ALCOHOLISM IN A SHOT GLASS

WHAT DO WE KNOW ABOUT ALCOHOLISM?

JEROME D. LEVIN PH.D.

What Do We Know About Alcoholism?

Jerome D. Levin Ph.D.

e-Book 2016 International Psychotherapy Institute

From *Alcoholism in a Shot Glass: What You Need to Know to Understand and Treat Alcohol Abuse* by Jerome D. Levin

Created in the United States of America

Table of Contents

What Do We Know About Alcoholism?

In terms of solid, empirically verified, replicated knowledge, surprisingly little is known about alcoholism. Aside from the physiological evidence and some imprecise demographic findings, there are few hard facts about alcoholism. Some studies strongly suggest that there is a genetic component or predisposition to some forms of alcoholism; there are a handful of replicated empirical psychological findings; there are fewer than half a dozen longitudinal studies; and there is a limited body of known fact about special populations suffering from alcoholism. This chapter takes a look at what is known in each of these areas.

EVIDENCE FOR A GENETIC FACTOR

Conceptually there are two basic questions that researchers ask about the heritability of alcoholism. The first is simply whether alcoholism, some forms of alcoholism, a predisposition to alcoholism, or a predisposition to some forms of alcoholism are inherited. The second is, if alcoholism or a predisposition to it is at least partially inherited, what is it that is inherited? An appetite for alcohol? Enjoyment of alcohol? Relief of some dysfunction or dysphoria by alcohol? Or, in terms of learning theory, more than normal reinforcement by alcohol? A capacity for alcohol (a “hollow leg”)? A deviant

reaction to alcohol by the nervous system or the liver? Some combination of the above? In other words, is it consumption, preference, metabolism, sensitivity and acute reaction to, tolerance (acute and chronic), physical dependency and withdrawal, absence of some protective factor, or some unknown aspect of the alcoholic's relationship to alcohol that is implicated in a predisposition to alcoholism?

It is well known that people have different responses to alcohol. We all know people who love it and people who cannot stand it. Some people really get a lift from a drink, and some people say, "Oh, I just get sleepy," or "I get a headache," from even a few drinks. Is the difference constitutional or is it a result of experience? Is it genetic or acquired? It is not surprising that there are differences in people's responses to alcohol. After all, people react idiosyncratically to all sorts of drugs, and some cats get high on catnip and some do not. For example, there is the phenomenon of the *alcohol flush*, characterized by reddening and other symptoms, including rapid pulse and difficulty breathing, occurring after low doses of alcohol. This phenomenon occurs in very few Caucasians but in a considerable number of Asians. This racial difference in response to alcohol suggests that a genetic factor is involved.

Scientists study the possible heritability of alcoholism in a variety of ways. One is animal studies, particularly those that attempt to breed an

appetite for ethanol. Yet another is the study of the prevalence of alcoholism in families, twins, and the relatives of alcoholics, particularly adoptees who are children of alcoholics. Another is studying the reaction to alcohol of nonalcoholic children of alcoholics. Much current research is focused on the search for *biological markers*, usually but not necessarily biochemical abnormalities that accompany or are antecedent to alcoholism. A biological marker can be anything assumed to be carried by the same chromosome as the trait or condition under investigation. For example, color blindness was once thought to be a biological marker for alcoholism. That does not mean that color blindness causes alcoholism, only that the two go together or, as statisticians say, are correlated because they are presumed to be transmitted by the same chromosome. A biological marker *can* have a causal relation to the condition it marks, but this is not necessarily the case.

Animal Studies

Since people are among the very few animals that naturally drink ethanol in more than minimal quantities, finding animal models for alcoholism is difficult. Elephants are apparently an exception, getting smashed on fermented palms and going on rampages, but it is not clear that it is the alcohol rather than the palms that attract them, nor are we certain whether alcoholic elephants see pink elephants. Alcohol does not appear to be strongly reinforcing for most animals. Psychologists define a *reinforcer* as

something that increases the frequency of a behavior; that is, if alcohol is reinforcing, drinking it should increase the frequency of drinking it. Animals generally do not want more alcohol to drink after drinking some; that is, they don't find it reinforcing. This is strikingly different from the animal response to cocaine. If a rodent is given a choice of pushing a lever that delivers cocaine and one that delivers food, he or she will choose the cocaine, continuing to do so until he or she collapses. Nevertheless, nonhuman subjects have been used to study drinking behavior and alcoholism. *Appetite for alcohol* and *preference for alcohol* are traits that occur in some but not all rodent individuals and strains. Strains of mice and rats have been bred to drink alcohol, and some in fact prefer it to water. There are even rodents that will voluntarily drink enough alcohol to have withdrawal symptoms when they stop. This is not a normal mouse predilection. The mouse souse is not found in nature; neither is the heavy-hitting rat. The way these anomalies are created is by breeding with each other those animals that show some appetite for alcohol and in turn selecting those of their offspring with the greatest appetite for alcohol to mate with those of the offsprings' generation that are similarly inclined. This process of selective breeding is continued over the generations until a strain of rodents with a distinct appetite for alcohol emerges. Surprisingly, Martini Mickey can be bred in as little as 10 generations. With some such strains, continued selective breeding results in animals that prefer alcohol and even in animals that show a physical dependence. These results argue strongly for

the existence of a genetically transmitted appetite for alcohol in rodents. Although extrapolating from animal models to humans is inferential and it is not certain that similar propensities are inherited by humans, the heritability of an appetite for alcohol in rodents is a striking finding that strongly suggests that similar mechanisms exist in humans. Additionally, the heritability of a tolerance for alcohol, as measured by the *righting response* (the ability to remain on or regain one's paws after a heavy dose of ethanol), has been demonstrated in rodents.

