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City University of New York PH.D. 1982

PSYCHIATRIC ILLNESS IN THE FAMILIES OF PATIENTS WITH DSM III ANTISOCIAL PERSONALITY DISORDER

by

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A dissertation submitted to the Graduate Faculty in Clinical Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

1982

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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ACKNOWLEDGEMENTS

I would like to thank Drs. Armand Loranger and Herbert Nechin for their invaluable guidance in designing and carrying through this research, Mr. Emmanuel Lerner for his generous help and advice concerning the statistical analysis, and Ms. Alice Watt for organizing my time and turning my illegible writing into a manuscript, Dr. William Eckerd for his comments, patience and support, and Ms. Lindsey Eckerd for at least sleeping at night.

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CHAPTER I

LITERATURE REVIEW

> "I believe there are monsters born in the world to human parents....Just as there are physical monsters, can there not be mental or psychic monsters born? The face and body can be perfect, but if a twisted gene or malformed egg can produce physical monsters, may not the same process produce a malformed soul?"
>
> J. Steinbeck, East of Eden, 1952

History

There has been a popular awareness of the concept of psychopathy, or sociopathy, or antisocial personality disorder (ASPD) for a very long time. Koch coined the term "psychopathy" in 1891 to include all mental abnormalities that neither produce psychosis nor allow mental "normality," a very broad definition of the term (Koch, 1891, cited in Reid, 1978). The syndrome was described clearly in case studies by Kraepelin (1904). Kraepelin conceived of psychopathic conditions as "circumscribed infantilism" or "circumscribed developmental inhibition." His case studies are very similar to those of Cleckley (1941) in what is cited as the first thorough descriptive study of psychopathy in The Mask of Sanity in 1941. In another early study, Schneider (cited by Schulsinger, 1972) attempted to use descriptive classification in a way similar to Kraepelin, but he struggled to avoid moral judgments of behavior. He pointed out the basic difficulty in defining psychopathy as an individual psychological process: it is inherently social, or antisocial, and one must struggle to de-

fine social norms of behavior in order to define deviation.

Freud (1916) published "Some Character Types Met in Psychoanalytic Work." While he did not address psychopathy directly, he referred to "criminals from a sense of guilt" and differentiated them from criminals without guilt. Following Freud, Franz Alexander (1930) elaborated on the distinction between individuals living out impulses with antisocial behaviors who nonetheless experience inner conflict and the "united" antisocial personality of the criminal. As did Freud, he described a crucial aspect of the dynamic approach to psychopathy: the need to recognize unconscious motives to differentiate the "pathological character" from true criminals. Fortunately or unfortunately, he took upon psychoanalysis an awesome task: to differentiate individuals neurotically driven by unconscious motives from true criminals, "with whom they are almost always confused in current judicial practice." Alexander admitted his belief that most criminals would turn out to be neurotic characters, substituting alloplastic acting out of neurotic impulses for autoplastic symptom formation.

The definition of psychopathy continued to be extremely broad. In a text by Sadler (1936) "constitutional psychopathic inferiority" is described, first listing the usual antisocial features, then adding a long list of physical abnormalities such as large or small brain, weakness or paralysis of body parts and gross physical defects. This view opposes that of Kraepelin, Cleckley and others who note that one of the psychopath's characteristics is his/her appearance of supernormality and the ability to mimic normal interpersonal functioning.

The notion of psychopathic personality, formalized in 1952 in DSM I, must have been used prior to that time, since Cleckley (1941) in 1941 complains that personality disorder as a diagnosis is a "cumbersome and altogether vague diagnostic category...including a whole variety of maladjusted people who cannot by the criteria of psychiatry be classified with the psychotic, the psychoneurotic, or the mentally defective." Cleckley also was responding to the impact of Freud and the psychoanalytic theorists, whose dynamic unconscious formulations were at odds with his descriptive phenomenological studies. He particularly disagreed with notions of unconscious guilt and early childhood trauma as an etiology of the disorder, criticizing analysts for mistaking "presumptions" for evidence. This split between dynamic and phenomenological descriptive approaches has continued to the present, with writings usually representing one view or the other depending upon the orientation of the writer.

As Cleckley's description is usually cited as classic, I will include its cardinal features here. The notable features of psychopathy include: superficial charm and good intelligence, the absence of delusions or irrational thinking, the absence of nervousness or neurotic manifestations, unreliability, untruthfulness and insincerity, lack of remorse or shame, antisocial behavior without compunction, a failure to learn from experience, an incapacity for love and pathological egocentricity, poverty in affective response, fantastic behavior with drink, an impersonal and poorly integrated sex life, and no life plan. The psychopath "mimics human personality but is unable to really feel," or

as Johns and Quay (1962) put it later, "he knows the words but not the music."

Greenacre (1945) criticized the diagnosis of psychopathy as having become a catchall for patients with antisocial behaviors who lacked obvious psychotic or neurotic symptoms. She felt that "constitutional psychopaths" had faulty structural development of the conscience. She describes these patients similarly to Kraepelin and Cleckley, and emphasizes their inability to learn from experience: "they repeat the same fiascos time and time again in an impressively self-destructive fashion" (Greenacre, 1945).

This theme is echoed in DSM II (American Psychiatric Association, 1968). Here psychopathy is renamed "sociopathic personality disturbance, antisocial reaction":

> This term refers to chronically antisocial individuals who are always in trouble, profitting neither from experience nor punishment, and maintaining no real loyalties to any person, group or code. They are frequently callous and hedonistic, showing marked emotional immaturity and lack of responsibility with a lack of judgment and an ability to rationalize their behavior so it appears warranted, reasonable, and justified (APA, 1968).

Following DSM II, the descriptive accounts of sociopathic personality focused upon further differentiation and elaboration of the syndrome as narrowed somewhat to the domain of personality functioning. Karpman (1955) differentiated aggressive-predatory and passive-parasitic personalities, although little has been done with that distinction. Much writing has focused on formulating the primary underlying features of the syndrome: Craft (1955) saw lack of empathy and

impulsiveness as equal; Foulds and Boss (Boss, 1966; Foulds, 1965; cited by Hare, 1970) saw egocentricity as primary; and Arieti (1967) focused on impulsive need for immediate gratification as the central feature determining all others. Another debate has focused on the presence or absence of guilt, anxiety, and neurotic symptoms in the syndrome. Robins (1966) cited the presence of neurotic symptoms in as much as one-third of her follow-up study of antisocial youths, and described a positive correlation between the classical syndrome of sociopathic personality and neurotic symptoms. This argued against seeing antisocial behaviors as an alternate (substitute) expression of neurotic symptoms. Several dynamically oriented authors argued the presence of unconscious anxiety and guilt in these patients (Holt, 1979; Ruotolo, 1979), while others including Karpman (1955) and Cleckley (1941) distinguished between those demonstrating anxiety and guilt as secondary psychopaths and primary psychopaths or aneopaths (Reid, 1978) who gave neither physiological nor psychological evidence of anxiety and remorse.

The Current Concept and Debate

The difficulty in clearly defining psychopathy has been evident in the research literature as well as in clinical settings. Most research prior to DSM III used checklists of behaviors based on Cleckley's description. Robins (1966) cites the difficulty of discerning unconscious motives or of using self-report data as a basis for diagnosis, giving the example of a psychopathic patient who repeatedly beat, exploited, and abandoned his wife while proclaiming his love for her.

She concluded that objective description of behavior was the only solution.

DSM III (APA, 1980) classifies psychopathy or sociopathy as antisocial personality disorder, one of several enduring disturbances in personality functioning.[1] According to DSM III, personality traits that are inflexible and maladaptive and cause significant impairment in social or occupational functioning or subjective distress are considered to constitute personality disorders. These disorders are characteristic of long-term functioning and not discrete episodes of illness, and are recognizable by adolescence or earlier. The traits themselves may be egosyntonic but interfering with effective functioning, or they may be egodystonic but resistant to the individual's efforts to modify them. Antisocial personality disorder is limited to individuals over 18-years old, with specified historical and behavioral criteria developed by Spitzer, Endicott and Robins (see p. 43 for DSM III criteria).

However, even with the development of DSM III, consensus has not been reached. A significant minority continues to argue that, to quote Valliant (1975), "Cleckley's psychopath is a mythical beast." Valliant studied patients in 1975 and concluded that if one limited acting out via external controls, one finds (other) severe but comprehensible personality disorders such as hysteria and depression.

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[1] DSM III Personality Disorders include Paranoid, Schizoid, Schizotypal, Histrionic, Narcissistic, Antisocial, Borderline, Avoidant, Dependent, Compulsive, Passive-Aggressive, and Atypical or Mixed Personality Disorders.

He refers to the "concealed anxiety" of these patients, to which staffs respond with high anxiety, stereotypy, and poor treatment. It is useful to address Valliant's point, and question whether psychopathy, or sociopathy, or ASPD exists as a valid diagnostic entity, and whether it is a clinically useful concept.

Grinspoon and Bakalar (1978) raise a common criticism of the current concept of ASPD, that it is practically equivalent to the subset of people arrested for crimes, and therefore combines a legal judgment with a clinical one. They argue that the personality disorder could include people without a history of arrests, and also point to the crucial distinction between antisocial and dyssocial behavior. (Dyssocial behavior violates the standards of the dominant culture, but does not necessitate the lack of any ethical or social code as suggested by the term "antisocial.") The confusion of legal, moral, and psychological judgments is a difficult problem yet to be clearly addressed. Alan Frances (1980) echoes this concern (he reports that 80% of all criminals would be diagnosed ASPD) and feels that the current criteria miss the most important clinical point regarding the assessment of the person's capacity for loyalty, guilt, and anticipatory anxiety.

Eysenck and Eysenck (1978) raise another criticism more endemic to psychological categorization as a whole. They conceive of personality functioning as a continuous spectrum of genetically determined traits and suggest a dimensional rather than a categorical approach. They describe secondary ASPD as high on the neuroticism and extraver-

sion dimensions, while primary ASPD is described as high on neuroticism, extraversion, and especially psychoticism. Frances (1980, 1981) concurs that a difficulty with personality disorders is their continuity with normality and each other, and also recommends a dimensional system, if not the one proposed by Eysenck and Eysenck.

Reference has been made previously to the distinction between primary and secondary (neurotic or symptomatic) psychopathy or ASPD. While as Frances pointed out, DSM III does not make this distinction by failing to include vital object relations criteria, it would seem reasonable to go further and question even the inclusion of secondary ASPD in the diagnosis. As Hare and Schalling (1978) point out, the inclusion of these cases in the diagnosis ASPD implies that these people are basically psychopaths, although their motivation is different, they are usually able to experience guilt and remorse, and they are able to form affectionate relationships. There is also empirical evidence (Fagan & Lira, 1980) to support the distinction between primary and secondary psychopathy.

Despite all the above criticisms and qualifications concerning the concept and diagnosis of ASPD, to quote Jenkins (1960), "whether we retain the name, the concept of the psychopathic personality will remain with us for it is founded in experience and we need it." In fact, empirical evidence does support the differentiation of a group corresponding to ASPD. Evidence on genetics, physiological correlates and multivariate studies of behavioral factors are compatible with the clinical concept, as will be discussed later. Also, MMPI profiles

have identified with prototypes of people with ASPD (high on psychopathic deviate and hypomanic scales) although this correlation is not always clear (Spielberger, Kling, & O'Hagan, 1978).

ASPD As a Personality Disorder: Dynamic Hypotheses

If for the time being we accept the concept of a diagnosis of ASPD as useful and valid, it is important to understand how it is "located" vis-a-vis other personality disorders in dynamic terms, to place it in terms of dynamic understandings of diagnostic categories. Kernberg (1975) discusses antisocial personality as a low-level character disorder within his delineation of borderline character syndromes. He reports that "all clear-cut antisocial personality structures I have examined presented a typical borderline personality organization," which is described as characterized by chronic diffuse anxiety, polysymptomatic neurosis, and primitive defensive mechanisms. Antisocial personality may fit Kernberg's more process- and organizationally-oriented definition of borderline syndromes, but according to DSM III and other authors, ASPD is distinct from borderline personality. Sarwer-Forer (1977) sees these patients as more similar to impulse-ridden characters and comments that they are not readily treatable by currently available therapies.

Eysenck and Eysenck (1978) and Frances (1980) have proposed a continuity between personality disorders and it is worth exploring whether ASPD is seen as dynamically distinct from other personality disorders. The core issues of interest to dynamic theorists are whether all those diagnosed ASPD have unconscious guilt as a core, and whether the su-

perego of those with ASPD is structurally different than that of others.

