Disinhibitory Psychopathology:
A New Perspective and a Model for Research

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The syndrome produced by lesion of the septum in animals can serve as a functional research model of human disinhibitory psychopathology. Disinhibitory psychopathology appears to span several traditionally separate psychological categories—psychopathy, hysteria, hyperactivity, antisocial and impulsive personality, and alcoholism. It is proposed that these categories are separate manifestations of the same genetic diathesis and that the "septal syndrome" may constitute a valid model of behavioral aspects of this diathesis. A program of experimentation utilizing this animal model is outlined.

The quest for a physiological explanation of syndromes of disinhibition or dyscontrol, especially psychopathy, is a current preoccupation among theoreticians of impulsive behavior (see Mawson & Mawson, 1977; Syndulko, 1978). One line of speculation in particular has focused on the limbic system as a possible site of central nervous system (CNS) dysfunction responsible for behavioral disinhibition (Gray, 1972; Hare, 1970). Evidence bearing on this hypothesis, however, is rather indirect, consisting mainly of psychophysiological anomalies open to a variety of interpretations. Among findings enumerated as possibly implicating limbic dysfunction are the electroencephalogram abnormalities discovered in psychopaths and impulsive children (Arthurs & Cahoon, 1964; Ehrlich & Keough, 1956; Knott, Platt, Ashby, & Gottlieb, 1953), including temporal lobe slow-wave activity (Bay-Rakal, 1965; Hill, 1952). Also cited are the abnormal positive spikes localized in the temporal lobe that have been observed in human syndromes of extreme violence, aggression, and impulsiveness (Kurland, Yeager, & Arthur, 1963). In addition to such psychophysiological evidence, reference has been made to the disinhibitory effects of medial-septal lesions in animals to support a limbic hypothesis (Hare, 1970). Gray (1972) in particular has drawn on the septal and hippocampal lesion data in developing a novel physiological interpretation of Eysenck's (1957, 1967) concept of "extraversion."

The recent attention to animal lesion data in interpreting human disinhibition has added a fresh dimension to theoretical development. Thus far, such data have been considered solely in the context of physiology, with an eye toward identifying neural structures or pathways that regulate impulsive behavior. Although it is an important finding that physical lesions of sites in the brain can produce syndromes of impulsive behavior, we propose that the behavioral effects rather than the anatomical loci of such lesions are the phenomena most relevant to advancing our understanding of disinhibition in humans.

Citing dysfunctional anatomical loci to account for human clinical syndromes is unsatisfactory for two reasons. First, given our present level of psychophysiological technology, anatomical hypotheses offer little prospect of advancing beyond the stage of primitive conjecture. This is because their confirmation ultimately depends on direct physiological evidence in humans, which re-
mains unavailable. Second, theories that simply invoke anatomical loci to account for behavioral phenomena merely provide a dry description of a chain of physical events and do not qualify as scientific explanations. Although it is granted that all behavior can ultimately be traced to physical causes, behavioral phenomena cannot adequately be explained by simple reference to a locus in the CNS because brain structures do not operate in a vacuum. Behavioral phenomena are the result of the interaction of anatomical structures with the environment. Consequently, dysfunctional anatomical loci must be understood in terms of their perceptual and motivational consequences (i.e., interaction with the environment) if a complete account of the resultant behavior is to be furnished.

Animals with limbic lesions are of interest to the study of human disinhibition not because of the site of their lesions but because of the perceptual, motivational, and other psychological characteristics entailed by limbic dysfunction that produce an impulsive animal. In this article we attempt to integrate some of the experimental evidence pertaining to syndromes of human disinhibition and propose that the "septal syndrome," considered as an analogical model, has potential for shedding new light on psychological precur- sors of disinhibited behavior in humans. In this regard, we shall focus on the perceptual, motivational, learning, and behavioral parallels between syndromes of disinhibition in humans and the syndrome produced by a lesion of the septum in animals. We intend to show that there is a valid behavioral analogy between the human and animal syndromes, but in suggesting such an analogy, we do not imply that human disinhibition can be traced to septal dysfunction. Rather, we suggest that behavioral analysis of the septal syndrome can elucidate basic psychological components of human disinhibition.

Human Disinhibition

As a theoretical concept, disinhibition has its origins in the Pavlovian tradition and has acquired a precise meaning in that context. Pavlov defined disinhibition as the process whereby an extraneous stimulus presented during extinction serves to increase the strength of a conditioned response (CR) on that trial (Mackintosh, 1974). The term disinhibition was intended to connote the disruption of active inhibitory processes regulating tendencies to respond. In the context of this article, the term disinhibition is used in another, primarily descriptive sense. It refers to human behavior that has been interpreted as arising from lessened controls on response inclinations. Among the behavioral syndromes characterized primarily by disinhibition are the personality construct of "impulsiveness" (Kipnis, 1971), psychopathy (Hare, 1970), antisocial behavior in adolescents, and, quite likely, hyperactivity in children.

Although the concept of disinhibition as it is used here is necessarily vague and merely descriptive, there is a common theme linking the syndromes to which it refers. The syndromes are usually characterized by patterns of behavior that succeed in obtaining immediate gratification at the expense of long-term and more enduring gains. Disinhibited individuals appear unable to control their immediate response inclinations as a means of achieving long-range goals. Similarly, they appear unable to control such inclinations as a means of avoiding long-range discomfort. They maintain this pattern of behavior, though recognizing verbally the overriding importance of its long-range detrimental effects. In short, immediate contingencies of reinforcement appear to outweigh the more global picture in determining behavior.

