth & Mailloux, 2000). Second, although a large percentage of psychopathic adults probably evinced significant externalizing behavior problems during childhood, it is a logical fallacy to argue that those diagnosed with CD will mature into adult psychopaths (Edens, Skeem et al., 2001; Hart, Watt, & Vincent, 2002). In fact, in the Epidemiological Catchment Area Study (ECA), 74% of participants with childhood CD did *not* mature into adults with APD (Robins, Tipp, & Przybeck, 1991), much less into psychopaths. At present, we have no longitudinal evidence that even the smaller class of individuals who manifest apparent traits of psychopathy during childhood or

early adolescence will mature into adult psychopaths (Edens, Skeem et al., 2001; Seagrave & Grisso, 2002; Vincent & Hart, 2002). Simply put, CD is far from a straightforward analogue of adult psychopathy.

To further bolster his claim of racial and ethnic differences in psychopathy, Lynn cites data on group differences in the prevalence of attention deficit hyperactivity disorder (ADHD) during childhood. He postulates "the close association between ADHD and conduct disorder in childhood and psychopathic behaviour and crime in adulthood leads to the expectation that the racial differences present in psychopathic personality and conduct disorder should also appear in ADHS (sic)" (p. 281). He then reviews several studies demonstrating higher rates of ADHD among Black children in comparison to White children. This analysis is problematic on two levels. First, Lynn overestimates the significance of ADHD in the etiology of APD. Although it is true that higher rates of antisocial behavior are seen in adulthood among those who were diagnosed with ADHD during childhood, this relationship seems to be driven more by the presence of comorbid conduct problems than by ADHD per se (Herrero, Hechtman, & Weiss, 1994; Lilienfeld & Waldman, 1990; see also Faraone, Biederman, Jetton, & Tsuang, 1997; Kosson, Cyterski, Steuerwald, Neumann, & Walker-Matthews, 2002). Moreover, the few studies that have found a modest relationship between ADHD and subsequent antisocial conduct among children without concurrent CD diagnoses have not controlled for the presence of Oppositional Defiant Disorder symptoms, which may be the more salient etiological risk factor in the development of CD and later APD (for a review, see Lahey & Loeber, 1997). Given such findings, some researchers have gone so far as to argue that ADHD alone and ADHD comorbid with CD may represent distinct disorders with differing etiologies (Lynam, 1996; see also Thapar, Harrington, & McGuffin, 2001).

Second, Lynn does not consider environmental explanations for ADHD, which may contribute to explaining any disproportionate prevalence among Black children. Aside from the known correlates of poverty, low SES, and single-parent homes, there also is some evidence to suggest that racial biases may exist in the identification of ADHD symptoms by schoolteachers (Reid, Casat, Norton, Anastopoulous, & Temple, 2001; Reid et al., 1998), and that social class biases may be evident among other professionals (e.g. school and child psychologists; Cuccaro, Wright, Rownd, Abrahamson et al., 1996).

### *1.5. Lynn's incomplete presentation of data on social deviance*

Notably, Lynn sometimes presents information that is consistent with his hypothesis that group differences in social deviance exist while omitting data that contradict this hypothesis. Two examples are provided here. First, despite his (inappropriate) emphasis on racial and ethnic group differences in antisocial personality, Lynn does not present data on differences in APD. These data are available in one of the most famous studies of mental disorder, the ECA Study. In this study, the estimated lifetime prevalence rate of APD for White participants was 3.9% and that for African-American participants was 3.7% (Robins et al., 1991). Across multiple methods of assessing prevalence, there were no meaningful differences between racial groups in APD. As explained earlier, APD is not psychopathy. Although these data do not speak to differences in psychopathy per se, they suggest that Lynn's review (given his focus on social deviance) is incomplete.

Second, Lynn offers several forms of evidence that Blacks are more prone to engaging in risk-taking and reckless behavior than Whites. Some of this evidence is derived from a national youth survey (Flint, Yamada, & Novotny, 1998) in which Blacks were more likely than Whites to endorse the statement, "I get a kick out of doing things every now and then that are a little risky or dangerous." Lynn does not indicate that this lone question was part of a much broader survey regarding race differences in the onset and progression of cigarette smoking, the results of which indicated that a significantly higher percentage of White teens progressed from experimentation with cigarettes to regular smoking. The lone survey question notwithstanding, Black teens arguably are less reckless than Whites, if cigarette smoking is considered an indicator of recklessness. Similar results also have been obtained in relation to race differences in illicit substance use and other forms of risk-taking behavior (Blum et al., 2000; Bray, Adams, Getz, & Baer, 2001; Shillington & Clapp, 2000). Lynn does not address these issues.

