

AUTONOMIC HYPERREACTIVITY AND RISK FOR ALCOHOLISM

R. O. PIHL, PETER FINN and JORDAN PETERSON

Department of Psychology, McGill University
Montreal, Quebec, Canada

(Final form, June 1988)

Contents

Abstract	489
1. Introduction	489
2. The At Risk Model	490
3. Experiment 1	492
4. Experiment 2	493
5. Experiment 3	494
6. Conclusion	494
References	495

Abstract

Pihl, R.O., Finn, Peter and Peterson, Jordan: Autonomic Hyperreactivity and Risk for Alcoholism. Prog. Neuro-Psychopharmacol. & Biol. Psychiat. 1989, 13:489-496

1. Studies which illustrate autonomic hyperreactivity in sons of alcoholics are reviewed and discussed. This response is elicited by a wide range of stimuli which vary from the stressful to the incidental. It also occurs independently of coping strategy. This hyperreactivity, which is particularly characteristic of multigenerational sons of alcoholics is significantly dampened by alcohol. This pattern of results supports a negative reinforcement model of alcohol abuse in these individuals.

Key words: Alcoholism, ANS, Hyperreactivity.

1. Introduction

Current debate over the classification of alcoholism as a disease or a problem in comportment is specious. Although apparently necessary for bureaucrats, lawyers and moralists, the definitional morass that engulfs the terms alcoholism and disease stifles meaningful discussion. In addition, alcoholism is generally conceived of as a homogeneous phenomenon whereas most researchers, regardless of orientation, seem to concur that the problem is heterogenous in nature and in etiology. The variability and general anemia which characterizes the treatment literature suggest that

such lack of differentiation also characterizes treatment. This neglect is hardly trivial as the direct treatment cost in the United States in 1983 was estimated at \$15 billion dollars. This figure explodes to \$116 billion dollars when all economic repercussions are considered (Harwood et al, 1984).

One subgroup of individuals at high risk for alcoholism are individuals with a familial history of the problem. Numerous family, twin and adoption studies support this conclusion (reviewed in Goodwin, 1979). In methodologically sound adoption studies the risk for offspring of alcoholics ranges from 4 to 9 times the risk of controls (Cloninger et al, 1981). Two distinct subtypes of familial alcoholism have been identified: type 1 and type 2. Each differ in terms of family pedigree and age of onset (Cloninger et al, 1981). Type 1 alcoholism is observed in both sexes and is typified by recurrent mild alcohol abuse. Type 2 alcoholism is male limited, has an early age of onset, tends to be associated with early anti-social behaviour and involves the individual in many treatment interventions over a life span. Positive family history predicts the course (Latcham, 1985; Schuckit, 1984), and severity (Hesselbrock, et al. 1985; Latcham, 1985; Templer, et al. 1974) of alcoholism. Type 2 familial alcoholics have also been differentiated from other alcoholics on biochemical (von Knorring et al. 1985) and personality measures (von Knorring et al. 1987).

2. The At Risk Model

Researchers are now specifying the nature of the mechanism (biological, psychological, and/or interactional) that places these individuals at increased risk. Biochemical hypotheses involving most neurotransmitter systems have been offered. The serotonergic system has received particular and recent emphasis (Murphy et al, 1987). The present paper focuses on heritable differences in autonomic hyperreactivity which, it is proposed, form the basis of the increased likelihood of drug abuse problems given the appropriate drug using cultural milieu. This proposal is illustrated in Figure 1. Given a biochemical or neuropsychological dysfunction, it is suggested these individuals are hyperreactive to a wide range of stimuli. This hyper-reactivity may lead to a wide range of behaviour problems, one of which may be drug abuse. Use of depressant drugs may prove negatively reinforcing since such use reduces the degree of hyperreactivity.