One ingenious, if highly speculative, hypothesis to account for the relatively low rate of alcoholism among Jews is as follows. Jews once had rates of drunkenness, problem drinking, and alcoholism as high as any other group. However, living as a persecuted minority in constant danger, the Jews who liked to drink heavily were more vulnerable and more likely to be killed before reproducing than Jews who did not. Over the centuries the genes that mediate a high appetite for alcohol diminished and became infrequent in Jews. For other peoples who did not share the special vulnerability of the Jews, drinking alcohol was safer than drinking the often contaminated water and had survival value, so these heavy drinkers reproduced, while the Jews who were heavy drinkers were less likely to reproduce. An ingenious hypothesis indeed, but the time scale—a mere few hundred years—seems too short. Be this as it may, there are rodents that like to drink and rodents that do not, and the ones that do are the descendants of many generations of

rodents with similar inclinations.

Biological Markers

Research on biological markers is "hot." There are several reasons why researchers are intrigued by the search for metabolic, neurophysiological, and other biological correlatives of alcoholism. One reason involves attempts to establish the heritability of alcoholism. If alcoholism, or a sub-type of it, is associated with a biological trait known to be heritable, then that association can be interpreted as evidence for the heritability of alcoholism. The establishment of biological markers for alcoholism raises the possibility of developing diagnostic procedures to identify alcoholics or those susceptible to alcoholism early in the disease process and of taking preventive or remedial actions to prevent or limit clinical manifestations of the underlying susceptibility. For example, if an elevation of enzyme X is found to be a biological marker for alcoholism, adolescents who test high on enzyme X could be warned that they are at high risk for alcoholism. Finally, the study of biological markers offers intriguing possibilities for understanding and illuminating the underlying metabolic and neurophysiological concomitants of alcoholism. As in all research on alcoholism, it is often difficult to tell what is the chicken and what is the egg, what is causative of and what is resultant from alcoholism. For example, if high levels of enzyme X are found in alcoholics, is the high level caused by the drinking itself, that is, is it a

consequence of the alcoholism? Or is it etiological, that is, did it antedate and contribute to the development of the alcoholism? Or is the elevation of enzyme X a genetically transmitted trait that shares a chromosome with a gene that transmits a trait that increases the susceptibility to alcoholism? That is, is the level of enzyme X a marker neither consequent to nor causative of alcoholism? These are difficult questions to answer. Determining what is cause and what is effect has long been a problem in purely psychological research on alcoholism, and the case is even worse in biological marker research. There are several ways researchers try to tease out cause from effect. The best method would be to use *longitudinal* research designs that follow subjects from childhood into adult onset of alcoholism and gather data on the status of biological markers antecedent to the development of the disease. The next best design is to study children of alcoholics, who are presumed to be at high risk for alcoholism, and see if they are distinguished from controls who are presumed normal on either baseline measures of various biological markers or in their reactions to challenge doses of ethanol. The least rigorous but easiest method is to simply look for correlations between traits and alcoholism.

Color Blindness The first biological marker discovered for alcoholism was thought to be color blindness (Cruz-Coke & Varela, 1966). However, Varela, Rivera, Mardones, and Cruz-Coke (1969) later demonstrated that the color blindness in the Cruz-Coke and Varela study was the *result* of severe

alcohol abuse. Interestingly, Varela et al. also showed that female relatives of alcoholics differed significantly from controls in the occurrence of blue-yellow color blindness, so it is possible that there is a connection between a recessive gene for blue-yellow color blindness and alcoholism. If there is such a connection, it would consist of the sharing of a common chromosome that carries both the gene for color blindness and a gene for some trait that predisposes to alcoholism.

Platelet Enzymes Researchers study the levels of various enzymes in blood platelets because they are easily assessable and because platelet levels are assumed to reflect brain levels of the same enzymes. Additionally, the base levels of these enzymes are known to be under genetic control.

The two platelet enzymes that have received the most attention are *monoamine oxidase* (MAO), which breaks down norepinephrine and other excitatory neurotransmitters, and *dopamine-betahydroxylase* (DBH), which converts dopamine to norepinephrine. The base levels of both are under genetic control, and both affect the level of the neurotransmitter norepinephrine, high levels of which are associated with anxiety. Tabakoff et al. (1988) found significant differences between alcoholics and nonalcoholics in the degree of MAO depression following drinking. In other words, drinking increases the level of a neurotransmitter, high levels of which are associated with anxiety, more in alcoholics than in nonalcoholics through a differential

lowering of the level of the enzyme MAO, which biodegrades norepinephrine and other monoamines. Alexopoulos, Lieberman, and Frances (1983) found depressed levels of platelet MAO in alcoholics and their relatives. This finding has been consistently replicated. Depressed platelet MAO is also associated with a variety of psychiatric illnesses.

Schuckit and Gold (1988) studied the difference between high-risk college students (those who had a family history of alcoholism) and those who had an average risk (they were free of a family history of alcoholism) on a number of biological markers including platelet MAO levels. Schuckit and Gold found no significant difference at baseline (that is, before drinking).[6]

Challenge doses equivalent to three to five drinks of ethanol (ETOH) increased the difference between the *family history positive* (FHP) and *family history negative* (FHN) groups on levels of platelet MAO, but they did not reach significance. The research on platelet MAO in abstinent alcoholics is inconsistent, but depressed levels persist for a long time. Some studies show no change in this trait with sobriety. It is likely that low platelet MAO is a biological marker for alcoholism. If so, this is important because high levels of norepinephrine are associated with sensation-seeking behavior, which is characteristic of Cloninger type 2 (male limited) alcoholics. Type 2 alcoholics have been found to have significantly lower platelet MAO than type 1 alcoholics (von Knorring, Bohman, von Knorring, & Oreland, 1985).

