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THE ASSOCIATION BETWEEN ANTISOCIAL PERSONALITY AND SOMATIZATION DISORDERS: A REVIEW AND INTEGRATION OF THEORETICAL MODELS

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ABSTRACT. *Evidence from several sources is consistent with the possibility that antisocial personality disorder (ASPD) and somatization disorder (SD) are sex-differentiated manifestations of the same underlying predisposition. Following a discussion of diagnostic issues and a brief review of data on the relation between ASPD and SD, four models of the association between the two syndromes are reviewed: the frontal lobe model, the efferent inhibition model, the behavioral disinhibition model, and the negative emotionality model. Behavioral disinhibition may lead to recurrent antisocial and risk-taking behaviors in some individuals, resulting in elevated negative emotionality and a propensity to develop somatic symptoms. Factors that might be responsible for channeling the diathesis to ASPD and SD into sex-differentiated alternative pathways are discussed.*

The etiology of antisocial personality disorder (ASPD) poses one of the foremost challenges to psychopathology researchers today (Lewis, 1974). Although it is known that ASPD is influenced by genetic factors (Schulsinger, 1972), the processes predisposing to its development largely remain a mystery. Equally puzzling is the finding that ASPD patients and their relatives are prone to a variety of psychiatric conditions (Guze, 1976).

Wender and Klein (1981), among others, have argued that ASPD and three other syndromes—somatization disorder (SD), attention-deficit hyperactivity disorder (ADHD), and alcohol dependence (alcoholism)—cluster within families more often than expected by chance. Wender and Klein have dubbed this group of illnesses "The Unnamed Quartet" to highlight its relative obscurity in the corpus of descriptive psychopathology, and have argued that these conditions may be manifestations of the same disease process. In this article I review the current status of theoretical models of the association between ASPD and SD, which is the link in Wender and Klein's quartet that has been the most extensively researched.[1]

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[1]Evidence bearing on the association among attention-deficit hyperactivity disorder and antisocial personality disorder and somatization disorder will be briefly reviewed later in the manuscript.

Although ASPD is primarily a disorder of males, and SD of females (American Psychiatric Association [APA], 1987), these two syndromes share a number of features. For example, both predominate among the lower social classes, begin early in life, typically run a chronic course, tend to be refractory to psycho- and pharmacotherapy, and are associated with marital discord, substance abuse, suicide attempts, and other complications (Cloninger, 1978b; Goodwin & Guze, 1985). These observations, as well as the literature to be reviewed here, have led to the conjecture that ASPD and SD are sex-differentiated expressions of the same predisposition (Cloninger, 1978b; Widom, 1984; Winstead, 1984).

I begin with a discussion of diagnostic issues relevant to ASPD and SD, and briefly review the evidence bearing on the relation between these syndromes. I then examine four models of etiology linking the two conditions, and end by reviewing factors potentially responsible for shaping their phenotypic expression.[2]

DIAGNOSTIC ISSUES

Antisocial Personality Disorder

The diagnosis of ASPD is a source of considerable controversy (Hare & Cox, 1978). Much of this controversy centers around the relative merits of using behavioral indicators versus personality attributes in the diagnosis of the syndrome. Specifically, two diagnostic approaches can be distinguished: "behavior-based" and "personality-based" (Lilienfeld, 1991a). The *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed. rev.; DSM-III-R) (APA, 1987), which primarily employs the former approach, defines ASPD as a condition characterized by an early (prior to age 15) onset of repeated delinquent, irresponsible, or criminal behaviors that persist into adulthood. The major criteria for ASPD include theft, vandalism, truancy, persistent lying, inconsistent work behavior, financial irresponsibility, recklessness, and physical aggressiveness. A major advantage of the behavior-based approach is its high interrater reliability (Cloninger, 1978a; Widiger & Frances, 1987).

By contrast, advocates of the personality-based approach contend that the syndrome is best operationalized in terms of a set of covarying personality traits. Cleckley (1976), for example, delineated 16 features of the "psychopathic personality," including superficial charm, lack of anxiety, dishonesty, guiltlessness, failure to learn from punishment, egocentricity, absence of deep emotions, and lack of forethought. Advocates of this approach generally contend that the behavior-based approach has sacrificed validity for the sake of reliability (Lykken, 1984). Specifically, they argue that only a subset of individuals with ASPD, so-called "primary" psychopaths (also called "psychopathic personalities" or "psychopaths") possess the traits outlined by Cleckley, and that the residual group of "secondary" psychopaths comprises several conditions with varied etiologies (e.g., neurotic conflict, allegiance to a culturally deviant subgroup). In addition, advocates of this approach

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Although the association between alcoholism and the other disorders in the quartet requires further investigation, adoption studies suggest that alcoholism and ASPD are largely if not entirely genetically independent (Cloninger, 1978).

[2]For the remainder of the manuscript, I will use the term *antisocial personality disorder* (ASPD) in all cases in which the disorder was defined on the basis of a chronic history of antisocial behaviors. I will use the term *psychopathy* or *psychopathic personality* in all cases in which the disorder was defined primarily on the basis of personality traits. Similarly, I will use the term *somatization disorder* (SD) in all cases in which the disorder was defined on the basis of a chronic history of multiple unexplained somatic symptoms.

contend that many psychopaths escape detection when behavioral criteria are employed (Widom, 1977); these "subclinical" psychopaths have been the subject of much conjecture, but little research. Thus, the behavior-based approach may be overinclusive in certain ways, and yet underinclusive in others (Lilienfeld, 1991a).

Somatization Disorder

Like ASPD, the diagnosis of SD is based primarily upon clearly defined, easily-agreed-upon criteria. Formerly known as hysteria or Briquet's syndrome, SD is a syndrome characterized by multiple and recurrent physical symptoms (e.g., pain, urinary problems, conversion symptoms) lacking a demonstrated organic basis. SD patients often have an extensive history of "doctor-shopping" as evidenced by multiple hospitalizations and unnecessary operations (Goodwin & Guze, 1984).

Like ASPD, SD is believed by many authors (e.g., Chodoff & Lyons, 1958) to be typically characterized by an underlying constellation of personality attributes. This latter syndrome, known as hysterical personality or histrionic personality disorder (APA, 1987), comprises such features as vanity, self-dramatization, attention-seeking, emotional lability, shallow affect, dependent behavior, and sexual provocativeness. Although the relation between histrionic personality disorder and SD has received little research attention, preliminary evidence suggests that these conditions covary substantially (Kimble, Williams, & Agras, 1975; Lilienfeld, VanValkenburg, Larntz, & Akiskal, 1986).