Freud (1916) discussed the criminal acts of patients under his care as reflecting the effort to relieve themselves of excessive guilt derived from the Oedipus complex: the criminal intent to kill the father and have sexual relations with the mother. Freud does acknowledge that among adult criminals there must be those who commit crimes with no sense of guilt "who have either developed no moral inhibition or...consider themselves justified." Alexander, following Freud's thought, went so far as to see the lack of patricide as a sign that "in this modern world, even criminality has been domesticated....I am convinced that most of our criminals will turn out to be neurotic characters and that the notion of pure criminality must be looked upon as a threshold concept akin to the theory of a limit in mathematics" (Alexander, 1930). Greenacre (1945) describes the familial interactions that would interfere with the development of conscience both in terms of the resolution of the Oedipal complex and in terms of pre-Oedipal development. She suggests the child is unable to identify with primitive parental images, is unable to separate due to parental narcissistic attachment, and develops from that attachment a sense of magical omnipotence that impairs reality testing and emotional reactivity. However, she writes, "it is said that the psychopath has no guilt feelings, no conscience, no psychic mechanisms of defense against anxiety....If this were true the psychopath would explode from the force of his own primitive aggression." She saw the conscience of the psychopath as isolated, magical, and unusable, though.

These writings seem to suggest that all psychopaths might be found to experience unconscious guilt and conflict. This is equivocated somewhat by Freud's reference to some who experience no guilt and Greenacre's notion of an unusable conscience. This idea is taken to its extreme in current literature by Holt (1979) who suggests that a 20-year-old multiple murderer who professes only remorse at being caught still is responding to intolerably perfectionistic standards giving rise to burdensome guilt and anxiety.

On the other side of this debate, Reid (1978) suggests that the core content of the unconscious of the true psychopath differs from that of neurotics and psychotics, for it is empty, more related to the emptiness and hopelessness of endogenous depression. He views the drive for stimulation of the psychopath as an effort to escape from this nothingness.

Kernberg (1975) writes that the more antisocial trends are present, egosyntonic, and integrated into the patient's characterological makeup, the less of an integrated depersonalized superego is present. This may follow Greenacre's original suggestion that the superego of those with ASPD is structurally different from that of other people. Kernberg views moral self-evaluation as a part of superego functioning and as a more subtle part of reality testing, which would therefore be significantly and uniquely impaired in a patient with ASPD.

The Antisocial Child

Many psychodynamic theorists cite Bowlby among others in suggesting that ASPD has its roots in early childhood and specifically in

early maternal deprivation. Bowlby (1940, 1946) described "affectionless" children, and suggested a correlation between prolonged separation prior to age 6, lack of relatedness in children and delinquency. Unlike some others, Bowlby did not go so far as to say that separations in and of themselves cause affectionless character or delinquency, but that the combination of deprivation of maternal care, multiple placements and multiple parenting caused further deprivation. Subsequent researchers (Miller & Dollard, 1950; Mowrer, 1960; Schoben, 1949) focused on the interaction of multiple variables and turned away from a single cause-effect paradigm. McCord (1979) used a sophisticated multiple regression analysis to derive six factors that have impact on subsequent antisocial behaviors: mother's self-confidence, father's deviance, parental aggressiveness, maternal affection, parental conflict and parental supervision.

Research has established some consistent findings regarding childhood behavior and antisocial personality. Antisocial behavior does not begin in adulthood; there is always a childhood history of antisocial behavior (Robins, 1978). However, childhood antisocial behavior does not necessarily predict adult outcome. The number of childhood antisocial symptoms is a much more powerful prognostic indicator than is the presence of any particular antisocial symptom. Even in children showing ten or more antisocial symptoms, only 43% went on to a diagnosis of ASPD (Robins, 1966). Boys outnumber girls by 4:1 in conduct disorders, and those girls with behavioral problems come from more troubled families.

Parents of antisocial children often have a higher level of behavior problems, including alcoholism, ASPD, and hysteria. Antisocial fathers have been found to have a greater impact on offspring than antisocial mothers, even when the fathers are out of the home (Robins, 1978). Effective parenting can help children at risk of ASPD; according to Cloninger, Christiansen, Reich, & Guze (1978), the risk of ASPD is reduced by strict and consistent discipline. While there is increasing evidence of the importance of genetic factors, it is clear that environmental experiences account for the substantial variance, even between monozygotic twins reared together. Criminality aggregates among siblings occur more often than can be accounted for by genetic factors alone. Also, siblings are often more alike than are parents and offspring.

Genetic Studies

Most current researchers see antisocial personality disorder as an interaction of genetic and environmental factors, taking into account genetic loading, the condition of the nuclear family, early models, peer and social learning, cultural norms, and organic experience (i.e., illness, injury) (Cloninger, Reich, & Guze, 1978). Cloninger, Reich, and Guze propose that the pathogenic factors relevant to the development of ASPD are multiple and additive, without assigning relative importance to genetic and environmental factors. These factors together constitute a continuous underlying variable which they suggest is normally distributed and which is termed the liability to develop the disorder. A second important concept in

their work is that of a threshold for the expression of the disorder, which may be defined independently for each sex. The fact that girls with conduct disorders come from significantly more disturbed families than do boys may relate to a higher threshold for females.

While Cloninger, Reich, and Guze propose an additive model in which genetic and environmental factors are independent, Mednick and Hutchings (1978) propose an interactional model, in which genotype has a different clinical presentation in different environments and the same environment has different impact on different genotypes. In their study, they found that if both the biological and the adoptive father of adopted-away sons were criminal, then more adoptive sons were criminal (36%) than if only the biological father were criminal (21%). However, if only the adoptive father were criminal, the percentage of criminal sons was not significantly different than if neither father was criminal (10.4% and 11.2%, respectively). Eysenck and Eysenck (1975, cited by Cloninger, Reich, & Guze, 1978) suggest that 50% of the total variance between primary psychopaths and controls is due to genetic factors, a higher percentage than other researchers. According to Hare (1970), all but two twin and adoption studies have supported the hypothesis that genetic (biological) factors are important, and of those two, one did not follow the offspring through the entire risk period while the other still found delinquency in 13% of the children of criminal parents and in 14% of the children of antisocial parents, compared with 4% of other children.

A problem with many early studies is the equating of criminality

with ASPD. Given this drawback, there are still impressive data from twin studies and adoption studies.

The first twin study was by Lange (1929, cited by Mednick & Hutchings, 1978) in 1929. He found 77% concordance for criminality in monozygous (MZ) twins and 12% concordance for dizygous (DZ) twins. Following studies through 1961 (Mednick & Hutchings, 1978) were in general agreement with the direction of these results, finding 60% concordance for MZ twins and 30% concordance for DZ twins. Sampling bias in these studies tended to favor concordance in MZ twins. Christiansen (1968) found 36% concordance in MZ twins and 12% concordance in DZ twins.

Studies of adopted-away offspring of antisocial parents (or biological parents of known adopted-away antisocial offspring) have yielded results similar to the Mednick and Hutchings study cited earlier. Schulsinger (1972) found more psychopathy in the biological relatives of adopted offspring with ASPD than in the relatives of controls. Notably, biological fathers showed the most psychopathy, followed by biological parents together, and then biological relatives taken together. The data on the fathers is most impressive, as shown in Table 1. Other studies (Cadoret & Cain, 1980; Crowe, 1974) yielded similar results.

Popular attention had been given to research on chromosomal abnormalities in prison populations which suggested that XYY and XXY men showed increased aggressive and violent behavior. However, in a population at large in Denmark, 4754 men yielded 12 XYY men, and 14

TABLE 1

PERCENTAGE OF ASPD IN FATHERS

	Biological Father	Adoptive Father
Index Cases (ASPD)	9.3	1.9
Control Cases	1.8	0

XXY men with little serious violent behavior on record (Mednick & Hutchings, 1978). It would seem that sampling bias had greatly influenced earlier findings.

Physiological Evidence

Neurological Evidence

Evidence for genetic transmission of ASPD implies some biological correlate(s) of the syndrome, and researchers have focused upon neurological and autonomic nervous system correlations. While there is continued debate concerning the interpretation and practical implications of this work, there seems to be increasing consensus about the existence of significant findings. Koch and Kraepelin were among the first to suggest that psychopathy had a "consitutional" basis, although this notion fell out of favor for a while.

Many clinical features of ASPD can be produced by disorders of the brain resulting from illness or trauma (Elliot, 1978). These organic disorders produce a partial syndrome, however. As an example, in patients having suffered head trauma, it is not unusual to see ex-

plosive rage, aggressiveness, lying, and stealing. These patients do not manifest any lack in interpersonal attachments, loyalty, guilt, or shame as would someone with ASPD. Similar problems can be seen in patients with post-encephalitic personality. Another disorder found in the background of people with psychopathic behavior is temporal lobe epilepsy, but similarly, epileptics are usually able to relate well to others and aggressive outbursts are usually followed by remorse. Children with minimal brain dysfunction often have short attention spans, hyperkinesis, and resulting inappropriate response to social situations. If the child's judgment is poor he may not forsee consequences of behavior, and he may be unable to control impulsive and/or aggressive outbursts. Any and all of the organic defects described can produce emotional problems by evoking the rejection and hostility of families. This in turn can provoke dynamically-based acting out by the child or patient and a self-fulfilling cycle can begin.

EEG studies have shown both antisocial subjects and their parents to have abnormal amounts of slow wave activity (Hare & Cox, 1978). EEG abnormalities have been found in as many as 31% to 58% of persons with ASPD. These findings have been interpreted as delayed cortical maturation, cortical dysfunction, or low cortical arousal.

The maturational lag hypothesis is based on the observation that the slow wave EEG activity in subjects with ASPD resembles EEG activity found in normal children (Elliot, 1978). The type and distribution of EEG abnormality suggests a disturbance in the function in the

diencephalic and mesencephalic components of the reticular activating or limbic mechanisms. This could be due to delayed myelination or development of dendrites. Hare (1970) suggests that the egocentricity, impulsivity, and inability to delay gratification of ASPD subjects are also found to some extent in children. A reasonable question raised by the maturational lag hypothesis is whether one outgrows the ASPD syndrome. Robins' (1966) data suggests that one-third of ASPD patients become less grossly psychopathic, especially around ages 30-40. The hypothesis does not explain the presence of the findings, though, and the resemblance to children may be superficial. Also, 15% of normal population have EEG abnormalities and are mentally and behaviorally normal (Hare, 1970). Syndulko (1978) questions whether EEG findings relevant to ASPD are specific enough to merit conclusions at this time.

Autonomic Nervous System Response

Physiological evidence of low cortical arousal of ASPD subjects is fairly consistent, and suggests that ASPD people have a chronic need for stimulation to increase reticular activity to achieve optimum affect and behavior (Blackburn, 1978; Hare, 1970). This low cortical arousal is interpreted by Quay (1965) as due to intrinsic hyporeactivity or rapid habituation processes and by Hare (1970) to a capacity to defensively "tune out" stimuli, particularly aversive stimulation. Research has shown that the tonic skin conductance (SC) of those with ASPD is lower than others, and that this initial difference may increase over testing as the SC responses of ASPD subjects gets lower

over repeated trials while non-ASPD subjects have an increased response. This difference was not found to change with age, which would not support the maturational lag hypothesis. This lowered electrodermal arousal has been taken as physiological evidence that those with ASPD lack fear anxiety, or apprehension. It may also reflect a cognitive process, a lack of motivation on the part of these subjects to involve themselves or please the experimenter. However, SC responses to aversive stimuli are considered classical conditioning responses, which suggests a physiological unavailability to aversive conditioning by these subjects.

Low cortical arousal would also reduce the effectiveness of the cortical inhibitory system, which inhibits responses in anticipation of aversive stimuli. Impending pain or disaster is not sufficient to generate a conditioned fear stimulus that would bring about inhibition in most people in an aversive situation. ASPD subjects are particularly good at "negative preception" (Hare & Blering, cited by Hare & Cox, 1978): tuning out or attenuating a noxious stimulus, so their autonomic underreactivity is augmented when a warning signal precedes the stimulus. Also, following aversive stimulation, those subjects show a long recovery time (Aniskiewicz, 1979), further supporting their ability to "tune out" cues and mitigate the impact of punishment.