Evidence supporting the view that sons of alcoholics with a multigenerational history are prone to autonomic hyperreactivity comes from personality studies, electrophysiological studies of brain wave and autonomic response to laboratory provocations, and from research on the effect of the consumption of alcohol on these behaviours. In terms of personality, the construct of impulsivity is frequently utilized when sons of alcoholics are studied (reviewed in Pihl et al., 1988). Terms such as hyperactive, antisocial and aggressive are often employed. Notably, these same descriptors appear in numerous longitudinal studies of individuals who later develop alcoholism (Zucker & Lisansky-Gomberg, 1986). We have proposed that

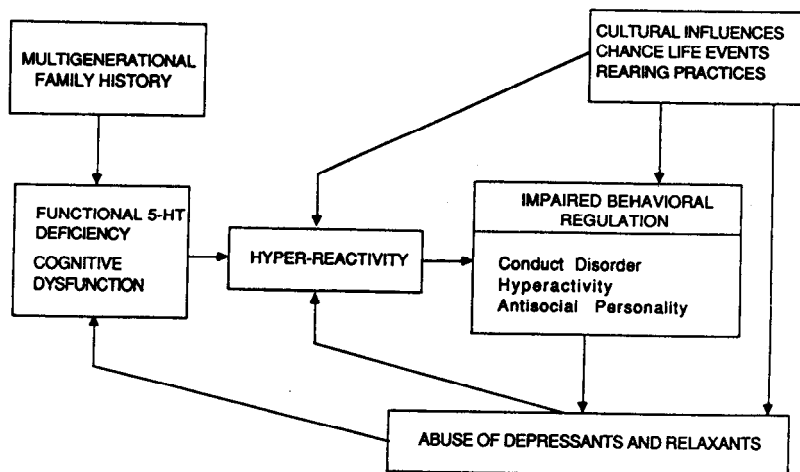


FIGURE 1: Figure one schematically describes the roles played by inheritance, culture and experience in determining the predisposition to alcohol abuse.

this pattern of personality and temperament might be more aptly described as lack of behavioral regulation.

Excessive fast EEG beta activity has been found to characterize the resting state of sons of alcoholics (Gabrielli et al., 1982). This excess of fast activity has also been observed in alcoholics (Mendelson & Mello, 1979) and has been found by some to signify the psychological states of tension and anxiety (Kiloh & Osselton, 1961). Increased latency and attenuation of the P300 component of the cortically evoked potential has also been found in sons of alcoholics (Elmasian et al., 1982; Begleiter et al., 1984) and in alcoholics (Porjesz & Begleiter, 1981; Begleiter, Porjesz & Tanner, 1980). In associated literature, sons of alcoholics and alcoholics have been described as stimulus augmenters. Stimulus augmenters consistently overestimate the dimensions of objects or manifest comparatively increased amplitude in cortically evoked response to increased stimulus intensities. Stimulus augmentation has been associated with increased sensitivity to pain (Petrie, 1978) to psychological events in general (von Knorring 1976) and with increased motivation to work for alcohol (Ludwig et al., 1977). Notably Buschbaum and Ludwig (1978) found that after consuming alcohol, alcoholics shifted from stimulus augmentation to reduction where a decreased cortical evoked response occurs to greater stimulus intensity. The general conclusion from these studies is that sons of alcoholics lack a certain degree of perceptual neuropsychological inhibition which renders them oversensitive to stimulation.

3. Experiment 1

The role of the autonomic nervous system in this pattern of vulnerability is illustrated in three experiments completed in our laboratory. In the first study (Finn & Pihl, 1987) non alcoholic men with a multigenerational history of male limited alcoholism (High Risk) were compared to men with a single generation of male alcoholism (Moderate Risk) and to subjects with no family history of the disorder (Low Risk). These subjects did not differ in age, years of schooling, socioeconomic status of their family, number of alcoholic beverages consumed per week nor in terms of a battery of psychological measures which factored into emotionality, extroversion activity and positive family environment scores. All subjects were connected to a polygraph so that heart rate and digital blood volume amplitude could be measured. A shock electrode was attached to the inside of the left arm. Subjects were then confronted with a countdown period from 10 to 1 after which a shock intensity of 1.85 ma's for 0.25 s was presented. This was done over 10 successive trials under two counterbalanced conditions, with and without alcohol. In the alcohol condition subjects consumed 1.32 millilitres per kilogram of body weight of 95% USP alcohol mixed with orange juice 20 minutes before the beginning of the shock procedure. The results for heart rate under these two conditions are presented in figure 2. In this figure multigenerational high risk subjects clearly display a heightened reactivity to the shock duration which is significantly dampened by alcohol. The reverse seems to be the case for the other two groups. A very similar result occurred on the measure of digit blood volume amplitude. High risk subjects were more reactive than the two other groups and unlike the others displayed significant dampening of that response when intoxicated.