The prototypical syndrome of disinhibition is probably psychopathy, since it appears to be the most extreme form. Consequently, psychopathy serves as a useful model for consideration of the general concept. As Cleckley (1976) has described the disorder, psychopathy involves a pattern of behavior that the individual does not find intrinsically unpleasant or abhorrent but which, by its nature, entails many negative consequences that the individual would prefer to avoid. Much of the behavior of psychopaths seems traceable to their disinclination to anticipate the aversive consequences of their activities or of events in general. Their actions, whether heinous or benign, are committed with total disregard for
any suffering or hardship that may arise for themselves or others as a result. Consequently, their behavior is often antisocial and amoral, and although they may offer convincing protestations of repentance, it is likely that they will sustain their pattern of antisocial or criminal behavior in the future. They adhere to no moral code, and their behavior appears to be regulated only by immediate gratification of their transient desires. Psychopaths are notable for an absence of neurotic anxiety or any normal anxiety at all for that matter. In fact, being unconcerned with aversive events of no immediate consequence, they are often poised and assured in situations that might cause anxiety or panic in normal individuals.

Psychological experimentation with psychopaths has tended to confirm Cleckley's clinical observations. Among the earliest of such experiments was that of Lykken (1957), who found that psychopaths were indeed deficient in avoiding the aversive consequences of their behavior. Three groups of subjects, psychopathic criminals, neurotic criminals, and noncriminals, performed a task described as a "mental maze." The task involved a series of 20 choice points; at each choice point the subject chose to press one of four levers. Only one lever was the correct one at a given choice point, and pressing any of the other three levers constituted an error. Thus, the manifest task was to learn the maze while committing as few errors as possible. Subjects were given 20 trials to do so. There was also a latent task, however, not explained fully to subjects. At each choice point, pressing one of the three incorrect levers resulted in an electric shock to the subject, and this lever was always the same for a particular choice point. This feature of the task was intended to test subjects' tendency to avoid those particular errors that entailed painful consequences. The results of the experiment indicated no differences among the groups of subjects in ability to learn the manifest task. Regarding the latent task, on the other hand, psychopaths exhibited no improvement in the ratio of shocked errors to unshocked errors as the experiment progressed. By contrast, neurotic criminals and noncriminals gradually reduced their proportion of shocked errors. These findings, since replicated (Schachter & Latané, 1964; Schmauck, 1970), support the hypothesis that one of the features of syndromes of disinhibition is a disinclination to alter one's behavior to avoid discomfort.

Psychophysiological studies by Hare have suggested a mechanism to account for psychopath's poor avoidance. In one such study (Hare, 1965c), psychopathic criminals, neurotic criminals, and noncriminals observed the numbers 1–12 which appeared through the window of a memory drum for six trials. Beginning with Trial 2, subjects were informed that a shock would be delivered each time the number 8 appeared. Subjects' skin conductance (SC) was monitored throughout the experiment, and it was found that psychopath's rise in SC did not occur until the shocked number 8 was imminent. For the other two groups, the rise in SC began considerably earlier in the number sequence. These results suggest that psychopaths do not anticipate a noxious event when it is temporally remote; only cues with close temporal association with pain appear to elicit fear. As Hare (1970, p. 82) described the finding, the psychopath appears to have a "steep temporal gradient of fear arousal."

The psychopath's diminished anticipation of aversive events appears to be part of a deficit in classical conditioning. Early temporal cues, as conditioned stimuli, fail to elicit an emotional response even though such cues are reliable predictors of a subsequent aversive event. As further evidence of a classical conditioning deficit, Hare (1965a) found that psychopaths were slower than nonpsychopaths to develop a reliable SC response to a tone that terminated in shock. Moreover, when conditioning did eventually take place, psychopaths showed significantly less generalization of SC response to other tones. These deficits in classical conditioning are observed in spite of the fact that psychopaths' SC response to painful shock is no different in magnitude from the response exhibited by normal individuals (Hare, 1965a, 1965c).

In view of these deficits in classical conditioning, Mowrer's (1947) two-factor theory of avoidance can serve as the basis for a tidy
account of psychopaths’ tendency not to avoid aversive events (Hare, 1970). According to two-factor avoidance theory (Mowrer, 1947), kinesthetic and other cues associated with a punished act constitute conditioned stimuli signaling an upcoming noxious event and acquire the capacity to elicit fear. Consequently, responses that are instrumental in reducing such conditioned fear, namely inhibition of the punished act, are reinforced. The individual succeeds in avoiding aversive consequences by inhibiting the punished act (i.e., reducing fear) prior to its termination in punishment. Psychopaths, who do not readily acquire conditioned fear or whose fear arousal does not occur until late in the response–punishment sequence, are not reinforced for inhibition of a punished act because there is no conditioned fear to provide the basis for such reinforcement. The ultimate result is that psychopaths fail to avoid the aversive consequences of their behavior.

Mednick and Hutchings (1978) have also invoked two-factor avoidance theory to account for psychopaths’ avoidance deficit. They, however, have cited the slow SC recovery rate of psychopathic subjects rather than their low anticipatory SC as the basis for poor avoidance. These authors contend that a slow recovery of SC in psychopaths represents delayed reduction of anxiety and, therefore, insufficient reinforcement for avoiding punishment.

