

A BIOBEHAVIOURAL MODEL FOR THE INHERITED PREDISPOSITION TO ALCOHOLISM

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ABSTRACT

Sons of male alcoholics are at increased risk for the development of alcoholism, and are characterized as well by other traits, that may serve as markers for the alcoholic predisposition. These other traits include mild cognitive abnormalities, cardiac hyper-reactivity to signalled electric shock, susceptibility to alcohol-intoxication elimination of that hyper-reactivity, and increased baseline heart-rate while drinking.

KEYWORDS

Alcoholism, heredity, psychophysiology, neuropsychology.

THE HIGH RISK PARADIGM

Figure 1 schematically presents a family pedigree, typical of the participants in the Douglas Hospital - McGill University Alcohol Research Program. These high-risk subjects are nonalcoholic males between the ages of 18 and 30 (according to DSM-III and Michigan Alcoholism Screening Test criteria) who have, at minimum, an alcoholic father, grandfather and uncle or brother. The high-risk paradigm has as its major advantage the potential to separate cause from consequence. By contrast, derivation of causal inferences from the study of seasoned alcoholics risks confusion of chronic intoxication-induced abnormalities with markers highlighting predisposition to alcoholism. Of course, the major disadvantage of studying high-risk individuals is that the probability any subject will actually become alcoholic cannot be determined. It must be said, as well, that high-risk studies are premised on the assumption that genetic factors play a role in the development of alcohol abuse. A significant part of the evidence supporting this assumption has been the subject of recent criticism (Searles, 1987; Lester, 1988) but claims that inheritance plays a significant role remain supportable (Murray, Clifford and Gurling, 1983; Pihl, Peterson and Finn, 1990).

Subjects are selected for participation in the Douglas-McGill project within extremely narrow parameters to decrease the possibility that noise due to variant subject selection will obscure small but significant differences between subject groups, and because it appears that

such variant selection has contributed unnecessary obscurity to the relevant present-day literature. Young males from heavily-loaded alcoholic families are chosen (1) because both youth and masculinity are associated with heavy alcohol consumption, which provides a necessary but insufficient precondition for the development of alcohol abuse, and (2) because the role played by females in determining the heritable predisposition to alcoholism remains unknown, and is difficult to determine, given the possible confound of fetal alcohol syndrome. Focus on multigenerational history exists because sparse familial involvement does not provide sufficient insurance that the determining familial factor is heredity. As well, research from the Douglas - McGill project (Finn and Pihl, 1987) suggests that distinctive psychophysiological patterns of reaction are characteristic of individuals from heavily-loaded pedigrees, but not of those whose familial alcoholism is confined to the father. The exclusive study of family-history positive university students must be considered problematic, as well, since individuals capable of above average academic achievement might be expected to be those who have not inherited cognitive deficits, for example, that might predispose them to alcohol abuse.

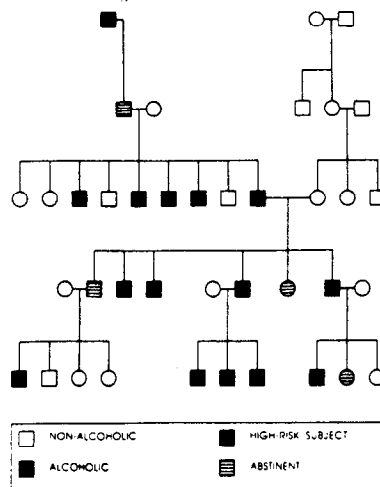


FIGURE 1: A pedigree from the Douglas-McGill project.

CHARACTERISTICS OF SONS OF MALE ALCOHOLICS (SOMAs).

SOMAs can be distinguished from the sons of non-alcoholics in terms of characteristics apart from their heightened likelihood of alcohol abuse. There are over twenty studies which describe SOMAs as deficient in what might be termed behavioural regulation: they are often labelled conduct-disordered, hyperactive and/or impulsive, in childhood, prior to the development of any problem drinking (Tarter, Alterman and Edwards, 1988; Pihl et al., 1990). A recent study from the Douglas-McGill project provides support for this general conclusion. Harden and Pihl (1990) studied 16 preadolescent males (mean age 12) from families with multigenerational histories of male alcoholism and 16 controls with no familial alcohol abuse, matched for age and IQ. Parents of the SOMAs who participated in this study rated their sons more highly in terms of conduct-disorder and learning problems, hyperactivity and anxiety than parents of the control subjects, when asked to complete the Conners Parent Rating Scale. The idea that this behavioural pattern is associated with the development of alcoholism

in adulthood is supported by evidence from longitudinal studies of children who become alcoholic as adults (Pihl et al., 1990), research demonstrating the existence of a relationship between sociopathy and alcohol abuse (Pihl et al., 1990), and studies estimating that 30-40 per cent of alcoholics qualify for a diagnosis of Attention Deficit Disorder, Residual Type (Wood, Wender and Reimberr, 1983).