### *1.6. Are there meaningful racial and ethnic differences in psychopathic personality?*

Unlike the Pd scale, childhood disorders, APD, and various indices of 'social deviance' cited by Lynn, the PCL-R and its derivatives are the most heavily researched and perhaps best-supported measures of psychopathy available. Lynn notes that the PCL-R "has not been used to any significant scale to investigate racial and ethnic differences" (p. 275). This statement neglects a growing body of research on potential test bias of the PCL-R with racial and ethnic minorities. Investigations of the construct validity of the PCL-R suggest that the measure's pattern of external correlates often differs in White and Black offenders (for a review, see Cooke, Kosson, & Michie, 2001). For example, Black psychopaths (as defined by the PCL-R) do not manifest the same passive avoidance deficits on laboratory tasks as White psychopaths (e.g. Kosson, Smith, & Newman, 1990; Newman & Schmitt, 1998). As observed by Cooke et al. (2001), this may mean that psychopathy is (a) more difficult to measure in Black than White samples, or (b) influenced by genetic and socio-cultural factors that differ across ethnic groups.

Cooke et al. (2001) effectively ruled out the former explanation by conducting confirmatory factor analyses and item response theory analyses of the PCL-R, based on a sample of 732 US jail and prison inmates. First, they found no significant difference between Black and White samples in the factor structure of this measure. Second, although several PCL-R items were significantly more discriminating in Black or in White groups, the authors concluded that these differences cancelled one another out such that the PCL-R was "similarly useful in diagnosing psychopaths in both ethnic groups" (p. 539). As noted by the authors, however, these results do not indicate that psychopathy is characterized by the same underlying mechanisms in Black and White individuals: individuals who manifest similar traits may have developed those traits via different routes (issue "b," above).

Lynn's (2002) assertion that the PCL-R has not been used to investigate racial and ethnic group differences in psychopathy may reflect that there have been relatively few simple comparisons of group differences in total scores. Although it is true that no normative data on the PCL-R or PCL:SV have been collected on representative community samples, sixteen studies of correctional and clinical samples provide the simple statistics required to compute the staple of Lynn's analysis, an effect size for group differences. Although a recent meta-analysis of all sixteen studies (Skeem, Edens, & Colwell, 2002) indicated that the difference between Black and White subjects

in psychopathy was small and insignificant ($d = 0.11$, ns), effect sizes were heterogeneous across studies, suggesting the presence of a moderator variable. Effect sizes for the most frequently studied group, male correctional inmates, were homogeneous across nine studies. A meta-analysis of these studies also indicated that racial differences in psychopathy were small and insignificant ($d = 0.14$, ns). In fact, across all 16 studies, Blacks obtained scores that were a sample-weighted average of 0.7 total PCL-R points higher than Whites, less than one-quarter the size of the PCL-R's standard error of measurement.

In summary, the first flaw in Lynn's argument lies in its equation of two constructs that have long been distinguished both in theoretical and empirical work: psychopathy and antisocial behavior. There is evidence that the chief measures of 'psychopathy' on which Lynn selectively relies are poor proxies for the core interpersonal and affective traits of this disorder. Data on measures that better assess these psychopathic traits indicate that Blacks obtain only marginally higher scores than Whites.

## 2. Omission of alternative explanations for purported racial differences

Even if there were substantial racial and ethnic differences in psychopathy, different mechanisms may explain these differences (Cooke et al., 2001). The second flaw in Lynn's (2002) argument relates to his assertion that differences across various indices of social deviance reflect *genetically based* differences in psychopathy among groups. Although he notes that it would be difficult to develop an 'environmentalist explanation' for the data he reviews based on such factors as 'white racism' (p. 309), Lynn provides relatively little evidence to support his genetic assertion. In this section, we construct alternative non-genetic explanations for some of the group differences that Lynn reports. To be clear, we do not contend that any group differences are necessarily wholly environmentally based. Our argument is that, given the state of the science, we cannot reasonably determine the extent to which etiological influences for putative racial differences in psychopathy are genetic, environmental, or both. We begin with the MMPI Pd scale and criminality.