An interesting point was made in research cited by Hare (1970). ASPD subjects did not give SC responses or learned responses to avoid shock, but did learn responses to avoid losing money. This suggests

that these subjects are not incapable of learning, but the physiological substrate of learning avoidance behavior is significantly different than that of control subjects. This increases the impact of motivation and highlights that what stimulates motivation for those people would not be the anxiety or fear responses that might impact on other people.

Trasler (1978) took the above literature and explored its implications from a sociological and social learning perspective. He suggests that what might be considered the conditioning equivalent of internalization of controls occurs via secondary aversive conditioning. The child's actions, initially controlled by the aversive responses of the parents, become controlled by secondary stimuli (inhibition responses) leading to internal inhibitory controls. This results in a shift in the balance of situational and internal controls in the direction of internal control. He further proposes that the internal controls most firmly established for most people are those relating directly to childhood experience, regarding such issues as not lying, stealing, control of aggression, control of sexual exhibitionism, etc. According to Trasler, normal people are able to more easily violate speeding laws than steal, for example, because speeding corresponds less immediately to childhood experience, so situational control (a patrol car with radar) is necessary. Persons with ASPD have an imbalance of situational and intrinsic controls, because the complex neurocortical system mediating aversive inhibitiory conditioning is not functioning normally. While these people are not

"driven" to break laws, they are "people whose decisions and choices, immediate options and alternatives are different from others" (Trasler, 1978). Trasler concludes that ASPD is due to either physical defects inhibiting conditioning, an environment that did not provide the proper stimulation to achieve internalized conditioning, or some combination of the two. The implications of these ideas in psychodynamic terms is fascinating; that these persons potentially are limited by a physiological substrate of the identification process, with subsequent impact on the development of ego and superego functions and on the potential for object relations.

Antisocial Personality Disorder and Related Syndromes

Alcoholism

The literature on alcoholism and ASPD would seem to lead to two conclusions: there is increased alcoholism in families of those with ASPD, but ASPD and alcoholism as primary disorders sort independently of each other genetically. Bohman (1978), Cloninger, Christiansen, Reich, and Gottesman (1978), and others have found a strong genetic component in the transmission of alcoholism from parents to children, but suggest a different genetic pattern than is seen with ASPD. While DSM I listed alcoholism under sociopathic disorders (APA, 1952), most contemporary writing seems to be in concordance with Rada's (1978) conclusion that one may find alcoholics with antisocial behaviors, "sociopaths" who drink, and only occasionally both disorders as primary. However, it is my clinical experience that a significant num-

ber of patients whose alcoholism (or substance abuse) brings them under the jurisdiction of courts find themselves with diagnoses of ASPD.

Hyperactive Child Syndrome

There is significant evidence linking hyperactive child syndrome and ASPD. Many authors (Mendelson, Johnson, & Stewart, 1971; Robins, 1966) have found hyperactive children at increased risk of becoming ASPD adults, even as many as 25%. According to Cantwell (1972) and Morrison and Stewart (1971), there is a significantly high incidence of psychopathy, alcoholism, and hysteria in the families of hyperactive children. Sixty to 70% of hyperactive children show low control nervous system arousal on three indicators of autonomic activity, underlying high behavioral activity similar to the stimulation seeking of ASPD subjects (Satterfield, 1978). Also, there is evidence of excessive slow wave EEG activity (Satterfield, Cantwell, & Satterfield, 1974). Satterfield's (1978) theoretical explanation for these data are similar to those proposed for ASPD: either delayed cortical maturation or low CNS arousal, although he prefers the latter hypothesis. Cantwell (1978) suggests a heterogeneity of etiology of hyperactive child syndrome producing a similar phenomenological picture. He proposes as a direction for future research a study of those hyperactive children who do and do not develop ASPD, perhaps using biological markers.

Hysteria

Research and clinical experience have indicated an "unholy triad" of hysteria (Briquet's syndrome), ASPD, and alcoholism in certain families. In a study of the families of female felons, 78 to 80 percent of whom were found to have ASPD, first-degree male relatives had a high incidence of ASPD, alcoholism and drug dependence, while first-degree female relatives were often diagnosed as having hysteria (Cloninger & Guze, 1973). In general 44% of the relatives of female felons received a psychiatric diagnosis with one of three diagnosed ASPD or hysteria. Guze had previously found similar results using a population of male felons (Guze, 1976; Guze, Woodruff, & Clayton, 1971).

A difficulty with other anecdotal literature supporting the link between ASPD and hysteria has been the lack of clarity of the diagnosis of hysteria. Warner (1978) argued that the male/female diagnostic differences cited are the result of sexual stereotypy in diagnosis by mental health professionals, so that a similar clinical presentation would be diagnosed hysteria in females and ASPD in males. However, in describing the presenting syndrome, Warner lists excitability, hyperemotionality, instability, self-dramatization, attention seeking, seductive behavior, immaturity, egocentricity, vanity, suggestibility, and dependence. This presentation does not resemble the DSM III criteria for ASPD, the research criteria used for ASPD by Guze (1976), nor the research criteria for hysteria used by Guze et al. (1971). It will clarify the connection of hysteria and ASPD to specify

the syndrome (Briquet's syndrome) described by Guze and his associates, based on criteria by Perley and Guze (Guze, 1976).

A. Patient presents dramatic or complicated medical history prior to age 35.

B. Twenty-five of these symptoms are necessary for a definite diagnosis and 20 to 24 symptoms for a probable diagnosis, divided among 9 of 10 groups of symptoms:

1. feeling sickly for most of life, headaches
2. blindness, paralysis, anesthesia, aphasia, fits or convulsions, unconsciousness, amnesia, deafness, hallucinations, urinary retention
3. fatigue, lump in throat, fainting spells, visual blurring, weakness, dysuria
4. breathing difficulty, palpitation, anxiety attacks, chest pain, dizziness
5. anorexia, weight loss, marked fluctuations in weight, nausea, abdominal bloating, food intolerances, diarrhea, constipation
6. abdominal pain or vomiting
7. dysmenorrhea, menstrual irregularity, excessive menstrual bleeding
8. sexual indifference, sexual frigidity, hospitalization for hyperemesis, gravidarium, vomiting for entire nine months of pregnancy
9. back pain, joint pain, extremity pain, burning pains of

the sexual organs, mouth or rectum, or other bodily pains

10. nervousness, fears, depressed feelings, need to quit work or inability to carry on regular duties because of feeling sick, crying easily, feeling life was hopeless, thinking a good deal about dying, wanting to die, suicide attempts

C. No other diagnosis to explain symptoms. Other differential diagnoses to rule out: obsessional neurosis, schizophrenia, primary affective disorder, organic brain syndrome.

Other Syndromes

The focus of this review has been those psychiatric syndromes suspected of being genetically linked to ASPD. Obviously, other psychiatric syndromes have been related to ASPD phenomenologically in clinical presentation, including sexual perversions, homosexuality, drug dependence, and narcissistic personality disorder, but inasmuch as these have not been genetically linked to ASPD, they are not central to the research in this study.

Comparative Data

Since this study will focus on the diagnosis of ASPD and data on psychiatric family histories of these patients, it will be useful to summarize briefly the quantitative research that will be used for comparisons.

Diagnosis of ASPD. In the DSM III criteria for ASPD, certain childhood history factors must be considered. Age of onset of anti-

social symptoms must be prior to 15 years of age. Robins' data (1966) indicates an even earlier onset, with 86% of boys and 36% of girls showing antisocial symptoms prior to age 11. Table 2 summarizes Robins' data and will be used as a comparison to study subjects.

TABLE 2

AGE OF ONSET OF ANTISOCIAL SYMPTOMS*

Age of onset	% Boys	% Girls
Less than 8 yr.	58	9
8-10 yr.	28	27
11-13 yr.	8	9
14-18 yr.	6	55

*(From Robins, 1966, 1978)

DSM III also specifies that at least two of a list of antisocial symptoms be present in childhood. Robins explained in greater detail the kinds of antisocial symptoms presented by a population of children at a guidance clinic, and, based on later follow-up, found the number of children with a given symptom who went on to an adult diagnosis of ASPD. Table 3 summarizes this data.

Robins also looked at the total number of antisocial symptoms as a variable, calculating the percentage of children with a given number of symptoms who later met criteria for an adult diagnosis of ASPD. DSM III requires two symptoms in childhood, but Robins' data indicates that of children with 3-5 antisocial symptoms, only 15% go on

TABLE 3

CHILDHOOD SYMPTOMS

Symptom	% Adults diagnosed ASPD (historically)	% Children later diagnosed ASPD
Theft	83	31
Incorrigibility	80	30
Truancy	66	34
Running away	65	33
Bad companions	56	30
Staying out late	54	30
Physical aggression	45	32
Poor employment record	44	32
Impulsivity	38	35
Reckless, irresponsible	35	29
Slovenly appearance	32	34
Enuresis	32	29
Lack of guilt	32	38
Early sexual experience	28	31
Pathological lying	26	39
Sexual perversion	18	37

to adult diagnosis of ASPD. Table 4 summarizes these results.

DSM III also specifies adult symptoms for the diagnosis of ASPD, including factors such as poor work history, arrests, divorce or sep-

TABLE 4

NUMBER OF ANTISOCIAL SYMPTOMS IN CHILDHOOD*

	% diagnosed ASPD as adults
10+ Antisocial symptoms	43
(+ 6+ other symptoms)	42
6-9 AS symptoms	27
(+ 6+ other symptoms)	22
3-5 AS symptoms	15
(+ 6+ other symptoms)	10
0-3 AS symptoms	4
(+ 6+ other symptoms)	6

*(From Robins, 1966, 1978)

arations, fighting, thefts, defaulting on debts, illegal occupation, and aimless travel. Robins examined a more detailed list of adult antisocial symptoms, and calculated the percentage of adults with a given symptom who were diagnosed ASPD. Table 5 illustrates this data.

Psychiatric Illness in Families

The literature on patterns of psychiatric illness in the relatives of people with ASPD is limited and often reports on the illness in families of felons (or criminality in the families of felons) as opposed to examining ASPD as distinct from criminality. Guze (1976)

TABLE 5

ADULT BEHAVIOR AND SYMPTOMS OF PATIENTS DIAGNOSED ASPD*

Symptom	% of patients with ASPD diagnosis with symptom
Poor work history	85
Poor mental history (% of those maimed)	81
Financial dependency	79
Arrests	75
Excessive alcohol	72
School problems	71
Impulsiveness	67
Sexual behavior	64
Wild adolescence	62
Vagrant	60
Belligerency	58
Social isolation	56
Armed forces difficulties (% of those serving)	53
Lack of guilt	40
Somatic complaints (more than 9 or disabling)	31
Use of aliases	29
Pathological lying	16
Use of drugs	15
Suicide attempts	11

*(From Robins, 1966)

estimated that 78%-80% of criminals in his samples of convicted felons would be given a diagnosis of ASPD; this claim has been challenged as an overestimate without any data provided. Cloninger, Christiansen, Reich, and Gottesman (1978) estimated the prevalence of ASPD in the general population as 3.3% of men and .94% of women based on observed data. (Their predicted estimates had been 3.6% ± .09 for men and .75% ± .28 for women.) Data on family criminality and ASPD can be compared to these figures.

Tables 6 through 8 summarize data on criminality, ASPD, and sociopathic spectrum disorders in relatives of ASPD patients or felons. Cloninger, Christiansen, Reich, and Gottesman (1978) looked at ASPD in first-degree relatives of patients with ASPD. Their results are summarized in Table 6.

TABLE 6

ASPD IN RELATIVES

Patients with ASPD	% observed men	% observed women
Men	16.8	4.4
Women	27.7	10.1

Schulsinger (1972) looked in more detail at psychopathic spectrum disorders in biological and adoptive relatives of ASPD patients and controls. Her data is summarized in Table 7 (A, B, C, & D). Clearly, the greatest incidence of ASPD is in biological fathers of ASPD patients.

TABLE 7

PSYCHOPATHIC SPECTRUM DISORDERS AND ASPD IN RELATIVES

	Biological	Adoptive
A. Psychopathic spectrum disorders in relatives		
ASPD patient	14.4% ± 2.0	7.6% ± 2.3
Control	6.7% ± 1.5	5.3% ± 1.9
B. Core psychopathy in relatives		
ASPD patient	3.9% ± 1.1	.8% ± .8
Control	1.4% ± .7	1.5% ± 1.0
C. Core psychopathy: Parents only		
Index	4.5% ± 2.0	.9% ± .9
Control	.9% ± .9	0%
D. Core psychopathy: Fathers only		
Index	9.3% ± 4.0	1.9% ± 1.9
Control	1.8% ± 1.8	0%

Mednick and Hutchings (1978) looked at criminality in biological and adoptive parents of criminals, and again results suggest the greatest concordance between criminal sons having committed a major offense and criminal biological fathers. Table 8 illustrates their data.

