

Inherited Predisposition to Alcoholism: Characteristics of Sons of Male Alcoholics

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Sons of male alcoholics (SOMAs) are at markedly heightened genetic risk for the development of alcohol abuse. Study of SOMAs could therefore conceivably increase the efficiency of research aimed at uncovering those heritable factors that predispose to alcoholism. SOMAs manifest observable behavioral, cognitive, and psychophysiological abnormalities while sober and react idiosyncratically to alcohol intoxication. They are most commonly described as conduct disordered and hyperactive, appear heir to a variety of deficits in verbal and abstract cognition, and perform more poorly in the academic environment. SOMAs are characterized by abnormal patterns of cued psychophysiological response, and appear more sensitive to the putatively reinforcing aspects of alcohol intoxication. Various methodological weaknesses permeate the relevant literature. Some straightforward improvements are suggested.

Very little is known about those factors that contribute to the development of alcoholism, although alcohol has been abused since the beginning of civilization (Austin, 1985). What has been discovered can nonetheless be utilized to provide useful topographical information to suggest where the further search for detailed knowledge is likely to prove most fruitful. For example, alcohol misuse is common. Five to 10% of adults might reasonably be described as abusers (Kamerow, Pincus, & MacDonald, 1986). Second, male alcoholics outnumber female alcoholics three to one (Adrian, 1984). A curious individual, armed only with these two facts, might determine that whatever factors make alcohol use appealing are far from rare, especially among males, and might therefore decide to preferentially study men, the more susceptible sex. Third, alcoholism runs in families. The degree to which this is a consequence of heredity or of environment is subject to debate, and the literature has been criticized for methodological error (Lester, 1988; Littrell, 1988; Murray, Clifford, & Gurling, 1983; Searles, 1988); however, it appears at least that genetic factors heighten the likelihood of abuse (Cloninger, Bohman, & Sigvardsson, 1981; Cloninger, Sigvardsson, & Bohman, 1988; Goodwin, 1985). This piece of information, in combination with the facts listed previously, make it appear that the study of sons of male alcoholics (SOMAs) might be particularly useful in analysis of the cause of alcohol abuse. An additional fact further narrows the field. It is not advisable to study those who already drink to excess; alcohol detrimentally affects every physiological subsystem, and the study of seasoned alcoholics risks confusion of cause and

consequence. Nonalcoholic SOMAs appear, therefore, to be ideal subjects in many respects for those interested in studying the underlying causes of alcohol abuse. This review is therefore devoted to those individuals.

The relevant studies in this review have been classified according to commonality of findings along five dimensions: behavior, cognition, psychophysiology, reaction to alcohol, and biochemistry. Each section contains an integrated summary statement and additional relevant information, including information derived from the study of the children (male and female) and other less closely related relatives of male and female alcoholics, from longitudinal studies of children who later develop alcoholism, and from research comparing familial and nonfamilial alcohol abusers.¹

Behavioral Characteristics of SOMAs

SOMAs have most consistently been described as conduct disordered and hyperactive (Alterman et al., 1989; Cantwell, 1972; Morrison & Stewart, 1971, 1973; Nylander, 1960 [part 1]; Stewart, de Blois, & Singer, 1978; Tarter, Hegedus, Goldstein, Shelly, & Alterman, 1984); solely as conduct disordered (Aronson & Gilbert, 1963; Cadoret & Gath, 1978; Harden & Pihl, 1988; Nylander, 1960 [part 2]; Nylander & Rydelius, 1982; Rydelius, 1978, 1981, 1983a, 1983b); and as impulsive (Aronson & Gilbert, 1963; Knop, Teasdale, Schulsinger & Goodwin, 1985; Saunders & Schuckit, 1981; Schulsinger, Knop, Goodwin, Teasdale, & Mikkelsen, 1986). These descriptors have been most frequently applied during childhood, but often remain applicable in adulthood as well. They appear as valid even within those

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¹ A detailed tabular summary of the studies of SOMAs reviewed in this article is available from the authors upon request. This summary describes the nature of the subjects studied in each investigation and outlines the major and minor findings.

studies that provide control for environmental influence (Cadoret & Gath, 1978; Morrison & Stewart, 1971, 1973; Nylander, 1960 [part 2]; Stewart et al., 1978). This conduct-disordered/hyperactive pattern of behavior apparently first presents a serious problem in the early years of school, when it disrupts the academic environment. The families of SOMAs are often overtly unstable (Knop et al., 1985; Nylander, 1960 [parts 1 and 2]; Rydelius, 1983a; Schulsinger et al., 1986; Tarter et al., 1984).