### 2.1. Alternative explanations for differences in Pd

Lynn suggested that racial and ethnic differences in MMPI Pd scores should be accepted as the best available estimates of differences in psychopathy. Aside from concerns about the extent to which Pd actually assesses psychopathic traits, there is reason to question whether these differences remain once other potential explanatory variables are controlled. Racial differences on the MMPI/MMPI-2 have been a focus of research for decades and a considerable body of research suggests that, once other sociodemographic factors are statistically taken into account, apparent racial and ethnic differences on the MMPI/MMPI-2 are significantly attenuated or disappear (Dahlstrom, Lachar, & Dahlstrom, 1986; Graham, 1999; Greene, 2000). For example, Timbrook and Graham (1994) compared scores of Black and White men ($n = 116$ for each group) from the MMPI-2 normative sample The authors found that the only scale that evinced a significant difference across the groups was the Schizophrenia (Sc) scale 8. The (non-significant) measure of effect size for Pd across these two groups was a modest 0.33. Interestingly, a significant difference

was noted for women ($n = 176$ for each group) on Pd even after controlling for age, education, and family income, although the disparity was only 3.2 T-score points ($d = 0.33$). We should note that differences of less than 5 T-score points on the MMPI/MMPI-II historically have not been considered to be 'meaningful' by experts in the field (e.g. Greene, 1987). Thus, what Lynn calls genetically based racial and ethnic group differences in psychopathy may also reflect differences in sociodemographic factors among racial groups.

Notably, statistical control in such studies can constitute overcontrol if race itself partially produces differences in such sociodemographic factors as education and income (see Meehl, 1971). *If* this were the case, controlling for these sociodemographic factors would suppress some of the variance of race itself. Although it is reasonable to conduct covariance analyses to explore hypotheses, promising hypotheses should be further investigated using behavior genetic (e.g. twin and adoption studies) or other more sound designs.

Lynn presents an adoption study to support a genetic explanation for putative racial differences in psychopathy. However, his analysis of this study is incomplete. Specifically, Lynn cites a secondary reference (Whitney, 1996) to an unpublished dissertation (DeBerry, 1991) to demonstrate that Black children adopted into White families displayed significantly higher than normal Pd scores in late adolescence. Lynn argues that, even when raised by White parents, Blacks' genetically driven psychopathic traits become readily apparent as they mature. There are two primary problems with this argument. First, research on the MMPI indicates that elevations on Pd are *normative* among adolescents. In fact, various investigators attempted to develop 'adolescent norms' for the MMPI because adolescents obtained pseudopathological scores on this instrument (for an overview, see Archer, 1987). Inspection of the DeBerry data indicates that the average Pd score for Black adolescents was 60.5 (SD = 12.9), which is essentially what one would expect in a sample where the modal age was 16 and the average age was 20 (Archer, 1987). Simply put, these relatively high scores merely reflect the developmental effects of adolescence.

Second and more importantly, Lynn does not discuss DeBerry's (1991) data on the non-adopted White children from these same families. The average Pd score for non-adopted White adolescents actually was slightly higher ($M = 61.1$, SD = 13.0) than that of the adopted Black adolescents. These data clearly contradict Lynn's argument that this study indicates a genetic effect for racial differences in psychopathy. Instead, this research indicates that when environments are held somewhat constant, Pd scores are comparable across racial groups.

### 2.2. Alternative explanations for differences in criminality

Lynn also cites a number of studies examining racial and ethnic differences in crime and imprisonment rates to support his hypothesis that differences in psychopathy are genetically based. A number of these studies, however, do not control for other variables that potentially contribute to differences across races. For example, Smith (1997) investigated rates of imprisonment per 10,000 population for Black, White, and Asian men in England in 1993 and found that Blacks were imprisoned at a significantly higher rate (211.8) than Whites or Asians (33.1 and 32.9, respectively). Smith explained that such discrepancies may reflect bias at various stages of the criminal justice system. For example, Black males were overrepresented among arrests for crimes that were open to police discretion (e.g. drug offenses), but were relatively infrequently arrested for crimes that tend to be investigated with absent knowledge of the perpetrator's ethnicity (e.g. burglary). Lynn

does not address the hypothesis of bias in the justice system (see Steffensmeier, Ulmer, & Kramer, 1998), apparently assuming that any racial differences in crime must be explained by genetic variation.