An account of the avoidance deficit in terms of two-factor theory is appealing both for its simplicity and its utilization of relatively well-defined concepts such as classical and instrumental conditioning. There have been other explanations of the deficit, however, most notably that of Quay (1965), who postulated that psychopaths fail to avoid because they actually seek the stimulation inherent in acts of transgression. According to this view, psychophysiological findings of underarousal in psychopaths (Hare, 1965a, 1965b, 1968) are indicative of an aversive state that the affected individual strives to remedy by engaging in acts of thrill seeking, thereby increasing arousal. There is, in fact, convincing experimental evidence that individuals who exhibit behavioral disinhibition of a sort, including psychopaths, antisocial preadolescents, and hyperactive children, do seek sensory stimulation to a greater extent than normals or neurotics (Skrzypek, 1969; Whitehill, Demyer-Gapin, & Scott, 1976; Zentall, 1975).

Thus, psychopaths’ low level of arousal and their deficit in classical conditioning may represent something larger than the mere underpinnings of an avoidance deficit.

What could possibly be involved? Unfortunately, progress in experimentation on psychopathy and other syndromes of disinhibition has not been so substantial that a coherent theory can be constructed. There have been many provocative findings, and they appear to follow a common theme, but they do not readily admit of conceptual integration.

Consider, for example, the time-honored clinical observation and experimental evidence to the effect that psychopaths and antisocial adolescents are unlikely to forego immediate gratification as a means of obtaining more attractive rewards later on. Given a choice, both antisocial adolescents and adult psychopaths are more likely than normals to accept a smaller reward immediately than wait for a larger one (Mischel, 1961; Unikel & Blanchard, 1973). Widom (1978) also reported preliminary evidence indicating that even among psychopathic subjects, those who fail to delay gratification tend to have higher scores on the Pd scale of the Minnesota Multiphasic Personality Inventory. This phenomenon in syndromes of disinhibition may be part and parcel of the same deficit that produces in psychopaths a tendency not to avoid shock. Both the disinclination to avoid shock and the disinclination to delay gratification constitute response styles that fail to take into account future consequences of present behavior. In the case of a deficit in shock avoidance, the individual is unlikely to alter his or her current behavior as a means of forestalling or eliminating painful consequences in the future; with respect to failure to delay gratification, the individual is unlikely to restrain his or her immediate response inclination to accept a tempting reward as a means of avoiding the loss of a more valuable reward later on.

This comparison notwithstanding, an ac-
count of disinclination to delay gratification utilizing two-factor theory of avoidance in conjunction with Hare's psychophysiological findings is difficult to justify at present. One would have to postulate that the steep temporal gradient of arousal that psychopaths exhibit in response to punishment by shock applies equally to punishment by loss of reinforcement.

In this regard, Schmauk (1970) has presented data indicating that not only do psychopaths show normal autonomic anticipation of loss of monetary reinforcement, but moreover, they avoid such loss as successfully as normals. The experimental task used by Schmauk was the Lykken maze, however, with loss of money substituting for shock as punishment for certain errors; consequently, the task differed from a delay of gratification paradigm where there is immediate incentive to abandon future prospects of reward and, furthermore, where all reinforcement obtained represents profit rather than the absence of loss. The finding that psychopaths succeed in avoiding loss of monetary reinforcement in an experimental situation that is patently an avoidance paradigm, but nevertheless fail to delay gratification, would indicate that their failure to delay gratification cannot properly be viewed as an avoidance deficit.

The notion that psychopathic behavior is not entirely derived from a low aptitude for avoidance is further supported by findings to the effect that syndromes of disinhibition are characterized by anomalies in time perception. Antisocial delinquents, for example, appear to have a restricted future time perspective, and their performance on time estimation tasks suggests that time passes more slowly for them than normals (Siegman, 1961). An important experiment by Painting (1961) suggests that psychopaths' deficit in avoiding noxious events as well as their disinclination to delay gratification may, in fact, be part of a general deficit in perceiving the connection between even neutral events that are related remotely in time.

In this experiment, primary psychopaths, neurotic psychopaths, and controls performed a task consisting of choosing to press one of two levers per trial. In one condition, the correct response on a trial was dependent on the response the subject had made on the immediately previous trial; in another condition, the correct response was dependent on the response the subject had made two trials previously; in a third condition, there was no relation between the correct response on a given trial and what the subject had done previously. Half of the subjects performed the task under contingencies of positive reinforcement, and half performed under contingencies of negative reinforcement; the former involved a gain of cigarettes for a correct response and the latter involved a loss for an incorrect response. Although there was essentially no difference between neurotic psychopaths and controls in any of the conditions, and virtually no effect of contingencies of positive versus negative reinforcement, primary psychopaths differed from other subjects in an important respect. When information concerning the correct response on a trial was temporally remote, that is, contained two trials back, primary psychopaths performed significantly worse than the other two groups of subjects. By contrast, when information concerning the correct response was contained in the immediately previous trial, primary psychopaths performed as well as the others and, in fact, somewhat better with positive reinforcement. Primary psychopaths were also noteworthy for a rigid response style in the independent trials condition; they tended to favor the response that had been correct on the previous trial.

The fact that psychopaths' performance deteriorates when information signaling the correct response is historically remote implies a deficit in perceiving the connection between events as the interval between them increases. This hypothesis is further supported by an experiment demonstrating that psychopaths' performance on a paired-associate task deteriorates relative to normals' as the delay between the subjects' response and presentation of the correct associate is increased (Gullick, Sutker, & Adams, 1976). Whether such time-related deficits form the basis of both disinclination to delay gratification and disinclination to avoid aversive events is an open question that invites further exploration.
Descriptively, then, human disinhibition, particularly psychopathy, presents mixed experimental results, but not so mixed that seemingly plausible speculation as to the theoretical connections cannot abound. In the next section, we elaborate a new and integrative perspective on human disinhibition that incorporates aspects of the construct usually considered in isolation.