Children of alcoholics (both male and female) have also been frequently described as conduct disordered (Chafetz, Blane, & Hill, 1971; Fine, Yudin, Holmes, & Heinemann, 1976; Haberman, 1966). Such children frequently manifest behavioral patterns associated with attention deficit disorder or hyperactivity or both (Fine et al., 1976; Sher & Alterman, 1988), appear hypersensitive to auditory and visual stimulation (Fine et al., 1976), and have difficulty in regulating excitement and mood (Lund & Landesman-Dwyer, 1979). They are often raised in severely disrupted families (Chafetz et al., 1971).

Various longitudinal studies make the same point. Children or adolescents who develop alcoholism later in life as adults are often antisocial or impulsive or both (Block, 1971; Hagnell, Lanke, Rorsman, & Ohman, 1986; Hechtman, Weiss, & Perlman, 1984; Hoffman, Loper, & Kammeier, 1974; Jones, 1971; Loper, Kammeier, & Hoffman, 1973; Magnusson & Bergman, 1988; McCord & McCord, 1960; Nylander, 1979; Nylander & Rydelius, 1973; Ricks & Berry, 1970; Robins, 1966; Vaillant, 1983), are characterized by impoverished interpersonal ties (Jones, 1968; McCord & McCord, 1960; Monnelly, Hartl, & Elderkin, 1983; Robins, 1966), and are hyperactive or aggressive or both (Hechtman et al., 1984; Jones, 1968; Magnusson & Bergman, 1988; McCord & McCord, 1960; Nylander, 1979). Heightened conflict between both parents or between parents and children (Jones, 1968, 1971; Hechtman et al., 1984; McCord & McCord, 1960; Monnelly et al., 1983; Ricks & Berry, 1970; Robins, 1966; Vaillant, 1983) is common. A comparatively high proportion of these vulnerable individuals have alcoholic parents (Nylander & Rydelius, 1973; Vaillant, 1983).

Familial alcoholism has been similarly associated with male sociopathy (Guze, Tuason, Gatfield, Stewart, & Picken, 1962; Guze, Wolfgram, McKinney, & Cantwell, 1967; Latham, 1985; Winokur, Rimmer, & Reich, 1971), childhood conduct disorder (Cadoret, Cain, & Grove, 1980; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1975; Rosenberg, 1969), childhood hyperactivity (Alterman, Petrarulo, Tarter & McGown, 1983; Goodwin et al., 1975), and overt familial disruption (Rosenberg, 1969).

Male-limited Type II alcoholism, theoretically characterized by heightened severity (Oreland, von Knorring, von Knorring, & Bohman, 1985; von Knorring, Bohman, von Knorring, & Oreland, 1985; von Knorring, Palm, & Andersson, 1985), early onset, and increased heritability (Cloninger, 1987), is often preceded by childhood conduct disorder (Cloninger et al., 1988; Hasin, Grant, & Endicott, 1988; Schaeffer, Parsons, & Errico, 1988; von Knorring, von Knorring, Smigan, Lindberg, & Edholm, 1987) and childhood hyperactivity (Tarter, McBride, Buonpane, & Schneider, 1977). The notion that some relationship exists between attention deficit disorder/hyperactivity and alcoholism is further buttressed by the findings of Horton (1985) and Wood, Wender, and Reimherr (1983), who estimated that 30% to 40% of alcoholics qualify for the *Diagnostic*

and Statistical Manual (third edition) diagnosis of residual attention deficit disorder.

Despite widespread differences in methodology and assumption across studies, there is general, unequivocal agreement on the elements of personality, temperament, and behavior that particularly characterize SOMAs, especially those at highest risk for later alcohol abuse. Regardless of the source of information, the same story emerges: SOMAs, and those more generally at risk for alcoholism, are characterized by conduct disorder/antisocial personality with or without hyperactivity/attention deficit disorder. Such descriptors seem to be generally applied to those who are unable or unwilling to adequately regulate their behavior in social situations in accordance with prevailing social standards.

