

Characteristics and Putative Mechanisms in Boys at Risk for Drug Abuse and Aggression

R. O. PIHL^{a,c} AND J. PETERSON^b

*Department of Psychology
McGill University
Stewart Biological Sciences Building
1205 Dr. Penfield Avenue
Montreal, Quebec, Canada H3A 1B1*

*^bHarvard University
Cambridge, Massachusetts*

The "at risk" paradigm is increasingly being used by researchers interested in understanding the reasons for the problematic behaviors of drug abuse and aggression. Mountains of descriptive statistics of characteristics of abusers and offenders exist, quantifying the extent of the problem. However, typically and unfortunately, this information does not clarify critically needed information regarding causes. This problem commonly emerges in studies of clinical populations, where the separation of cause from consequence is difficult. Drug abuse often results in bidirectional interactions, where the abuse leads to physiological changes and to new behaviors that are unrelated to and obscure important etiological mechanisms. This problem, and others, makes it necessary to study individuals who do not yet display the behaviors but are putatively at risk for developing them. The high-risk approach target also allows for the study of events as they occur, awareness of the multiplicity of potential outcomes, subdivision by age of onset, and developmental perspective of change over time. Furthermore, there exists the potential to observe complex circular-feedback mechanistic processes.

Risk factors in selecting target populations tend to be more focused in the area of drug abuse than aggression. Specifically, genetic models are common. Sons of male alcoholics, for example, are characterized by 4 to 9 times increased risk for developing alcoholism.¹ Offspring of aggressive individuals have been studied less often, although hereditary contributions to impulsivity and aggression have been reported² (as has risk information from numerous longitudinal studies).³⁻⁷ Risk factors evident in populations of drug abusers and aggressors include gender; time of problem behavior onset; and various behavioral, social-psychological, biochemical, psychophysiological, and neuropsychological/cognitive factors. For example, boys develop drug abuse problems more frequently than girls and are more likely to have problems with physical aggression.⁸ Furthermore, behavior patterns for both disorders differ between the sexes, and heritability of vulnerability may be sex linked. In addition, early onset of drinking problems is important in determining type and degree of abuse.⁹ The same is true of aggression.¹⁰ Finally, there appear to be clusters of characteristics in particular subpopulations that may relate to similarities in type of drug abuse and patterns of aggressive behavior.

^c Tel: (514) 398-6100; fax: (514) 398-4896.

The focus of the first part of this paper is on the commonalities in individual characteristics between the groups. The second part of the paper presents a speculative explanation of why these phenomena may occur together.

BEHAVIORAL CHARACTERISTICS

Boys at risk for either or both drug abuse and aggression are impulsive, hyperactive, and attentionally deficient.^{11,12} These conditions appear in frequent association with early onset of aggression and possible diagnosis of conduct disorder. It is true that families of these individuals are often overtly unstable. Nonetheless, occurrence of these associated behaviors has been reported for boys at risk for drug abuse, with environmental factors controlled.¹³ A fundamental problem in behavioral regulation apparently forms a core of vulnerability. A related issue is the frequently debated overlap between attention deficit hyperactivity disorder (ADHD) and conduct disorder.¹⁴ Although, important differences between the two conditions exist, commonalities abound. Children afflicted with conduct disorder and ADHD both manifest problematic attention, poor control of motor behavior in structured situations, and a proclivity to respond with aggression. It is additionally clear that individuals beset with both problems suffer the worst consequences.¹⁴ Sons of male alcoholics, for example, constitute one population frequently characterized by this specific pair of problems.¹¹

Youth at risk for drug abuse have been described as emotionally reactive,¹⁵ particularly to negative affect,¹⁶ and as characterized by high levels of anxiety.¹⁷ "Hot tempered,"¹⁸ "callous," "unemotional," and "ubiquitously impulsive,"¹⁹ by contrast, are terms commonly applied to highly aggressive adolescents. The application of a multidimensional view of personality to individuals genetically at risk for alcoholism has resulted in the creation of two typologies: a male-limited, more severe, early-onset variant often with prior antisocial personality (ASP) (type II), and a less severe, late-developing heterogenous disorder (type I).⁹ Young males at risk for type II alcoholism are characterized as low in harm avoidance (anxiety), high in novelty seeking (impulsivity), and low in reward dependence (detached, tough minded). Each of these characteristics has been linked theoretically to postulated different brain systems.⁹ Although data have been far from confirmatory (there are, for example, individuals clearly impulsive and anxious and even mixed impulsive-anxious in the type II sample), these characteristics also emerge in models of conduct disorder.^{20,21} Such models often borrow from Gray's^{22,23} ideas of a behavioral activation incentive reward system, a behavioral inhibition system (anxiety), and a fight/flight system (response to punishment). We²⁴ recently tested Cloninger's model on a large sample of boys assessed for temperament in kindergarten, and at age 13 for delinquency. The strongest predictor of delinquency was novelty seeking/impulsivity, followed by the relatively weaker predictors of low anxiety and low-reward dependence. Further, at age 13, 71% of the boys who were early-onset delinquent reported having been drunk, compared to 25% of the remainder of the sample.