EVIDENCE SUGGESTING AN ASSOCIATION BETWEEN THE TWO DISORDERS

The evidence suggesting an association between ASPD and SD has been reviewed elsewhere (e.g., Cloninger, 1978b; Widom, 1984; Winstead, 1984), and thus will be only briefly recapitulated here. This evidence derives from three major sources: studies of intra-individual association, family studies, and adoption studies.

Studies of Intra-Individual Association

Informal clinical observations (e.g., Blacker & Tupin, 1977; Carothers, 1975; Chodoff, 1982; Halleck, 1967), as well as uncontrolled studies and case reports (e.g., Cloninger & Guze, 1970a, 1970b; Forrest, 1967; Guze, Woodruff, & Clayton, 1971b; Liskow, Othmer, Penick, DeSouza, & Gabrielli, 1986; Moravesik, 1894) suggest that ASPD and SD (as well as conversion disorder) overlap substantially within individuals. Moreover, the results of several studies using both psychiatric and normal comparison groups (Guze, Woodruff, & Clayton, 1971a; Lewis, Rice, & Helzer, 1983; Lilienfeld et al., 1986; Liskow, Penick, Powell, Haefele, & Campbell, 1986; Robins, Purtell, & Cohen, 1952; Spalt, 1980) indicate that ASPD and SD covary significantly within both males and females. In addition, Robins (1966) found that adolescent females with conduct disorder, which is often a precursor of ASPD (APA, 1987), had a much higher rate of SD in adulthood relative to a normal comparison group. Thus, there is some suggestion that ASPD and SD may be associated not only cross-sectionally, but longitudinally (also see Maddocks, 1970).

Family Studies

The results of several uncontrolled studies suggest high rates of ASPD among the first-degree relatives of SD patients (Arkonac & Guze, 1963; Ljungberg, 1957; Woerner & Guze, 1968), and high rates of SD among the first-degree relatives of criminals (Guze,

Goodwin, & Crane, 1969; Guze, Wolfgram, McKinney, & Cantwell, 1967), although it is not known how many of these criminals had ASPD. Cloninger and Guze (1975) reported an elevated rate of SD among the daughters of felons with ASPD (78%) compared with the daughters of felons without ASPD (26%), and Lilienfeld et al. (1986) reported an elevated rate of ASPD among the first-degree relatives of SD patients compared with the first-degree relatives of other psychiatric patients. In addition, Guze et al. (1971a) reported that SD patients, compared with anxiety neurotics, had a significantly higher percentage of first-degree relatives with antisocial behavior; nevertheless, they did not assess family history of ASPD per se.

Cloninger, Reich, and Guze (1975) have posited a multifactorial threshold model in which female SD, male ASPD, and female ASPD represent increasingly severe manifestations of a shared diathesis (male SD, the rarest and presumably most severe phenotype, was excluded from the model because of an absence of family data). A chi-square goodness-of-fit test revealed good agreement with the values predicted by this model: Female SD, male ASPD, and female ASPD were found in decreasing order of prevalence in the population, and were associated with increasing risk for all three syndromes among first-degree relatives. In addition, positive assortative mating between ASPD fathers and SD mothers (see Guze et al., 1970; Woerner & Guze, 1968) contributed slightly to the familial association between the conditions, but did not substantially worsen the fit of the model. It should be noted, however, that the authors utilized a maximization procedure in which parameter values were iterated until the point of best fit was ascertained; thus, their results require replication.

An additional line of evidence for the ASPD–SD association derives from family studies of children with ADHD. Several research teams comparing ADHD children with normal (Cantwell, 1972; Morrison & Stewart, 1971) or psychiatrically disturbed (Morrison, 1980) children have reported an elevated prevalence of ASPD in the fathers, and of SD in the mothers, of the former group. Nevertheless, more recent evidence (Lilienfeld & Waldman, 1990) suggests that the familial association among ADHD, ASPD, and SD is largely mediated by conduct disorder, which covaries substantially with ADHD (Hinshaw, 1987). For example, August and Stewart, (1983) and Lahey et al. (1988) found that ADHD with conduct disorder confers a high familial risk of ASPD and SD, whereas ADHD alone does not. Because a large percentage of conduct-disordered children develop ASPD, these findings provide further support for a familial association between ASPD and SD.

Adoption Studies

Crowe (1974) reported no cases of either SD or hysterical personality among the adopted-away offspring of 46 female felons; nevertheless, he did not assess whether these felons met criteria for ASPD. Cadoret, Cunningham, Loftus, and Edwards (1976) found that the female adoptees of biological parents with ASPD had a significantly higher rate of unexplained somatic symptoms compared with the female adoptees of both normal and psychiatrically disturbed parents; male adoptees exhibited a nonsignificant trend in this direction. In a separate sample, Cadoret (1978) reported an elevated rate of psychosomatic symptoms (similar to those found in SD patients) among the female adoptees of biological parents with ASPD or antisocial behavior compared with the female adoptees of biological parents without ASPD or antisocial behavior. In neither study, however, were adoptees assessed for SD per se.

Bohman, Cloninger, von Knorring, and Sigvardsson (1984) reported that the biological fathers of female adoptees who were "somatizers" (i.e., who had repeatedly missed

work days due to illness) had a substantially higher rate of criminality compared with the biological fathers of nonsomatizers. Nevertheless, it is not known how many of the adoptees met criteria for SD, or how many of the fathers met criteria for ASPD.

Finally, Morrison and Stewart (1973) and Cantwell (1975) compared the rates of ASPD and SD in the adoptive parents of ADHD children with rates of these conditions in the biological parents of these children. In both studies, prevalence of ASPD and SD was significantly higher among biological parents, prompting the investigators to suggest that ADHD, ASPD, and SD are genetically associated. Nevertheless, in neither study were interviewers blind to the parents' adoptive versus nonadoptive status: moreover, as adoptive parents are typically screened for severe psychopathology (Lewontin, Rose, & Kamin, 1984), these studies do not provide a strong test of environmental influences.

Summary and Implications

Evidence indicates that ASPD and SD are associated within both individuals and families more often than expected by chance. Family studies of ADHD children also are consistent with a familial association between ASPD and SD. In addition, results of adoption studies raise the possibility that the link between ASPD and SD is influenced by genetic factors, although absence of diagnostic criteria in most of these studies precludes any definitive conclusions.