Schulsinger (1972) reported finding psychiatric illness in 19% of biological relatives of psychopathic patients (compared with 12.0% -

TABLE 8

CRIMINALITY IN ADOPTIVE AND BIOLOGICAL PARENTS

Criminal record of adoptee	% Criminal adoptive father	% Non-criminal adoptive father	% Criminal biological father	% Non-criminal biological father
Not registered as criminal	9.2	90.8	9.5	90.5
Minor offense	14.0	86.0	37.7	62.3
Criminal offense	21.7	78.3	48.8	51.2

13.7% of controls). She reported psychopathic disorders in 14% of biological relatives of these patients (compared with 5.3%-7.6% of controls) and ASPD in 3.9% of biological relatives (compared with .8%-1.5% of controls). This estimate is for men and women combined, and therefore appears low compared to Cloninger's estimate of 3.3% ASPD for men in the general population (Cloninger, Christiansen, Reich, & Gottesman, 1978). More elaborated data on psychiatric illnesses in families is limited to two sets of studies, Robins (1966, 1978) and Cloninger et al. (Cloninger, Christiansen, Reich, & Gottesman, 1978; Cloninger & Guze, 1978).

Robins' data is based on the follow-up of children seen in a guidance clinic, where families were interviewed or researched as part of case histories. Her data is summarized in Table 9.

Cloninger and Guze (1973) and Cloninger, Christiansen, Reich, and Gottesman (1978) looked at families of male and female criminals.

TABLE 9

PROBABLE DIAGNOSES OF PARENTS OF

CHILDREN LATER DIAGNOSED ASPD

Probable diagnosis	Fathers (%)	Mothers (%)
Sociopathic or alcoholic	36	11
Psychotic	4	8
Schizophrenic	1	2
Chronic brain syndrome	1	Less than .5
Manic depressive	Less than .5	1
Undiagnosed hospitalization	1	5
Neurotic	Less than .5	3
Feeble minded	1	6
Undiagnosed ('very nervous"; "nervous breakdown," suicide)	5	11
No evidence of disease or senility only	54	61

Their data on psychiatric diagnosis of first-degree relatives is based on interviews or reported data. (Data based on interview is separated from second-hand reports.) Their results are summarized in Tables 10 and 11.

These studies by Cloninger and Guze (1970, 1973), Cloninger, Christiansen, Reich, & Gottesman (1978), and Cloninger, Reich, and Guze (1978) are limited by their use of criminal populations instead of clinically diagnosed subjects. The data on male and female crim-

TABLE 10

PSYCHIATRIC ILLNESS IN THE FAMILIES OF FEMALE CRIMINALS

Psychiatric diagnosis	% Men interviewed	% Men not interviewed	% Women interviewed	% Women not interviewed
Alcoholism	61	37	27	19
Hysteria	0	0	34	0
ASPD	36	18	11	4
Drug dependence	14	3	4	1
Anxiety neurosis	19	0	16	0
Homosexuality	11	1	6	0
Primary affective disorder	0	0	6	0
Mental retardation	0	1	4	0
Dementia	0	0	1	0
Schizophrenia	3	0	0	1
No special diagnosis but ill	6	9	23	26
Some psychiatric illness (ASPD or hysteria)	36	18	39	4
Alcoholism or drug dependence (and not hysteria or ASPD)	31	27	14	19
Other illness	19	11	37	28
Total	86	56	90	51
No illness	4	44	10	49

TABLE 11

PSYCHIATRIC ILLNESS IN FAMILIES OF MALE CONVICTED CRIMINALS

Diagnosis	% white men	% black men	% all men	% white women	% black women	% all women	% total
Psychiatric diagnoses in interviewed relatives							
Anxiety neurosis	7	6	7	11	17	13	10
Manic depression	2	0	2	8	0	5	4
Homosexuality	3	0	2	0	0	0	1
Hysteria	0	0	0	6	5	5	4
ASPD	14	29	19	5	0	3	9
Alcoholism	27	49	34	4	6	5	15
Drug addiction	2	6	4	0	0	0	2
Psychiatric hospitalization	0	6	2	4	0	3	2
Suicide attempt	1	0	1	2	0	1	1
Suspected psychiatric diagnosis in noninterviewed relatives							
Dementia	0	0	0	1	0	1	<1
Schizophrenia	0	0	0	1	0	1	<1
Manic depression	2	0	1	1	0	1	1
ASPD	19	26	21	0	0	0	13
Alcoholism	21	10	18	1	7	3	12
Psychiatric hospitalization	<1	0	<1	4	0	3	<2
Suicide attempt	<1	0	<1	1	0	1	<1

inals are consistent in suggesting that alcoholism and ASPD are the most common psychiatric problems in these families. Hysteria, drug dependence, and anxiety neurosis are present frequently, although hysteria is limited to female relatives. Major psychoses are rarely found in these families. These data are consistent with the literature on syndromes related to ASPD discussed earlier.

CHAPTER II

INTENT OF THIS STUDY

The intent of this research is to study the patterns of psychiatric illness in the families of patients with a diagnosis of antisocial personality disorder (ASPD). As was evident in the literature review, the criteria for ASPD and even the inclusion of this diagnosis as a psychiatric disorder have been controversial. Etiological theories have echoed this controversy, and attempts at treatment of these patients have reflected confusion over the "status" of this disorder. In The Diagnostic and Statistical Manual (DSM) III of the American Psychiatric Association (1980) ASPD has been included as a personality disorder, defined by specific historical and behavioral criteria. This study will compare one aspect of ASPD, the psychiatric illness in families of these patients, with patterns of psychiatric illness in the families of patients with major psychoses (schizophrenia and bipolar disorder) and families of patients with a different personality disorder as defined by DSM III, borderline personality disorder. The resulting data will add to our understanding of ASPD in the context of psychiatric illness.

As this study will be among the early attempts to utilize the DSM III criteria for ASPD, the collection of this data will also comment on the utility of this concept as defined by DSM III, and the extent to which these criteria seem to reflect a clinically meaningful group of patients. Data will be collected regarding the age of

onset of antisocial symptoms, the numbers of symptoms, the kinds of symptoms, and adult behavior to compare this group of patients meeting DSM III criteria of ASPD to those persons studied in the past as sociopathic or psychopathic (Robins, 1966).

Hypotheses

Hypothesis 1: Prevalence of major psychotic illness.

A. The morbid risk for schizophrenia, bipolar disorder and unipolar depression in the ASPD relatives will approximate the morbid risk for these illnesses in the general population.

B. The morbid risk for schizophrenia, bipolar disorder and unipolar depression in the ASPD relatives will be significantly less than the risk for those illnesses in the relatives of schizophrenic and bipolar patients when the risk factor for the latter groups exceeds the risk for the general population.

C. The morbid risk for schizophrenia, bipolar disorder and unipolar depression in the ASPD relatives will not differ significantly from the morbid risk for those illnesses in the borderline relatives.

Hypothesis 2: Prevalence of personality disorder.[1]

A. The morbid risk of personality disorder in the ASPD relatives will be significantly greater than the risk of personality disorder in relatives of schizophrenic and bipolar disorder patients.

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[1]"Personality disorder" will be used here in a generic sense. Persons whose personality functioning has resulted in maladaptive behaviors in occupational or social settings and/or experience subjective distress are described by DSM III as manifesting personality dis-

B. The morbid risk of personality disorders in the ASPD relatives will not differ significantly from the morbid risk for personality disorder in borderline relatives.

Hypothesis 3: Prevalence of alcoholism.

A. The morbid risk for alcoholism in the ASPD relatives will be descriptively greater than the estimated risk of alcoholism in the general population.

B. The morbid risk for alcoholism in the ASPD relatives will be significantly greater than the risk of alcoholism in the relatives of schizophrenic and bipolar disorder patients.

C. The morbid risk of alcoholism in the ASPD relatives will not differ significantly from the risk of alcoholism in the borderline relatives.

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order. Disturbances of mood including anxiety or depression are also frequent presenting symptoms. For the purposes of this study, individuals treated by a mental health professional for enduring difficulties or dissatisfactions in effective functioning not due to psychosis, organic difficulties or acute situational stress are considered to have "personality disorders." (Such treatment was usually outpatient treatment.)

This category is comparable to Tulis' category of "neurosis or personality disorder" (Tulis, 1979). Loranger, Oldham, and Tulis (1982) specified the following criteria for "borderline personality," which corresponds well with the description of the individuals characterized as "personality disorders." Loranger's criteria include: A) No evidence of toxic or organic brain disease; B) Does not meet the criteria for schizophrenia or bipolar disorder; C) At least two of the following traits or behaviors: 1) labile, moody, emotional; 2) dramatic, histrionic, hysterical, attention seeking, manipulative; 3) demanding, dominating, inconsiderate, selfish, egocentric; 4) anger, rages, tantrums; 5) promiscuity or casual sexual relationships, homosexuality, incest, sexual perversions; 6) lying, stealing, truancy, vandalism, arrest, "acting out"; 7) impulsive, suicidal, reckless; 8) alcohol or drug abuse; 9) assaultive, violent. These criteria are broadly descriptive

Limitations

This study will be necessarily limited by the use of hospital charts. Only hospitalized patients will be available for study, and it is obvious that many people with ASPD will not be hospitalized, either being "successful" in their antisocial life style, or in jail. In practice, many of those hospitalized with this diagnosis chose hospitalization as an alternative to jail, or as an attempt to use psychiatric illness as a defense in court. The hospital in this study is a private psychiatric hospital in an affluent suburb, which suggests that an ASPD patient there probably has economic or other resources at his or her disposal. While a jailed population meeting DSM III criteria might serve as an interesting control group, it would be impossible to find a group of "successful" persons with this disorder.

In addition, the charts themselves present limiting factors. Only patients hospitalized long enough to assemble case history material can be considered. Patients with ASPD are poor and unreliable historians, a difficulty often shared by their families. Completeness of records also presents a difficulty beyond the lack of cooperation of patients and families. Treating staff, involving therapist, social workers, nurses, aides, and others do not always write extensive and detailed notes, particularly about patients who can evoke such negative countertransference (and justifiable rage) as do these patients. Ob-

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of four personality disorders clustered by DSM III: histrionic, narcissistic, antisocial and borderline. I chose to use the broader terminology to more closely follow DSM III criteria.

jective reporting can also be influenced by the theoretical biases of the treating staff in the material considered relevant to research and record.

CHAPTER III

METHOD

Subjects

Data for this study is derived from psychiatric hospital charts in the Medical Records Department of the Westchester Division of The New York Hospital-Cornell Medical Center in White Plains, New York. Charts of patients carrying a discharge diagnosis of antisocial personality disorder (301.7) were reviewed. Both males and females were included in this study--although in keeping with the literature on this disorder, there were significantly more males than females. It is anticipated that the patterns of familial illness would be similar for males and females, although it has been suggested that more severe family pathology is needed for the expression of antisocial traits by females. Charts dating from 1969 to 1981 were reviewed to compile a sample of 35 charts. Basic information about each patient including hospital number, last New York Hospital admission and discharge, age at first psychiatric treatment and at first psychiatric hospitalization, date and place of birth, age, education, marital status, occupation, religion, and previous inpatient and outpatient treatment were recorded. Review of the charts included the psychiatric case study (history, mental status, and formulation), social service and psychological testing summaries, progress notes by the therapist, social worker, nursing staff, occupational and recreational therapists, discharge summary, and reports from other professionals or facilities

regarding previous hospitalizations or treatment.

Charts were reviewed for concordance with the DSM III criteria for antisocial personality disorder, as follows:

DSM III Diagnostic Criteria for Antisocial Personality Disorder

A. Current age at least 18 and a history of continuous and chronic antisocial behavior in which the rights of others are violated.

B. Onset before age 15 as indicated by a history of two or more of the following:

   1. Truancy (positive if at least five days per year for at least two years, not including the last year of school).
   2. Expulsion from school.
   3. Delinquency (arrested or referred to juvenile court because of behavior).
   4. Running away from home overnight at least twice while living in parental or parental surrogate home.
   5. Persistent lying.
   6. Unusually early or aggressive sexual behavior.
   7. Unusually early drinking to excess, or substance abuse.
   8. Thefts.
   9. Vandalism.
   10. Required to repeat school grades or grades markedly below what would be expected on basis of estimated or known IQ.
   11. Chronic violations of rules at home and/or at school (other than truancy).