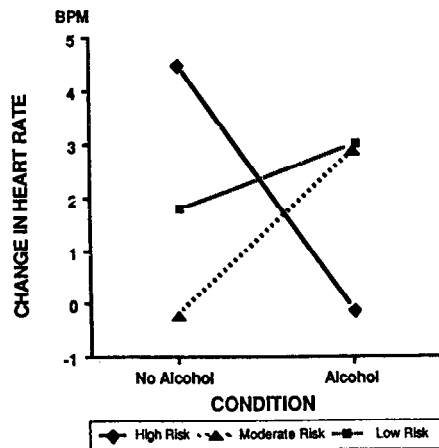


FIGURE 2: Mean change in heart rate in beats per minute (BPM) from resting baseline for the three groups under both no-alcohol and alcohol conditions.

Specifically, without a multigenerational history of the disorder, alcohol appears to increase the significance of the stress response. The reduction of heart rate to alcohol in the stress situation is consistent with the findings of Sher & Levinson, (1982) and Shuckit et al (1981) although in these studies risk was defined somewhat differently. Similarly, increased autonomic nervous system reactivity has been reported in alcoholics (Chandler et al., 1975; Coopersmith, 1964) and there are reports of a significant reduction in this activity after alcohol consumption (Coopersmith & Woodrow, 1967; Garfield & McBreearty, 1970). Another significant result of this study (Finn & Pihl 1987) is the demonstration that stringent criterion of at least two preceding generations of male alcoholism are needed before delineating high risk sons of alcoholics. The lack of hyperreactivity and dampening displayed by the uni-generational moderate risk subjects supports this argument. Previous high risk studies in general have failed to go beyond one generation and have failed to control for the effects of the alcoholic family environment on their measures.

4. Experiment 2

A second study (Finn & Pihl, 1988) was designed as a replication of the study just discussed. New multigenerational high risk subjects were compared to new moderate risk sons of alcoholics. The procedure was identical to the study discussed above with the exception that a lower alcohol dosage (1 ml per kg) was utilized. The results replicated the previous study and consistently differentiated between the two groups. Again dramatic dampening of cardiovascular reactivity to an unavoidable shock after consuming alcohol was noted. This is illustrated in Figure 3.

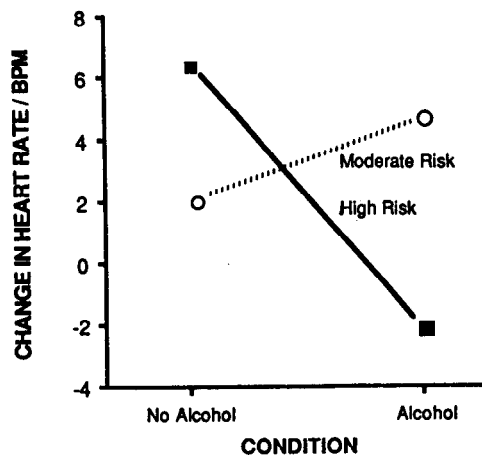


FIGURE 3. Mean change in heart rate in beats per minute from resting baseline for the high and moderate risk groups under both no alcohol and alcohol conditions.

5. Experiment 3.

In both of the above studies a painful unavoidable stressor was the stimulus. Given the procedure of these studies, a plausible hypothesis might be that multigenerational high risk individuals are simply more sensitive to painful stimulation. Brown & Cutter (1977), for example, have noted that alcoholics are more sensitive to painful stimulation than non alcoholics. Furthermore, in the above studies the high risk men were cardiovascularly reactive when anticipating the shock. This suggests that there is a cognitive component to their reaction. These individuals seem to be responding as if they are attempting to cope with the situation (Obrist, 1976) which is, in fact, non avoidable. The purpose of the third study was first to assess responsivity to a painless stimulus and second to attempt to measure the effect of whether the stressor is avoidable or unavoidable. In this third study (Finn, Zeitouni & Pihl 1988) multigenerational high risk men were compared to family history negative men. In addition to heart rate and digital blood volume amplitude, skin conductance and muscle tension were also measured. Non aversive stimuli in the form of a 1 KHZ tone with a duration of .25 s, (which subjects were told to ignore) were played through headphones. In addition during both alcohol and non alcohol sessions subjects received in counterbalanced fashion avoidable and non avoidable shocks. The multigenerational high risk subjects overreacted in terms of skin conductance to tone stimuli. They displayed greater amplitude, a shorter latency and a longer habituation rate than did the family history negative men. Alcohol significantly reversed this effect, as in the previous two studies. These results suggest that the reactivity of these subjects is not restricted to patently aversive situations. High risk men were also equally reactive to both avoidable and unavoidable shock whereas individuals without a genetic history of alcoholism differentiated in their autonomic response to the two tasks.