Studies on platelet DBH are conflicting, and no clear correlation between platelet DBH levels and alcoholism has been established. There is, however, considerable albeit not consistent evidence that plasma, spinal fluid, and platelet levels of DBH are significantly depressed in alcoholics. However, this could be caused by the subjects' alcoholism. If this finding proves to be generally true of alcoholics, it would mean that they have higher levels of dopamine and less dopamine converted to norepinephrine than nonalcoholics. Since high levels of dopamine are associated with serious mental illness, depressed levels of DBH may contribute to the relatively poor reality testing characteristic of active alcoholism and the early stages of recovery from it. Paradoxically, there are studies (Schuckit & Gold, 1988) that show significantly higher DBH levels in high-risk (FHP) than in low-risk (FHN) groups of young men. High levels of DBH are believed to be associated with less subjective feelings of intoxication after alcohol consumption. This could contribute to heavy drinking by the high-risk group. None of this is certain, and it is far from clear what the findings on DBH levels in alcoholics and high-risk groups mean. However, there does appear to be an association between DBH levels and alcoholism.

Tabakoff et al. (1988) also found abnormalities in the stimulation of the enzyme called platelet *adenylate cyclase* by various metabolites in alcoholics and thought this might be a biological marker of alcoholism. The abnormality consisted of less stimulation. This is interesting because stimulation of

adenylate cyclase enhances the synthesis of cyclic adenosine monophosphate (cAMP), one of the second messengers with which, it is hypothesized, ethanol interferes. The depression of platelet MAO, DBH, and adenylate cyclase activity persists into abstinence in recovering alcoholics and is postulated to contribute to the prolonged withdrawal syndrome (discussed in chapter 2) and to neurological complications of alcoholism.

Blood Levels of Acetaldehyde Schuckit and Gold (1988) also found significantly higher levels of acetaldehyde, the first breakdown product (metabolite) of alcohol in FHP students after drinking. This finding is intriguing since high levels of acetaldehyde are aversive and presumably would discourage drinking, yet apparently such is not the case in high-risk subjects. Additionally, acetaldehyde is postulated to interact with neurotransmitters to produce the morphine-like *tetrahydrosioquinolines* (TIQs), a mechanism that some investigators believe is involved in the development of alcohol addiction; it is possible, therefore, that high levels of acetaldehyde could be implicated in addiction to alcohol. However, there has been difficulty in replicating this finding either in college-age FHPs or younger ones. It is known that those who are frankly alcoholic tend to produce high levels of acetaldehyde when they drink. But the evidence is not clear-cut. There is, however, no question that people vary in the ways they metabolize alcohol and that the resultant levels of acetaldehyde have something to do with drinking behavior. Presumably acetaldehyde levels

interact with a host of other biochemical, cultural, and psychological variables to determine one's risk for alcoholism. High blood levels of acetaldehyde could result from more rapid conversion of ETOH (ethyl alcohol) into acetaldehyde or from the less rapid conversion of acetaldehyde into acetate. The biological substrate (underlying mechanism) of high acetaldehyde levels may be the presence of atypical or *isoenzyme alcohol dehydrogenase* (ADH) or atypical or isoenzyme aldehyde dehydrogenase (ALDH), both of which are genetically controlled. An isoenzyme has the same atomic constituents as the enzyme, but they are somewhat differently configured. The reader will recall the relationship between propyl and isopropyl alcohol illustrated in Figures 1.11 and 1.12, which is a simple isomeric relationship, not different in principle from the isoenzyme relationship in which the molecules are far larger and more complex. The heritability of these liver enzymes and their variants is well established. High levels of acetaldehyde are associated with the alcohol flushing syndrome, and, not surprisingly, Schuckit and Gold's high-risk FHP subjects had significantly higher occurrence rates of flushing after drinking. Again, this is a non-replicated finding.

Static Ataxia Also known as upper-body sway, *static ataxia* is a measure of unsteadiness that presumably is related to underlying neurological status. To what degree such unsteadiness is genetically controlled or environmentally determined is unknown. Ataxia is the medical term for staggering; static ataxia is staggering while standing still. Some

degree of body sway is found in everyone and each of us has a baseline measure of it. Schuckit and Gold (1988) found no difference in baseline measurements of body sway between FHPs and FHNs. They did find a significant difference in the effect of alcohol on body sway in FHPs and FHNs. The high-risk subjects showed significantly less increase in static ataxia after a challenge dose of ETOH. Other research (National Institute on Alcohol Abuse and Alcoholism, 1988) has demonstrated significantly higher baseline body sway in children of alcoholics, and it has been suggested that static ataxia could serve as a biological marker for alcoholism.

Serum Hormone Levels Schuckit and Gold (1988) also found significantly lower blood plasma levels of the hormones *prolactin* and *cortisol* after challenge doses of ETOH in FHP subjects. The significance of these possible biological markers for alcoholism is not clear; however, persistently low levels of serum prolactin and serum cortisol are found in abstinent alcoholics and may play a part in the protracted withdrawal syndrome discussed in Chapter 3.