Lynn also presents statistics from secondary references (Amar, 1998; Taylor & Whitney, 1999) to argue that Blacks have higher crime and imprisonment rates than Whites. These studies are based on national crime data (e.g. Uniform Crime Reports) that simply *describe* imprisonment as a function of the number of offenders per racial population. The studies do not *explain* any racial differences. Several relatively sophisticated studies have revealed that apparent racial or ethnic differences in crime or imprisonment disappear after controlling statistically for such confounding variables as socioeconomic status. For example, Neapolitan (1998) examined International Crime Statistics for Blacks, Asians, and Whites, after controlling for a number of demographic variables (e.g. SES, household size, youth population). He found that, although Blacks had higher crime rates, the effect of race was no longer significant once these variables were controlled. Similarly, Centerwall (1995) examined domestic homicide data (i.e., number of victims per 10,000 race-specific population), controlling for household crowding. He found that the 6.3 times greater risk for Blacks than Whites dropped to a non-significant 1.2 when groups were stratified by household crowding. Moreover, Paschall, Flewelling, and Ennett (1998) found that racial differences in self-reported violent behavior existed only for those of low socioeconomic status, with Blacks being twice as likely as Whites to engage in such behavior. However, further examination of the data revealed that Blacks' greater *exposure* to violence completely accounted for the racial differences in violent behavior. This body of research suggests that factors such as poverty are plausible alternative sources of influence on individuals' crime rates.

### 2.3. Alternative explanations for deficits in moral reasoning

Lynn (2002) departs from his primary focus on socially deviant behaviors as putative indices of psychopathy to present data on racial and ethnic differences in children's moral reasoning. Lynn notes that "[w]eakness of moral understanding is a central feature of psychopathic personality" (p. 282). Although affective deficits and emotional detachment often are regarded as core features of psychopathy, Lynn references measures of moral reasoning that consist "entirely of verbal reflective judgments about hypothetical moral dilemmas" (Jurkovic, 1980, p. 716) that are unlikely to tap these core features.

Specifically, Lynn presented the scores of high school children from different racial and ethnic backgrounds on a self report, vignette-based measure of moral judgment, the Defining Issues Test (DIT; Rest, Cooper, Coder, Masanz, & Anderson, 1974; Rest, Narvaez, Thoma, & Bebeau, 1999). Based on these scores, he concluded that there are racial and ethnic group differences in moral reasoning and suggested that these signify differences in psychopathy. There is little support for this argument.

First, there is little evidence that there are group differences in moral reasoning after controlling for such factors as intelligence and SES. Several investigators have found that scores on the DIT are significantly associated with various indices of intelligence, aptitude, and achievement, with correlations in the 0.20–0.50 range (Rests, 1979; see also Dollinger & LaMartina, 1998; Murk & Addleman, 1992; cf. Thoma, Narvaez, Rest, & Derryberry, 1999). Although one might argue that higher intelligence facilitates more sophisticated thinking about moral matters, a strong correlation

between intelligence and measures of moral reasoning raises issues about the discriminant validity of the latter measures.

The DIT's overlap with verbal ability (e.g. Sanders, Lubinski, Benbow, & Persson, 1995) raises the more immediate concern that poorer verbal facility in disadvantaged racial and ethnic groups may "obscure underlying capability" (Jurkovic, 1980, p. 723). Despite Lynn's central thesis that psychopathy (as indicated by impaired moral reasoning) accounts for group differences in socially deviant behavior above and beyond those attributable to intelligence, the effect sizes he presents on the DIT do not control for verbal ability.

Second, moral reasoning (as assessed by measures like the DIT) cannot be equated with psychopathic personality. Although Lynn notes that delinquents have consistently been shown to possess more impaired moral reasoning than nondelinquents (but see Jurkovic, 1980), juvenile delinquency is not synonymous with psychopathy. Moreover, based on a sample of 60 juvenile delinquents, Chandler and Moran (1990) found that PCL scores were only significantly associated with one of six measures of moral reasoning. Similarly, based on a sample of 44 adolescents, Trevethan and Walker (1989) found that PCL-defined psychopaths, delinquents, and normals did not differ in their responses to Kohlberg's hypothetical moral vignettes, although the groups (delinquent/psychopathic versus normal) could be distinguished by a measure of real-life moral reasoning. Notably, measures like the DIT have been criticized because they inadequately assess individuals' construction of and coping with real-life moral dilemmas (e.g. Locke & Tucker, 1988; Trevethan & Walker, 1989; Wygant, 1997).