A number of studies have also suggested that SOMAs are heir to a variety of mild cognitive deficits, although the evidence for this characteristic is not as consistent as that suggesting their behavioral deviance. Two studies from the Douglas-McGill project, one involving young adult males (Peterson, Finn and Pihl, 1990) and one the preadolescents described previously (Harden and Pihl, 1990) add credence to the hypothesis that SOMAs manifest mild abnormalities in cognitive functions often associated with the prefrontal-temporal cortices - categorization of complex visual and verbal information, planning, and organization of memory (Tarter et al. 1988; Pihl, Peterson and Finn, 1987; Gorenstein, 1987). This hypothesis can also be applied usefully, to provide a neuropsychological explanation for the existence of the previously-described behavioural abnormalities, characteristic of SOMAs.

The most intensive research to date, conducted upon SOMAs, concerns their psychophysiological response to various stressors, while sober and while intoxicated. The majority of these studies have concentrated upon various components of the cortical event-related potential (ERP). Reduced amplitude or increased latency of the P300 component is most commonly reported, at least in those studies that select non-university students from highly loaded families as subjects (Pihl et al., 1990). The P300 ERP component varies with subjective evaluation of relevance (Porjesz and Begleiter, 1981); increased latency and/or reduced amplitude of this component, during tasks that require voluntary maintenance of attention, could conceivably be one psychophysiological concomitant of the neuro-psychological deficits characteristic of SOMAs, and described above.

Three studies from the Douglas - McGill Project have demonstrated that SOMAs manifest cardiac hyper-reactivity to stimuli that might be described as involuntarily motivating (threat of and signalled electric shock) (Finn and Pihl, 1987, 1988; Finn, Zeitouni and Pihl, 1990). Alcohol consumption essentially eliminates this reactivity. The combined results of these three studies are graphically presented in Figure 2. SOMAs also manifest cardiac hyper-reactivity to participation in a mental arithmetic task (Harden and Pihl, 1990) and are characterized by heightened orienting response to novel non-aversive tones (Finn et al., 1990). Begleiter, Porjesz and Tenner (1980) have suggested that SOMAs may be less able to distinguish between what is relevant and what is irrelevant; the combined results of the psychophysiological work suggests that they may hyporeact to stimuli that are boring, so to speak, while hyper-reacting to stimuli of intrinsic motivational significance (Pihl et al., 1990).

Pihl and Peterson (1991) have attempted to provide a theoretical framework within which the phenomena described so far in this paper can be understood. This framework, or model, has to date centred primarily on the impaired behavioural regulation, cognitive deficits, psychophysiological hyper-reactivity and alcohol-induced dampening of that reactivity characteristic of the heavily-loaded family-history SOMAs in the Douglas-McGill project. Figure 3 presents a schematic view of the interaction between these characteristics. This model is based upon the assumption that the cognitive deficits are primary; that it is the difficulty in abstractly classifying novel and/or

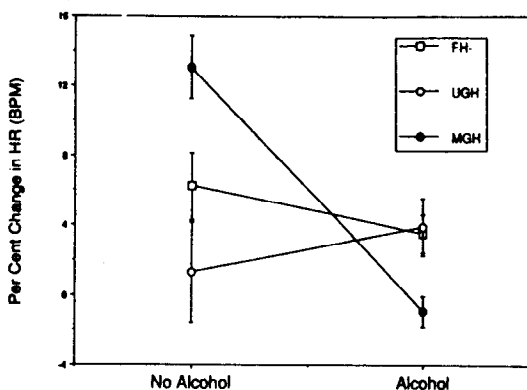


FIG. 2: Heart rate change (%) to signalled shock. (FH-)= no family history; (UGH)= unigenerational family history; (MGH)= multigenerational family history.