Subjective Experience and Objective Measures of Effects of Drinking There is a whole set of research findings suggesting that those who are at high risk for alcoholism have a "hollow leg" (that is, they experience fewer adverse effects than do other drinkers from the same dose of ETOH). They also feel less drunk. Here science supports the folk wisdom of AA, most

of whose members report that they could really "sock it away" early in their drinking careers. It makes sense that someone who can easily drink a lot of an addictive drug will be more likely to become addicted to it. Schuckit and Gold's (1988) research supports this. It is one of their findings about the differential response of FHPs and FHNs to challenge doses of ETOH. In fact, the FHPs reported feeling not only less impaired but also better. That is to say that alcohol is highly reinforcing for these subjects. Surprisingly, Schuckit and Gold found objective correlates for these subjective reports. The FHPs either suffered less impairment or improved on a variety of objective tests of cognitive functioning and motor performance. This may be because alcohol is highly anxiety reducing for high-risk subjects. If so, this is at variance with the research of Mello and Mendelson (1970) showing that alcoholics are more, not less, anxious on objective measures of anxiety after drinking, although the alcoholics themselves report the opposite. Apparently, alcoholics react differently to alcohol after they develop their disease than they did before, at least with regard to anxiety. This may explain one mechanism of addiction. If those who "benefit" most from drinking drink more to obtain those benefits but lose them in the process, they may nevertheless continue to search for the old rewarding experience by continuing to drink long after such reinforcement is obtainable. This is congruent with AA folk wisdom that points to the already mentioned high capacity for drink—the hollow leg—and the futile search for the old magic as antecedents of alcoholism. Both

conditions would set one up for alcohol addiction.

Electroencephalograph Studies The electroencephalograph (EEG) records brain waves, usually by using scalp electrodes to detect the electrical activity of the brain. What is recorded are averages of the electrical activity of millions of neurons. Sometimes the averaging is done by the brain itself, so what is present at the electrodes is the averaged potential. Sometimes the raw input to the EEG is further processed by computer averaging, so that the output is interpretable. There are two main EEG findings about alcoholics: the first concerns *alpha waves* and the second *event-related* or *evoked* potentials.

It has been shown (Pollock et al., 1983; Propping, Kruger, & Mark, 1981) that Cloninger's type 1 milieu-limited alcoholics have low rates of slow alpha waves, high rates of fast alpha waves, and poor synchrony of those waves when they are abstinent. This may be true of other alcoholics as well. Alpha waves are characteristic brain wave patterns found in everyone. Subjectively, minimal poorly synchronized slow alpha activity and excessive poorly synchronized fast alpha activity are experienced as dysphoric. Such a pattern is a neuroelectrical correlative of tension. Type 1 alcoholics show a marked increase in slow alpha activity, which also becomes better synchronized, and a marked decrease in fast alpha activity when they drink ETOH. Again, there is evidence that this may be true of other alcoholics as well. Subjectively, these changes are experienced as calm alertness and relief of tension. In other

words, their anxiety levels drop when they drink. Cloninger (1987b) calls this type of anxiety *cognitive anxiety*. Cognitive anxiety is characterized by anticipatory worry and guilt. This suggests that for some alcoholics excessively high levels of anxiety are antecedent to their alcoholism and that alcohol was a particularly effective antianxiety drug for them, at least before their disease progressed. Nonalcoholic FHP subjects have a greater increase in slow alpha tracings and a greater decrease in fast alpha tracings when they drink than do controls (that is, alcohol is more reinforcing for them). Women FHPs were found to have minimal slow alpha activity, suggesting that they would be more subject to type 1 alcoholism, which is exactly what epidemiological research shows.

Evoke (or *event-related*) potentials are spikes in the EEG that reflect brain activity in response to a visual or auditory stimulus that is either unpredictable or task relevant but not usual. Porjesz and Begleiter (1983) found that both sons and daughters of alcoholic fathers have significantly higher amplitudes of event-related potentials. They are *stimulus augmenters* —they who experience stimuli with particular intensity. This is a neuroelectrical measure of stimulus augmentation, but alcoholics have also been found to be stimulus augmenters on other measures of this trait. Cloninger type 2 (male limited) alcoholics are stimulus augmenters in terms of the amplitude of their evoke potentials when abstinent. Alcohol either decreases their augmentation or changes it to *stimulus attenuation*, a

decrease in *stimulus reactance* that is assumed to be pleasurable and reinforcing. Cloninger (1987b) speaks of his type 2 alcoholics as suffering from *somatic anxiety* (bodily tension) in contrast to the cognitive anxiety of type 1 alcoholics. In both cases, alcohol consumption reduces anxiety: in the first by reducing stimulus augmentation (amplitude of the evoke potential) and in the second by reducing fast alpha brain activity and synchronizing it. Anxiety reduction is reinforcing, and such mechanisms could predispose to alcoholism. Female relatives of type 2 alcoholics have significantly higher rates of somatic anxiety but not of alcoholism than do controls.

An evoke potential known as the *P3 (or P300) wave* has received special attention. There have been several important findings, including the nonreversible flattening in the amplitude of P3 in alcoholics and decreased P3 in the sons of alcoholics (Begleiter, Poijesz, Bihari, and Kissen, 1984). Since P3 is an orienting response, this may correlate with findings (Tarter, 1981) of *attention-deficit disorder* (ADD) in alcoholics shown to have been hyperactive in childhood. Hyperactivity is believed to be antecedent to alcoholism in a considerable number of male alcoholics who are probably Cloninger type 2s for the most part. Tarter thinks their hyperactivity may be etiological or at least predisposing to alcoholism.

The National Institute on Alcohol Abuse and Alcoholism (NIAAA) (1988) found cognitive deficits of various sorts (for instance, impaired problem-

solving ability) in nonalcoholic sons of alcoholics. It is hypothesized that these deficits may be manifestations of the same underlying factor that manifests itself in hyperactivity and abnormal P3 waves. Since the NIAAA (1988) also reports studies that it found no significant difference between the cognitive abilities of children of alcoholics and those of children whose parents are not alcoholics, these studies are difficult to interpret. It is not known if the deficits found are the result of environmental or genetic factors or an interaction of the two.

Most of the biological marker research demonstrates something about high-risk males. Research currently is beginning to show similar differences in response to ETOH in daughters of alcoholics.