Syndromes of Disinhibition: An Integrative Perspective

It is generally assumed that progress in the understanding of human disinhibition will be achieved as competing hypotheses are sorted out in empirical investigation. However, most psychological experimentation in psychopathy has adhered to an implicit concept of the primary research question, and this may be hampering theoretical development. Rather than exploring the totality of the construct, research has focused on a limited, though admittedly salient aspect, the phenomena of moral, social, and legal transgression. This trend is most evident in the extensive investigation of psychopaths' unresponsiveness, both behavioral and autonomic, to punishment, and it is undoubtedly true that this aspect of disinhibition has undergone the most advanced theoretical development to date. This topic, moreover, continues to be the subject of new experimentation and interpretation (see Hare, 1978). Perhaps as a consequence of the intriguing advancements in this area, other aspects of psychopathy have been relatively neglected, particularly those aspects in which unresponsiveness to aversive sanctions is not an apparent issue. Among such aspects are several of the clinical indicators suggested by Cleckley (1976): the failure to follow any life plan, involving inability to delay gratification; fantastic reaction to alcohol; lack of insight; poor judgment; unreliability; general poverty in major affective reactions; and unresponsiveness in interpersonal relations. The scattered experimental findings that psychopaths exhibit learning deficits in the absence of punishment (e.g., Gullick et al., 1976; Painting, 1961) also suggest important aspects of the construct that have not been significant elements in current theorizing.

In addition to the behavioral aspects of psychopathy just alluded to, current concepts of disinhibition have failed to incorporate into a coherent research model the accumulating evidence that there is a genetic component to psychopathy and related syndromes. Schulsinger (1972), investigating psychopathy in the biological and adoptive relatives of psychopathic and control adoptees, found the highest prevalence of psychopathy among the biological relatives of psychopathic probands.

Not only does there appear to be a genetic diathesis underlying psychopathy, but there is evidence that this same diathesis plays a role in the development of other disorders. For example, Cloninger, Reich, and Guze (1975) present evidence that hysteria and psychopathy are separate manifestations of the same etiological process. Also, hyperactivity in children appears to bear a fundamental relationship to psychopathy. Although hyperactivity was long assumed to be a matter of excess motor output, careful analysis has suggested that the term hyperactivity is really a misnomer (Whalen & Henker, 1976). Children's hyperactivity appears to be situation specific; accurately characterized, the syndrome consists, in part, of an inability to sustain attention as well as impulsivity on structured tasks. Moreover, it has been found that childhood hyperactivity is often followed by antisocial behavior in adolescence (Mendelson, Johnson, & Stewart, 1971), and that the syndrome bears a genetic relationship to psychopathy as indicated by investigation of psychiatric illness among relatives of hyperactive children (Cantwell, 1972; Morrison & Stewart, 1971). Thus, behavioral analysis of hyperactivity, its developmental trends, and its genetic relationship to other disorders indicate that it is part of a broader constellation of disinhibitory syndromes. According to evidence presented by Tarter (1979), primary alcoholism may also be included among such syndromes. Psychopathic traits are evident in the personality profiles of a majority of alcoholics; psychopaths and alcoholics exhibit similar psychophysiological irregularities; finally, as youngsters, both groups are characterized by hyperactivity and signs of minimal brain dysfunction. In view of this,
Tarter (1979) suggested that "hyperactivity may be the behavioral substrate for what later emerges in a significant proportion of persons as sociopathy and alcoholism" (p. 59).

Research approaches to human disinhibition must begin to accommodate the broader aspects of the construct that have thus far been considered mainly in isolation. The construct, as seen here, possibly spans several psychological categories—psychopathy, hysteria, hyperactivity, classes of antisocial behavior, impulsivity, and alcoholism—and involves a genetically determined diathesis modified by differential experience. The similarity between this formulation and Meehl's (1962) diathesis-stress model of schizophrenia is not accidental. Meehl proposed that there is a specific genetic etiology of schizophrenia, called schizotaxia, and that this diathesis underlies the various schizotypes brought about by environmental moderation. Given the genetic as well as the behavioral links among the various syndromes just enumerated, it is possible that the formal characteristics of Meehl's diathesis-stress model of schizophrenia may be profitably applied to disinhibition as well. If so, it is the task of researchers in the areas of psychopathy, hysteria, hyperactivity, antisocial and impulsive personality, and alcoholism to coordinate efforts in discovering the common diathesis as well as the differential moderating factors that produce these separate manifestations. The search for the diathesis in particular must look beyond the phenomenon of antisocial transgression, which characterizes only some disinhibitory syndromes, and only partly so at that. It is proposed here that such a diathesis is subtly manifested in all syndromes of disinhibition, including some "normal" personality types, and moreover, exhibits heritable variation. What is inherited is likely to be a disposition to a particular behavioral style, which may or may not be rooted in a neuropathological irregularity, and which entails more or less serious consequences for psychological adjustment depending on environmental circumstances.

There are, certainly, several research strategies that may be adopted to discover the diathesis and basic psychological components of the syndromes described. Our own attention has been drawn to the disinhibitory effects of bilateral septal lesions in animals as a means of elucidating disinhibited behavior in humans. Other authors have already alluded to the septal lesion literature in the endeavor to identify neural structures or pathways that might be dysfunctional in psychopaths (Hare, 1970), extraverts (Gray, 1972), and hyperactive children (Rosenthal & Allen, 1978). However, as proposed earlier, the attempt to identify dysfunctional CNS structures in disinhibited humans is unlikely to advance beyond the level of speculation. Moreover, dysfunctional anatomical loci do not explain complex behavioral phenomena. To state, for example, that "septal dysfunction causes impulsive behavior" does little more than juxtapose two distinct conceptual spheres, anatomy and behavior, whose connection remains to be explained.