C. At least three of the following since age 15:

1. Poor occupational performance over several years as shown by either:

   a. frequent job changes (three or more jobs in five years not accounted for by nature of job or economic or seasonal fluctuation)

   b. significant unemployment (six months or more in ten years when expected to work)

   c. serious absenteeism from work (average three days or more per month--late or absent)

   Note.--Poor academic performance for the last few years of school may substitute for this criterion in individuals who by reason of their age or circumstance have not had an opportunity to demonstrate occupational adjustment.

2. Three or more non-traffic arrests, or a felony conviction.

3. Two or more divorces and/or separations (whether married or not).

4. Repeated physical fights or assault (not required by one's job or to defend someone or oneself).

5. Repeated thefts, whether or not caught.

6. Illegal occupation (e.g., prostitution, pimping, selling drugs).

7. Repeated defaulting on debts or other major financial responsibilities, such as child support.

8. Traveling from place to place without a prearranged job or clear goal for the period of travel or clear idea when the travel would terminate.

D. No period of five years or more without antisocial behavior between age 15 and the onset of adult antisocial behavior, when the individual was not bedridden, confined in hospital or penal institution, or under treatment.

E. Antisocial behavior is not symptomatic of either severe mental retardation, schizophrenia, schizoaffective, or paranoid disorder.

Patients meeting these criteria who were not adopted children were included in this study.

At the same time, charts were examined to determine any overlap between the group of ASPD patients and patients with borderline personality disorder. The DSM III criteria for borderline personality disorder are as follows:

DSM III Diagnostic Criteria for Borderline Personality Disorder

At least six of the following are characteristic of the patient's long-term functioning and are not limited to discrete episodes of illness:

1. Impulsivity or unpredictability in at least two of the following areas which are potentially self-damaging:
   a. spending
   b. sex
   c. gambling
   d. shoplifting
   e. drug or alcohol use
   f. overeating
   g. physically self-damaging acts
   h. other _____
2. A pattern of unstable and intense interpersonal relationships characterized by:
   a. marked shifts of attitude

b. idealization

c. devaluation

d. manipulation (consistently using others for one's own sake)

e. other _____

3. a. Inappropriate intense anger

b. Lack of control of anger

4. Identity disturbance manifested by uncertainty about several of the following:

a. self-image
b. gender identity
c. values
d. loyalties
e. long-term goals or career choice
f. friendship patterns
g. other _____

5. Affective instability: Marked shifts from

a. normal mood to depression

b. normal mood to irritability

c. normal mood to anxiety

usually lasting hours and only rarely for more than a few days, with a return to normal mood.

6. Problems tolerating being alone, manifested by:

a. efforts to avoid being alone

b. depressed when alone

7. Physically self-damaging acts, for example:

a. suicidal gestures

b. self-mutilation

c. recurrent accidents

d. physical fights

e. other _____

8. Chronic feelings of emptiness or boredom.

The Patient Sample

Based upon the review of available charts, a sample of 36 cases meeting DSM III criteria for ASPD were selected. None of these cases met DSM III criteria for borderline personality disorder. These cases included 33 men and 3 women, all hospitalized at some time between 1969 and 1981 at The New York Hospital-Cornell Medical Center, Westchester Division. The mean age at admission of these patients was 23.7. Table 12 illustrates the age on admission of this patient group.

TABLE 12

AGE ON ADMISSION OF ASPD PATIENTS

Age	Number of cases
15-19	11
20-24	11
25-29	10
30-34	2
Over 35	2

The three women were of ages 18, 23, and 31, yielding a mean age of 24, comparable to the mean age of the entire sample. Of these pa-

tients, 5 were married, 6 divorced, 1 was single, and 24 never married.

Sixty-nine percent of these patients had at least one prior hospitalization. Table 13 shows the number of hospitalizations for patients in the sample.

TABLE 13

NUMBER OF HOSPITALIZATIONS OF ASPD SAMPLE

Number of hospitalizations	Number of cases
1	10
2	10
3	7
4	3
5	3
6 or more	3

Interestingly, the age of the patient did not predict the number of hospitalizations: two of the patients with more than 6 hospitalizations were 20 years old.

Robins (1966) had collected data on the age of onset of antisocial symptoms, showing 86% of boys and 36% of girls manifesting symptoms by age 11. Ages of onset of symptoms for this current sample were widely spaced, from age 3 to age 15. Table 14 shows age of onset of antisocial symptoms.

Forty-two percent of these patients developed antisocial symptoms by age 11. The 11-13 age group also represents 42% of these cases, as

TABLE 14

AGE OF ONSET OF ANTISOCIAL SYMPTOMS

Age of onset	Number of cases
3-5	5
6-7	5
8-10	5
11-13	15
14-15	6

compared with only 8% of boys and 9% of girls having an onset of symptoms in that age span in Robins' data.

Age at first treatment was varied, but 50% of these patients were first treated between ages 16-20. Table 15 illustrates the age of first treatment for this group.

TABLE 15

AGE AT FIRST TREATMENT OF ASPD CASES

Age at first treatment	Number of cases
3-5	1
6-10	2
11-15	7
16-20	18
21-25	5
26-30	3

It is interesting to note that while 84% of these patients manifested symptoms by age 13, 72% did not receive any kind of treatment until age 16-20.

DSM III (1980) requires that patients diagnosed ASPD manifest at least two of the following symptoms by age 15:

1. Truancy (positive if at least five days per year for at least two years, not including the last year of school).
2. Expulsion from school.
3. Delinquency (arrested or referred to juvenile court because of behavior).
4. Running away from home overnight at least twice while living in parental or parental surrogate home.
5. Persistent lying.
6. Unusually early or aggressive sexual behavior.
7. Unusually early drinking to excess, or substance abuse.
8. Thefts.
9. Vandalism.
10. Required to repeat school grades or grades markedly below what would be expected on basis of estimated or known IQ.
11. Chronic violations of rules at home and/or at school (other than truancy).

Robins (1966) compiled a more comprehensive list of childhood symptoms of her sample. Table 16 illustrates the number of patients manifesting particular childhood symptoms in the current sample. This list is based on description of patients by family and hospital staff.

(Symptoms are listed in order of most common occurrence.)

TABLE 16

CHILDHOOD ANTISOCIAL SYMPTOMS OF ASPD SAMPLE

Childhood antisocial symptoms	Number of patients
Stealing	21
Poor school work	18
Drug use	18
Fights/assaults	17
Truant	14
Expelled/suspended	10/2
Early sexual activity or promiscuity	8
Lying	8
Chronic violation of rules	8
Drinking	8
Violent temper/tantrums	7
Referred to juvenile court	7
Disruptive in school	6
Runaway	6
Drop out of school	5
"Out of control"/rebellious	5
Arson	4
Manipulative/"con artist"	4
Torturing animals	3
Bad crowd	3

TABLE 16, Continued

Childhood antisocial symptoms	Number of patients
Threaten parents	3
Selling drugs	3
Gambling	2
Cursing	2

This patient group also demonstrated childhood symptoms not necessarily antisocial in nature. Since the presence or absence of neurotic symptoms in antisocial children and adults is often a topic of debate, it is significant to note the presence of these symptoms in this sample (Table 17).

TABLE 17

NEUROTIC CHILDHOOD SYMPTOMS OF ASPD SAMPLE

Symptom	Number of cases
Stutters	2
Night terrors	2
Nightmares	2
Bedwetting	2
Hyperactive	2
Problems with peers	2
Enuretic	1

TABLE 17, Continued

Symptom	Number of cases
Dyslexic	1
Homosexual activity	1

While DSM III only requires two childhood antisocial symptoms, Robins' data indicated the presence of more symptoms and suggested that it was the number of symptoms, as opposed to the nature of the given symptom, that predicted adult antisocial outcome of childhood disorders. Table 18 illustrates the number of childhood symptoms described historically for this patient sample.

TABLE 18

TOTAL NUMBER OF CHILDHOOD ANTISOCIAL SYMPTOMS

Number of antisocial symptoms	Number of cases
2	3
3	4
4	3
5	7
6	9
7	4
8	3
9	3

Sixty-seven percent of this patient sample had childhood histories of five or more symptoms of antisocial behavior.

Poor early home environment and early losses are often blamed for the acting-out behaviors of children and adolescents. Not surprisingly, therefore, 61% of this sample came from "broken" homes in which one or more parent was absent or deceased. It may be more surprising to consider that the remaining 39% came from homes at least superficially intact, described as adequate by mental health personnel, and often producing other nondisturbed siblings. Table 19 illustrates more fully the early family losses of this sample group.

TABLE 19

EARLY LOSSES OF ASPD SAMPLE

	Number of cases
Overall	
Early loss or broken home	22
Stable home	14
Divorces	
Patient's age at parents' divorce:	
Under 5	7
5-10	4
10-15	6
Patient's age at loss of parent due to death or illness:	
Under 5	1

TABLE 19, Continued

	Number of cases
5-10	4
10-15	1

DSM III requires three adult antisocial behaviors to meet criteria for ASPD. Four of these adult behaviors are criminal arrests, thefts, illegal occupation, fights or assaults (whether or not detected) and three are "predisposed towards criminality": poor occupational performance (unemployment, job changes, absenteeism), defaulting on debts, and aimless traveling). Table 20 illustrates the adult antisocial behaviors meeting DSM III criteria manifested by the 36 ASPD patients.

TABLE 20

ADULT ANTISOCIAL BEHAVIORS

Behavior	Number of cases
Three or more nontraffic arrests	26
Illegal occupation (often drug-related)	23
Repeated thefts	21
Frequent job changes	20
Unemployment	9
Poor academic performance	9

TABLE 20, Continued

Behavior	Number of cases
Repeated fights	8
Aimless traveling	5
Defaulting on debts	4
Absenteeism at work	3
Two or more divorces	2
Felony conviction	1
(Not in DSM III) AWOL	1

Psychopathology of Family Members

The methodology of assessing the psychopathology of first-degree relatives (parents and full siblings) was developed by A. W. Loranger, Ph.D., in research on the families of schizophrenic and bipolar disorder patients (Loranger, 1981) and was used by E. Tulis, Ph.D. in research on the families of borderline patients (Tulis, 1979). The data on families of ASPD patients will be compared to the data on the other groups previously studied, and therefore the same methodology for assessing family pathology will be employed.

Information about first-degree relatives (natural parents and full siblings) was derived from the psychiatric case studies, social service reports, discharge summaries, and when possible, from clinical data or other corroborating information (i.e., letters from out-

side treating therapists). Ages and occupations of family members were noted, as was history of psychiatric treatment. Any other outstanding and relevant information was recorded, such as history of prison record or drug abuse, personality characteristics, time frame of illnesses and treatments, and past and present relationship with the patient.

On the basis of treatment history and other correlating material, the family member was categorized as: 1) normal: no history of psychiatric treatment and no obvious psychiatric disorder; 2) impaired: no history of treatment but evidence of psychiatric disorder (i.e., drug abuse or alcohol abuse, periods of depression or explosive outbursts); 3) outpatient treatment: current or past outpatient treatment; and 4) hospitalized: current or past hospitalizations for a psychiatric disorder.

The data was further evaluated to determine whether the relatives in categories 3 and 4 had ever been treated for bipolar disorder or schizophrenia. Only treated cases were used in comparisons with other groups. DSM III criteria as well as clinical evaluation of existing data were used in this evaluation. In addition, information about other treated relatives was examined for evidence of personality disorders or neurosis.

The sample size of relatives was corrected for age, based upon whether they had completed the period of risk for the particular illness. This assignment of weighted scores was on the basis of the Weinberg Shorter Proband Method, described in Slater and Cowie (1971)

and used by Tulis (1979) and Loranger (1981). Family members who had completed the period of risk were assigned a weight of 1. Those family members still within the period of risk were assigned a weight of 1/2, while those who had not entered the risk period were excluded. Relatives who had been treated for the illness or disorder in question were considered as having completed the risk period and were given a weighted score of 1. When an exact age was unavailable, it was estimated based upon chart data, and if the relative had died, the age at death was used. Therefore, a sample size corrected for age and risk was obtained for each illness.