It should be noted that all of the investigations reviewed thus far have employed behavior-based, rather than personality-based, criteria for ASPD. As discussed earlier, behavior-based criteria may result in considerable heterogeneity at the etiological level. Thus, it is conceivable that secondary psychopathy rather than primary psychopathy is associated with SD. Because all of the current criteria sets for ASPD are primarily behavior-based, however, this issue cannot be resolved at present.

In view of the adoption data, it seems reasonable to treat the notion of a genetic relation between the two syndromes as a working hypothesis for heuristic purposes. If ASPD and SD share a common genetic etiology, this putative genotypic dysfunction might be manifested at some intermediate point in the gene-behavior pathway. This in turn might allow researchers to uncover neuropsychological, psychophysiological, or neurophysiological markers shared by both conditions.

PREVIOUS RESEARCH ON MODELS OF SHARED ETIOLOGY

Some of the psychophysiological evidence linking ASPD to SD has been reviewed by Cloninger (1978b). As he noted, these data do not readily conform to models positing a common liability to these conditions. Whereas most investigators have found psychopaths to exhibit low resting skin conductance levels (SCLs), low autonomic variability, few spontaneous skin conductance fluctuations, and normal or rapid habituation to repetitive sounds, most researchers have found the *opposite* pattern among SD patients. Although many of these studies are based upon patients with isolated conversion symptoms, the balance of the evidence suggests that, in contrast to psychopaths (Hare, 1978), SD patients tend to be autonomically overaroused.

Cloninger has attempted to resolve this discrepancy by pointing out that most of the hysterics upon which this conclusion is based were hospitalized shortly prior to testing for agitated depression or conversion reactions, and had recently undergone increases in anxiety. In contrast, most of the psychopaths tested in these studies had been imprisoned for many years and may have been in states of relative calm. Thus, the measures employed

in these studies may be too state-dependent to serve as adequate biological markers of the diathesis to ASPD and SD. If so, researchers will need to identify markers that are temporally stable and present during symptom remission (Iacono, 1983).

There have been few attempts to synthesize the literature on etiological factors in ASPD and SD into a coherent theoretical model. I will review the evidence for four such efforts—the frontal lobe model, the efferent inhibition model, the behavioral disinhibition model, and the negative emotionality model— and conclude with a discussion of factors potentially responsible for channeling the diathesis to ASPD and SD into sex-differentiated alternative pathways. Cloninger's (1978b) chapter represents the only major previous attempt to explain the association between ASPD and SD in terms of an interaction between a biological predisposition and gender-related modifying factors. Cloninger's review, however, was written before much of the research reviewed here was carried out; consequently, it contains little discussion of the four models presented below. Other reviews of the ASPD–SD link (e.g., Widom, 1984; Winstead, 1984) have generally mentioned theoretical models of this association only briefly.

It should be pointed out that, in contrast to most of the literature reviewed thus far, most of the remaining studies employ personality-based criteria for psychopathy. Consequently, their relevance to studies of ASPD must remain somewhat speculative. Nevertheless, as a substantial proportion of prisoners with ASPD have been found to score highly on measures of personality-based criteria (Hare, 1983), cautious extrapolation from these studies to those on ASPD may be justified.

THE FRONTAL LOBE MODEL

One promising line of evidence relevant to the ASPD–SD link is the performance of patients with these conditions on tests of frontal lobe functioning. Lezak (1976) described four symptom clusters resulting from prefrontal damage: (a) perseveration—failure to curtail activities after they have ceased to be adaptive; (b) deficient self-awareness—inability to appreciate one's impact upon others, tendency toward self-satisfaction, low anxiety, impulsivity, and reduced concern for cultural conventions; (c) concrete attitude—inability to plan or sustain goal-directed behavior; and (d) slowing—apathy, indifference, and inability to sustain attention. With the possible exception of the fourth category, these clusters comprise features strongly reminiscent of primary psychopathy (Gorenstein, 1982). This has led some authors to suggest that frontal lobe dysfunction may be a useful model for psychopathy and related syndromes (e.g., Gorenstein, 1982). It is perhaps noteworthy that criminal behavior and unexplained somatic symptoms, among other psychiatric symptoms, have been found to result more frequently from damage to the frontal lobes than from damage to other lobes (Lishman, 1968).

Moreover, Blumer and Benson (1975) have argued that lesions of the orbital surface of the prefrontal cortex produce a "pseudopsychopathic" syndrome marked by impulsivity, self-centeredness, and insensitivity to others. This suggests that ASPD and similar syndromes may be characterized by abnormalities in the orbital-frontal cortex, a possibility to which I shall return.

Gorenstein (1982) found that, compared with other psychiatric patients and normal college students, psychopaths differed significantly on three tests previously found to discriminate patients with prefrontal lesions from patients with other brain lesions—the Wisconsin Card-Sorting Task (WCST; psychopaths committed more perseverative errors), Sequential Matching Memory Task (SMMT), and a measure of Necker Cube reversals (psychopaths reported more reversals). Moreover, performance of psychopaths

on these tests was comparable with that of frontal lobe patients. Hare (1984), however, failed to replicate these findings, despite using the same tests as Gorenstein and selecting a more extreme group of psychopaths. Hare's results may have been due to low power (there were only 14, 16, and 16 subjects in his high, medium, and low psychopathy groups, respectively), as most trends were in the same direction as Gorenstein's. Alternatively, Gorenstein's results may have been artifact of greater substance use among psychopaths relative to normals; Hare found that severity of alcohol use was positively correlated with errors on the SMMT and perseverative errors on the WCST.

Sutker, Moan, and Allain (1983) also failed to find differences between psychopaths and other prisoners on the WCST. Nevertheless, they identified psychopaths solely on the basis of elevated scores on the MMPI Psychopathic Deviate scale, which yields a very heterogeneous group of conditions (Lachar, 1974). The MMPI, unlike the Cleckley criteria, does not appear to adequately distinguish primary from secondary psychopaths (Lykken, 1957). In addition, two recent groups of investigators (Hoffman, Hall, & Bartsch, 1987; Sutker & Allain, 1987) have failed to replicate Gorenstein's findings, casting doubt upon the association between psychopathy and frontal lobe dysfunction.