6. Conclusion

The findings of these three studies suggest that male multigenerational family history subjects have difficulty assessing and responding to a range of environmental demands. Elsewhere (Pihl et al 1988) we have suggested that a cognitive deficit involving the analysis of meaning underlies the hyperreactivity. In effect the subjects react as if many stimuli are threatening, and potentially aversive. This threat-reaction is significantly ameliorated by alcohol. While there is a good deal of evidence of a neuropsychological deficit in both alcoholics and sons of alcoholics (Tarter et al, 1985) much experimentation is called for in examining the relationship between autonomic hyperreactivity and these hypothesized cognitive deficits.

References

- BEGLEITER, H., PORJESZ, B., and TANNER, M. (1980). Neuroradiological and neurophysiological evidence of brain deficits in chronic alcoholics. *Acta Psychiat Scand*, 62, (Suppl. 286), 3-13.
- BEGLEITER, H., PORJESZ, B., BIHARI, B., and KISSIN, B. (1984). Event-related brain potentials in boys at risk for alcoholism. *Science* 225, 1493-1496.
- BROWN, R.A., and CUTTER, H.S.G. (1977). Alcohol, customary drinking behavior, and pain. *J. Abnorm. Psychol.*, 86, 179-188.
- BUCHSBAUM, M.S., and LUDWIG, A.M. (1978). Effects of sensory input and alcohol administration on visual evoked potentials in normal subjects and alcoholics. In, Biological Effects of Alcohol (Begleiter, H. and Kissin, B. pp 561-577) Plenum Press: New York.
- CHANDLER, B.C., PARSONS, O.A., and VEGA, A. (1975). Autonomic functioning in alcoholics: A study of heart rate and skin conductance. *J. Stud. on Alcl.*, 36, 566-577.
- CLONINGER, C.R., BOHMAN, M. and SIGVARDSSON, S. (1981). Inheritance of alcohol abuse: Cross-fostering analysis of adoptive men. *Arch Gen Psychiat*, 38, 861-868.
- COOPERSMITH, S. (1964). Adaptive reactions of alcoholics and non-alcoholics. *Q. J. Stud. Alcohol*, 25, 262-278.
- ELMASIAN, R., NEVILLE, H., WOODS, D., SCHUCKIT, M. and BLOOM, F. (1982). Event-related brain potentials are different in individuals at high and low risk for developing alcoholism. *Proc. Nat. Acad. Science*, 79, 7900-7903.
- FINN, P.R., and PIHL, R.O. (1987). Men at high risk for alcoholism: The effect of alcohol and cardiovascular response to unavoidable shock. *J. Abnorm. Psychol.*, 96, 230-236.
- FINN, P.R. and PIHL, R.O. (1988). A comparison between two groups of sons of alcoholics on cardiovascular reactivity and sensitivity to alcohol. *Alcoholism: Clin. and Exper. Res.*, in press.
- FINN, P.R., ZEITOUNI, N.C., and PIHL, R.O. (1988). The effect of alcohol on psychophysiological hyper-reactivity to non-aversive and aversive stimuli in men at high risk for alcoholism. *J. Abnorm. Psychol.*, in press.
- GABRIELLI, W.F. Jr., MEDNICK, S.A., VOLAVKA, J., POLLOCK, V.E., SCHULSINGER, F. and TILFILL, T.M. (1982). Electroencephalograms in children of alcoholic fathers. *Psychophysiol*, 19, 404-407.
- GARFIELD, Z.H. and MCBREARTY, J.F. (1970) Arousal level and stimulus response in alcoholics after drinking. *Q. J. Stud. on Alcohol*, 31, 832-838.
- GOODWIN, D.W. (1979). Alcoholism and heredity: A review and hypothesis. *Arch. Gen. Psychiat.*, 36, 57-61.
- HARWOOD, H.J., NAPOLITANO, D.M. and KRISTIANNSEN, H. (1984). Economic Cost to Society of Alcohol and Drug Abuse and Mental Illness: 1980, Research Triangle Institute, Triangle Park, NC.
- HESSELBROCK, V.M., HESSELBROCK, M.N. and STABENAU, J. (1985). Alcoholism in men patients subtyped by family history and antisocial personality. *J. Stud. Alcoh.*, 46, 59-64.
- KILOH, L.G. and OSSELTON, J.W. (1961). Clinical Electroencephalography, Butterworths: London.
- von KNORRING, L. (1976). Visual averaged evoked responses in patients suffering from alcoholism. *Neuropsychophysiol.*, 2, 233-238.
- von KNORRING, A.L., BOHMAN, L., von KNORRING, L. and ORELAND, L. (1985). Platelet MAO activity as a biological marker in subgroups of alcoholism. *Acta Psychiat. Scand.*, 72, 51-58.
- von KNORRING, L., von KNORRING, A.L., SMIGAN, L., LINDBERG, U. and EDHOLM, M. (1987). Personality traits in subtypes of alcoholics. *J. Stud. on Alcohol.*, 48, 523-527.