SOMAs appear impaired in the ability to concentrate, to pay attention, and to control their motor behavior sufficiently when required to, at least in structured situations, and they appear comparatively quick to resort to aggression in social situations. SOMAs often break rules or act out frequently, and although they may appear and may in fact be gregarious, they often get into trouble with others. This combination of traits, contradictory at the surface, is not uncommon among children described by their peers as aggressive (Hinshaw, 1987). Such characteristics put them into conflict with authority, and this trouble undoubtedly causes its own second-order problems. There may be a heritable tendency for this pattern of behavior to emerge (Cloninger et al., 1988). It is possible that such a tendency is exacerbated by parental separation and neglect, which are frequently associated with parental alcoholism (Jenkins, 1968; McCord, 1972; Rydelius, 1981) and which may form part of the same long-term behavior pattern.

In the general literature, hyperactivity and conduct disorder overlap, although subgroups of children within these domains appear to differ in important ways (Hinshaw, 1987). Low socioeconomic status, intrafamilial hostility, parental alcoholism or drug abuse, antisocial personality, and hysteria tend to characterize the families of conduct-disordered children, while cognitive and achievement deficits (which are also typical of SOMAs) are often associated with hyperactivity (Hinshaw, 1987). It is of interest to note that August and Stewart (1983) and August, Stewart, and Holmes (1983) described the relationship between alcoholism and childhood hyperactivity as secondary to that which exists between alcoholism and childhood conduct disorder. However, children displaying a combination of hyperactivity and aggression suffer from the worst consequences of both in terms of observed behavior, peer status, and outcome (Hinshaw, 1987; Magnusson & Bergman, 1988). It appears possible that SOMAs are often characterized by such a combination. The emergence of these behavioral patterns in childhood appears to be strongly associated with increased risk for alcohol abuse later in life. This association may be causal in that all or some of the personality/temperament/behavioral abnormalities may themselves predispose to alcohol abuse, or it may be correlational, in which case such abnormalities are merely associated with alcohol abuse and are therefore, like alcoholism itself, consequential to the true cause.

Cognitive Characteristics of SOMAs

Although the picture is far from clear, SOMAs are most frequently characterized by comparatively poor performance on

tests of linguistic ability (Drejer, Theilgaard, Teasdale, Schulzinger, & Goodwin, 1985; Gabrielli & Mednick, 1983; Harden & Pihl, 1988; Hegedus, Alterman, & Tarter, 1984; Knop et al., 1985; Noll & Zucker, 1983; Peterson, Finn, & Pihl, in press; Schulzinger et al., 1986; Tarter et al., 1984; Whipple, Parker, & Noble, 1988). Reduced performance during tests involving abstraction or problem solving or both are also commonly reported (Drejer et al., 1985; Harden & Pihl, 1988; Knop et al., 1985; Peterson et al., in press; Schaeffer, Parsons, & Yohman, 1984; Schulzinger et al., 1986; Tarter, Jacob, & Bremer, 1989). Comparative deficits in total IQ (Gabrielli & Mednick, 1983), performance IQ (Whipple et al., 1988), memory (Harden & Pihl, 1988; Hegedus, Alterman, & Tarter, 1984; Peterson et al., in press; Tarter et al., 1984), visuospatial ability (Hegedus, Alterman, & Tarter, 1984; Tarter et al., 1984), perceptual-motor capacity (Hegedus, Alterman, & Tarter, 1984; Schaeffer et al., 1984; Tarter et al., 1984), and auditory/visual attention span (Hegedus, Alterman, & Tarter, 1984; Tarter et al., 1984) have been described with less consistency.

It is also important to note that SOMAs perform less well academically and have more trouble with school (Drejer et al., 1985; Hegedus, Alterman, & Tarter, 1984; Knop et al., 1985; Rydelius, 1981; Schulzinger et al., 1986; Tarter et al., 1984). Problems related to academic achievement are also reported in many of the longitudinal studies among children and adolescents who later develop serious problems with alcohol (see, for example, the review by Zucker & Lisansky-Gomberg, 1986). Such children are frequently characterized by poorer school performance, higher rates of school truancy, and completion of fewer years of school. The degree to which this reduced performance can be attributed to impaired cognitive ability per se is subject to debate. It could also conceivably arise as a consequence of the behavioral abnormalities discussed previously or for other reasons, such as poor family life or frequent transfer from school to school (Knop et al., 1985).