Performance on the Porteus Mazes Test is highly sensitive to prefrontal lobotomy (Porteus, 1965). Milner (1965), for example, found that patients with frontal lesions made significantly more qualitative errors than did patients with other lesions on a visually guided maze like that of Porteus. Some investigators have reported similar findings with psychopaths. Schalling and Rosen (1968), for example, found that criminals with high ratings on a scale of Cleckley psychopathy committed more qualitative errors (e.g., crossed lines, changed directions) than did other criminals. Similarly, Sutker and Allain (1987) reported that nonincarcerated psychopaths, defined on the basis of the MMPI, had higher (i.e., poorer) qualitative scores (Q scores) than did nonpsychopaths. In contrast, Sutker, Moan, and Swanson (1972) found that MMPI-defined psychopaths had significantly lower Q scores than did either antisocial psychotics or prisoners with no significant scale elevations. The results of these studies by Sutker and colleagues are difficult to interpret, however, in light of their subject selection criteria (see above).

These studies on psychopaths are of interest in light of an evaluation of 10 SD patients on a battery of neuropsychological tests (Flor-Henry, Fromm-Auch, Tapper, & Schopflocher, 1981). These researchers found that SD patients exhibited significantly greater impairment than psychotic depressives, schizophrenics, and normals (all four groups were matched on IQ) on several tests of frontal lobe functioning, including the Porteus Mazes Test, WCST, Category Test, and Trail-Making Test. The findings are particularly impressive given the small sample sizes, and point to the possibility of frontal lobe dysfunction among SD patients.

Foulds (1951) reported that, compared with dysthymics, both "hysterics" and "psychopaths" made more qualitative errors, particularly pencil lifts and crossed lines, on the Porteus Mazes Test. Nevertheless, he reported neither total Q scores nor diagnostic criteria. Similarly, Minski and Desai (1955) found that "hysterics" committed significantly more qualitative errors than did a group of ulcer patients matched for age, IQ, and socioeconomic status. Again, however, no diagnostic criteria were reported.

Thus, although the results of several studies suggest that psychopaths and SD patients perform poorly on tests of frontal lobe functioning, a number of replication failures and the questionable (or absent) diagnostic criteria in many of the studies preclude clear-cut conclusions. Two additional caveats should be mentioned. First, many of the measures of frontal lobe dysfunction employed in these investigations are of questionable validity. The WCST and Necker Cube reversals, for instance, have not consistently differentiated

patients with frontal lesions from other organic patients (Hare, 1984). In addition, Porteus Q scores, unlike the Porteus test quotient, are not consistently affected by frontal lobe surgery (Kerr, 1979). A second point, frequently reiterated but also frequently forgotten, is that poor performance on neuropsychological tests is not equivalent to neuropsychological dysfunction. Performance on these tasks is multiply determined, and may reflect personality variables (e.g., impulsivity, apathy) unrelated to structural brain damage.

These reservations notwithstanding, there remains one aspect of frontal lobe dysfunction that merits consideration vis-à-vis ASPD and SD. Specifically, prefrontal lobotomies have been found to relieve intractable pain (Barber, 1959; Valenstein, 1973). Following the operation, there is apparently a dissociation between the perception of, and the subjective response to, pain. This distinction between the sensory and reactive components of pain has received substantial empirical support (Tursky, 1974). Most postlobotomy patients report that although they still feel pain, pain no longer bothers them (Barber, 1959). Consistent with this observation is the finding that prefrontal lobotomy generally results in increased pain tolerance (Hall & Stride, 1954).

It may therefore be relevant that, when given sufficient incentives, Cleckley psychopaths show higher pain tolerance than do nonpsychopaths (Hare & Thorvaldsen, 1970). Moreover, psychopaths do not differ from nonpsychopaths in their ratings of the painfulness of shocks (Schacter & Latane, 1964), suggesting that this effect is not due to decreased pain sensitivity. It may be significant that Janet (1929) and other authors had observed that many hysterics display "*la belle indifference*," a lack of concern regarding disabling and often painful symptoms. The classic MMPI pattern of patients with conversion reactions is the "conversion valley," a configuration in which the Depression scale is markedly reduced relative to the Hypochondriasis and Hysteria scales (Hanvik, 1951). This pattern is commonly associated with low scores on the Psychasthenia scale, suggesting low manifest anxiety (Dahlstrom & Welsh, 1960). Such findings raise the intriguing possibility that the higher pain tolerance of psychopaths and the belle indifference of hysterics are mediated by the same mechanisms as the pain tolerance of patients with frontal lobe damage. These phenomena may even bear some relation to the "hidden observer" effect sometimes observed in highly susceptible subjects during hypnosis (Nogrady, McConkey, Laurence, & Perry, 1983), in which the sensory qualities of pain are apparently registered outside of awareness. As hysterics score highly on indices of suggestibility (Bendefeldt, Miller, & Ludwig, 1976), this possibility seems worth pursuing.

Summary

The evidence that psychopaths perform poorly on neuropsychological tasks of frontal lobe dysfunction is inconsistent at best, while the evidence that SD patients perform poorly on these tasks must be qualified by the absence of standard diagnostic criteria in several studies. Circumstantial evidence points to the possibility that psychopaths (and perhaps SD patients) have elevated tolerance for pain, a characteristic common among patients with frontal lobe dysfunction.

THE EFFERENT INHIBITION MODEL

One explanation for the increased pain tolerance of psychopaths and the belle indifference of many SD patients is that both groups have an increased capacity to ignore or tune out aversive stimulation. For example, Yochelson and Samenow (1976) referred to the "cutoff" process by which some criminals screen out both internal (e.g., fear) and external deter-

rents of antisocial behavior. Some provocative, albeit circumstantial, evidence that psychopaths may be able to reduce their subjective reactions to noxious stimuli derives from the work of Lykken (1968).

Lykken has proposed that fearless individuals, including psychopaths, may be adept at "negative preception,"[3] a centrally mediated efferent tuning mechanism that attenuates the psychological impact of painful and other noxious input. Lykken and Tellegen (1974) postulate that negative preception involves an effortful, phasic inhibition of the ascending reticular activating system (ARAS) via cortical efferents, and is most effective when stimuli are temporally predictable. One of the few investigations linking negative preception to psychopathic traits was conducted by Jordan (1975), who found that Psychoticism and Aggression scores (taken from an early version of Tellegen's [1982] Multidimensional Personality Questionnaire, or MPQ) were among the best predictors of negative preception capacity (operationalized as the difference in phasic autonomic responsivity between predictable and unpredictable shock). Psychoticism appears to relate to a number of psychopathic personality traits (Hirschfeld, 1978), while aggression is a cardinal feature of the antisocial personality (APA, 1987).