SOCIAL-PSYCHOLOGICAL FACTORS

The willingness to try a drug and its effect appears powerfully affected by the expectation one has of that drug's effect. For example, it is the case that different

alcoholic beverages seem to result in differential proclivities to aggression (at identical blood alcohol levels).²⁵ Similarly, group-specific expectations seem to dictate the saliency of provocative stimuli and thus the probability of aggression.

At the familial level, individuals at risk for drug abuse are exemplified by high levels of family adversity.²⁶ A similar pattern is commonly noted in individuals at risk for aggression.²⁷ Of course, most individuals from such backgrounds do not become drug abusers or aggressors. It thus becomes important to first recognize this fact and second explore how abuse and neglect interact to increase risk status. For example, Cadoret and Stewart's² adoption study showed that socioeconomic status and adoptive family pathology interacted with biological parent antisociality to predict aggressiveness in 283 male adoptees. Family stress, variously defined, is implicated in the development of both drug and aggression problems. Consequently, calls for promoting family and parental competence are frequently heard (with target age of intervention, "the younger the better").

Peer influences similarly are of profound import for both problems. They are among the strongest predictors of drug use among the young.²⁸ An absence of strong peer relationships, in fact poor social skills and rejection, seems to characterize highly aggressive children.²⁹ This is particularly true if the aggressive behavior is highly condemned. However, where the behavior is viewed ambiguously or even expected, peer influence can provide powerful reinforcement for aggression.

BIOCHEMICAL CHARACTERISTICS: THE SEROTONIN (5-HT) STORY

It seems that almost every neurotransmitter, neuromodulator, and neurohormone has been implicated as important in determining risk for both drug abuse and aggression. However, comparisons between specific populations remain difficult, due to the lack of a critical mass of study for a particular chemical and a lack of study comparability. Where findings generally do concur, other methodological issues unfortunately become relevant. Regarding low platelet monoamine oxidase activity, for example, a rather recurrent finding in both populations, the measurement often lacks both specificity and accuracy when assessing central functioning.¹¹

The study of 5-HT levels and functioning has drawn a great deal of recent attention, and, in consequence, the preceding caveats have become somewhat less applicable. Two recent reviews of clinical³⁰ and animal studies³¹ have detailed the role of 5-HT in alcohol intake, abuse, and dependence. These reviews conclude that decreased 5-HT levels appear to increase alcohol intake, whereas increased 5-HT levels decrease intake. There also exists substantial support for the idea that some alcoholics have lowered central 5-HT neurotransmission. A small number of studies of high-risk individuals have been completed. These individuals also appear characterized by reduced 5-HT neurotransmission.^{32,33}

A growing volume of human and animal studies have similarly implicated reduced 5-HT functioning in violent behavior. In humans, reduced cerebrospinal-fluid (CSF) 5-HIAA (a major metabolite of 5-HT) characterizes a number of impulsive and aggressive populations, including children who display severe cruelty toward animals (and who are otherwise aggressive),³⁴ men with poor impulse control,³⁵ criminal recidivists who commit violent crimes,³⁶ and successful suicide victims.³⁷ One technique used to study 5-HT effects on behavior is to manipulate the 5-HT precursor, tryptophan. In one study we completed³⁸ male and female vervet monkeys were fed tryptophan-supplemented, -balanced or -depleted diets.

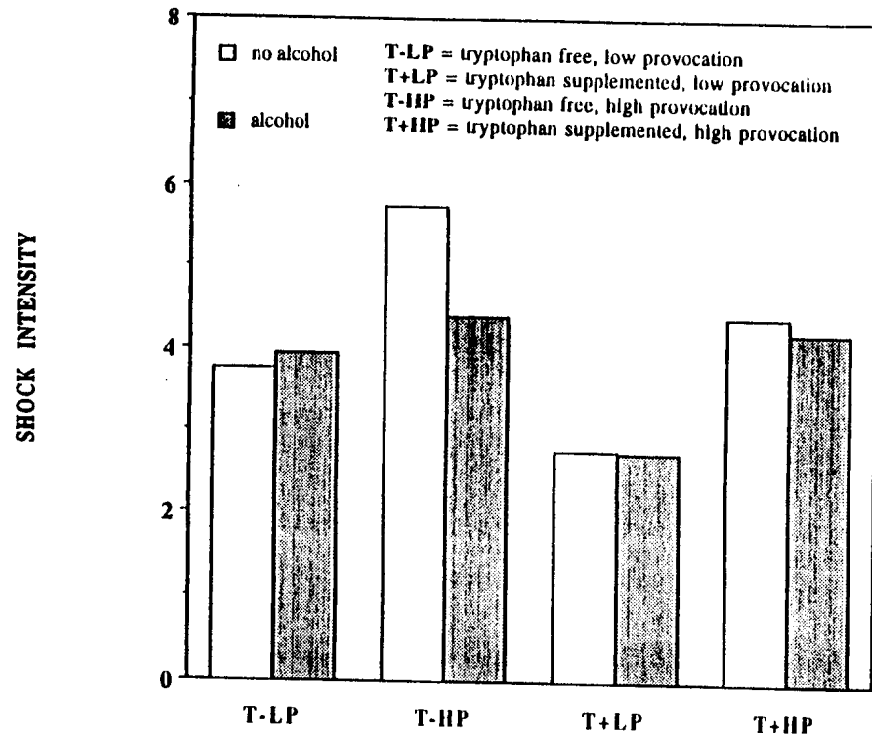


FIGURE 1. The effect of altered tryptophan levels and alcohol under high and low provocation on the intensity of shock administered to subjects.