There are several studies that do not provide consistent support—either directly or indirectly—for the notion that SOMAs are characterized by poorer cognitive abilities. These studies can be grouped into three categories. First, there are those that include a preponderance of females as subjects or as the alcoholic parent and do not offer breakdown of results by sex (Hesselbrock, Stabenau, & Hesselbrock, 1985; Wilson & Nagoshi, 1988; Workman-Daniels & Hesselbrock, 1987). These can be eliminated from consideration in this review by definition. Second are those that look for cognitive deficits among SOMAs and controls drawn from a university population (Alterman, Bridges, & Tarter, 1986a, 1986b; Alterman et al., 1989; Schuckit, Butters, Lyn, & Irwin, 1987). With regard to these studies, it might be said most briefly that SOMAs enrolled in college might well be individuals self-selected for academic achievement, not likely to be characterized by evident cognitive deficits. Finally, comparison of familial and nonfamilial alcoholics occasionally highlights weak group differences attributable to family status (Schaeffer et al., 1984), particularly within studies using a large number of subjects (Schaeffer et al., 1988), and sometimes fails to provide such differentiation (Alterman, Gerstley, Goldstein, & Tarter, 1987; Reed, Grant, & Adams, 1987).

Tarter, Alterman, and Edwards (1985, 1988), Pihl, Peterson, and Finn (1987), Pihl, Finn, and Peterson (1989), and Goren-

stein (1987) suggested that the pattern of cognitive impairment typical of men at risk for alcoholism is reminiscent of that demonstrated by individuals who have suffered some form of mild prefrontal cortical trauma. Authors of two recent articles designed specifically to test this hypothesis concur (Harden & Pihl, 1988; Peterson et al., in press). The fact that the noncognitive characteristics of SOMAs and individuals with minimal prefrontal damage share certain additional features supports this suggestion. These features include increased impulsivity, attentional deficit, impairments in the voluntary regulation of social behavior, heightened levels of activity, abnormalities of the orienting response, and poor emotional modulation (Eslinger & Damasio, 1985; Luria, 1980; Tarter et al., 1985, 1988). This hypothesis appears plausible and could serve heuristically to guide further research.

Psychophysiological Characteristics of SOMAs

It is more difficult to draw straightforward conclusions from the literature describing the psychophysiology of SOMAs because of its technical complexity, methodological heterogeneity, and general contentiousness. The following text is therefore somewhat more detailed than that offered in the preceding sections.

SOMAs have frequently been characterized by attenuation or delay of various components of the cortical event-related potential (ERP) response to stimulus presentation requiring voluntary directed attention or higher order cognitive or linguistic processing. The ERP waveform, with a duration of approximately one-half second, is produced by the electrical response of the brain to a brief sensory stimulus of any modality, and is derived from the electroencephalogram (EEG) by signal-averaging techniques, which extract the time-locked electrical activity. The early components (< 100 ms) appear to vary with change in "objective" stimulus characteristics; the later components (100–500 ms) conceivably vary with change in "subjective" evaluation (Porjesz & Begleiter, 1981).

Six studies (Begleiter, Porjesz, Bihari, & Kissin, 1984; Begleiter, Porjesz, Rawlings, & Eckardt, 1987; Elmasian, Neville, Woods, Schuckit, & Bloom, 1982; O'Connor, Hesselbrock, & Tasman, 1986; Steinhauer, Hill, & Zubin, 1987; Whipple et al., 1988) specifically refer to reduced amplitude of the P300 component. Two more (Hill, Steinhauer, Zubin, & Baughman, 1988; Whipple & Noble, 1986) refer to its increased latency. Increased latency, attenuation, or both of the P300 component during tasks that require voluntary directed attention and cognitive processing also characterize abstinent alcoholics (Begleiter, Porjesz, & Tenner, 1980; Porjesz & Begleiter, 1981). Such abnormalities remain noticeable even after periods of abstinence of up to five years, unlike deficits in brain-stem auditory response conduction velocity, which characterize abstinent alcoholics (Porjesz & Begleiter, 1985) but not SOMAs (Begleiter, Porjesz, & Bihari, 1987). These results, in combination, suggest that P300 deficits alone may predate the onset of alcoholism, and that they may serve as a marker for the inherited risk of alcoholism (Begleiter, Porjesz, & Bihari, 1987).