Hare (1978) has reported that primary psychopaths, in contrast to nonpsychopaths, experience marked heart rate acceleration prior to an anticipated electric shock or loud tone. Heart rate acceleration has been interpreted by some authors (e.g., Lacey, 1967) as reflecting a cortically initiated defensive reflex (Sokolov, 1963) resulting in sensory rejection (i.e., decreased attention to the environment). Thus, psychopaths may be able to adaptively tune out noxious stimulation and thereby attenuate its subjective impact. Other authors (e.g., Obrist, 1976) have contended that premonitory heart increases represent a sympathetically mediated attempt at "active coping" (i.e., active avoidance). This may mean that psychopaths are prone to utilize adaptive coping mechanisms in anticipation of aversive input.

In a reanalysis of Valins' (1967) data, Lykken (1967) found that, whereas the heart rates of high scorers on a self-report index of fearfulness, the Activity Preference Questionnaire (APQ; Lykken, Tellegen, & Katzenmeyer, 1973), decelerated prior to stimuli of all intensities, the heart rates of low APQ scorers decelerated prior to weak stimuli but increasingly accelerated prior to increasingly noxious stimuli. This implies that less fearful subjects respond more adaptively to anticipated stressors, in that they better discriminate among stimuli with varying threat values. Such enhanced discrimination may allow these individuals to buffer themselves against potentially disruptive stimuli in proportion to their expected aversiveness. Lykken, Macindoe, and Tellegen (1972) similarly reported that low APQ scorers exhibited larger heart-rate accelerations prior to electric shocks than did high scorers. Moreover, less fearful subjects showed smaller heart-rate accelerations following shock, suggesting effective diminution of stimulus impact. Nevertheless, the relation between the APQ and primary psychopathy is unclear (Hare & Cox, 1978); moreover, Jordan (1974) found that the APQ did not relate to negative preception scores. Thus, the relation between psychopathy and negative preception is unclear.

As noted earlier, primary psychopaths have been reported to possess higher pain tolerance compared with nonpsychopaths when given sufficient incentives. Although there are several explanations for increased tolerance (e.g., psychopaths may be more motivated to demonstrate that they can withstand strong shocks), one plausible explanation is that

[3]Lykken (1968) also posited a "positive preception" mechanism whereby the impact of pleasurable stimuli is augmented. Little research has been conducted on this phenomenon, however.

psychopaths have efficient mechanisms for inhibiting their emotional reaction to pain and are thus better able to endure high pain intensities.

There is also evidence that some of the features of SD may be due to cortifugal inhibition (Ludwig, 1972). Whitlock (1967) has theorized that la belle indifference is a form of selective inattention to discomfort resulting from cortical inhibition of afferent input to the ARAS. The similarity between Whitlock's hypothesis and Lykken's concept of negative preception is intriguing. Hernandez-Peon, Chavez-Ibarra, and Agular-Figueroa (1963) stimulated both the affected and normal legs of a patient with hysterical hemianesthesia. They found that the somatosensory-evoked potentials following stimulation of the anesthetic side were smaller than those elicited from the normal side. Although this result has not been consistently replicated (Ludwig, 1972), it points to the possibility that cortifugal inhibition is involved in the genesis of at least some hysterical symptoms.

The notion of efferent inhibition as underlying ASPD and SD is not new. Eysenck (1955) initially argued that both primary psychopaths and hysterics are "extraverted neurotics." According to Eysenck, extraverts, and extraverted neurotics in particular, rapidly develop cortical inhibition, presumably over the ARAS, and dissipate it slowly, compared with introverts. In a series of studies, Eysenck (e.g., 1955) and others found that, relative to introverts, extraverts show larger and more persistent kinesthetic aftereffects (Petrie, 1967) and greater reminiscence in motor learning, suggesting a larger accumulation of inhibition during original learning. Nevertheless, this literature has been plagued by numerous replication failures (e.g., Meier, 1960), and questions regarding the reliability and validity of these measures have not been satisfactorily resolved (Zuckerman, 1978).

Psychopaths and criminals have longer half-recovery times of the skin conductance response (SCR) than other individuals (Hare, 1978). Venables (1975) has argued that the length of the SCR recovery limb is a marker of a dimension of "openness–closedness" to the environment. According to Venables, the long recovery times of psychopaths indicate a defensive "gating out" of external stimuli analogous to the sensory rejection posited by Lacey (1967) to result from cardiac acceleration. As Bundy and Fitzgerald (1975) have demonstrated, however, long SCR recovery times are directly related to the amount of preceding electrodermal activity. As psychopaths produce fewer spontaneous skin conductance fluctuations than other subjects (Hare, 1978), it is uncertain whether the association between psychopathy and SCR recovery time per se is of psychological significance.

Summary

Although the evidence for the efferent inhibition hypothesis is equivocal, this hypothesis appears to furnish a parsimonious explanation for many of the features of ASPD and SD. Several circumstantial lines of evidence seem compatible with the possibility that patients with ASPD, and perhaps those with SD, possess efficient mechanisms for lessening the impact of noxious stimuli. Nevertheless, further research on the relation of these hypothesized inhibitory processes, such as negative preception, to ASPD and SD is necessary.

THE BEHAVIORAL DISINHIBITION MODEL

Gray (1982) has formulated a neurophysiological model of personality that comprises three underlying dimensions. The behavioral inhibition system (BIS), consisting of the

medial septal area, hippocampus, and orbital-frontal cortex (SHF), is the putative substrate of anxiety. The behavioral activation system (BAS), which Gray views as isomorphic with Olds' mesolimbic reward system, facilitates behavior in response to classically conditioned signals of reward. The BIS inhibits the BAS in response to classically conditioned signals of punishment and uncertainty. In addition, in these situations it triggers increases in cortical arousal via the fight–flight system (see below) and causes heightened attention to the environment. The fight–flight system mediates escape and aggressive behavior in response to unconditioned punishment, and increases cortical arousal via inputs to the ARAS.

Gorenstein and Newman (1980) contended that a broad spectrum of "disinhibitory syndromes," including ASPD, SD, ADHD, and substance abuse, may be due to weakened inhibitory control of the BIS over the BAS, resulting from SHF dysfunction. Their hypothesis unifies a variety of disparate findings concerning psychopaths and hysterics, including two already discussed—the scattered (although inconsistent) reports of their poor performance on frontal lobe tasks and their similarities in some respects to patients with orbital-frontal lesions (Blumer & Benson, 1975). It also may explain the low levels of arousal observed in psychopaths and perhaps hysterics, and is consistent with the conjecture that these patients exhibit decreased attention to the environment in response to aversive stimuli. Cloninger (1987) has similarly argued that antisocial and histrionic personalities are characterized by low harm avoidance, a personality dimension that may result from individual differences in BIS strength.