Tryptophan depletion increased aggression among males, particularly during social interactions (such as competition during feeding), which tend to naturally elicit aggression. In another study,³⁹ using a similar procedure with humans, the tryptophan manipulation was crossed with alcohol, and subjects were then tested on a competitive laboratory aggression task under two levels of provocation. FIGURE 1 illustrates the results of this study. Tryptophan depletion and alcohol significantly increased the intensity of shock delivered to an opponent.³⁹ These results offer laboratory support to the idea that low 5-HT levels may be related to increased aggression.

The general conclusion of a number of reviews^{40,41} is that 5-HT plays a constraining or governance role on the CNS. We have previously⁴² compared the ascending serotonergic projections to a conductor of an orchestra, who is responsible for the organization and control of the orchestra's sometimes fractious instrumental sections (composed of talented but individualistic soloists). As "conductor," the serotonergic system seems to regulate the CNS in at least three primary ways. These are portrayed schematically in FIGURE 2. First, the 5-HT system appears to modify sensitivity to incoming sensory stimuli. The system is densely innervated in cortical areas devoted to primary sensory processing. Furthermore, it has been shown that reductions in 5-HT potentiate startle to the

unexpected and theoretically dangerous.⁴³ This startle may serve as a nonspecific primer, enhancing CNS capability to select and engage in specific behaviors. Reductions in 5-HT appear to decrease the focus but increase the breadth of sensory response. Second, a decrease in the functioning of the 5-HT system seems to decrease control and extend duration of psychomotor response (frequency and amplitude to primary reinforcers).⁴¹ The 5-HT and dopaminergic psychomotor systems interact in determining response to incoming sensory information and appear to engage in reciprocal homeostatic modulation. Dopaminergic activity, for example, in the nucleus accumbens, appears to facilitate behavior motivated by relevant sensory input by reward, punishment, and signals of reward. Decreased 5-HT enhances, and increased 5-HT constrains, this facilitation. Furthermore, 5-HT depletion appears to maintain reward-driven behavior for longer periods of time regardless of variations and external contingencies. Thus, it appears that decreased 5-HT concentrations increase sensory, affective, and behavioral sensitivity to (unconditioned) stimuli that drive reward and punishment-driven behavior. Third, the functioning of the 5-HT system seems to modulate sensitivity to cues that regulate driven behavior (such as cues of punishment defined as threat). High levels of 5-HT in primates are associated with social potency, low levels with decreased dominance and heightened aggression.⁴⁴ Decreases in 5-HT activity may alter the sensitivity to signals that normally suppress social behaviors; therefore, abnormally low levels of 5-HT may be associated with inappropriate social behavior. 5-HT reduction similarly reduces decreased inhibition of social interaction in

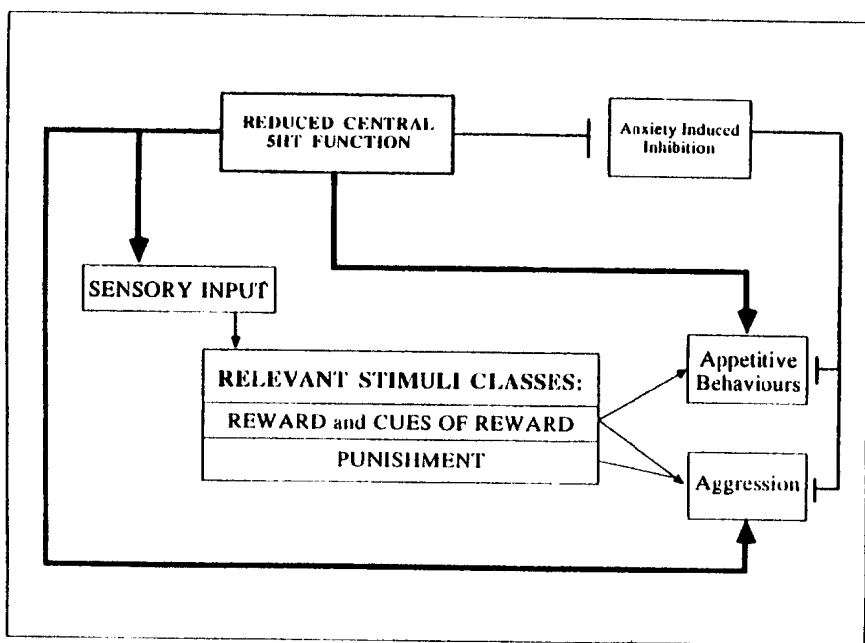


FIGURE 2. A speculative model of how reduced central 5-HT function effects appetitive behaviors and aggression.

novel, brightly lit, and thus fear-inducing environments and shares characteristics with a number of GABA agonists, including benzodiazepines, barbiturates, and alcohol. However, the effect of GABA potentiation and 5-HT depletion are far from identical. It appears possible that potentiated GABA inhibition inhibits anxiety at the affective level, whereas reduced 5-HT activity reduces the inhibitory control of anxiety on behavior (without necessarily reducing the affective aspect of anxiety).