Authors of two additional studies discussed an ERP component whose production has been related to language. Schmidt and Neville (1984) recorded ERPs evoked during a visual language task from 10 SOMAs and 10 controls matched on several

variables including alcohol intake. SOMAs were characterized by decreased amplitude of the N430 component, which has been linked in the past to semantic processing. In addition, the latency of that component correlated positively with the personal drinking habits of the SOMAs but not with those of the controls. Neville and Schmidt (1985) essentially replicated this study, eliciting ERPs from 10 sober SOMAs and 10 matched controls during the course of a visual letter-rhyming task. SOMAs were once again characterized by decreased amplitude of the N430 component, independent of personal drinking habits. These group differences were more pronounced in readings taken from the right hemisphere than in those from the left.

Seven studies (Neville & Schmidt, 1985; Polich & Bloom, 1986, 1987, 1988; Polich, Burns, & Bloom, 1988; Polich, Haier, Buchsbaum, & Bloom, 1988; Schuckit, Gold, Croot, Finn, & Polich, 1988) reported little or no differences in P300 production (amplitude and/or latency) between SOMAs and matched controls. The majority of these (Neville & Schmidt, 1985; Polich & Bloom, 1986, 1987, 1988; Polich, Burns, & Bloom, 1988) reported, however, that such production is correlated with self-reported alcohol consumption, or by such alcohol consumption in interaction with family history.

The apparent contradiction between these studies and those described earlier might lead one to conclude, like Polich, Burns, & Bloom (1988), that "the P300 ERP component is not a reliable biological marker for the inheritability of alcoholism" (p. 252). Before this conclusion is accepted as definitive, it should be noted that the subjects in all those studies reporting essentially negative results with the P300 have been college undergraduates who reported familial alcoholism limited at best to the father. As noted previously, college students might be expected to have escaped from a serious heritable cognitive deficit (like that which might underlie the P300 abnormalities, e.g., Begleiter et al., 1980). By contrast, subjects in those studies reporting positive results were not college students, and often came from families characterized by Cloninger's Type II alcoholism (Begleiter & Porjesz, 1988; Cloninger, 1987), which is severe, male-limited, highly heritable, and of early onset. It seems at least possible, and parsimonious as well, to posit that the ERP studies differ in outcome primarily because of variant subject selection. If more attention was paid to standardizing task nature as well, the combined results of the P300 studies would likely be less contradictory.

The significance of the P300 component, and of the deficits associated with its production, has been the subject of considerable general discussion, summarized briefly here. The P300 component can be elicited only when active attention is directed toward discrimination, when decision or response depends upon that discrimination, and when the targets occur unpredictably and infrequently (Porjesz, Begleiter, & Samuelli, 1980). The amplitude of the P300 has been related to the subjective determination of the meaning of an event, and not to its sensory modality or inherent qualities (Halgren, Squires, Wilson, Rohrbaugh, Babb, & Crandall, 1980). Its amplitude is therefore logically reduced in anhedonic individuals (Simons, 1982) and in those with aberrant levels of motivation (Porjesz et al., 1980; Roth, Pfefferbaum, Horvath, Berger, & Koppel, 1980). The process underlying manifestation of the P300 is initiated only whenever the stimuli presented to the individual is "moti-

ationally significant" (Begleiter, Porjesz, Chou, & Aunon, 1983). Unexpected or uncertain events, which are motivationally significant because of their unexpected or novel quality (Gray, 1982) therefore elicit large P300 amplitudes (Duncan-Johnson & Donchin, 1977; Tueting, Sutton, & Zubin, 1971). Begleiter et al. (1980) suggested that it is the inability to differentiate between what is relevant and what is irrelevant which distinguishes abstinent alcoholics' (and, by implication, the sons of alcoholics') P300 component from that of nonalcoholic controls. It therefore may be misleading to assume that it is a general reduction in P300 amplitude or a delay in latency per se that characterizes the predisposition to abuse alcohol. It appears possible instead that SOMAs manifest abnormalities in P300 specifically during situations where they must voluntarily allocate attentional resources to maintain the motivational significance of the target stimuli. This predisposition may also manifest itself in the attention deficit disorder/hyperactive behavior and cognitive abnormalities characteristic of these individuals. This appears to mean that SOMAs may have difficulty with voluntary modulation or control of the orienting response, which involves the inhibition of ongoing behavior, increased sympathetic arousal, and redirection of attention (Gray, 1982; Sokolov, 1969).