Gorenstein and Newman drew a number of parallels between the performance of septal-lesioned rats and psychopaths on laboratory tasks, such as poor passive avoidance learning, slow acquisition of classically conditioned fear responses, and failure to anticipate temporally remote aversive events. Newman, Gorenstein, and Kelsey (1983) showed that, compared with controls, rats with septal lesions are less likely to delay gratification when presented with a choice between an immediate but only periodically delivered reward and a delayed but assured reward. Psychopaths seem to show a similar preference (Unikel & Blanchard, 1973).

Newman and his associates have extended their model to humans with putative SHF dysfunction, including psychopaths. Newman, Widom, and Nathan (1985) found that primary psychopaths committed more passive avoidance errors than did nonpsychopaths on a task involving monetary rewards and punishments, but performed as well as nonpsychopaths under conditions of punishment alone. Newman, Patterson, and Kosson (1987) reported that, in the presence of prominent cues of monetary reward during a card-playing task, psychopaths, relative to nonpsychopaths, perseverated (i.e., continued to play cards) despite steadily increasing monetary loss. Results of these studies suggest that, like septal rats, psychopaths are relatively insensitive to signals of punishment, particularly when reward cues are salient.

Other features of ASPD and SD are potentially interpretable within the context of the SHF model. Primary psychopaths give diminished SCRs to tones presaging shock (Hare, 1965; Lykken, 1957), which is consistent with the hypothesis of an underresponsive BIS. Moreover, psychopaths show normal SCRs to unwarned aversive stimuli (Hare, 1978), indicating that this effect is not due to generalized electrodermal hyporesponsivity. Fowles (1980) has argued that electrodermal activity in response to classically conditioned signals is a marker of BIS strength. It may thus be relevant that Franks (1956) reported that, relative to dysthymics and normals, hysterics exhibited slower SCR conditioning to tones signaling delivery of air puffs to the right eye (although only the difference from

dysthymics reached significance). Because eye-blink conditioning is aversive to almost all subjects (Gray, 1970), this finding may mean that, like psychopaths, hysterics are underresponsive to punishment cues.

Moreover, many of the clinical features of SD can arguably be conceptualized as resulting from an underactive BIS. For example, the perpetual doctor-shopping (Goodwin & Guze, 1984) and repetitive somatic symptoms of SD patients can perhaps be viewed as perseverative behaviors that persist in spite of long-term maladaptive consequences. The development of physical symptoms following stress may be an expression of a tendency to forego long-term gains for short-term gratification (in this case, immediate anxiety reduction). Moreover, the histrionic personality's craving for excitement, seductiveness, and proclivity toward angry outbursts (APA, 1987) might be interpretable in terms of diminished inhibitory control over BAS-mediated behaviors, which include novelty seeking, sexuality, and irritative aggression, respectively (Depue & Spoont, 1987).

Nevertheless, the behavioral disinhibition model appears inconsistent with the efferent inhibition model, because the former posits *deficient* inhibition over brain centers mediating goal-oriented behavior. A possible resolution of this paradox hinges on the constituent of the SHF I have heretofore neglected: the hippocampus. Several authors have proposed that the principal function of the hippocampus is the tuning out of irrelevant or redundant sensory input (Douglas & Pribram, 1966). Thus, hippocampal lesions produce a failure to ignore unimportant stimuli, leading to overattention to the environment (Crider & Solomon, 1982). Interestingly, hippocampectomized monkeys show abnormally brief SCR recovery times (Venables, 1975), the opposite pattern from that found in psychopaths.

Moreover, Gray, based upon neurophysiological data, has proposed that the hippocampus contains a gating mechanism (at the dentate gyrus) for filtering out stimuli not entailing punishment or uncertainty, and that this mechanism determines which stimuli trigger the BIS to inhibit reward-seeking behavior. Thus, "excessive" or overinclusive gating should result in diminished sensitivity to punishment or uncertainty cues, and thus to the weakened behavioral inhibition hypothesized by Gorenstein and Newman (1980) to underly ASPD and SD. As conceptualized by Gray, this gating process appears to involve a process of efferent inhibition similar to that proposed by Lacey (1967), Lykken (1968), and others. Thus, increased efferent inhibition (read: gating) of punishment signals in the hippocampus may lead to diminished BIS strength. This in turn would lead to decreased attention to the environment and decreased arousal (see above) and perhaps many of the features of ASPD and SD.

It must be emphasized that this integrative hypothesis is speculative, and will require considerably more physiological data before it can be adequately corroborated. Nevertheless, the analysis offered above suggests that a rapprochement between the efferent inhibition and behavioral disinhibition models may be possible. Interestingly, several psychodynamic writers have posited somewhat similar "inhibition-leading-to-disinhibition" models for the etiology of antisocial behaviors. Fenichel (1945), for example, spoke of "isolation of the superego" as a cause of psychopathic behavior. Thus, the framework proposed here may eventually serve to unite seemingly incompatible theoretical models, and generate testable predictions.

Summary

The hypothesis of an underactive BIS, although based on provisional data, seems to account for many of the clinical and psychophysiological features of ASPD and SD. Further neuro-

physiological research will be necessary, however, to determine if the diminished sensitivity to signals of punishment posited by this model can be explained by inhibition of these signals via cortical mechanisms, as per the efferent inhibition hypothesis.

THE NEGATIVE EMOTIONALITY MODEL

One final possibility that has received little or no attention is that the ASPD–SD association is mediated by high levels of *negative emotionality* (NE) (Lilienfeld, 1991b). NE, which is very similar to what has been called neuroticism or general maladjustment, is a dimension that taps a pervasive tendency to view oneself and the world negatively, and to become easily upset and distressed (Watson & Clark, 1984). In addition, individuals with high NE are prone to experience a variety of unpleasant affects, including anxiety, anger, mistrust, and resentment.

Although there has been little research on the relation between NE and psychopathy (Lilienfeld, 1991a), there is some evidence that ASPD patients are characterized by high scores on this dimension. DiLalla and Gottesman (in press), for example, found that patients with ASPD were characterized by elevated scores on the NE higher order factor of Tellegen's (1982) MPQ; ASPD patients also exhibited elevations on several lower order NE scales, including Alienation and Aggression. In addition, Harpur, Hare, and Hakstian (1989) reported that measures of neuroticism and anxiety were generally weakly positively correlated with a factor closely related to ASPD, whereas these measures were negatively correlated with a factor closely related to psychopathy. This raises the possibility that, whereas ASPD may be characterized by high NE, psychopathy may be characterized by low levels on this dimension, a point to which I will return shortly.