Using Loranger's previous works (1978; Loranger & Levine, 1978) the period of risk for bipolar disorder is 15 to 60 years of age for both males and females. The period of risk for unipolar depression is 15 to 70 years of age. For schizophrenia, the period of risk is 15 to 50 for females and 15 to 40 for males. The risk period for neurosis and personality disorder was estimated to be between 15 and 40 years of age. The number of relatives treated divided by the adjusted sample size yielded the morbid risk for that illness.

Following Loranger (1981), Loranger and Levine (1978), and Tulis (1979), the morbid risk for the group under investigation was compared to the morbid risk or expected prevalence of that illness for the population at large. Based on Zerbin-Rudin's (1967) compilation of 20 studies, the morbid risk for schizophrenia in the general population is 1.17% (SE = 0.0187). The morbid risk for bipolar disorder is estimated at 1% in the general population combining data on bi-

polar and unipolar disorders, and at .3% for bipolar disorder only. Morbid risk for personality disorders in the general population is not available at this time.

The morbid risk of these illnesses in the relatives of ASPD patients was then compared to the data compiles by Loranger on the morbid risk for these illnesses in the immediate relatives of 100 hospitalized females and 200 hospitalized males and females diagnosed using DSM III as manic-depressive or bipolar (Loranger, 1978; Loranger & Levine, 1978), and the morbid risk of relatives of patients diagnosed as schizophrenic, and the morbid risk of relatives of borderline patients (Tulis, 1979). The statistic used was the standard error of the difference between two proportions, and tests of significance were based on the Z normal curve distribution.

Comparison data will also be collected on morbid risks for alcoholism. Family report of alcoholism was used as sufficient evidence for diagnosis, since documentation of treatment of alcoholism was often unavailable. Several studies have indicated that this family history method underreports cases of alcoholism when compared to other research methods of diagnosis (Andreasen, Endicott, Spitzer, & Winokur, 1977; Mendlewicz, Fleiss, Cataldo, & Rainer, 1975; Rimmer & Chambers,

1969; Thompson, Orraschel, Brusoff, & Kidd, 1982). It will be assumed that the morbid risk for alcoholism in this population will be slightly underestimated. Tsuang and Winokur's (1978) estimate of 6.7% of alcoholism in the general population will be used (12% males, 1.8% females). According to Winokur, Cadoret, Dorzab, and Baker's (1971) data, age of onset of alcoholism is between 10 and 49 for males, while for females the age range is slightly later. However, for purposes of comparison the same figures as were used by Tulis (1979) will be used in this study: 15 to 40 for males, and 15 to 50 for females.

Another focus of this study is to empirically compare the population of ASPD patients derived using DSM III criteria and the populations of sociopathic patients previously studied. In particular, these patients will be compared descriptively with Robins' (1966) subjects regarding age of onset of antisocial symptoms, number and kind of antisocial symptoms, and adult behavior.

Statistical Method

The statistical procedure utilized will be the difference between two proportions (the morbid risks for illness) as based upon sample data, and tests of significance will be based upon the *Z*, normal curve distribution.

CHAPTER IV

RESULTS

Families of ASPD Patients

The first-degree relatives of the 36 ASPD patients in this sample include 36 fathers, 36 mothers, and 65 full siblings (35 male siblings and 30 female siblings). Table 21 presents the makeup of the families of this sample.

TABLE 21

FAMILIES OF ASPD PATIENTS, INCLUDING AGE, SEX, ORDINAL POSITION OF SIBLINGS AND DEGREE OF IMPAIRMENT (I=INPATIENT, O=OUTPATIENT)

Case #	Patient and siblings	Father	Mother
1	M 27, M 29*, M 30	59	55
2	M 29*, F 17	53(0)	51
3	M 25, M 20*, M 15(0)	43	39(0)
4	M 30*	58	52(0)
5	M 18*, M 15	43	38
6	F 29, M 27*	60	51
7	F 29(I), M 21*, M 19(0)	63	52(0)
8	M 28, M 25*, F 17(I)	53(0)	49
9	M 18*, M 17	54	51(I)
10	M 24*, M ?	53	54
11	F 20, M 19(0)(D), M 18*	45	45

TABLE 21, Continued

Case #	Patient and siblings	Father	Mother
12	M 24(0), M 19*, M 11(0)	47	46
13	M 23*, F 22, M 18, M 13	48(0)	44
14	M 21*	40's(0)	40's(0)(D)
15	M 27*, M 21	5_(0)(D)	49
16	M 21, M 18*, F 18(1)(DZ twin)	54	51
17	M 29*, F 27, F 25	59	53
18	M 18*, M 15, M 11, F 7	45	41
19	F 19(0), M 18*, F 12	49(0)	49(0)
20	M 38, F 31*	66(D)	62(0)
21	M 23*, F 20	55	51
22	M 27* (other sibs adopted)	53	49
23	M 50's(0), F 42, F 40, M 38*, F 30	76	73(0)
24	F 18*	40's(0)	30's(D)
25	M 38(0), M 29*	52(0)(D)	62
26	M 21(0), M 19*, M 17, F 15, F 14(0)	46(0)	45(1)
27	F 25, M 22, M 19*	53	47
28	M 24, M 23*, M 14	49	44(0)
29	M 20*	40's	40's(0)
30	M 27*, M 23	54	52
31	M 22, M 19*, M 18, M 14, F 12, M 8	56(1)	43
32	F 31, F 29, M 28(0), M 26*, F 19	40(0)(D)	51
33	M 38, M 36*, F 32, M 21(1)	62(1)	late 50's(1)

TABLE 21, Continued

Case #	Patient and siblings	Father	Mother
34	F 23*, M 22(0), F 19, F 17(0), F 15, F 10, M 18	45(0)	39
35	M 23*, F 23(1), M 18	50	51
36	F 22, M 20*, F 20(DZ twin)	54	53

*index ASPD patient
0 = received outpatient psychiatric treatment
I = received inpatient psychiatric treatment
D = deceased

Of the 32 families with full siblings of the patient, 18 families (56%) had no behavioral or psychiatric problems in other children, while 14 families (44%) had other children with treated psychiatric difficulties. Two patients (both male) had female dizygous twins. Of these two, one twin had no history of psychiatric illness, while the other twin was hospitalized for drugs, promiscuity, and other acting-out behavior.

Psychiatric Illness in ASPD Relatives

Table 22 summarizes the number of first-degree relatives of the 36 ASPD patients, and the prevalence of psychiatric treatment. Thirty-three percent of these families had no immediate family member in psychiatric treatment other than the index patient.

Table 23 illustrates the psychiatric illnesses diagnosed and treated in immediate family members of the ASPD sample.

TABLE 22

INPATIENT AND OUTPATIENT TREATMENT FOR PSYCHIATRIC ILLNESS IN 137 ASPD RELATIVES

	Total N	In treatment total	Outpatient	Inpatient
Fathers	36	13	11	2
Mothers	36	11	9	3
Total parents	72	24	20	5
Brothers	35	11	10	1
Sisters	30	7	3	4
Total siblings	65	18	13	5
Total relatives	137	42	33	10

The most frequent diagnosis of ASPD parents was alcoholism (N = 19, 26%), followed by personality disorders (N = 9, 12%), and unipolar depression (N = 3, 4.2%). Schizophrenia and bipolar disorder each only represented one case (N = 0/1, 0/1%).

The suggestion has been made that women with ASPD came from more disturbed families than do men with the same diagnosis, possibly due to a higher threshold for expression of ASPD in females. There were too few women (N = 3) to provide significant findings but the direction of the findings is still of interest. In the first case, the

TABLE 23

TREATED PSYCHIATRIC ILLNESS IN ASPD RELATIVES

	Fathers (N=36)	Mothers (N=36)	Both Parents (N=72)	Male Sibs (N=35)	Female Sibs (N=30)	All Sibs (N=65)	All Rel (N=137)
Bipolar disorder	0	0/1?	1	0	0	0	1
Unipolar depression	2	1	3	0	0	0	3
Schizophrenia	0	0/1?	1	0	0	0	1
Personality disorder	7	2	9	7	3	10	19
Alcoholism/drugs*	11	8	19	10	2	12	31
Suicide	0	0	0	0	0	0	0

*Alcoholism and drug addiction were combined since several of the siblings had drug addiction problems.

Note. A slash score indicates a questionable case. The left hand figure is the more rigorous estimate; the right hand figure includes the questionable case.

father was treated for personality disorder, the mother was treated for alcoholism, and a brother had no psychiatric illness. In the second case, the father was both alcoholic and treated for a personality disorder, the mother died when the patient was very young and there were no siblings. In the third case, neither father, mother nor brother had a history of psychiatric illness.

In the overall sample, 25% of cases had both parents with no history of psychiatric disorder, 25% had one parent with a personality disorder, 41% had one alcoholic parent, and 11% had some psychiatric disturbance in both parents.

To meaningfully assess the risk of psychiatric illness in these relatives, the number of cases must be weighted to take into account the number of relatives still at risk for developing the illness in question. Table 24 indicates the size of the absolute sample, the weighted sample and the number of treated relatives for each psychiatric disorder studied. Because the age range of the risk period differs for each illness (and, occasionally, by gender), the weighted sample size is different for each illness considered.

Morbid risk for a given illness is calculated based upon the frequency of treated cases in the weighted sample. Table 25 demonstrates the morbid risk for psychiatric illnesses in the sample of 137 ASPD relatives.

TABLE 24

ABSOLUTE SAMPLE SIZE, WEIGHTED SAMPLE SIZE, AND NUMBER OF TREATED CASES AMONG ASPD RELATIVES

Bipolar disorder (age of risk: 15-60)			
	Fathers	Mothers	All parents
Absolute sample	36	36	72
Weighted sample	21	20.5	41.5
Treated cases	0	0(1?)	0(1?)
	Male siblings	Female siblings	All siblings
Absolute sample	35	30	65
Weighted sample	14.5	12.5	27
Treated cases	0	0	0
	All male relatives	All female relatives	All relatives
Absolute sample	71	66	137
Weighted sample	35.5	32.5	68.5
Treated cases	0	0(1?)	0(1?)
Unipolar depression (age of risk: 15-70)			
	Fathers	Mothers	All parents
Absolute sample	36	36	72
Weighted sample	19	18.5	37.5
Treated cases	2	1	3

TABLE 24, Continued

Unipolar depression (age of risk: 15-70)			
	Male siblings	Female siblings	All siblings
Absolute sample	35	30	65
Weighted sample	14.5	12.5	27
Treated cases	0	0	0
	All male relatives	All female relatives	All relatives
Absolute sample	71	66	137
Weighted sample	33.5	31.0	64.5
Treated cases	2	1	3
Schizophrenia (age of risk: females 15-50; males 15-40)			
	Fathers	Mothers	All parents
Absolute sample	36	36	72
Weighted sample	34.5	30.5	65
Treated cases	0	0(1?)	0(1?)
	Male siblings	Female siblings	All siblings
Absolute sample	35	30	65
Weighted sample	15	12.5	27.5
Treated cases	0	0	0
	All male relatives	All female relatives	All relatives
Absolute sample	71	66	137
Weighted sample	49.5	42.5	92.5
Treated cases	0	0(1?)	0(1?)

TABLE 24, Continued

Personality disorder (age of risk: 15-40)			
	Fathers	Mothers	All parents
Absolute sample	36	36	72
Weighted sample	36	34	70
Treated cases	7	2	9
	Male siblings	Female siblings	All siblings
Absolute sample	35	30	65
Weighted sample	20.5	14	34.5
Treated cases	7	3	10
	All male relatives	All female relatives	All relatives
Absolute sample	71	66	137
Weighted sample	56.5	48	104.5
Treated cases	14	5	19

Alcoholism/drugs (age of risk: females 15-50; males 15-40)			
	Fathers	Mothers	All parents
Absolute sample	36	36	72
Weighted sample	36	29	65
Treated cases	11	8	19
	Male siblings	Female siblings	All siblings
Absolute sample	35	30	65
Weighted sample	19.5	13.5	33.0
Treated cases	10	2	12

TABLE 24, Continued

Alcoholism/drugs (age of risk: females 15-50; males 15-40)			
	All male relatives	All female relatives	All relatives
Absolute sample	71	66	137
Weighted sample	55.5	42.5	98
Treated cases	21	10	31

Researchers had speculated concerning the relative amounts of pathology to be found in mothers and fathers. This study supported the hypothesis that fathers show more disturbance, with fathers having a 19% morbid risk of personality disorder compared with a 5% morbid risk in mothers. Seven of the fathers manifested marked antisocial traits (19%) compared with only one (3%) of the mothers. Both fathers and mothers were very likely to be alcoholic (30.5% and 27.6% respectively) which is notable considering the usual predominance of males in alcoholism.