In what way, then, do we propose that the effects of septal lesions in animals be utilized to elucidate human disinhibition?

We advocate the formulation of the neurophysiological consequences of a septal lesion in psychological terms—terms that have clear and testable implications for behavior. Such terms include perceptual, motivational, cognitive, and other psychological peculiarities inferred from experimentation with the lesioned animals. Taken together, such terms denote a hypothetical construct, or "septal lesion process," that supplants the anatomical description of what is happening in the brain of a septal-lesioned animal.

"Disinhibitory psychopathology" is also a hypothetical construct. It details psychological peculiarities of disinhibited humans, for example, steep temporal gradient of fear arousal, deficit in mediation of temporal intervals, and so on. Whether disinhibitory psychopathology involves a neurological defect is not a question we are prepared to address. Nevertheless, we shall presently compare the behavior of disinhibited humans to the behavior of septal-lesioned animals in whom there is no question as to a neurological defect. In so doing, however, we do not imply that disinhibitory psychopathology can be traced to septal dysfunction. What we imply
is that comparable perceptual, motivational, cognitive, and other psychological processes account for the disinhibited human's and the septal-lesioned animal's behavior.

The thesis of this article, then, is that disinhibitory psychopathology and septal dysfunction refer to functionally equivalent hypothetical constructs; that is to say, the behavioral consequences of septal dysfunction are comparable to the behavioral consequences of disinhibitory psychopathology in theoretically relevant respects. We advance this notion while acknowledging the obviously different origins of septal dysfunction and disinhibitory psychopathology. In the former case, the construct is "induced" through physical lesion of a portion of the brain. In the latter case, a comparable psychological state arises in humans by other, as yet unknown, means—genetics or early life experiences or some combination thereof. In any event, we present the functional equivalence of these animal and human syndromes as more than a curiosity. We suggest that the comparison entails important implications for research. Through experimental analysis of the behavior of septal-lesioned animals, we can infer the perceptual, motivational, cognitive, and other psychological peculiarities—the hypothetical construct—accounting for these animals' behavior. This, we suggest, can provide clues to basic psychological components constituting the common denominator of several disinhibitory syndromes in humans.

In the next section we review evidence suggesting that there is a valid analogy between the behavior of septal-lesioned animals and that of disinhibited humans. First, however, we must acknowledge that to compare disinhibitory psychopathology with the septal syndrome is to risk certain pitfalls. The septal syndrome itself is by no means clearly understood, and there are major differences of opinion as to the proper explanation of septal-lesioned animals' behavior in various experimental paradigms. Consequently, the interpretation of a particular pattern of septal behavior that we choose to cite will occasionally be at odds with other interpretations. Moreover, the interpretation that we are likely to select from the literature will tend to serve our particular purpose of demonstrating a behavioral analogy between the animals and disinhibited humans.

For example, we discuss the fact that septal-lesioned animals' deficit in performance on DRL, a schedule involving differential reinforcement for a low rate of responding, can be overcome by providing an external stimulus to indicate the interval when responses must be withheld. In this context we have deliberately selected the interpretation that such a finding suggests loss of normal ability to mediate temporal intervals, an interpretation similar to explanations found in the literature on psychopaths. There are other interpretations of the DRL finding, and there is no doubt that this and other features of septal behavior are still in need of empirical analysis. Needless to say, aspects of disinhibitory psychopathology are no less in need of empirical analysis. Nevertheless, we do venture interpretations of the literature as it now stands in the interest of demonstrating that a case can be made for an analogy between the lesioned animals and disinhibited humans. Although our presentation of the literature is selective with respect to both the data and the interpretations we cite, we do cover a number of behavioral dimensions—avoidance learning, anticipation of noxious events, classical aversive conditioning, inhibition of appetitive responding, mediation of temporal intervals, and stimulation seeking—and maintain that the similarities between septal-lesioned animals and disinhibited humans on these dimensions cannot be dismissed lightly but are, in fact, compelling.

Last, in spite of our exclusively functional conceptualization of septal dysfunction, we acknowledge that anatomy cannot be thrown entirely by the wayside. In the pages to follow we refer to "septal" dysfunction but might well have spoken of dysfunction within an entire system composed of the medial septum, the hippocampus, and orbito-frontal cortex (SHF) (Gray, 1972), not to mention other structures. This is because many of the behavioral effects of septal lesions can be duplicated by lesions elsewhere in the limbic system. Moreover, specific behavioral components of the septal syndrome can be dis-
associated and localized in different parts of the septum and in the various neighboring structures of the limbic system. Thus, although we have chosen to ignore anatomical details to facilitate exposition of the functional analogy, it is apparent that the problem of delineating a septal syndrome is itself quite complex, and the question of anatomy must eventually be addressed at more sophisticated levels of theoretical development. Admittedly, it is unlikely that the typical gross bilateral lesion of the septum, the type produced in our own and most other labs, could actually induce a functional psychological state equivalent to what could ever occur naturally in either humans or animals. Our analogy between disinhibitory psychopathology and septal dysfunction would be on much safer ground if we could be assured that the systems disrupted by electrical destruction of the septum were those likely to be disrupted by factors that would, in nature, modify the central nervous system to produce impulsive behavior—genes, the intrauterine environment, nutrition, toxins, sensory experience, trauma. Nevertheless, a septal lesion, disrupting the SHF system, does produce a reasonably coherent syndrome worthy of a certain level of analysis and, pending other developments, it shall form the basis of our functional analogy to disinhibitory psychopathology.