In addition to providing little evidence that there are racial and ethnic differences in moral reasoning that signify psychopathy, Lynn provides little evidence that any deficits in moral reasoning are genetically-based. Sociocultural factors are as plausible a basis for differences in moral reasoning as genetic factors. For example, the DIT is significantly associated with political values (Emler, Palmer-Canton, & St James, 1998; Murk & Addleman, 1992; Thoma et al., 1999). Lynn presented data on the DIT for individuals from countries with considerably different norms, values, and cultures. Such sociocultural factors "are assumed from a Piagetian perspective to help determine the rate of children's development of moral structures, the final stage attained, and the content of their value orientation" (Jurkovic, 1980, p. 710, citations omitted). In fact, Moon (1986) reviewed evidence that this rate of development differed across cultures. Similarly, a variety of studies suggest that different cultures do not have equivalent models of and motives for moral and prosocial behavior, with some cultures preferring cooperative behaviors more than others (for reviews, see Carlo, Fabes, Laible, & Kupanoff, 1999; Wygant, 1997). Although some of these cultural differences conceivably could reflect genetic differences, it is highly unlikely that cultural systems are entirely genetically based. Nevertheless, Lynn does not consider cultural factors (of any sort) as potential bases for putative racial and ethnic differences in moral reasoning.

When attempting to predict social deviance based on moral reasoning, one arguably should consider the social learning that takes place within an individual's perceived "functional justice structure" (Jurkovic, 1980, p. 722). If, for example, an individual lives in a social structure where the rules are seen as morally illegitimate, he or she is less likely to support conventional solutions to moral problems (Jurkovic, 1980). If any direct relations are found among race and ethnicity, measures of real-life moral reasoning, and psychopathy, we should ask:

> Is the thinking of [psychopaths] largely preconventional because their moral notions have turned them into [psychopaths]? Or has the preconventional world many of them grew up in caused them to form the only intelligently realistic theories about the way the world works? (Brown & Herrnstein, 1975, p. 325).[3]

### 2.4. Alternative explanations for differences in psychopathy

Because Lynn equates various indices of antisocial behavior with psychopathy, his analysis leaves open the question of the extent to which the small differences in *psychopathy* (as assessed by the PCL measures) among racial and ethnic groups are attributable to genes. Because research on the genetic and environmental determinants of psychopathy is in its infancy (see Skeem, Poythress, Edens, Lilienfeld, & Cale, in press), we cannot fully address this question. Nevertheless, there is evidence of an association between environmental variables and psychopathic traits (Marshall & Cooke, 1995; Weiler & Widom, 1996). More importantly, as is the case with social deviance and moral reasoning, apparent racial and ethnic differences in psychopathy are substantially attenuated once even basic environmental factors are taken into account.

In the MacArthur Violence Risk Assessment Study (Monahan et al., 2001), over 900 civil psychiatric patients were assessed for psychopathy using the PCL:SV and followed in the community for one year. For a subset of 270 patients who participated at one of the study's three sites, Silver (2000) used census tract data to identify each patient's neighborhood and characterize the extent to which it was disadvantaged. He computed single "neighborhood disadvantage" scores by factor analyzing indicators of each neighborhood's rates of public assistance, poverty, unemployment, managerial employment, vacant dwellings, female-headed households, and average household wage. Neighborhood disadvantage scores were strongly correlated with patients' race ($r = 0.63$).

To determine the degree of association between psychopathy and race after controlling for neighborhood disadvantage, we completed analyses based on 253 patients in Silver's sample who had complete PCL:SV data. First, a direct regression analysis was performed in which neighborhood disadvantage was entered before patient race to predict patients' total PCL:SV scores. This analysis indicated that there was a good fit based on the effect of neighborhood disadvantage alone, $R^2 = 0.11$, $F(1, 251) = 31.96$, $P < 0.001$, which increased little after the addition of race, $R^2 = 0.13$, $F(1, 250) = 6.38$, $P < 0.05$. Although the improvement in fit was statistically significant, race added very little to neighborhood disadvantage in predicting psychopathy ($R^2$ change = 0.02). Second, the zero-order correlation between race and PCL:SV total scores ($r = 0.34$) decreased substantially after controlling for neighborhood disadvantage (partial $r = 0.16$). In short, race explained little of the variance in psychopathy once neighborhood disadvantage was taken into account. Because