threatening stimuli that is the cause of the psychophysiological hyper-reactivity (Peterson et al., 1990; Pihl and Peterson, 1991); and that the behavioural dysregulation typical of SOMAs is a consequence of the interaction between the abstracting deficit and subsequent hyper-reactivity, and the social environment. Abstract cognition allows for rapid, risk-free categorization and planning in the face of threat and/or novelty (Gray, 1987). Theoretically, deficits in abstraction/planning may be associated with increased cardiac output, associated with muscular preparation-for-activity (Obrist, 1976), when approach or active avoidance is utilized in threatening situations as an alternative to abstraction. There is evidence that activity in the threat/novelty detection system is associated with anxiety, and that reduction of this activity might be negatively-reinforcing (Gray, 1987). Figure 3 is predicated upon the assumption that alcohol-induced reductions in heart-rate reactivity are associated with reductions in cued anxiety, and therefore with such negative reinforcement. Of course, cultural and environmental factors (most blatantly, the availability of alcohol) play a role in determining the final outcome of this process. This general model is described in more detail in Pihl and Peterson (1991). Its most obvious weakness is failure to consider the potential role of direct positive reinforcement, putatively reflected in increased baseline heart-rate.

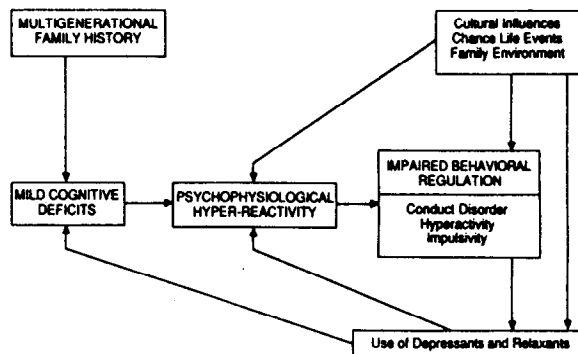


FIGURE 3: Partial schematic model for the inherited predisposition to alcoholism.

With regards to positive reinforcement: SOMAs also appear significantly more sensitive to the excitatory effect of alcohol upon resting baseline heart rate (Figure 4) (Finn and Pihl, 1987, 1988; Finn et al., 1990; Levenson, Oyama and Meek, 1987), as well as to its reactivity-dampening properties. Wise and Rompre (1989) have suggested that all drugs of abuse, including alcohol, might share psychomotor stimulant properties in common. Fowles (1983) claims that size of heart-rate increase can, under some circumstances, be associated with magnitude of positive reinforcement. In keeping with this line of reasoning: it appears that approximately 20 per cent of the variance in weekly alcohol consumption among non-alcohol males (SOMAs and controls) can be accounted for by the interaction between alcohol-induced heart-rate reactivity dampening and alcohol-induced baseline heart-rate increase (Peterson, Pihl, Seguin, and Finn, 1990).

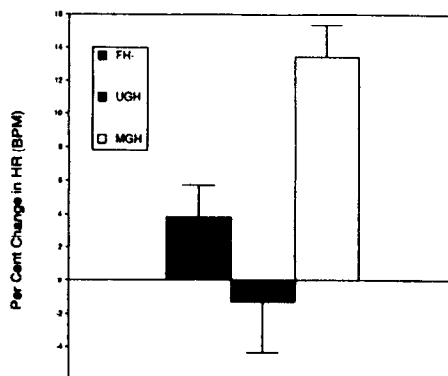


FIGURE 4: Alcohol-induced change (%) in baseline HR, by family history.

CONCLUSION

Sons of male alcoholics differ from sons of non-alcoholics in a variety of manners. They are more likely to become alcoholic, to be labelled conduct-disordered and/or hyperactive, appear prone to a particular pattern of mild cognitive dysfunction, and manifest abnormal psychophysiological reactions to various stimuli, and to ingestion of alcohol. SOMAs from families with extensive histories of male alcoholism appear particularly sensitive to two potentially rewarding effects of alcohol. Alcohol's hyper-reactivity-dampening effect might plausibly be considered negatively reinforcing, while its ability to accelerate baseline heart-rate might be indicative of direct positive reinforcement. It appears possible that sensitivity to the former might be a function of mildly atypical cognitive functioning, while sensitivity to the latter lacks explanation at present. Those who are prone to both phenomena consume significantly more alcohol, weekly, than those who are not, while still drinking at non-alcoholic levels. It appears possible that the presence of both factors might vary with genetic influence.

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