The "Septal Syndrome"

The effects of septal lesions are neither debilitating nor readily apparent. The notorious "septal rage," which sometimes appears immediately after surgery, disappears altogether within several days. Septal-lesioned animals exhibit no universal learning impairment; in fact, they are more astute learners than controls in several learning paradigms. However, these lesioned animals are profoundly impaired on tasks that might be construed as requiring a capacity for "impulse control."

McCleary (1966) has characterized the septal lesion deficit as consisting, in part, of an apparent inability to suppress certain response tendencies when situational contingencies of reinforcement impel the normal animal to do so. In one experiment (McCleary, 1961), both normal and septal-lesioned cats were trained to approach a food trough from which they obtained a small portion of food. When the response was well learned, an electric shock became contingent on such approach for two trials. Whereas normal animals quickly learned to inhibit their approach response, septal animals were almost unaffected, returning to eat immediately or only a few trials after being shocked. Similar results were obtained with septal-lesioned rats, which failed to inhibit their tendency to approach a water dish that had been electrified (Kaada, Rasmussen, & Kveim, 1962).

The deficit that these animals exhibit has been termed one in passive avoidance, meaning the animal fails to suppress or inhibit those particular responses that result in punishment. In this respect, the deficit is similar to that observed by Lykken (1957) in psychopaths, who also fail to inhibit those responses that entail painful consequences. Although this parallel is not in itself remarkable, it merits particular attention when considered in conjunction with findings suggesting that the same explanatory account applied by Hare (1970) to avoidance deficits in human psychopaths also applies to the passive-avoidance deficits observed in septal-lesioned animals. As the reader will recall, psychophysiological and conditioning studies have suggested that psychopaths have a steep temporal gradient of fear arousal and are deficient in classical aversive conditioning. Furthermore, these results have provided the basis for a two-factor avoidance account of psychopaths' tendency not to avoid noxious events. Interestingly, septal-lesioned rats also appear to have a steep temporal gradient of fear arousal. Although the evidence has not been obtained from psychophysiological studies, the behavior of septal-lesioned rats on a Sidman avoidance task recommends such an interpretation. Septal-lesioned rats acquire Sidman avoidance more rapidly than normal animals. Most importantly, however, subsequent to acquisition by both groups of animals, septal-lesioned rats succeed in avoiding the same number of shocks as normals while emitting fewer responses (Morgan & Mitchell, 1969; Sodetz, 1970). To do this, the lesioned
animals tend to wait until the end of the interval, as potential shock nears, before making the response to postpone punishment.

At first glance, this response style appears contrary to that of psychopaths, who are deficient in avoiding shock. However, it must be noted that Sidman avoidance differs from the Lykken maze paradigm. In the latter case, the subject is actively engaged in problem solving and the shock contingency is relatively peripheral. In the case of Sidman avoidance, the avoidance response is the sole requirement for the subject. Psychopaths' performance on simple operant avoidance has not been assessed; however, their steep temporal gradient of fear arousal suggests that their behavior on Sidman avoidance would parallel that of septal-lesioned animals, failing to emit an avoidance response until faced with temporal cues closely associated with shock. Psychopaths' avoidance deficit is probably evident only in situations in which response requirements other than simple avoidance are imposed, specifically, prior to psychopaths' typically delayed rise in fear arousal. Septal-lesioned animals' performance on Sidman avoidance, therefore, suggests that they resemble psychopaths in being immune to fear arousal when an upcoming noxious event is relatively remote. Both humans and animals appear to anticipate a noxious event only when it is imminent. As a further point of interest, the finding that the septal-lesioned rat is relatively placid and efficient on a Sidman avoidance task is reminiscent of Cleckley's (1976) observation that the psychopath is less disposed to panic and more poised than the normal individual in stressful situations.

As noted before, psychopaths not only exhibit a steep temporal gradient of fear arousal, they also are deficient in acquiring conditioned fear to a conditioned stimulus (CS) that signals shock. The same appears to be true of septal-lesioned animals, which do not readily acquire a conditioned emotional response (CER) as measured by suppression of operant responding (Brady and Nauta, 1953; Duncan, 1971; Harvey, Jacobsen, & Hunt, 1961). In such experiments, both septal-lesioned and normal animals are trained to press a lever to receive a reward. As the animal responds, a CS (such as a buzzer) terminating in electric shock is then presented. After several CS-US (unconditioned stimulus) pairings, the normal animal sharply decreases its rate of lever pressing in the presence of the CS. The septal-lesioned animal, on the other hand, is relatively unaffected by CS presentation, continuing to respond for food in the presence of a stimulus that virtually immobilizes the normal animal with fear. The septal-lesioned animal's lack of response suppression appears to represent a specific inability to learn the connection between the CS and the shock. As Duncan (1971) demonstrated, animals exposed to the CS-US pairings during temporary septal dysfunction and subsequently presented with the CS while performing an operant task in the normal state also fail to suppress operant responding.

In summary, then, septal-lesioned animals bear a remarkable resemblance to disinhibited humans, particularly psychopaths, in their disinclination to inhibit punished responses, their steep temporal gradient of fear arousal, and their deficit in classical aversive conditioning. The close correspondence between animals and humans in all three aspects of functioning suggests that the same theoretical account, a two-factor theory of deficit in avoidance, is applicable in both cases. More generally, with respect to aversive learning at least, we are presented with the most interesting possibility that syndromes of human disinhibition and the behavior of septal-lesioned animals are derivable from analogous hypothetical constructs.