Analysis of the role serotonin plays in modulating motivational states can help clear up a pervasive mystery: How is it that impulsive/substance-abusing children can be simultaneously anxious and impulsive? The different motivational systems that make up the human psyche might be considered analogous to motors. The systems responsible, respectively, for response to punishment, consummatory reward, threat, and incentive reward each can gain access to motor output and capacity for abstraction. What one system wants, so to speak, is not necessarily what is best for another: an object may be simultaneously threatening and promising, or rewarding in the short-term and punishing over the longer course. The ancient serotonergic system appears capable of moderating and integrating the operation of the different "motors," insuring that the person is not working at cross-purposes. Low levels of 5-HT appear associated with the independent working of the different motivational systems, and with their increased activation; higher levels are associated with harmonization and less activation. An individual characterized by low serotonin can therefore be simultaneously anxious, in terms of experienced emotional state, and impulsive, in terms of behavioral output, because he/she cannot use anxiety to govern behavior. It is interesting to note—and relevant to the latter sections of this paper—that the frontal cortex and the ascending serotonergic systems are mutually regulating, and that the abstract capacity of the anterior portion of the brain is actively involved in the processes that underly the serotonergic system's ability to modulate and control the intensity and direction of motivational drive.

PSYCHOPHYSIOLOGICAL CHARACTERISTICS

Sons of alcoholics display, when sober, a distinctive cortical evoked-potential response (EPR), which is a brief waveform to a specific stimulus derived from the electroencephalogram by signal-averaging techniques. Polich *et al.*⁴⁵ in a meta-analytic review of this literature found that overall these individuals displayed smaller P300 waveform amplitudes than controls. This was particularly true on tasks that used visual stimuli and were more difficult. This response, which has predictive value, has also been shown to be heritable, that is, found in approximately one third of boys genetically at high risk for alcoholism, although only in one fifth of genetically at-risk girls. An increased incidence of alcohol and drug abuse characterizes individuals who manifest abnormal EPR response at young ages.⁴⁶ This abnormal response pattern might be associated with reduced frontal lobe functioning.⁴⁷ Similarly, EPR studies in individuals at risk for aggression have found the characteristic reduced amplitudes.^{48,49} Recent studies⁵⁰ have looked at frontal P300 decrements and examined EPR correlates of selective attention in aggressive subjects and concluded that, as in the studies of alcoholics' sons, frontal lobe dysfunction is likely.

A second psychophysiological characteristic typical of individuals at risk for alcohol and drug abuse is heightened autonomic response to novel, threatening,

and rewarding stimuli, all of which might be considered motivationally significant. Logically, this response has been particularly noted in individuals at risk for the abuse of depressants and anxiolytics, because reactivity is often dampened by ingestion of these drugs. This phenomenon has been labeled "stress dampening"⁵¹ and is negatively reinforcing.¹¹ Dampening appears to be associated only with the rising limb of the drug response curve⁵² and appears dose dependent.⁵³ There is some lack of consistency in the literature regarding dampening, which could be accounted for by time of assessment, dose effects, and population and expectancy differences.⁵⁴ It is interesting to note, and may be relevant, that certain strains of animals sensitive to alcohol (*e.g.*, high alcohol-sensitive rats and the long-sleep mice) are differentially sensitive to the sedative effects of alcohol.⁵⁵

The arousal response of individuals at risk for aggression has also been studied, with varied results. Both hypo- and hyperarousal have been reported. Findings of hypoarousal support the notion that a deficient inhibition system typifies psychopathic adults and antisocial children.⁵⁶ The idea of general hypoarousal, however, does not seem consistent, as conduct disordered children who were nonreactive to neutral high intensity stimuli have been shown to exhibit increased reactivity to pleasant stimuli.⁵⁷ Furthermore, disruptive children (antisocial, noncooperative, disobedient, aggressive) are characterized by higher resting heart rate⁵⁸ and can be divided into anxious and nonanxious subgroups. FIGURE 3 illustrates the heart rate response of such subdivided boys to a math stress test involving both wins and losses.⁵⁹ The marked reactivity of the anxious disruptive subjects is apparent.