In addition, there is circumstantial evidence that SD patients may be characterized by high levels of NE. Coppen, Cowie, and Slater (1965) reported that "hysterics" exhibited substantially elevated scores on a neuroticism measure compared with normals, although it is not known how many of these patients met criteria for SD. Lilienfeld (1991b) reported that a self-report measure of ASPD loaded moderately highly (.33) on a factor comprising anxiety, depression, aggressiveness, and feelings of alienation, which appeared to represent NE. Interestingly, the Health Concerns scale of the MMPI-2 (Butcher, Graham, Williams, & Ben-Porath, 1990), which assesses a number of physical symptoms (e.g., fainting spells, nausea) similar to those of SD patients, also had a substantial loading (.57) on this factor.

Finally, Watson and Pennebaker (1989) reported that self-report measures of health (many of which contain items assessing symptoms common among SD patients) are substantially saturated with NE. Moreover, they found that NE is related to physical complaints, but not to objective physical health. They argued that this association is probably attributable to heightened perception of, and sensitivity to, physical symptoms on the part of high NE individuals, and suggest that NE "is a . . . general trait of *somatopsychic distress*" (p. 248).

Thus, there is some suggestion that both ASPD and SD are associated with high NE, although it must be emphasized that much of this evidence (particularly that on SD) derives from studies lacking formal diagnostic criteria. Nevertheless, if this finding were to be upheld by more systematic research, it would raise the question of how NE might mediate the ASPD–SD association.

Fowles (1987) has conjectured that behavioral disinhibition (i.e., an underactive BIS and/or an overactive BAS) produces high NE in some individuals, because this disinhibi-

tion may lead to recurrent impulsive and risk-taking behaviors that create difficulties and distress for the individual. Moreover, high NE would be especially likely in individuals whose behaviors involve repeated transgressions against society (e.g., those with ASPD) because such individuals would be likely to experience adverse consequences (e.g., imprisonment, occupational difficulties) from their behaviors. Thus, although psychopaths may be less anxiety-prone than other individuals (e.g., Lykken, 1957), they may actually experience *more* anxiety than other individuals in some cases because of the recurrent stressors that they bring upon themselves. This may account for Harpur and colleagues' (1989) finding that a factor related to ASPD is (slightly) negatively correlated with anxiety and other NE measures, whereas a factor related to psychopathy is positively correlated with these measures.

Thus, the behavioral disinhibition model may be compatible with the negative emotionality model. Specifically, a propensity towards behavioral disinhibition may lead to recurrent legal, interpersonal, and occupational difficulties in certain individuals (i.e., those with ASPD and closely related phenotypes), which in turn may result in high NE. Such high NE might then lead to SD by means of heightened perception of symptoms (Watson & Pennebaker, 1989), as well as to other syndromes characterized by high NE, such as anxiety disorders. This conjecture is consistent with the findings of Boyd et al. (1984), who reported that individuals with ASPD were at greatly elevated risk for a number of conditions apparently characterized by high levels of NE, including panic disorder, simple phobia, and obsessive-compulsive disorder. Thus, the overlap between ASPD and other forms of psychopathology may not be specific to SD, but may instead extend to a variety of syndromes characterized by high NE, although this possibility will need to be examined more extensively.

Although this causal model (behavioral disinhibition → antisocial and risk-taking behavior → high NE → SD and perhaps other conditions characterized by high NE) is speculative, it engenders a number of falsifiable predictions:

1. The conditional probability of having ASPD given SD should exceed the conditional probability of having SD given ASPD, because most SD patients would have had a history of antisocial and risk-taking behavior.
2. ASPD symptoms would more often precede SD symptoms than the reverse.
3. SD would be positively correlated with ASPD, but neglibly (or perhaps even negatively) correlated with primary psychopathy.
4. The association between ASPD and SD would no longer hold after NE levels are controlled for statistically.

Although none of these hypotheses has apparently been tested, confirmation of them would provide relatively strong corroboration for the negative emotionality model.

Summary

Preliminary evidence points to the possibility that both ASPD and SD patients are characterized by high levels of NE. The negative emotionality model may be compatible with the behavioral disinhibition model in that impulsive and risk-taking behaviors may lead to psychological distress and interpersonal difficulties in certain individuals, and in turn to high NE and conditions characterized by high NE (e.g., SD). Nevertheless, considerably more research using standardized diagnostic criteria is necessary to corroborate these conjectures.

FACTORS RESPONSIBLE FOR MODIFYING THE EXPRESSION OF THE DIATHESIS

The question remains, however, as to how a single diathesis can be manifested in two distinct phenotypes, depending largely upon the sex of the individual. Two traits that appear especially important for differentially channeling the expression of this diathesis in the two sexes are aggression and dependency.

Sex Differences in Aggression and Dependency

The most likely mechanisms for this hypothesized channeling process are prepubertal hormonal influences and differential sex-role socialization, both of which have substantial effects upon the levels of overt aggression, which is strongly associated with ASPD (APA, 1987). There is also evidence that dependency, which is associated with histrionic personality (APA, 1987), is influenced by differential sex-role socialization. Widom (1984) and Cloninger (1987) have argued that a major difference between antisocial and histrionic personalities is their degree of dependency. It is thus relevant that dependent traits are associated with increased risk for a broad spectrum of somatic symptoms (Greenberg & Bornstein, 1988).

Results of over 100 studies demonstrate that, beginning at age 2, males are more physically aggressive than females; moreover, this sex difference has been observed across a variety of cultures (Maccoby & Jacklin, 1974). Intraspecific aggression is greater among males than females in most mammalian species (Gray & Buffery, 1971), including rodents and nonhuman primates, making it plausible that this dimorphism is at least partially due to biological factors. Although studies of sex differences in dependency have generally yielded inconsistent results, the overall trend is in the direction of somewhat greater dependency among females (Lazarus & Monat, 1979).

What factors might account for the virtually ubiquitous sex difference in physical aggression? Androgenization decreases the threshold for aggressive behavior in primates over the course of both short- and long-term development. Rhesus monkeys administered testosterone during pregnancy bear female offspring that engage in increased amounts of rough-and-tumble play (Young, Goy, & Phoenix, 1964). Administration of testosterone to infant female rodents increases their fighting in adulthood (Edwards, 1970). Girls exposed to excessive male hormones in utero exhibit more "tomboyish" behaviors in childhood (Money & Ehrhardt, 1972).