[3] Sampson and Bartusch (1998) reported data relevant to this issue. Based on a large sample of urban community residents ($N = 8782$), these authors found that Blacks endorsed more legal cynicism (e.g. "laws are made to be broken," "there are no right or wrong ways to make money") and more tolerance for deviance (e.g. it is okay for a teenager to…"smoke cigarettes," "have fist fights") than Whites. However, these relationships were effectively eliminated after statistically controlling for the extent to which residents' neighborhoods were disadvantaged (see later). This suggests that neighborhood context is as plausible a basis for "normative orientations toward law and deviance" as a "racially induced subcultural system" (p. 101).

these analyses are subject to the caveats noted earlier about the risk of "overcontrolling" for race with the use of statistical controls, we are not necessarily arguing that any racial differences in psychopathy are explained away entirely by neighborhood disadvantage. However, these results suggest that the threatening living environments in which Blacks often live are a plausible source of influence on psychopathic traits (see Swartz et al., 1998) that warrant further investigation.

Notably, these results have important implications for key correlates of psychopathy including violence. In the above MacArthur study, psychopathy was the strongest predictor of community violence, compared to a formidable array of over 100 violence risk factors (Monahan et al., 2001). However, the amount of concentrated poverty in patients' neighborhoods significantly predicted violence even after taking into account such factors as psychopathy and race (Silver, Mulvey, & Monahan, 1999). In fact, the effect of race alone in predicting violence disappeared after statistically controlling for neighborhood disadvantage: individuals living in highly disadvantaged neighborhoods were more likely to be violent, whether White or Black (Silver, 2000). In brief, these preliminary analyses suggest that contextual variables that vary across races and are omitted by Lynn may be key risk factors for socially deviant behavior.

In addition to contextual factors, it is possible that the typically small group differences in measures of psychopathy reflect rater bias on the part of examiners (Kosson et al., 1990). As noted by Cooke et al. (2001), White examiners may be prone to rating Blacks as more generally psychopathic than Whites. Although the research on this topic is limited, one unpublished dissertation (Mack, 1999) reported such a finding. Using a case vignette design, White psychologists were asked to make several clinical ratings of an individual portrayed as exhibiting various symptoms of APD. When the examinee was identified as Black, White psychologists rated him as considerably more "psychopathic" ($d = 0.49$) than when he was identified as White. Given that there are no meaningful racial differences in observed base rates of APD (Robins et al., 1991), the magnitude of this effect seems at least partially attributable to clinician bias.

In summary, Lynn's presentation of data to support genetic explanations for apparent differences in psychopathy appears selective and incomplete. Lynn often omits alternative explanations for group differences in criminality and other indices of social deviance. Substantial evidence suggests that group differences in criminality, the MMPI Pd scale, moral reasoning, and even psychopathy per se often are significantly attenuated once such sociodemographic variables as poverty and social bias are taken into account. In fact, taking these factors into account often renders trivial already small racial and ethnic group differences in psychopathic personality disorder.

## 3. Presentation of a speculative evolutionary explanation for purported differences

Lynn (2002) concludes his analysis by offering an evolutionary explanation for his conclusion that there are genetically-based differences among racial and ethnic groups in psychopathy. From the perspective of an evolutionary biologist or behavioral geneticist, there are three primary flaws with Lynn's explanation. First, Lynn (2002) "assumes a genetic basis for race differences in psychopathic personality" (p. 308). This assumption is far from trivial. Superficial physical differences are insufficient for demonstrating that two groups are genetically and evolutionarily divergent. Although there is ample genetic evidence that human populations show differences in a wide range of gene frequencies, no meaningful definition of "race" corresponds to the populations in question.

In a section titled, ''Scientific Failure of the Concept of Human Races,'' Cavalli-Sforza, Menozzi, and Piazza (1994) endorsed the practice of using genetic evidence to cluster humans into populations, but flatly reject the notion that ''race'' is a useful evolutionary category because it does not correspond to any genetically defined population. This is consistent with Boyd's (1950) earlier observation that, as ''data on the physical characteristics of the human race have accumulated... it has gradually become clear that whatever races we choose to distinguish will be almost entirely arbitrary, and their distribution will depend on the particular characteristics on which we choose to base them'' (p. 207). Even if natural selection is acting to distinguish two populations, gene flow between the populations can inhibit their divergence (e.g. Brandon, 1996; McNeilly, 1968). Some societies historically have responded to the mixing of races with social stigma or even criminal penalties, but Lynn does not provide evidence that the races are sufficiently genetically isolated to allow for the genetic divergence that his theory requires. Thus, there is little reason to grant Lynn's assumption that any differences in psychopathic personality are the result of genetic differences between races.