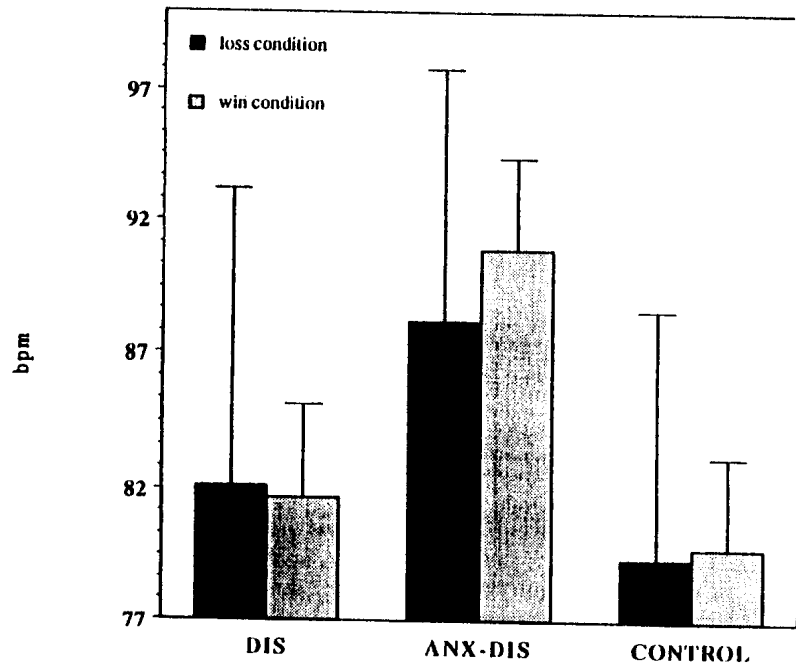


FIGURE 3. Heart-rate response in beats per minute to wins and losses in disruptive, anxious-disruptive, and control 10-year-old boys.

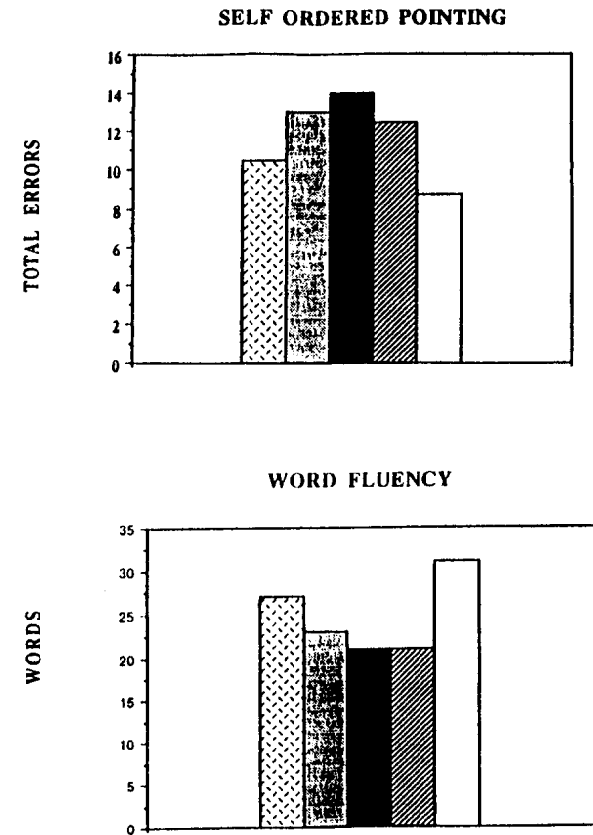


FIGURE 4. Scores on the Self-Ordered Pointing and Word Fluency neuropsychological tests for stable and unstable aggressive and control boys, and for boys with family histories of male-limited alcoholism (FH+) and those with negative family histories (FH-). ▨, nonfighters; ▩, unstable fighters; ■, stable fighters; ▤, FH+; □, FH-.

In this context, it is worth noting that animals most reactive to stress are most aggressive⁶⁰ and that aggressive outbursts are often seen in cardioreactive humans.⁶¹

COGNITIVE/NEUROPSYCHOLOGICAL CHARACTERISTICS

Distinctive aspects of cognitive functioning of males at high risk for the development of alcoholism have been reported. There have been two types of relevant studies: those that focus on school performance and academic variables and those that rely on specific, primarily neuropsychological, testing. FIGURE 4 schematically portrays a pattern of deficient performance for sons of male alcoholics between the ages of 8 to 15, with IQ controlled.⁵⁹ This FIGURE demonstrates their deficiency in cognitive abilities known to be mediated by the prefrontal cortex. The specific tests illustrated have been shown both in clinical populations following lesions and in normal populations on MRI/pet studies to involve differential areas of the frontal lobes.⁶² The performance of 13-year-old individuals with stable histories

of aggression existent over an 8-year period is also portrayed.⁶² The similarity between the two at-risk populations is striking. The idea that a general neuropsychological deficit can alter the regulation of aggressive behavior has growing acceptance.^{10,63} The presence of a specific neuropsychological deficit, however, and the precise nature of such a deficit has remained debatable. Peterson and Pihl⁶⁴ have theorized that the cognitive deficit, partially illustrated in the data in FIGURE 4, is central to the behavioral, psychophysiological, and increased risk for drug abuse characteristic of sons of multigenerational male alcoholics. This dysfunction is thought to be primarily reflected in atypical psychophysiological reactivity to threat and novelty and increased exploration. We have previously postulated a basic problem in classification of the meaning of stimulus events and associated difficulties in verbal reasoning, abstraction, and problem solving. These deficits are also seen as leading to a decreased likelihood of responding to nonintrinsic, nonindividually, relevant stimuli and to an increased likelihood of responding affectively to nonspecific novelty and threat. This situation likely stems from a problem in assigning specific affective relevance to these latter events. There is some evidence that alcohol dampens this system in some individuals at risk for alcoholism and concomitantly reduces aggression²⁵ from comparatively high sober levels. This model seems just as applicable to aggressive individuals per se who display similar cognitive characteristics.