Family Studies

Family studies (Bleuler, 1955; Amark, 1951; Pitts & Winokur, 1966) consistently show an increased incidence of alcoholism in relatives (parents and siblings) of alcoholics compared with various control groups or the general population. Such studies show a greater risk for male relatives (fathers and brothers) than for female relatives (mothers and sisters), and a higher risk for both male and female relatives than for control groups. These findings do not shed light on how alcoholism is transmitted, whether by culture, learning, or genetic factors. They do establish beyond doubt that

children of alcoholics are at risk for alcoholism.

Studies of Twins Studies of twins have contributed evidence for a genetic factor in alcoholism. Such studies are conducted by calculating the concordance between identical (*monozygotic*) and fraternal (*dizygotic*) twins for alcoholism; the *concordance rate* is the percentage of twins sharing a given trait or condition. In this case, the percentage of alcoholic twins with an alcoholic twin is calculated for populations of identical and fraternal twins. The concordance rates are then compared. Since identical twins are the product of the same fertilized egg, or *zygote*, and fraternal twins are the product of different fertilized eggs, a higher concordance between identical than fraternal twins is taken as evidence of a genetic factor in the transmission of the trait or condition. The results consistently show that identical twins of alcoholics have a statistically significantly higher incidence of alcoholism than do fraternal twins of alcoholics. In a typical study, Kaij (1960), using male twins, found a concordance of 53.5% in identical twins and a concordance of 28.3% in fraternal twins. However, too much should not be made of this evidence for a genetic factor. Environmental factors, including the fact that identical twins are more likely to be treated alike, confound such studies.

Studies of Adoptees A promising research design is to study the children of alcoholics who were adopted very early in life by nonalcoholic

adoptive parents, following them into adulthood and determining their rates of alcoholism and comparing those rates to their generational peers. The first and one of the most important of these adoption studies was conducted by Goodwin, Schulsinger, Hermansen, Guze, and Winokur (1973) in Denmark. Goodwin et al. followed children of alcoholics who were adopted at or shortly after birth and raised by nonalcoholic parents. An early study (Roe, 1945) found almost no alcoholism in children of alcoholics who were raised by nonalcoholic adoptive parents. This result may be confounded by the disproportionate number of girls in the study. Goodwin et al.'s results were the opposite. In their study, chronic alcoholism was four times more common in 55 adopted-out sons of alcoholic fathers than among 78 adopted-out sons of nonalcoholics. The sons of alcoholics had a 25% rate of alcoholism—higher than the 17% rate Goodwin et al. found for male children of alcoholics raised by those alcoholics. This, of course, means that 75% of the sons of alcoholics raised by nonalcoholic adoptive parents did not become alcoholic; therefore, simple Mendelian inheritance of alcoholism (that is, that alcoholism is caused by a gene) is not the case. Moreover, alcoholism was not found to be significantly more prevalent in adopted-out daughters of alcoholics raised by nonalcoholics. It is of considerable interest that Goodwin et al.'s adopted-out male children of alcoholics had significantly higher rates of hyperactivity, shyness, sensitivity, and aggression than adopted-out male children of nonalcoholics. Adoptees whose biological parents were not alcoholics but

who were raised by alcoholic parents did not have significantly higher rates of alcoholism. In contrast to their findings on alcoholism, Goodwin et al. found no correlation between problem drinking (alcohol abuse that did not qualify as alcoholism as they defined it) and alcohol abuse in biological parents.

A Swedish study conducted by M. Bohman (1978) and reported by Cloninger (1983) extended and confirmed Goodwin et al.'s findings. It was based on a much larger sample (862 men and 913 women) of known paternity born to single women between 1930 and 1949. As previously noted, Cloninger found a high correlation between what he called male-limited susceptibility (or type 2 alcoholism) in biological fathers and type 2 alcoholism in adopted-out sons of these fathers who were raised by nonalcoholic adoptive parents. It is early-onset severe alcoholism associated with antisocial and even overtly criminal behavior. Type 2 alcoholism was found only in males; the sons of type 2 fathers raised in nonalcoholic families had *nine* times the rate of alcoholism of the sons of all other fathers in the study. Approximately half of the adopted-out sons of type 2 alcoholics became alcoholic, but half did not, so once again the evidence does not support direct Mendelian inheritability but, rather, indicates that in one form of alocholism *biological vulnerability* to alcoholism is inherited. Goodwin (1988) speculates that what makes for the vulnerability may be low levels of the neurotransmitter serotonin, citing evidence that alcoholic rats have low levels of serotonin in certain parts of their brains and that serotonin reuptake

blockers like Prozac decrease appetite for alcohol in these rodents. Since alcohol increases serotonergic activity initially, it might be highly appealing to a person with a serotonergic activity deficit, while its biphasic impact on serotonin levels results in its ultimately decreasing serotonergic activity, setting up a vicious cycle resulting in addiction. Goodwin acknowledges the highly speculative nature of this theory of what is inherited. None of the environmental variables measured by the researchers significantly influenced the appearance of this type of alcoholism, increasing the likelihood that it is highly heritable. The Swedish data also indicated that type 2 alcoholism is extremely treatment resistant. An American longitudinal study (Vaillant, 1983) did not find any worse outcome for what it defined as sociopathic alcoholics, at least with regard to recovery from alcoholism, than for its nonsociopathic alcoholics. This is puzzling, since Cloninger's type 2s are presumably similar to Vaillant's sociopaths.