Second, even if Lynn could establish that there has been selection for different levels of psychopathic personality in different races (a very big ''if''), Sober (1984) has demonstrated that this is insufficient to demonstrate selection acting on genes for this trait. Lynn does not provide data that demonstrates the extent to which any association between race and psychopathy is based on genetic factors, cultural transmission, or undocumented environmental factors. Although language is heritable in the sense that the languages spoken by children and their parents are highly correlated, a geneticist would not assume that a Spanish-speaking child inherited genes for speaking Spanish from her parents. To prove a genetic basis for a trait, appropriate controls must be used to rule out environmental and cultural influences on the development of the trait. Moreover, it is desirable to address the specific causal role of genes in the developmental process that gives rise to behavioral differences, even for traits normally assumed to be genetic. For example, the established correlation between the sexual preference of fathers and their offspring in birds is not enough to demonstrate that sexual preference is genetically determined. Vos (1995) demonstrated differences in the acquisition of sex recognition cues among zebra finches (*Taeniopygia guttata*). Vos found that males reared by females whose beaks had been painted to look like those of males were prone to attempt copulation with other males. In fact, for these males, maternal beak color was a better predictor of mate preference than the sex of the stimulus bird. Although this study cannot be taken as conclusive evidence against a genetic basis of homosexuality in zebra finches, it demonstrates that one must provide evidence to support a claim that genes control complex behavioral traits.[4]

Twin and adoption studies may be the best tools available to behavioral geneticists when studying humans; however, it is irresponsible to uncritically assert that simply because identical twins raised separately display similarities, those similarities must be genetic. This is not to imply that no conclusions can be drawn from twin studies; rather, it is a reminder that separate environments are not necessarily different environments and unless we at least consider the range of developmentally relevant variables, we will never know.

[4] One might contend that genes control the underlying processes that allow the mother's beak color to play its part in the development of sex recognition in zebra finches; however, this simply shifts the problem to one of identifying the role of genes in those processes.

The third flaw in Lynn's argument involves his use of an uninformed concept of environment and dichotomization between genetic and environmental factors. Lynn argues that the stable prevalence of psychopathy across races in different geographic locations supports his claim that there is a genetic basis for race differences in this disorder. He contends that it would be difficult for an environmentalist hypothesis to account for the presence of these group differences across such a wide variety of locations. His claim that the presence of a character (psychopathy) in many geographic locations indicates that the trait is genetic has little basis in biology. An evolutionary biologist would not assume that different geographic locations represent different developmental or selective environments. As demonstrated by Brandon (1990), any attempt to reject an environmental explanation for complex behavioral traits must reference more than geographical location. Lynn's argument fails to acknowledge the possibility that racism, poverty, slavery, religious persecution, and other environmental factors may have similar effects regardless of where they occur.

Lynn also appears to assume that there are only two choices for explaining behavior—it is *either* genetically or environmentally induced. This false dichotomization between genetic and environmental factors ignores the non-additive ways that genes interact with environment factors to produce behavior. This dichotomization rests on a relatively simplistic model of evolution that has dominated this type of "nature versus nurture" discussion for the past century (Boyd & Richerson, 1985; Kitcher, 1985, 1996; see also Gabbard, 1992). In short, Lynn's genetic explanation reflects a relatively uninformed concept of the environment and its interaction with genetic factors, does not define "race" as an evolutionarily meaningful concept, contains little evidence that the races are evolutionarily divergent, and presents little evidence that the heritability estimates offered reflect genetic rather than environmental causal factors.

To those unfamiliar with genetics and evolutionary research, biological explanations may be appealing because they appear to move beyond the contingencies of psychological development. However, the appeal may fade once one glimpses the complexity and contingency of biological development. As Elster (1983) observed:

> In the cabinet of horrors of scientific thought the biological excesses of many social scientists around the turn of the century have a prominent place. The situation is less disastrous today, but the biological paradigm retains an importance out of proportion with its merits (p. 404).

As evidenced by the increasing and generally enthusiastic publicity about the Human Genome Project and genetic medicine, the language of genes promises "real" explanations. And in this enthusiasm, social scientists may continue to find refuge for research that can be molded to fit with superficially plausible, but practically baseless, biological explanations.