POTENTIAL COMMONALITIES AND MECHANISMS

FIGURE 5 is a schematic model of the interrelationship of three possible explanatory mechanisms as they relate to the high correlation to being violent and intoxicated by alcohol. Over 50% of murders, assaults, and rapes occur when the perpetrator (and, almost as frequently, when the victim) is intoxicated.⁶⁵ The model also has applicability to understanding commonalities in risk for both drug abuse and aggression. Specifically, the model postulates that drugs alter at least three systems that modulate the likelihood of aggressive behavior. The hypothesized systems are the threat system, the exploratory system, and the cognitive control system. These systems in turn each act on what is called a general expectancy set, which is a socioculturally determined theoretical dynamic encompassing all existent expectancies, providing the context in which objective stimuli are individually interpreted.^{66,67} The fundamental assumptions of the model are that (1) threat, specifically threat of punishment for one's own aggressive behavior, is soluble in alcohol (thus the inhibition of aggression in specifically retaliatory situations is reduced), (2) increased exploration subsequent to alcohol intake increases the likelihood of aggressive contact, along with increased sensitivity to cues for positive reinforcement and thus increased instrumental aggression; and that (3) cognitive control reduced by intoxication not only modulates the previous two systems but in itself can readily produce or fail to produce alternative solutions to a singular aggressive response. We have detailed elsewhere,²⁵ evidence supporting how each of these systems is involved in this alcohol/aggression relationship. What follows is an attempt to argue how these systems are simultaneously involved in increasing the risk for both drug abuse and/or aggressive behavior.

THE THREAT SYSTEM

This motivational system responds to cues of punishment and inhibits ongoing behavior in the presence of information, indicating that punishment is about to

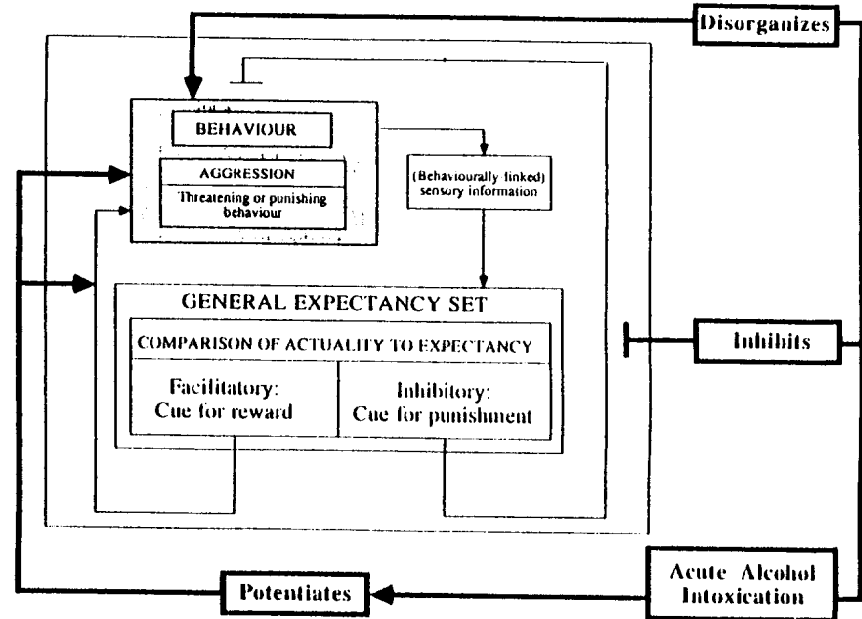


FIGURE 5. The effects of acute alcohol intoxication on aggression: inhibition of fear, potentiation of reward, and the disorganization of behavior.

take place. The operation of this system produces anxiety, an annoying state we are motivated to avoid. Individuals with a high threshold for anxiety are likely to engage in motivationally significant (satisfying or promising) activities that others might regard as too dangerous to be fun. Drugs with high abuse potential, such as alcohol, dissolve anxiety, and their capacity to do so allows individuals to engage in behaviors that might, for example, result in their own demise. The fundamental survival of the organism depends on identifying and responding to stimuli previously associated with danger, or possessing the possibility of representing danger. To avoid calamity or hurt, threat inhibits ongoing behavior. The affective component can be specific (fear) or general (anxiety). The importance of the amygdala, hippocampus, and prefrontal cortex in this system has been detailed. Sensory experience becomes labeled as threatening or promising through amygdaloid functions⁶⁸ unless obviated by other systems involved in imaginative or verbally mediated memory. Individual variability within the threat system may be a function of structure and/or level of naturally occurring endogenous anxiolytics or anxiogenics. GABAergic neurotransmission seems to modulate these systems and is readily potentiated by alcohol, benzodiazepines, and barbiturates. The increased behavioral sensitivity to these drugs in certain animal strains has been shown to be related to increased sensitivity to ethanol-potentiated chloride ion influx at GABA A receptors.⁶⁹