Adopted-out sons and daughters of type 1, late-onset alcoholic parents, who tended to be approval seeking as well as to worry and be guilt prone, were also significantly more likely to develop alcoholism. The type of alcoholism they developed was the same as that of their biological parents. However, this occurred *only* if they were reared in adoptive homes where heavy drinking was the norm. The actual environmental variable measured was working-class, lower socioeconomic status which the researchers argued went with a heavy drinking lifestyle or at least with approval of intoxication

as recreation and relaxation. This type of alcoholism was called *milieu-limited susceptibility* and was far less heritable. A reanalysis of the Swedish data (Cloninger, Bohman, & Sigvardsson, 1981) indicated a correspondence between type 1 alcoholism in the mothers and type 1 alcoholism in the adopted-out daughters.

Like Goodwin et al., the Swedish investigators found that children whose biological parents were not alcoholic but whose adoptive parents were did not develop alcoholism at rates significantly higher than children of nonalcoholics raised by nonalcoholics. They concluded that alcoholism in children of alcoholics is not transmitted by learning, modeling, or unconscious identification, let alone by the maladaptive use of alcohol to ameliorate the pain of being raised in an alcoholic home, although environmental provocation is necessary for milieu-limited alcoholism to occur. However, there may be a confounding variable here. It is known that many children of alcoholics become teetotalers, and it may be that the environmental, emotional, and interpersonal, as opposed to the genetic, influences of parental alcoholism may be to increase the likelihood of *either* alcoholism or total abstinence, while decreasing the likelihood of becoming a social drinker. This hypothesis was not tested in either the Danish or the Swedish studies. A more recent American study (Cadoret, O'Gorman, Troughton, & Heywood, 1984) using the same design found that adopted-out sons of alcoholics were three times as likely to develop alcoholism as adopted

sons of nonalcoholics. An important additional finding was that these Iowa children of alcoholics had a significantly higher rate of *conduct disorder* than their peers, a finding congruent with the high rates of hyperactivity in the Danish study and with retrospective evidence (Tartar, 1981) of childhood hyperactivity in clinical alcoholic populations.

Goodwin's isn't the only theory of biological vulnerability, and alcohologists have argued about what, if anything, is inherited in alcoholism. A variety of suggestions have been made. The alcoholics in the Danish study were Winokurian primary alcoholics, and Winokur (1974) has argued that there is a genetically transmitted *depressive spectrum* illness in which women are at risk for unipolar depression and men are at risk for alcoholism or sociopathy. He seems to argue that a common mechanism predisposes them to these diseases, but he does not specifiy what that mechanism might be. Others have pointed to the association of childhood hyperactivity and adult alcoholism found in the Danish study and elsewhere as a clue to what might be transmitted. However, hyperactivity has not been consistently found in the childhoods of alcoholics.

What conclusion can be drawn from this? The best evidence we have, which is fragmentary and based on small samples, shows that a predisposition to some forms of alcoholism is inherited. Alcoholisms can probably be arranged in a continuum ranging from those in which

constitutional factors play little or no role to those in which constitutional factors play a vital role. One third of alcoholics report no family history of alcoholism. From a clinical standpoint, the most important finding of these studies is that children of alcoholics are at extremely high risk for alcoholism, even if this predisposition is not necessarily exclusively genetic. Treatment is not importantly affected by the presence or absence of constitutional factors in the etiology of a particular person's alcoholism, although familial alcoholism, particularly if it is early onset, increases the odds that the patient can never drink "safely."

EMPIRICAL PSYCHOLOGICAL FINDINGS

There are few consistent empirical psychological findings in alcoholic populations. However, the few facts that have been determined do hold up across studies and populations. Unfortunately, almost all of the studies are about male alcoholism. Recent and current research seeks to remediate this but has thus far produced limited data. Furthermore, for the most part the existing studies are about the characteristics of men *after* they have become alcoholic; that is, insofar as the studies are descriptive of an "alcoholic personality," they describe the clinical alcoholic personality, not the prealcoholic personality). Given these limitations, it is nevertheless known that alcoholics have elevated psychopathic deviance scores on the Minnesota Multiphasic Personality Inventory (discussed below). They are also field

dependent (a concept discussed later in this chapter) on a variety of measures, have low self-esteem and impoverished self-concepts, manifest various symptoms of ego weakness, frequently have a confused or weak sense of identity, including sexual identity, and are stimulus augmenters (discussed both in this and in the previous chapter).

Minnesota Multiphasic Personality Inventory

The *Minnesota Multiphasic Personality Inventory* (MMPI) is a 550-item self-report widely used in both personality assessment and research. Subjects respond to each item by indicting if it is true of them. Like all self-reports, it is limited by the subjects' self-knowledge and by their conscious and unconscious desires to "fake good" or "fake bad." Subjects' responses to the items are reported as scores on 11 scales, including psychopathic deviancy, depression, hypomania (a mild mania), masculinity-femininity, hypochondriasis, paranoia, and psychasthenia (neurosis). The most consistent and frequently replicated MMPI finding with alcoholic populations is significant elevation of the *psychopathic deviate* (Pd) scale score. This finding goes back to Hewitt's 1943 study of an early AA group in Minneapolis. Subsequent MMPI studies of a wide variety of alcoholic populations have also reported elevated Pd. What does this mean? An examination of the Pd items reveals that a number of them refer to excessive drinking and others to situations likely to be associated with heavy drinking. Are the elevated Pd