What about behavior in situations in which reward is the only motivator? As noted before, disinhibited individuals such as psychopaths and antisocial adolescents are known both clinically and experimentally as less disposed than normals to forego immediate gratification as a means of obtaining a larger reward later on. Although analogous experiments have not hitherto been done with septal-lesioned animals, the behavior of such animals in appetitive DRL situations indicates that they have difficulty in suppressing tendencies to respond immediately as a means of
gaining a reward subsequently (Ellen & Butter, 1969; Ellen, Wilson, & Powell, 1964; Hothersall, Alexander, & Slonaker, 1972). In such experiments, both septal-lesioned and normal animals must refrain from making an operant response for a set interval of time in order to receive a reward for responses occurring after the interval has elapsed. Compared with normal animals, animals with septal lesions have trouble withholding responses during the required interval. Although this deficit is not the same as inability to delay gratification as seen in disinhibited humans, which involves accepting a small reward now at the expense of a larger one later, it seems likely that an animal that responds impulsively on a DRL schedule, thus not getting any compensation for its efforts, would likewise tend to respond impulsively when supplied with a small incentive for doing so. Experimentation is described later indicating, in fact, that septal-lesioned animals do exhibit a disinclination to delay gratification analogous to that displayed by psychopaths and antisocial adolescents. Further evidence of septal-lesioned animals' poor impulse control in the face of rewards can be seen in their tendency to respond at higher-than-normal rates on a fixed interval (FI) schedule for positive reinforcement (Ellen & Powell, 1962).

It was suggested earlier that the avoidance deficits and the deficits in delay of gratification seen in syndromes of human disinhibition may in fact represent a general inability to mediate the connection between even neutral events that are related remotely in time. Experiments with disinhibited individuals on time perception, alternation tasks, and paired-associate learning were cited as evidence supporting such an interpretation. There is experimental evidence to indicate that septal-lesioned animals also exhibit deficits in mediating temporal intervals. For example, the deficit that septal-lesioned animals exhibit on a DRL schedule can be overcome by providing an external stimulus signaling the duration of the interval when the animal must withhold responding (Ellen & Butter, 1969; Kelsey & Grossman, 1971). This finding suggests the possibility that animals with septal lesions have lost the ability to mediate temporal intervals via internally generated cues and must therefore depend on external signals to mediate successfully.

A study by White (1974) supports the hypothesis that septal-lesioned animals have difficulty mediating the connection between events that are related remotely in time. In this experiment, it was found that animals with medial septal lesions could not learn to bar press for reinforcement consisting of hypothalamic stimulation when a 10-sec delay was interposed between the bar press and delivery of the reinforcing electrical pulse. These animals could learn to bar press, however, if reinforcement delivery was immediate. Interestingly, animals with lateral septal lesions were able to learn to bar press whether hypothalamic reinforcement was delayed or not. These results suggest the possibility that the pathways coursing through the medial septum may in fact be the very tracts involved in mediating delayed reinforcement. Thus, in addition to parallels between syndromes of human disinhibition and the behavior of septal-lesioned animals in aversive and appetitive responding, there is the likelihood that deficits in mediating temporal intervals are an important component of functioning in both cases.

Discussion

It appears, therefore, that human disinhibition and the syndrome produced by lesion of the septum bear a fundamental resemblance to one another across several important dimensions of psychological functioning—avoidance learning, anticipation of noxious events, classical aversive conditioning, inhibition of appetitive responding, and mediation of temporal intervals. In addition, septal-lesioned animals, like psychopaths, antisocial adolescents, and hyperactive children, engage in stimulation seeking to a greater degree than their normal counterparts (Nielson, McIver, & Boswell, 1965; Zuromski, Donovick, & Burright, 1972).

These facts suggest a new research paradigm in the area of human disinhibition. Lesion of the septal/hippocampal/frontal (SHF) system cited by Gray (1972), viewed as a hypothetical construct rather than a
physical insult, can serve as a functional research model for perceptual, motivational, learning, and other psychological aspects of disinhibitory psychopathology. The model may be embellished by the additional (and certainly intriguing) hypothesis that psychological characteristics of SHF-lesioned animals represent the basic psychological components from which a variety of genetically related disinhibitory syndromes are derived when subjected to environmental influence. In short, it is proposed that experimental analysis of the behavior of SHF-lesioned animals may shed light on a basic dimension of human psychopathology.

The analogy between SHF dysfunction and human disinhibition can, in fact, be pursued in two directions. One involves testing disinhibited humans for attributes characteristic of SHF-lesioned animals. The other involves testing SHF-lesioned animals for attributes characteristic of disinhibited humans.