Differences between parents and children (siblings of the ASPD patients) were also striking. Male and female siblings combined had a 29% risk of personality disorders compared to a 12.9% risk for parents. Combined siblings had a 36.4% risk of alcoholism compared to 29.2% for all parents. However, the sibling figures are even more remarkable looking at male-female differences. Male siblings manifested a 51.3% risk of personality disorder compared to 21.4% risk

TABLE 25

MORBID RISK FOR AFFECTIVE DISORDERS, SCHIZOPHRENIA,

PERSONALITY DISORDER AND ALCOHOLISM OF 137 ASPD RELATIVES

	risk		risk		risk
Bipolar Disorder					
Fathers	0	Mothers	0/.0465	All parents	0/.0241
Male siblings	0	Female siblings	0	All siblings	0
All male relatives	0	All female relatives	0/.0308	All relatives	0/.0146
Unipolar Depression					
Fathers	.153	Mothers	.054	All parents	.80
Male siblings	0	Female siblings	0	All siblings	0
All male relatives	.0597	All female relatives	.0323	All relatives	.0469
Schizophrenia					
Fathers	0	Mothers	0/.033	All parents	0/.015
Male siblings	0	Female siblings	0	All siblings	0
All male relatives	0	All female relatives	0/.023	All relatives	0/.011

TABLE 25, Continued

	risk		risk		risk
Personality Disorder					
Fathers	.194	Mothers	.059	All parents	.129
Male siblings	.513	Female siblings	.214	All siblings	.290
All male relatives	.248	All female relatives	.104	All relatives	.182
Alcoholism/Drugs					
Fathers	.305	Mothers	.276	All parents	.292
Male siblings	.557	Female siblings	.148	All siblings	.364
All male relatives	.378	All female relatives	.235	All relatives	.316

for female siblings, and male siblings had a 55.7% risk of alcoholism compared with only 14.8% risk for females. Overall, 24.8% of male relatives as compared with 10.4% of all female relatives manifested personality disorders, while 37.8% of all male relatives and 23.5% of all female relatives were alcoholic. The group most at risk were male siblings, which is supportive of the research suggesting that females have a higher threshold for expression of ASPD and alcoholism. The data also supports the theory that personality disorder and psychopathic spectrum symptomatology are found predominantly in males in these families.

Comparison of Morbid Risk Figures

Morbid risk figures for treated affective disorders and schizophrenia in 137 relatives of ASPD and in the relatives of patients with affective disorders, schizophrenia and borderline personality disorder are presented in Table 26. These figures compare the relatives of the 36 ASPD patients with the first-degree relatives of 83 DSM III borderlines, 100 DSM III schizophrenics, and 100 DSM III bipolar disorder patients studied by Loranger, Oldham and Tulis (1982).

TABLE 26

MORBID RISK OF TREATMENT FOR MAJOR PSYCHOTIC ILLNESS IN IMMEDIATE FAMILIES OF 36 ASPD CASES, 83 BORDERLINES, 100 SCHIZOPHRENICS AND 100 BIPOLARS

Disorder	Number at risk	Treated	Morbid risk (percentage)
Relatives of ASPD			
Bipolar	68.5	0/1?	0/1.46
Unipolar	64	3	4.69
Schizophrenia	92.5	0/1?	0/1.08
Relatives of Borderlines*			
Bipolar	184	1	.54
Unipolar	171.5	11	6.41
Schizophrenia	239	0	0
Relatives of Schizophrenics*			
Bipolar	276	1	.36
Unipolar	241	5	2.07
Schizophrenia	344	10	2.91
Relatives of Bipolars*			
Bipolar	343.5	8	2.33
Unipolar	299.5	22	7.35
Schizophrenia	393.5	1	.25

*Morbid risk data for relatives of DSM III borderline, DSM III scnizophrenic, and DSM III bipolar patients are based upon Loranger, Oldham & Tulis (1982) and is reprinted with their permission.

Hypothesis 1: Prevalence of Major Psychotic Illness

A. As hypothesized, the morbid risk of schizophrenia in ASPD relatives (0%/1.08%) (slash scores indicate possible case: conservative estimate/all possible cases) closely resembles the estimated risk of schizophrenia in the general population of 1.17%. The morbid risk of affective illness for ASPD relatives of 4.69/6.15% overall (0/1.46% bipolar disorder, 4.69% for unipolar depression) is apparently greater than the estimated risk of all affective illness in the general population of 1-2% (Loranger, Oldham & Tulis, 1982). The risk of bipolar disorder in ASPD relatives does not greatly exceed the predicted value of .3%. However, the risk of unipolar depression of 4.69% would appear substantially larger than the general population estimate of 1%. It is not possible, given the size of the ASPD sample, to meaningfully determine the significance of these comparisons with the general population. This is due to the great discrepancy in sample sizes and to the extremely small proportions being considered. However, the direction of the findings could be considered strongly suggestive and meriting further research.

B. The morbid risk of schizophrenia in ASPD relatives of 0/1.08% did not differ significantly from the risk of schizophrenia in the sampled relatives of schizophrenics (morbid risk 2.91%; $z = .996$, p less than .40) or in the sampled relatives of bipolar patients (morbid risk .25%; $z = 1.13$, p less than .30).

The morbid risk of bipolar disorder in ASPD relatives (0/1.46%) also did not significantly exceed the risk of bipolar disorder in the

sampled relatives of schizophrenics (morbid risk .36%; $\underline{z}$ = .50/1.07; $\underline{p}$ less than .70/.30) or in the sampled relatives of bipolar patients (morbid risk 2.33%; $\underline{z}$ = 1.28/.45, $\underline{p}$ less than .03/.70).

The morbid risk of unipolar depression in ASPD relatives (4.69) was descriptively larger but did not differ significantly from the risk of unipolar depression in relatives of schizophrenics (morbid risk 2.17%; $\underline{z}$ = 1.16, $\underline{p}$ less than .3), and was descriptively smaller but did not differ significantly from the risk of unipolar depression in the relatives of bipolar patients (morbid risk 7.35%; $\underline{z}$ = .76, $\underline{p}$ less than .50).

C. As hypothesized, the morbid risk of major psychoses in ASPD relatives did not differ significantly from the risk of major psychoses in borderline relatives. The morbid risk of schizophrenia in ASPD relatives of 0/1.08% was not significantly larger than the risk of 0% for borderline relatives ($\underline{z}$ = 1.6, $\underline{p}$ less than .20). The risk of bipolar disorder in ASPD relatives (0/1.46% was not significantly different than the risk of .54% in borderline relatives ($\underline{z}$ = .611/.731, $\underline{p}$ less than .60/.50).

Following a similar pattern, the morbid risk of unipolar depression in ASPD relatives (4.69%) and in borderline relatives (6.41%) also did not differ significantly ($\underline{z}$ = .499, $\underline{p}$ less than .20).

Table 27 demonstrates the morbid risk of personality disorders in ASPD relatives as compared with the morbid risk of personality disorders in relatives of DSM III schizophrenics, DSM III bipolar disorders and DSM III borderlines. Again, the comparative data is based

upon the work of Loranger, Oldham and Tulis (1982).

TABLE 27

MORBID RISK OF PERSONALITY DISORDER IN RELATIVES OF 36 DSM III ASPD, 83 DSM III BORDERLINES, 100 DSM III SCHIZOPHRENICS, AND 100 DSM III BIPOLARS

Relative group	Number at risk	Treated relatives	Morbid risk	z and p compared to ASPD relatives
ASPD	104.5	19	18.18%	--
Schizophrenics	353.5	5	1.41%	$z = 6.76$ $p = .001$
Bipolars	413.5	3	.73%	$z = 7.91$ $p = .001$
Borderline	249	29	11.65%	$z = 1.63\%$ $p < 0.20$

Hypothesis 2: Prevalence of Personality Disorder

A. The risk of personality disorders in ASPD relatives of 18.18% is significantly higher than the risk of personality disorders in relatives of schizophrenic patients, 1.41% ($z = 6.76$, $p < .001$) and in relatives of bipolar patients, .73%)$z = 7.91$, $p < .001$).

B. The risk of personality disorders in ASPD relatives, 18.18%, is not significantly greater than the risk of personality disorder in borderline relatives at 11.65% ($z = 1.636$). The trend is strongly in the direction of a significant difference, with ASPD relatives having a higher morbid risk of personality disorder.

Table 28 demonstrates the morbid risk for alcoholism in ASPD rel-

TABLE 28

MORBID RISK OF ALCOHOLISM IN RELATIVES OF 36 DSM III ASPD, 83 DSM III BORDERLINES, 100 DSM III SCHIZOPHRENICS AND 100 DSM III BIPOLARS

Relative group	Number at risk	Number treated	Morbid risk	z/p
ASPD	98	31	31.63%	--
Schizophrenics	650.5	32	4.92%	8.90/ < .001
Bipolars	785	35	4.46%	9.70/ < .001
Borderlines	141.5	33	23.32%	1.43/ns; p < .20

Note. Data from Tulis, 1979; reprinted with permission.

atives as compared with the risk for alcoholism in relatives of schizophrenic, bipolar and borderline patients. When the morbid risk figures are compared to the figure for ASPD relatives, z and p are given.

Table 29 further elaborates the risk of alcoholism in ASPD rela-

TABLE 29

RISK OF ALCOHOLISM IN ASPD RELATIVES BY GENDER

	Weighted n	Treated cases	Risk
Males only	55.5	21	37.8%
Females only	42.5	10	23.53%
Males and females	98	31	31.63%

tives by delineating male/female differences.

Hypothesis 3: Prevalence of Alcoholism

A. The morbid risk of alcoholism in ASPD relatives of 37.8% for males, 23.5% for females and 31.6% overall is much higher than the general population estimate of 9.5% for men, 4.0% for women, and 6.7% overall (Tsuang & Winokur, 1978).

B. The morbid risk of alcoholism in ASPD relatives is significantly greater than the risk of alcoholism in relatives of schizophrenics (morbid risk 4.92%, $z = 8.90$, $p < .001$) and bipolars (morbid risk 4.46%, $z = 9.70$, $p \ll .001$).

C. The morbid risk of alcoholism in ASPD relatives is not significantly greater than the risk of alcoholism in borderline relatives, although there is a trend in that direction.

CHAPTER V

CONCLUSIONS

Hypothesis 1: Prevalence of Major Psychotic Illness

It was hypothesized that ASPD relatives would be comparable to the general population in morbid risk for schizophrenia and affective illnesses. As was predicted, the rates of schizophrenia (0/1.08%) and bipolar disorder (1.46%) in ASPD relatives closely resembled those rates in the general population.

The comparative samples of relatives of schizophrenics, bipolar and borderline patients did not manifest rates of schizophrenia or borderline disorder greater than would be expected in the general population. These rates of schizophrenia and bipolar disorder did not differ significantly between ASPD relatives and schizophrenic, bipolar and borderline relatives.

The data on unipolar depression is more difficult to evaluate. The risk of unipolar depression in ASPD relatives (4.69%) was descriptively higher than in the general population. The rate of treated depression in ASPD relatives was also higher than in the relatives of schizophrenic patients (2.07%). This difference was not found to be statistically significant. The difference in the rate of treated depression between bipolar and borderline relatives and ASPD relatives was also not found to be statistically significant. However, this morbid risk of unipolar depression in bipolar

and borderline relatives (7.35% and 6.41% respectively) was found to be significantly greater than in the general population (Loranger, Oldham & Tulis, 1982). This is suggestive that there could also be a trend for a higher risk in ASPD relatives as well.

Hypothesis 2: Prevalence of Personality Disorders

It was hypothesized that ASPD relatives would be at significantly greater risk for personality disorders than were relatives of schizophrenic or bipolar patients. This was clearly demonstrated with high levels of significance. ASPD relatives were 13 times as likely to have personality disorders as were relatives of schizophrenic patients and they were 25 times as likely to have treated personality disorders as did relatives of bipolar patients.

It was also hypothesized that ASPD relatives would not differ significantly from borderline relatives in the morbid risk of personality disorders. While the difference was not statistically significant in a rigorous statistical test, the z value of 1.636 was very close to the value necessary for significance of 1.64. This is strongly suggestive of a tendency for ASPD relatives to have more personality disorders than do borderline relatives.