findings trivial? Not necessarily. Later investigators (MacAndrew & Geertsma, 1963; MacAndrew, 1965) modified the Pd scale to eliminate these items, and the findings of elevated Pd held. The most reasonable interpretation of the elevated Pd scores is that alcoholics tend to have a "devil may care" attitude, or at least they say that they do. This could be seen as a tendency toward mildly sociopathic behavior. Interestingly, Pd scores fall, but not to the average range, with sobriety. It is the abnormal personality measure most resistant to change with continuing sobriety, psychotherapeutic treatment, and participation in AA. Within limits this can be a strength in our society. Furthermore, it is known that at least some prealcoholics also show elevated Pd scores on the MMPI. The University of Minnesota once required entering freshmen to take the MMPI, and Loper, Kammeier, and Hoffman (1973) back-checked the MMPIs of the University of Minnesota graduates admitted to the university hospital for treatment of alcoholism. These alcoholics showed significantly elevated Pd scores 13 years earlier when they were college freshmen. Their average Pd scores were significantly higher than the usual high scores of young males. Thus, there is something extremely persistent and characteristic of (male) alcoholics that is measured by this scale. It is antecedent to their alcoholism, accompanies it in its active phase, and persists with recovery. It is not known whether the presence of type 2 alcoholism accounts for the elevation of Pd scores. The Pd scores of female alcoholics are also elevated relative to nonalcoholic females, but neither their absolute

scores nor the differential is as high as that of male alcoholics. (The Pd scale is reproduced in Appendix 6A.)

The other consistent MMPI finding is elevation in the depression (D) scale in alcoholics. The elevation of depression is generally not as high as the elevation of Pd, but it is still at abnormal levels. Unlike elevated Pd, elevated depression does remit with enduring sobriety. The prealcoholic University of Minnesota students scored high on the hypomanic (Ma) but not the depression scale as freshmen; however, they had elevated scores on the depression scale when they were admitted to the hospital for treatment. This suggests that an acting-out hypomanic lifestyle may serve as a *manic defense* against an underlying depression. This hypothesis is controversial and much debated in the field—it is generally held by clinicians and rejected by researchers.

Clinical alcoholics also manifest elevated scores on scale 7, *Psychasthenia* (Pt), of the MMPI. The Pt scale is a measure of neuroticism indicative of high levels of anxiety, irrational fears, ruminative self-doubt, and self-devaluation. Obsessive worry, tension, indecisiveness, and concentration difficulties also correlate with high Pt scores. This finding is corroborated by the consistently high scores of alcoholics on the neuroticism scale of the *Eysenck Personality Inventory* (Cox, 1985). Alcoholics also score high on Zuckerman's (1979) *Sensation Seeking Scale*, a finding that corroborates the

widely reported elevation on Pd.

MacAndrew (1965) empirically derived a scale that distinguishes alcoholics from nonalcoholics by determining which MMPI items were responded to differently by alcoholic and nonalcoholic psychiatric inpatients. In that way he identified 49 items (exclusive of two that refer specifically to drinking), which became the *MacAndrew Alcoholism Scale* (MAC). Subsequent research has shown the MAC to be a highly sensitive measure of alcoholism, accurately identifying 85% of male alcoholics. MacAndrew called those who score high on the MAC *primary alcoholics*, using the term slightly differently than Winokur (see page 103). The MAC primaries are reward-seeking, bold, aggressive, impulsive, and hedonistic, sharing traits with high Pd scorers who are characterized by anger, resentment, complaints against family, rebellion against convention, and moodiness. (MacAndrew's items are given in Appendix 6B.)

MacAndrew also developed a scale of 18 MMPI items that identifies the 15% of male alcoholics not identified by the MAC scale. He called them *secondary alcoholics*. They are tense, fearful, depressed punishment avoiders. MacAndrew concluded that there are two types of male alcoholics: high rolling, devil take the hindmost, hell raisers; and depressed neurotics. You, the reader, may then say with Hamlet, "Who shall 'scape whipping?"; between the high rollers and the sad sacks, who is left. The answer is, among active

alcoholics, very few; among pre-alcoholics, we are not sure, and even if mild antisocial behavior and depression predispose to alcoholism, that still leaves multitudes who are neither acting out extroverts nor depressed introverts.

Field Dependence

The second important and consistent empirical psychological finding is that alcoholics tend to be *field dependent*. Field dependence refers to the way a person organizes his or her perceptive field. The concept of field dependence-field independence as a relatively enduring individual difference was developed by Witkin, Karp, and Goodenough (1959). Field dependence-field independence is a *cognitive style*, a way of structuring the experiential world. Cognitive styles manifest themselves in the characteristic ways in which a person establishes his or her spatial orientation and in the acuity of his or her figure and ground discrimination. The field-dependent person relies on the environment and on external cues, rather than on introceptive, internal cues, in orienting himself or herself in space. The field-independent person does the opposite. Witkin and Oltman (1967) argued that field dependence was one manifestation of a *global cognitive style*, while field independence was one manifestation of an *articulated* (that is, finely discriminated) style. The field-dependent person experiences events globally and diffusely, with the surrounding field determining the way those events are organized. The field-dependent person is less differentiated from the

environment than the field-dependent person, at least in terms of perceptual organization. Although field dependence-field independence is a dichotomous distinction, it is actually a continuous variable, with field dependence at one extreme and field independence at the other. Most individuals fall somewhere in between.

Field dependence-field independence is measured in several ways: by the *Rod and Frame Test* (RFT), the *Body Adjustment Test* (BAT), and the *Embedded Figure Test* (EFT). In the RFT, the subject sits in a darkened room and is asked to adjust a lighted rod to a true vertical position. The rod is surrounded by a lighted square that can be tilted to any angle. The field-dependent person will position the rod parallel to the square. Each subject has a limit (degree of tilt) beyond which they will not align the rod with the square. The field-independent person will position the rod vertically regardless of the tilt of the square. The degree of tilt to which the subject will rotate the rod is a measure of the person's field dependence-field independence. In the BAT, the subject, who is placed in a tiltable chair, must adjust his or her body to an upright position. The chair is positioned in a small room that can also be tilted. The degree from true vertical that the subject will say is upright is taken as a measure of field dependence. Clearly the BAT is similar to the RFT, with the body being the rod and the room the frame. In the EFT, the subject is asked to look for a simple geometric figure within a more complex figure in which it has been embedded. The score is the time taken to

find the embedded figure. All three tests measure the ability to differentiate a figure from an organized field, although the degree to which the RFT and BAT measure the same cognitive orientation as the EFT has been questioned.