- LATCHAM, R.W. (1985). Familial alcoholism: Evidence from 237 alcoholics. *Brit. J. Psychiat.*, 147, 54-57.
- LUDWIG, A.M., CAIN, R.B. and WIKLER, A. (1977). Stimulus intensity modulation and alcohol consumption. *J. Stud. Alcohol.*, 38, 2049-2056.
- MENDELSON, J.H. and MELLO, N.K. (1979). Biologic concomitants of alcoholism. *New England J. Med.*, 301, 912-921.
- MENDELSON, J.H. and MELLO, N.K. (1985). Diagnostic criteria for alcoholism and alcohol abuse. In J.H. Mendelson & N.K. Mello (Eds.), The Diagnoses and Treatment of Alcoholism, 2nd Edition, McGraw-Hill: New York.
- MURPHY, J.M., MCBRIDE, W.J., LUMENG, L., and LI, T.K. (1987). Contents of monoamines in forebrain regions in alcohol-preferring (P) and nonpreferring (NP) lines of rats. *Pharmac. Biochem. Beh.*, 26, 389-392.
- OBRIST, P.A. (1976). The cardiovascular-behavioral interaction - as it appears today. *Psychophysiol.*, 13, 95-107.
- PETRIE, A. (1978). *Individuality in Pain and Suffering*. University of Chicago Press: Chicago.
- PIHL, R.O., PETERSON, J. and FINN, P. (1988). An heuristic model for the inherited predisposition to alcoholism. *J. Abnorm. Psychol.*, in press.
- PORJESZ, B. and BEGLETTTER, H. (1981). Human evoked brain potential and alcohol. *Alcoholism: Clin. Exper. Res.*, 5, 304-317.
- SCHUCKIT, M.A., ENGSTROM, D., ALPERT, R. and DUBY, J. (1981). Differences in muscle-tension response to ethanol in young men with and without family histories of alcoholism. *J. Stud. Alcohol.*, 42, 918-924.
- SCHUCKIT, M.A. (1984). Relationship between the course of primary alcoholism in men and family history. *J. Stud. Alcohol.*, 45, 918-924.
- SHER, K.J. and LEVENSON, R.W. (1982). Risk for alcoholism and individual differences in the stress-response-dampening effect of alcohol. *J. Abnorm. Psychol.*, 95, 159-167.
- TARTER, R.E., ALTERMAN, A.I. and EDWARDS, K.L. (1985). Vulnerability to alcoholism in men: a behavior-genetic perspective. *J. Stud. Alcohol.*, 46, 329-356.
- TEMPLER, D.L., RUFF, C.F. and AYERS, J. (1974). Essential alcoholism and family history of alcoholism. *Q. J. Stud. Alcohol.*, 35, 655-657.
- ZUCKER, R.A. and LISANSKY-GOMBERG, E.S. (1986). Etiology of alcoholism reconsidered. The case for a biopsychosocial process. *Amer. Psycholog.*, 41, 783-793.

Inquiries and reprint requests should be addressed to:

Dr. R. O. Pihl
Department of Psychology
McGill University
Montreal, Quebec, Canada
H3A 1B1