Four additional studies (Finn & Pihl, 1987, 1988; Finn, Zeitouni, & Pihl, 1990; Peterson et al., in press) provided further evidence that abnormalities associated with the orienting response might characterize sober SOMAs. These investigations, conducted on individuals with extensive multigenerational family histories of severe male-limited alcohol abuse, suggested that SOMAs are characterized by heightened psychophysiological response to novel or aversive stimuli. These studies used stimuli whose motivational significance might be described as inherent: countdown to shock, electric shock, and presentation of novel tones. Such stimuli are different by nature than those used during the evoked-response investigations described previously. This distinction is of crucial importance. It is worthwhile to consider, given such a distinction, the clear possibility that SOMAs hyperreact to stimuli whose motivational significance is inherent or involuntary and hyporeact during situations that require the voluntary maintenance of attention. This line of reasoning is in keeping with (a) Gorenstein and Newman's (1980) theory, which suggests that those who are predisposed to alcoholism have difficulty in suppressing dominant response tendencies; (b) the explanations for P300 reductions provided by Begleiter and his colleagues; (c) the behavioral reports, summarized previously, that describe SOMAs as emotional, excitable, and as hypersensitive to sensory stimuli; and (d) the studies described earlier that detail cognitive and attentional deficits as typical of SOMAs.

Hennecke (1984) noted that the incidence of stimulus augmentation, characteristic of alcoholics (Buchsbaum & Ludwig, 1978; Coger, Dymond, Serafetinides, Lowenstam, & Pearson, 1976; von Knorring, 1976), was significantly higher among children of alcoholics, male and female, than among matched controls, while the incidence of field dependence, also characteristic of alcoholics (Karp, Witkin, & Goodenough, 1965), was not. Stimulus augmentation might be considered a form of perceptual hyperreactivity. Two studies have described EEG abnormalities as characteristic of sober SOMAs. Gabrielli et al.

(1982) demonstrated that the EEGs of SOMAs were characterized by an significant excess of high-frequency beta activity. This pattern of response is also typical of alcoholics (Mendelson & Mello, 1979). An excess of fast beta activity has been associated with psychological states of tension and anxiety (Kiloh & Osselson, 1961).

Begleiter, Porjesz, Rawlings, and Eckhardt (1987) also reported that various EEG abnormalities characterize SOMAs. It has been commonly reported that alcoholics can be differentiated from controls by their poorly synchronized brain wave pattern (Begleiter & Platz, 1972), and Naitoh (1973) argued as well that individuals with such poorly synchronized EEGs are predisposed to alcoholism. Propping, Kruger, and Mark's (1981) work, which showed that alcoholics and their first-degree relatives shared EEG patterns, provides some support for this notion; it suggested that the EEG patterns characteristic of alcoholics may be genetically determined. However, the exact significance of these somewhat abnormal patterns remains unspecified.

Finally, three studies (Hegedus, Tarter, Hill, Jacob, & Winsten, 1984; Lipscomb, Carpenter, & Nathan, 1979; Tarter et al., 1989) suggest that sober static ataxia characterizes SOMAs, and three others (O'Malley & Maisto, 1985; Schuckit et al., 1987; Schuckit & Gold, 1988) do not. It is difficult to draw any final conclusions from these studies. A pronounced lack of theory describing the relationship between alcoholic predisposition and increased sober static ataxia complicates interpretation and integration of these contradictory findings.

Reaction to Alcohol Among SOMAs

SOMAs appear hypersensitive to the dampening effect of alcohol on cardiovascular and electrodermal reactivity to average or novel stimulation (Finn & Pihl, 1987, 1988; Finn et al., 1990; Levenson, Oyama, & Meek, 1987; Peterson et al., in press). Alcoholics also seem to be characterized by some form of susceptibility to the stress-response dampening effect of alcohol intoxication. Alcoholics manifest significant reductions in electrodermal reactivity (Coopersmith & Woodrow, 1967; Garfield & McBrearty, 1970) and in pain reactivity (Brown & Cutter, 1977) after consuming alcohol. In addition, alcohol consumption results in a shift from stimulus augmentation to stimulus reduction in alcoholics (Buchsbaum & Ludwig, 1978; Petrie, 1978), which might be considered a form of dampening, and alcoholics who are augmentors, like the offspring of alcoholics (Hennecke, 1984), work harder for alcohol than those who are normalizers or reducers (Ludwig, Cain, & Wikler, 1977). SOMAs are also characterized by significantly increased baseline heart rate when intoxicated (Finn & Pihl, 1987, 1988; Finn et al., 1990; Levenson et al., 1987). The significance of this response, which deserves further investigation, remains unexplored.