In the first strategy, testing humans for attributes characteristic of SHF-lesioned animals, one draws on theoretical development in the animal literature for purposes of generating novel and potentially important hypotheses about human disinhibition. For example, as we have already seen, many psychopathic behavior patterns have typically been explained in terms of deficient anticipatory fear. As a result, research on psychopathy has delved most deeply into the psychopath's disinclination to anticipate and avoid punishment (Hare, 1965c; Hare, 1978; Lykken, 1957; Schmauk, 1970). Such a deficit has also been observed in septal-lesioned animals (McCleary, 1966), but further inquiry has shown that the septal syndrome is characterized, in addition, by significant deficits on purely appetitive tasks such as DRL (Ellen et al., 1964), two-lever counting (Flaherty, Hamilton, & Capobianco, 1974), reversal shifts (Hamilton, 1970), and others. In view of these pronounced appetitive deficits, which seem to involve hypersensitivity to reward, one line of theorizing has it that such hypersensitivity is the basic dysfunction in the septal lesion syndrome (Beatty & Schwartzbaum, 1968; Carlson & Norman, 1971; Henke, 1976). In fact, the animals' avoidance deficit may simply be derived from heedless pursuit of rewards. In terms of the analogy between SHF-lesioned animals and disinhibited humans, one may hypothesize that hypersensitivity to rewards, or some form of hedonism, likewise characterizes human disinhibition and in turn plays a significant role in sustaining the known avoidance deficits. Confirmation of this hypothesis might have the effect of changing the theoretical emphasis in human disinhibition from "deficient avoidance" to "heightened sensitivity to reward."

The second research strategy utilizing the analogy between SHF-lesioned animals and disinhibited humans involves exploring the animal syndrome for attributes already known to characterize human disinhibitory syndromes. Once such attributes are demonstrated in SHF-lesioned animals, not only does the analogy gain further credence, but the animal syndrome provides a model subject to rigorous in-depth examination of learning, motivational, attentional, perceptual, and other processes accounting for the behavioral attributes in question. In this respect, the animal model serves as a sort of experimental crucible, permitting thorough, controlled examination of a basic behavioral style from which a variety of disinhibitory psychopathologies are hypothesized to originate.

The latter approach was adopted by Newman, Gorenstein, and Kelsey (Note 1) in exploring one behavioral style characteristic of human disinhibition, the failure to delay gratification (Hare, 1970; Mischel, 1961; Unikel & Blanchard, 1973; Widom, 1978; Wishnie, 1977). The authors demonstrated that septal-lesioned rats, like disinhibited humans, prefer to accept an immediately available alternative rather than wait for a more attractive one. This finding lends some support to the validity of the analogy between the SHF syndrome and human disinhibition, but more importantly, the finding suggests the possibility that the failure to delay gratification observed in disinhibitory psychopathology is a fundamental, rather than epiphenomenal, aspect of the human condition.

Pragmatically, an experimental paradigm demonstrating failure to delay gratification
in septal-lesioned animals provides the opportunity for more detailed investigation into the mechanism by which an organism fails to delay rewards. Consequently, the investigators (Note 1) sought to elucidate the process of failure to delay gratification in septal-lesioned rats through systematic variation of stimulus conditions in the original experiment. Among the findings that emerged was the discovery that septal-lesioned animals were as capable as controls of comprehending the difference in value between the two rewards employed; moreover, septal-lesioned animals were able to learn the connection between a response and the delivery of a delayed reward; finally, findings indicated that the act of waiting itself was no more aversive to septal than control animals. These and other results pointed to the conclusion that septal-lesioned animals' failure to wait for reinforcement could be traced to an abnormally strong attraction to the immediately available, but less valuable, reward. It appeared as though septal-lesioned animals' consideration of a delayed alternative, quite normal in general, vanished almost completely when an opportunity for immediate gratification was introduced.

Such findings shed new light on the process of failure to delay gratification. Specifically, when an organism fails to wait for a more attractive alternative, we look to the irresistible and exaggerated hold that the immediate reward has gained on the organism's attention; explanations that invoke impairment in contingency learning, aversion to waiting, or related notions appear to be invalid.

Whether this view of failure to delay gratification applies to humans is, of course, a matter of empirical verification. Indeed, establishing the validity—and the limits—of the analogy between the SHF syndrome and human disinhibition resides quite plainly in the continuing verification of cross-predictions. In this regard, a special note may be offered concerning physiology. Throughout our discussion we have shunned speculation concerning physiological mechanisms involved in human disinhibition, proposing the SHF-lesion syndrome as a behavioral analogy only. This approach, of course, does not preclude eventual consideration of the SHF system as a likely site of physiological dysfunction in disinhibitory psychopathology. On the contrary, the adoption of the behavioral model will be crucial in establishing the role of this physiological mechanism. As cross-predictions suggested by the behavioral model continue to be confirmed, the notion that SHF dysfunction could play a role in disinhibitory psychopathology gains more credence.

Nevertheless, our primary interest in the SHF syndrome resides in its use as a functional research model. We have endeavored to show that human disinhibition encompasses a variety of behaviorally and genetically related syndromes including psychopathy, hyperactivity, hysteria, antisocial and impulsive personality, and alcoholism. We have postulated that these and possibly other disorders share basic psychological components, which, subject to environmental circumstances, produce these separate manifestations. In this connection, evidence was presented indicating close behavioral correspondence between the septal lesion syndrome and the most extreme form of human disinhibition, psychopathy. Consequently, it was proposed that the SHF syndrome, as a basic manifestation of disinhibition uncontaminated by environmental influence, may provide a functional research model of perceptual, motivational, learning, and other psychological peculiarities constituting the diathesis of disinhibitory psychopathologies. Thus, our exposition of the analogical relationship of the SHF syndrome to such conditions as psychopathy, impulsivity, and hyperactivity is intended to advance a conceptual framework from which an integrative research approach can be launched. Such an approach entails cross-referenced experimentation in animal and human behavior and the coordination of such research endeavors through the use of comparable experimental paradigms. A program of this kind will promote solutions to the problem of disinhibition and, moreover, will elucidate general principles of organismic adaptation that transcend speciation.

Reference Note
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