The abuse of drugs that readily dampen this threat system intuitively explains the high level of abuse found in patients with panic disorders,⁴² as well as in individuals with anxiety sensitivity.⁴² Stress dampening is also characteristic of

sons of alcoholics when intoxicated⁴¹ (however, just what is being dampened is not as apparent). Reactivity dampening and reduced aggression in these sons of alcoholics when intoxicated have lead us to theorize that the drug is somehow reducing the consequences of their sober cognitively mediated dysfunction in the classification of stimuli. This particular sober cognitive problem appears quite unlike the cognitive content problem characteristic of individuals who are anxiety or panic sensitive.⁴² In fact, when not intoxicated, the cognitive problem in sons of alcoholics is seen as resulting in increased aggression and the other academic and behavioral difficulties displayed by these subjects.

If appropriately socialized, one's own aggressive behavior is a cue for punishment, and even contemplation of aggression in a nonwarranted situation evokes in such individuals fear and anxiety. Unfortunately, this system is poorly developed in many individuals where the definition of unwarranted is very narrow or nonexistent. The usual culprits for this condition, a cacophony of values, a lack of socialization training, and the absence of meaningful external controls, particularly during periods of natural developmental rebellion, likely contribute heavily. In a sense, it is not so much that aggression is learned but rather that control of aggression is not learned and does not become a fixture of one's general expectancy set.

THE PSYCHOMOTOR EXPLORATORY SYSTEM

The motivational system that responds to cues of reward is also activated, directly, by the more addictive substances. Drugs that activate this system potentiate motivated behavior; if the motivation is towards aggression, it is aggressive behavior that may be potentiated. Individuals who are temperamentally predisposed to respond to cues of reward (*e.g.*, sensation seekers) are more likely to abuse abusable substances. This proclivity is likely enhanced if they are simultaneously immune to anxiety.

The effect of activating the psychomotor system, primarily dopaminergically innervated, is to promote exploration and interaction with biologically relevant stimuli in the environment.⁷⁰ Direct stimulation of this system through the implantation of electrodes or cannula is intrinsically motivating, as other potent reinforcers will be ignored and second order learning easily occurs.⁷¹ This effect is dependent on the density of dopamine neurons and is blocked by dopamine antagonists. Drugs that activate this system by various means may heighten sensitivity to cues of reward. Indeed, given this effect, the question should be not why these drugs are abused but why not?

Stimulants such as cocaine and amphetamines, and alcohol (at moderate doses on the rising limb of the curve) produce a reverse tolerance, which means that the use of one drug makes the others operate more effectively on the psychomotor exploratory system.⁷⁰ This may explain why there is such a high comorbidity among certain classes of drug abuse. For example, 84% of cocaine abusers also abuse alcohol.⁷² Yet, contrary to the general stereotype, there appears to be a great deal of individual variability in the stimulating effect of these drugs. Two rat strains, high alcohol drinking and P alcohol preferring, have been shown to have unusually dense GABA innervation (inhibitory function) of the dopaminergic system.⁷³ This state may account for the heightened alcohol preference that would stimulate this system. These rats after being administered alcohol, for example, manifest more locomotor activity,⁷⁴ maintain bar pressing longer, and are more sensitive to alcohol-induced sedation.⁷⁵ Perhaps representing a similar effect, alco-

holics suffer intense craving when alcohol is withdrawn, and sons of male alcoholics display a heightened heart rate response to the ingestion of alcohol.⁴² Heavy drinking sons of male alcoholics, in fact, peak faster on the blood alcohol curve than heavy drinking nongenetically related individuals⁴² and display EEG wave forms associated with states of well being and pleasure.⁷⁶

Individuals at risk for aggression are often described as seekers of highly stimulating situations, a behavior pattern that conforms to basal low arousal theories of psychopathy. As stated previously, this is true of some highly aggressive individuals, but probably not even the majority. For example, most of the aggressive subjects in the Montreal longitudinal study are both anxious and aggressive. An intriguing question remains: Does this differentiation, based on the presence or absence of anxiety, account for differential typologies of aggressive youth, for example, lifetime resistant versus adolescent limited¹⁰ and/or later adult diagnoses of ASP and/or psychopathy?

THE COGNITIVE CONTROL SYSTEM

A major function of the prefrontal cortex is to deal with abstractions and, more significantly, to help us make abstractions and to allow us to use our abstractions to govern our behavior. Furthermore, its operations are critical to our capacity to generate and consider alternatives, and to understand how we might successfully avoid those places where unpleasant things are most likely to happen. Impairments in this system produce disinhibited, aggressive, short-term-motivated behavior. Such impairments may occur as a consequence of endogenous events; alternatively, they may be drug induced. The neuropsychological deficits often found in both individuals at risk for alcohol and drug abuse, and aggression are highly suggestive of just such a problem. We have referred to this as a deficit in classification-oriented cognitive processing.⁶⁴ The deficits in these populations of voluntary attention, particularly under conditions of social demand, verbal reasoning, abstraction, problem solving, regulation of response to threat and novelty, and amount of exploratory behavior place the individual in a position of diminished control. This of course need not be independent of a reduced sensitivity to cues of punishment or heightened sensitivity to cues of reward that could well be additive.