SOMAs tend to self-report reduced intoxication at low to moderate doses of ethanol (O'Malley & Maisto, 1985; Pollock, Teasdale, Gabrielli, & Knop, 1986; Schuckit, 1980b, 1984b), and during the descending limb of the blood alcohol concentration curve (Moss, Yao, & Maddock, 1989), although this effect disappears at higher doses (Finn & Pihl, 1987, 1988; Finn et al., 1990; Peterson et al., in press; Schuckit, 1984a). Schuckit,

Parker, and Rossman (1983), Schuckit (1984a), Schuckit, Gold, and Risch (1987a, 1987b), and Schuckit (1988) also suggested that SOMAs manifest greater decreases in plasma cortisol and plasma prolactin after moderate alcohol consumption, although Moss et al. (1989) did not concur. These larger decreases are in logical accordance with SOMAs' reduced subjective intoxication (Pollock, Gabrielli, Mednick, & Goodwin, in press). The cause of these reduced self-reported low-dose alcohol effects has not been established, although one might be tempted to posit that increased experientially based tolerance among SOMAs might play a role.

Other studies suggest that SOMAs may be more generally sensitive to the reinforcing effects of alcohol. Alcohol intoxication reduces their baseline muscle tension (Schuckit, Engstrom, Alpert, & Duby, 1981), heightens their production of EEG waveforms associated with states of well-being (Pollock et al., 1983; Pollock et al., in press), and normalizes their visual ERP response to pattern reversal (Pollock, Volavka, Goodwin, Gabrielli, Mednick, Knop, & Schulsinger, in press). Finally, there have been two positive reports (Schuckit, 1980a; Schuckit & Raynes, 1979) and one negative report (Behar et al., 1983) that elevations in plasma acetaldehyde characterize SOMAs after alcohol consumption. These results may be unreliable because of difficulties in the accurate measurement of blood acetaldehyde (Eriksson, 1980), although elevated levels of blood acetaldehyde after alcohol consumption are also characteristic of alcoholics (Korsten, Matsuzaki, Feinman, & Leiber, 1975).

Biochemical Functioning and SOMAs

Six studies describe what little is directly known about the normal biochemistry of those with a positive family history of alcoholism. Three of these six studies support the most common finding that family history-positive subjects are characterized by lower platelet monoamine oxidase (MAO) activity than controls (Alexopoulos, Lieberman, & Frances, 1983; Schuckit, Shaskan, Duby, Vega, & Moss, 1982; Sullivan, Cavenar, & Maltby, 1979). The low platelet MAO activity characteristic of such subjects has been correlated with the frequency of alcoholism in their family pedigree (Major, Hawley, Saini, Garrick, & Murphy, 1985; Major & Murphy, 1978; Sullivan, Stanfield, Schanberg, & Cavenar, 1978), generally associated with alcoholism (Alexopoulos et al., 1983; Brown & Cutter, 1977; Gottfries, 1980; Major & Murphy, 1978; von Knorring, Bohman, von Knorring, & Orelund, 1985), and specifically linked to Type II alcoholism, which is considered highly heritable (von Knorring, Palm, & Andersson, 1985). However, relatively decreased platelet MAO activity also characterizes those suffering from other psychiatric disorders (Buchsbaum, Coursey, & Murphy, 1976) and may not provide accurate measure of central MAO function (Stahl & Kravitz, 1986). Its precise significance is therefore difficult to determine.