If, as seems likely, the sex difference in physical aggression among humans is rooted at least partially in biological factors, it seems no less likely that sex-role socialization serves to maintain or accentuate this difference. In Western societies, aggressiveness if frequently accepted or even encouraged among boys, whereas dependency is often reinforced among girls (Kagan & Moss, 1962). Serbin and O'Leary (1975), for example, found that teachers paid closer attention to boys when they exhibited aggression, but paid closer attention to girls when they exhibited clinging and other dependent behaviors. Longitudinal studies of aggression reveal that aggressive behaviors are more stable from childhood to adulthood for males than for females; conversely, dependent behaviors are more stable from childhood to adulthood for females than for males (Kagan & Moss, 1962). Perhaps the most plausible explanation for these findings is that sex-role expectations curtail the expression of aggression among females and of dependency among males.

Some research indicates that females are more likely than males to express aggression passively. When introduced to a newcomer, girls showed more indirect hostility (e.g., ignoring, excluding) than did boys (Feshbach, 1969). Despite being less physically aggres-

sive, girls are about as likely as boys to engage in verbal attacks (Frieze, Parsons, Johnson, Ruble, & Zellman, 1978). A meta-analysis of sex differences in studies of aggression (Eagly & Steffen, 1985) corroborated the greater aggressiveness of males, but revealed that this difference was smaller for acts producing social or psychological harm than for acts producing physical injury. It is likely that these patterns are in part a result of social prohibitions against the expression of physical aggression among females. It is worth noting in this regard that some authors (e.g., Halleck, 1967) have commented that somatic symptoms can be a means of indirectly retaliating against or manipulating others.

Finally, differential sex-role socialization may also be partially responsible for the tendency of females to report more physical symptoms than males (Mechanic, 1976). Although females appear to have a higher rate of genuine physical illness than males (Gove, 1984), social factors may make females more likely to adopt the "sick role." For instance, women in Western society have until recently had fewer fixed role obligations (e.g., occupational demands) than men, which may have given them greater flexibility to restrict their activities due to illness (Gove, 1984), and perhaps made them more likely to define symptoms as indicative of illness. In addition, traits such as dependency, anxiety-proneness, and affiliativeness may lead females to seek out medical care more often than males (Mechanic, 1976).

Secular Trends in SD and ASPD

Based upon clinical and anecdotal observations, some authors have suggested that SD and related conditions (e.g., conversion disorder) are declining in prevalence (Jones, 1980). According to the Epidemiological Catchment Area (ECA) survey (Robins et al., 1984), the lifetime prevalence rate for SD in the general population is only 0.1%, a substantial decrease from previous estimates based upon roughly comparable criteria. Moreover, SD appears to be more frequent among the less educated (Guze, Woodruff, & Clayton, 1971b). The ECA study similarly disclosed consistent trends for the prevalence of SD to vary inversely with educational level (Robins et al., 1984). Others have observed that SD is still frequently encountered in pockets of American culture that are relatively isolated from the medical profession (Weinstein, Eck, & Lyerly, 1969).

One explanation for these findings is that prevalence of SD varies as a function of its social acceptability, as the increased sophistication of the general public (and perhaps physicians) regarding both psychology and medicine may preclude its expression. In this regard, one is reminded of Freud's (1910/1957) prediction that when the "secret" (p. 148) of a neurosis is disclosed to others the neurosis will cease to exist. In addition, the greater freedom of women in modern Western society to openly express anger (Williams, 1977) may have rendered indirect routes, such as somatization, less essential. Finally, the recent increase in females' fixed-role obligations (Gove, 1984) may have limited their opportunities for sick role behavior.

Concomitant with the apparent decline of SD among women in Western society has been a marked increase in the rate of female criminality (Wilson & Herrnstein, 1985). This trend is almost entirely due to a rise in female petty property crimes (Wilson & Herrnstein, 1985), which are characteristically committed by patients with ASPD (Bohman, Cloninger, Sigvardsson, & von Knorring, 1982).

Equally intriguing is the apparent sudden "appearance" in recent decades of new forms of character pathology, such as borderline and perhaps narcissistic personality disorders, both of which covary with antisocial and histrionic personality disorders. A study of secular trends in diagnostic practices in Denmark (Simonson & Mellergard, 1988) re-

vealed that, from 1975 to 1985, prevalence of the borderline diagnosis increased from 5% to 20%, while prevalence of psychopathic, hysterical, and "immature" diagnoses declined. Although it is unclear whether this shift is due to changes in diagnostic practices or to symptom presentation, Millon (1987) has proposed that social learning factors, such as the absence of role models, the disappearance of unifying cultural traditions, and the loss of meaningful aspirations, are responsible for the increase in borderline personality disorder over the past several decades.

Thus, one explanation for the apparent secular trends in SD, female petty criminality, borderline personality disorder, and perhaps other conditions is that sociocultural factors have altered the phenotypic expression of a similar underlying predisposition. Further longitudinal research with uniform diagnostic criteria, however, will be necessary to disentangle the relative contributions of changing diagnostic habits and/or systems versus changing modes of symptom expression to these trends.

CONCLUDING REMARKS

The literature reviewed here is consistent with the thesis that a single neurophysiological predisposition, perhaps toward diminished sensitivity to signals of punishment and uncertainty (which in turn may lead to high NE and syndromes characterized by high NE), may be manifested in a multitude of phenotypes depending upon the sex, socialization, and modifying personality characteristics of the individual. The current diagnostic system considers each of these syndromes to be a distinct disease entity; this approach will remain defensible in the absence of valid markers of their genotypes. Nevertheless, it seems possible that an overreliance upon behavioral criteria, as evidenced in the current classification of ASPD, will result in nosologic schemes that sacrifice validity for reliability. Moreover, if the expression of the diathesis to ASPD and SD is as time- and culture-bound as I have hypothesized, our diagnostic system may reflect secular trends in socially acceptable behaviors more than the underlying state of nature. What will the personality disorders section of the *Diagnostic and Statistical Manual* look like a century from now? If this diathesis is as protean in its manifestations as I have suggested, psychologists and psychiatrists in 2092 may be discussing personality disorders whose names would sound as foreign to us today as "borderline personality disorder" would have sounded to Freud or Kraepelin.

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