Particular frontal cortical and limbic structures are implicated in these deficits based on neuropsychological and electrophysiological findings. Bidirectional interconnections between these areas and other brain areas, such as sensory association areas, however, counsels reserve in specifying which structures may be involved. Basically, the prefrontal cortex appears involved in the human capacity to make the abstract meaningful,⁷⁷ so that the individual is capable of attaching emotional significance to distal stimuli and using that stimuli to govern motivated behavior in the present. We generally describe this ability as empathy, which is the capacity to "feel the pain" of the abstract other, and to behave accordingly, or to use the (hypothetical) consequences of our actions to govern our actions. Empathy and the capacity to identify with a potential future self appear as capacities that are integrally related. In this perspective, the antisocial personality can neither empathize with the person one will become nor with the others one constantly encounters. These functions of the prefrontal cortex are thus involved in the construction of verbal and motor responses to novelty and/or threat and provide strategies that become the building blocks of the general expectancy set. Consequently, both the production and the inhibition of a response is affected.

Recent findings are relevant. Certain individuals at risk for aggression and drug abuse are highly reactive, and this reactivity is linked to a cognitive problem.^{62,78} One concomitant of this problem may be a change in saliency of stimuli so that sensitivity is heightened. It has recently been shown that sons of alcoholics, like alcoholics, are more sensitive to pain or threat of pain (electric shock) than controls that are dampened by alcohol.⁷⁹ Stable aggressive boys with high cognitive functioning show a more complex picture in their response to pain. Central processing is also important in response to inhibition. The characteristics of impulsivity for both at-risk groups have been previously detailed. Recently, we⁸⁰ assessed individuals who scored high and low on a putative frontal test and then ran these subjects on an aggression task under inhibited and noninhibited conditions. Notably, low cognitive functioners did not inhibit their level of aggression even under high response cost conditions. In another study,⁸¹ high functioners were also paid for reduced aggression and clearly were able to inhibit even when intoxicated, unlike low functioners. It would seem important to learn how these individuals preserved control and what methodologies might produce a similar response in those we now know to be vulnerable to responding with aggression.

CONCLUSIONS

There are profound similarities in the characteristics of boys at risk for drug abuse and boys at risk for aggression. These are found in behavioral, social, psychological, biochemical, and neuropsychological/cognitive measures. The patterns of overlapping characteristics suggest that there are a number of at-risk groups for both drug abuse and aggression, likely because of divergent etiologies. Differential mechanisms that may be operative include a diminished response to threat, an accelerated exploratory system, and lessened cognitive control. It appears the functioning of these, not necessarily independent, systems contributes markedly to risk.

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Considerations Regarding Biosocial Foundations of Personality and Aggression

JOAN McCORD^a

*Department of Criminal Justice
Temple University
Philadelphia, Pennsylvania 19122*

We stub a toe and feel pain. We see a friend enter the room and rise to greet her. These are ordinary actions that exemplify the interaction between thoughts (mind) and biological processes (body). Nevertheless, the notion that biological and social or mental processes interact has tended to produce more heat than light when the actions in question pertain to aggressive behavior in children.

Much of the heat has been generated by the mistaken view that biological processes determine outcomes. This view is wrong. A tulip cannot be a tulip without an appropriate bulb; but neither will any bulb become a tulip without sufficient light and water. Similarly, to be an outstanding athlete requires certain biological propensities; but the propensities will not result in superior athletic performance without adequate environmental and mental conditions.

It would be foolish to assign a number representing the degree to which biological factors determine variation in plant or human health, though biology is clearly influential. The degree to which biological processes determine outcomes varies in relation to environmental conditions as well as the variability in biological factors being considered.

Variance is a measure of groups, changing in relation to what and how objects are measured. The degree to which a set of properties (*e.g.*, genetic factors) accounts for the variance among groups does not explain why individuals have particular characteristics. An emphasis on evaluating the proportion of variance attributable to genetics has tended to divert attention away from necessary studies of the way in which different environments interact with various biologically differentiated individuals.

In order to understand how bulbs grow to become healthy plants, horticulturists study varieties of bulbs under varying conditions. They do not expect all plants to respond similarly to direct sunlight or torrential rains. Nor should behavioral scientists expect that all people will be similarly responsive to the same environmental stimuli.

What is important about the interactions between biology and social or mental conditions is understanding how these affect development. Craig Ferris and Thomas Grisso organized a conference designed to stimulate discussions about these interactions. That conference incorporated reports by biologists and social scientists. Often, they talked past one another. Yet the knowledge conveyed by the biologists had tremendous potential for understanding development of antisocial behavior, a social classification.

^a Address for correspondence: 623 Broadacres Road, Narberth, PA 19072. Tel: (610) 667-6197; fax: (610) 667-0568; e-mail: mccord@vm.temple.edu.