The three remaining studies variously describe the biochemistry of individuals with alcoholic relatives. Moss, Guthrie, and Linnoila (1986) offered evidence that adolescent SOMAs manifest an enhanced thyrotropin response to thyrotropin-releasing hormone. Swartz, Drews, and Cadoret (1987) demonstrated that nonalcoholic adoptees with alcoholic biological first- or second-degree relatives were characterized by significantly re-

duced mental stress-induced epinephrine release. This pattern is apparently also characteristic of those with aggressive or hyperactive traits (Swartz et al., 1987). Rosenthal, Davenport, Cowdry, Webster, and Goodwin (1980) demonstrated that depressed first-degree relatives of alcoholics are characterized by lower cerebrospinal fluid levels of the serotonin metabolite 5-hydroxyindoleacetic acid. Although the full significance of these findings is not yet clear, the latter study may be of particular interest given the wealth of indirect evidence suggesting that the dysfunctions of the serotonergic system may be involved in the predisposition to alcoholism (Buydens-Branchey, Branchey, Noumair, & Lieber, 1989; Friedman, Krstulovic, Severinghaus, & Brown, 1988; Murphy, McBride, Gatto, Lumeng, & Li, 1988; Naranjo et al., 1987; Roy, Virkunen, Guthrie, & Linnoila, 1986).

Conclusions

Nonalcoholic SOMAs have been most consistently described as conduct disordered and hyperactive. They have difficulty in maintaining concentration and focus of attention, and cannot or do not regulate their overt behavior in accordance with age-appropriate structured social norms. They appear heir to a number of mild cognitive deficits, and manifest decrements in performance on tests of linguistic ability, abstract thinking, and problem solving. Their academic performance is often reduced. It has been demonstrated, with less consistency, that SOMAs are characterized by abnormal patterns of cued psychophysiological response. They appear to hyporeact to stimuli that require the allocation of directed voluntary attention and hyperreact to stimuli that might be considered intrinsically motivating. Alcohol intoxication may be differentially reinforcing to SOMAs: It decreases their exaggerated response to novelty and aversive cues and increases their baseline heart rate.

It is somewhat surprising that these diverse studies are relatively consistent in their findings, given the high degree of heterogeneity among SOMAs and the variety of approaches adopted by those who classify and study them. However, more careful standardization of experimental approach would definitely increase the utility of future research into the heritability of the predisposition to alcoholism. Several specific alterations in methodology could improve such research considerably.

Probands (and controls) should be selected according to more stringent criteria, and their family pedigrees reported in more detail. The presence of male-limited alcoholism in at least two concurrent generations of a given proband's pedigree would significantly increase the possibility that the proband is at increased genetic risk. More extensive delineation of and reference to the family pedigree of subjects would be of invaluable assistance in more accurately determining the effects of purely heritable, rather than environmental, factors on alcohol use and abuse, and in discovering what other conditions coexist with alcoholism. Sex, age, and degree of relationship among affected family members should also be examined and reported. Accurate family histories dating back at least two generations should always be taken from experimental probands and controls and described in published studies.

The nature of alcoholism in the family, and the patterns of alcohol use among experimental subjects, probands and con-

trols, must be more carefully specified. Description of alcohol use should involve investigation of consumption and consequences. Frequency, duration, intensity, and onset of alcohol use are critical variables. The nature of complications arising from alcohol use must be clarified in as much detail. Duration and severity of detrimental effects upon physical, familial, social, and occupational functioning should be described.

In studies that use both males and females as subjects, data on the daughters of alcoholics should always be analyzed separately from that which pertains to the sons. This would enable further description of sex differences and allow cross-study comparability. In addition, it would remove the possibility that mixed-sex studies, reporting negative results, are masking true differences among groups. Subjects with alcoholic mothers should be placed in a separate experimental group if they are used at all. The effects of alcohol on fetal development are well known, and the inclusion of those whose mothers may have abused alcohol during pregnancy only confuses the issue at hand.

Demographic variables must be given careful consideration. With regard to age, for example, the likelihood that a given subject is actually characterized by an inherited predisposition to alcoholism declines as he or she ages without actually abusing alcohol. Younger subjects are therefore more suitable. It might be noted as well that absolute level of education is a critical variable in studies that consider comparative cognitive ability, and that searching for cognitive deficits in a university population is likely to be a thankless task.

Finally, comparison of SOMAs to sons of fathers with psychiatric disorders other than alcoholism would aid in determining the specificity and therefore the utility of any distinguishing markers. Implementation of these methodological improvements would eliminate much of the confusion that presently obscures the true nature of the inherited predisposition to alcoholism.

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