## Psychopathy, science, and public policy

There is increasing pressure to take psychopathy "out of the ivory tower" and apply the construct in a variety of real world settings (Hare, 2001). In part because of the established association between the PCL-R and various forms of aggression and crime (Gendreau, Goggin, & Smith, in press; Hemphill, Hare, & Wong, 1998; Salekin et al., 1996), the consequences of being

diagnosed 'a psychopath' are becoming more serious. In Canada and the United Kingdom, psychopathy is used to support indeterminate detention of certain classes of offenders (Fitch & Ortega, 2000). In several US states, psychopathy is used to support the determination that individuals are "predatory sex offenders" and therefore subject to civil commitment (Civil Commitment of Sexually Violent Predators Act, 1999). There is evidence that psychopathy increasingly is being used as an aggravating factor in the sentencing phase of US death penalty cases, where it has been argued that the presence of these personality traits renders a defendant a "continuing threat to society" (Cunningham & Reidy, in press; Edens, 2001; Edens, Petrila, & Buffington-Vollum, 2001). In US and Canadian courts, diagnoses of psychopathy also are being used to justify decisions to try and sentence juvenile offenders in the adult criminal justice system, typically based on the presumption that psychopathy is untreatable (Skeem, Cauffman, & Nold, 2002; Zinger & Forth, 1998). Similarly, adult offenders have been denied admission into treatment programs because they have been deemed psychopathic (e.g. *US v. Stitt*, 1998). In short, the legal ramifications of being identified as psychopathic may be considerable, typically to the detriment of those so identified.

Given the increasing legal significance of psychopathy, any differences in this construct related to race or ethnicity would raise complex criminal justice and public policy issues. Some might even contend that any differences would legitimate such controversial practices as racial profiling by the police (e.g. where 'driving while Black' is used as a pretext for criminal investigation) and selective prosecution and disproportionate sentencing of Black individuals in the criminal justice system (see Bortner, Zatz, & Hawkins, 2000; Wrightsman, Greene, Nietzel, & Fortune, 2002). Historically, Blacks have faced considerable stigmatization and prejudice within the legal system, particularly with respect to imposition of the death penalty (Baldus & Woodworth, 1998). Psychopathy and the instruments used to assess it could become powerful vehicles for exacerbating these disparities, particularly if genetically based group differences were identified.

Although science should not be driven by personal or political values (Lilienfeld, 2002), social scientists and their theories do not operate in a vacuum. Theories that rest on little support, whether politically incendiary or not, should be promulgated with some degree of temperance. Lynn (2002) makes such claims as "[w]e can *conclude* that differences in marriage rates are almost entirely a function of differences in psychopathic personality" (p. 306, emphasis added) and

> these residual differences [in rates of low birth weight babies] are *likely* to be due to some degree, to Black and Hispanic women behaving less responsibly during pregnancy by poor nutrition, alcohol and drug abuse, and can be interpreted as secondary effects of group differences in psychopathic personality (p. 307, emphasis added).

These claims are not supported by evidence or solid reasoning. Although we do not assert that Lynn's *ideas* must be untrue because they are politically incendiary, using such words as 'conclude' in the absence of credible data to support one's claims is inappropriate. Researchers are responsible for critically evaluating their work and carefully considering alternative explanations for their findings. Speculation derived from theory should be presented as tentative hypotheses, not as scientific fact.

Our argument against Lynn's (2002) conclusion that racial differences in social deviance are attributable to group differences in psychopathy is driven more by concern about the scientific

merit of this work than by its political implications. At present, the theory proposed by Lynn does not meet the minimal standards that one would apply to judge the scientific merit of any research, regardless of the nature of the topic under consideration. We have argued that Lynn equates psychopathy with antisocial and deviant behavior, rendering some of his claims tautological, and often neglects viable potential environmental explanations including poverty and racial discrimination for purported differences in antisocial and deviant behavior. The product of these processes is an assertion that there are meaningful, genetically based racial differences in psychopathy. Lynn provides an evolutionary explanation for these differences that does not meet methodological and interpretive standards in behavioral genetics and evolutionary biology. Although research on topics relevant to Lynn's theory is in its infancy, extant data suggest that racial differences in psychopathy are not large enough to have theoretical or practical significance. Thus, Lynn's conjecture about their genetic and evolutionary etiology is premature. If researchers choose to continue investigation of the issues raised by Lynn (compare Hunt, 1999; Kitcher, 2001), they will need to address the considerable scientific limitations of his model in its present form.

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