Both ASPD relatives and borderline relatives were at significantly higher risk for treated personality disorders than were the relatives of psychotic patients; they therefore more closely resembled each other than they resembled the other groups.

Hypothesis 3: Risk of Alcoholism

It was hypothesized that ASPD relatives would have a significantly higher rate of alcoholism than is found in the general population or in the relatives of psychotic patients. While a test of significance of the comparison to the general population would not be meaningful due to the variation in sample size, the risk of alcoholism in ASPD relatives greatly exceeds the risk of alcoholism in the general population; it is four times higher than the predicted rate for men, almost six times higher than the predicted rate for women, and almost five times higher than predicted rate overall. The rate of alcoholism in ASPD relatives was greater than the alcoholism rates in relatives of schizophrenic and bipolar patients with a high level of significance.

It was also hypothesized that ASPD relatives would have a rate of alcoholism that was not significantly different from the rate of alcoholism of borderline relatives. Although the rate of alcoholism in ASPD relatives was descriptively higher, this difference was not shown to be statistically significant.

Summary

The data supports the hypothesis that ASPD relatives more closely resemble borderline relatives in terms of morbid risk of major psychosis, personality disorder and alcoholism than do they resemble relatives of patients with major psychoses or the general population on these dimensions. This is consistent with the delineation of DSM III personality disorders as distinct from other categories.

CHAPTER VI

DISCUSSION

DSM III ASPD Diagnosis

This study explored the viability of the DSM III diagnosis of ASPD as a research tool, and determined whether a sample of ASPD patients meeting DSM III criteria resembled previously researched groups of "psychopaths," "sociopaths," etc.

Clinically, this current sample of ASPD patients often closely resembled Cleckley's (1941) classic description: they lacked obvious delusions, thought disorder or anxiety; they were irresponsible and exploitative; they were able to perform antisocial behaviors without compunction; they manifested no coherent life plan; and they were often violent under the influence of alcohol or drugs. Despite all of this, these patients appeared to be able to elicit support and repeated efforts at treatment, notwithstanding all evidence that they had lied, manipulated and abused previous chances at help.

The questions raised regarding diagnosis of the current group had two sources: DSM III had no provision for evaluating interpersonal functioning, and the current sample was much more frequently involved with drugs than were previous groups. Such drug use between 1969 and 1980 usually predicted other behaviors considered antisocial (thefts, drug sales and arrests) so that drug users often met DSM III criteria for ASPD adult behaviors without any nondrug-related antisocial behavior. This deficit is usually compensated for by the requirement

of a childhood history of antisocial behaviors, but the ASPD diagnosis of a few patients remained questionable vis-a-vis their supposed lack of capacity for empathy and intimate relationships. Also, there were several cases that had to be rejected from the sample due to their lack of criminal involvements. The patient's management of daily life and relationships made it clear that these persons were candidates for ASPD who simply got other people to do their dirty work for them and suffer the consequences. These "successful" sociopaths lacked the ego deficits necessary to meet DSM III criteria, but they plainly manifested major superego deficits.

Adult antisocial behaviors of the current sample resembled Robin's (1966) group. Many subjects had poor work histories and arrest records. The current group was more involved in drugs (use and sales) and less involved in sexual acting out than was Robin's sample.

Based upon childhood histories, the current sample and past groups had many similarities. Stealing, truancy, fighting and incorrigibility were salient features of these children then and now. In the current sample, there were fewer runaways and more children using drugs and alcohol. Age of onset was earlier in Robin's data; in her sample 58% of boys and 9% of girls manifested antisocial symptoms by age 8, and 83% of boys and 36% of girls showed antisocial behaviors by age 10. In the current sample, only 42% were involved in antisocial behavior by age 11, and 84% by age 13. It is notable, however, that in the social and political climate of the 1970's many children were rebellious towards authorities without being defined as antisocial until

such behavior escalated significantly.

The DSM III criteria did produce a sample clinically distinguishable from the DSM III borderline group. While ASPD patients were frequently impulsive and unpredictable in self-damaging ways as is characteristic of borderlines, there was little or no evidence of identity disturbance, affective instability, problems being alone or reported chronic feelings of emptiness. Self-damaging acts were rare in this sample, and these acts were obviously manipulative as opposed to depressive in nature. In clinical descriptions of hospital course, borderline patients seemed more responsive to inner affect storms while ASPD patients responded more to environmental influences.

In summary, DSM III ASPD criteria do appear to yield a meaningful group distinguishable from DSM III borderlines and comparable to past groups of antisocial cases. The criteria appeared overinclusive in cases involving drugs and underinclusive in cases where the criminal system was sidestepped.

This raises Grinspoon and Bakalar's (1978) criticism of the diagnosis of ASPD that it confuses a legal with a psychological judgement. They cautioned of the need to distinguish dyssocial behavior (violating social mores but maintaining a subgroup's values) and antisocial behavior. Concerns over legal culpability (are these people "sick" or to be held responsible?) obscure more psychological concerns regarding the extent of ego and superego weaknesses. By ignoring the patient's capacity for loyalty, guilt and anticipatory anxiety, DSM III does fall into the trap of only using criminal or nearly criminal

behavior as its adult criteria, thus seemingly making the subset of criminals and the subset of ASPD cases almost identical.

Physiological research (Hare, 1970) as well as past theoreticians (Cleckley, 1941; Karpman, 1955) have supported the concept of primary and secondary sociopaths. Secondary sociopaths manifest the impulsivity and inability to delay gratification, antisocial behaviors and poor judgements, without necessarily demonstrating the lack of capacity for relationships, loyalty and guilt thought characteristic of "psychopaths." Such patients might include Freud's (1914) and Alexander's (1930) "criminals out of a sense of guilt," as well as dyssocial individuals and primitive oral characters whose drug use, for example, predicated an antisocial life style. Primary sociopaths or aneopaths (Reid, 1978) manifest the cold ruthlessness, total lack of relatedness and total lack of usual conscience. A valid question would be this: Are ASPD patients simply primitive characters who get in trouble with the legal system, or is severe superego pathology necessary, as manifested in interpersonal functioning, whether or not ego functioning is equally primitive?

Valliant's (1975) criticism and rejection of the concept of the ASPD diagnosis would be subsumed by this line of criticism. He studied hospitalized drug addicts, and found that if acting out was limited via external controls, other personality disorders such as hysteria and depression as well as concealed anxiety became evident. This is an example of a case in which patients with primitive personality disorders became involved in an antisocial life style and were considered

to be ASPD patients, but in which the ASPD diagnosis in terms of their psychodynamics (as opposed to their lifestyles) would be questionable.

Personality Diagnoses and ASPD

The kinds of pathology and rates of pathology in ASPD relatives was not significantly different than those found in the families of borderline patients, suggesting some ground of commonality between these two groups. In addition, these two groups were clinically distinguishable from each other, although obviously there will be borderline patients with significant antisocial traits and ASPD patients with identity pathology like borderlines. This raises the question of the continuity or discontinuity of categories within the domain of personality disorders, especially if further research supports the distinctions separating personality disorders from other diagnostic groups.

Antisocial traits are not the exclusive domain of ASPD. Antisocial traits are found in "neurotics," personality disorders and psychotics. In neurotics, such traits are thought to represent the acting out of guilt and the wish to be punished. In psychotics, such behaviors are reflective of a loss of self-object distinctions in reality testing. Considering the presence of antisocial traits in personality disorder, it is intuitively evident that several personality disorders might also manifest antisocial behaviors. The self-damaging acts and impulsiveness of borderlines frequently can take antisocial forms, and the relationship between borderline personality disorder and ASPD has been considered.

Another personality disorder meriting consideration would be narcissistic personality disorder. Domash and Balter (1979) point out that narcissists can manifest superficiality, ruthlessness, manipulativeness and the dependence on external resources characteristic of antisocial characters. They speculate upon the psychodynamics of narcissistic patients who repair narcissistic injury by "putting one over," "screw or be screwed." Such patients use antisocial acts as a restitution of narcissitic injury by reasserting omnipotence and the supremacy of the grandiose self over the disappointing object, thus warding off disappointment and feelings of emptiness. Domash and Balter distinguish such patients from ASPD patients by seeing these behaviors as focused upon wants and not superego deficits. Yet it would seem that the dynamics described might be useful in thinking of ASPD patients as well. In an extreme case, a narcissistic patient may experience the good object as so depleted as to be ruined, therefore unavailable as a source of supply and self-stability. This is reminiscent of the suggestions by learning theorists that ASPD patients cannot profit from negative or aversive experience. This learning deficit might be equally seen as an inability to profit from positive experience--that the good object is ruined and cannot be taken in as a good part of the self, so it is not a meaningful influence upon behavior. ASPD patients might represent an extreme case in which the balance of self and good object is never sustaining and therefore constant vulnerability and acting out in restitution become egc syntonic.

The issue of depressive affect in ASPD patients is difficult, since there is no evidence that ASPD patients described themselves as depressed. Reid (1978) describes them as "empty," and their drive to stimulation as an effc t to ward off emptiness. Blackburn (1978) and Hare (1970) describe physiological evidence that ASPD patients require stimulation to increase reticular activity in order to achieve optimum affect and behavior. It is unclear from a phenomenological view how this lack of optimum affect is experienced consciously or unconsciously by these patients, and whether there is any resemblance to the affects of disappointment and emptiness being warded off by narcissists. There may be some physiological substrate of affect related to feeling "good enough," stable or unstable. Winokur's (1973) data suggested that depressed parents of depressed children had daughters who were depressed and sons who were alcoholic or sociopathic. It is conceivable that some physiological substrate of depression manifests itself differently in ASPD patients: whether this would be a genotypic difference or a phenotypic difference would be a matter for further research. In this current sample, there was evidence of some elevation of the rate of unipolar depression and In the high rate of alcoholism in the parent group, with high rates of personality disorders and alcoholism in the patients' male siblings.

These past paragraphs have considered the possibility that the psychodynamics described in narcissistic patients might well be relevant to ASPD patients as well. Where then, lies the distinction between narcissism and ASPD? In answer, DSM III and most theorists

point toward the antisocial behaviors indicative of major superego pathology. In DSM III specifically, this takes the form of criteria including arrests, illegal occupation, defaulting on debts, or thefts. Yet it would seem that the most meaningful aspect of severe superego pathology would be on an interpersonal level--the inability to experience guilt and the related inability to identify, empathize, and form meaningful relationships. Domash and Balter (1979) suggest that narcissists maintain a symbiotic relationship with objects: an object is "needed" in a primitive sense, and the self joins with this object to feel omnipotent, or at least good. Even such a primitive object might be mourned in response to loss. The physiological and learning theory data that ASPD patients cannot internalize controls via negative (or positive) conditioning might suggest that an early substrate of basic identifications as well as of later superego formation might be damaged. Therefore, it would be in terms of capacity for and quality of relationships that ASPD might most reliably be distinguished on a psychodynamic level.

Recommendations for Further Research

Frances (1980) has suggested a dimensional rather than a categorical approach to personality disorders, and this might be the most salient way of further refining the understanding of ASPD and other personality disorder diagnoses vis-a-vis each other. Such a dimensional analysis of traits might separate antisocial groups corresponding to the primary-secondary distinction, and might also clarify the distinctions in constellation of traits characteristic of narcissistic

and antisocial personality disorders. Clearly, further research on personality disorders as a group would be of great interest in refining the DSM III diagnostic system.

In addition, an important direction for future research would be to collect data on the patterns of psychiatric illness in the families of patients with other personality disorders. DSM III groups personality disorders into three clusters: borderline, antisocial, histrionic and narcissistic; paranoid, schizoid, and schizotypal; and avoidant, dependent, compulsive, and passive-aggressive. The data from this and preceding studies (Loranger, Oldham, & Tulis, 1982; Tulis, 1979), suggest certain commonality in patterns of illness between borderline and ASPD relatives, and some indication of increased unipolar depression in these families. However, further research would indicate whether other personality disorders, particularly those in the paranoid, schizoid and schizotypal cluster, follow this pattern, or whether they relate more closely to schizophrenic spectrum illness.

This kind of research would help clarify whether the current system of classifying schizophrenia, affective disorders and personality disorders more closely reflect research data than a system similar to that suggested by Stone (1980), classifying, for example, different personality disorders within schizophrenic spectrum disease or affective spectrum disease. Such distinctions would elaborate our understandings of these disorders and alert us to important clinical considerations in the families of such patients.

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