Ever since Witkin et al.'s 1959 study, alcoholic populations have consistently been found (Goldstein, 1976) to score on the field-dependent side of the field dependent-field independent continuum. In most studies they have been found to be highly field dependent. Further, field dependence persists into recovery. Stably sober alcoholics also have been found to be field dependent, although the degree of dependency tends to decrease with continuing sobriety. It is important to note that these findings are statistical averages. They do not measure the field independence-dependence of any particular alcoholic, who may be field independent.

Shelden Pisani (personal communication, April, 1994) has recently compared four groups of alcoholism counseling students at the New School for Social Research using the EFT. Her groups were recovering males, recovering females, nonrecovering males, and nonrecovering females. Contrary to expectations, recovering females scored most field independent, while both male and female recovering students scored higher than their nonrecovering counterparts. The presence of presumably codependent women living with active alcoholics in the nonrecovering part of the sample may bias these findings. Although the differences in this small sample just

missed reaching statistical significance, they suggest that field dependence has psychosocial as well as organic determinants.

Unfortunately, there are no longitudinal studies in which field dependence-field independence was measured prior to the onset of alcoholism; it is not known, therefore, whether field dependence is a factor in the etiology of alcoholism, a consequence of it, or both. It is known that field dependent subjects have less-articulated and less-differentiated body concepts, as measured by the Draw-a-Person Test in which the subject is asked to draw a human figure, than do field-independent subjects and that this is consistent with other data on the personalities of alcoholics. Further, field dependence is correlated with susceptibility to social influence. The field dependent person looks to the social environment to determine what he or she is feeling. It is probably part of what AA is talking about when it speaks of alcoholics being "people pleasers." Witkin and Oltman (1967) believe that field dependence is correlated with interpersonal dependency. Other researchers disagree. Some researchers think that field dependence in alcoholics is the result of brain damage from drinking, since organically brain-damaged people are field dependent. However, there is no proof that brain damage is the primary cause of field dependence in alcoholics. It is of some interest that hyperactive children, who are postulated to be minimally brain damaged, are field dependent, and the few longitudinal studies that are available suggest that alcoholics tend to have been hyperactive children.

Researchers have argued from this and other evidence that field dependence is characteristic of the prealcoholic personality. People grow more field independent as they mature and then become less so as they age; it is as if life were a process of progressive differentiation that eventually reverses itself as dedifferentiation ensues. However, the field dependence of alcoholics is independent of age. What can be concluded is that alcoholics, whether as a cause or as a consequence, whether through a failure to differentiate or through dedifferentiation, are relatively undifferentiated from their physical and social environments and that this trait persists into sobriety.

Impoverished Self-Concept

Elevated MMPI Pd scores and field dependence are the most consistently replicated findings in alcoholic populations. There are also several other findings that surface with considerable regularity in the research literature. One is impoverishment of the *self-concept*. The self-concept is a person's conscious image of himself or herself. It is related but not identical to self-representation, which is an endopsychic (that is, mental) structure that may be unconscious, preconscious, or conscious. Because it lacks an unconscious dimension, the self-concept is an empirical psychological, rather than psychoanalytic, construct. It is usually measured by some form of self-report.

One of the most illuminating self-concept studies is that of Conner (1962). His form of self-report was an adjective checklist. Active alcoholics who were studied early in treatment checked very few adjectives as descriptive of themselves. Thus, the self-concepts of these barely sober alcoholics could be characterized as either not extensive or as impoverished. The adjectives they did check were either primary traits, those that are functional in primary groups such as adolescent peer groups, or neurotic traits such as "anxious" or "depressed." They did not check secondary characteristics, those necessary to function successfully in the impersonal organizations of the modem marketplace. The primary traits are global and diffuse, such as "nice guy" or "soft hearted" The secondary traits are specific and delimited, such as "active," "wide interest," and "logical." In other words, alcoholics thought of themselves as having traits that would enable them to enter into relationships characterized by lack of differentiation but not as having traits that would enable them to enter into the segmental differentiated relationships characteristic of the workplace. Thus, their self-concepts were impoverished, depressed, and diffuse. It could be argued that the primary relationship they sought was that of infant to mother. However, when Conner tested a group of recovering alcoholics who had been sober for three years and in AA, he found that their self-concepts were radically different from those of the active alcoholics with whom they were matched on demographic variables. The recovering alcoholics checked many adjectives;

that is, their self-concepts were far more extensive than those of the active alcoholics. The neurotic traits found in the actives' checklists were not present. Secondary traits were included, but the primary traits characteristic of the active alcoholics' self-concepts also appeared in the self-concepts of the recovering alcoholics. In my own research (Levin, 1981) I found a similar persistence of diffuseness, into sobriety, operationalized in several ways, in a sample of well-educated, middle-class alcoholics.

Impoverishment of self-concept is a finding that appears with great regularity in the literature on the alcoholic personality. There is no doubt that it is characteristic of the clinical alcoholic personality, but whether it is true of the prealcoholic personality is not known. Constriction of self-concept in alcoholics may be the result of either a regression in personality development or a *premorbid deficit*, that is, a lack that existed prior to the manifestation of alcoholism. Although impoverishment of self has been replicated in many studies, no good evidence exists that it is a premorbid trait of alcoholics; it may be a consequence of the alcoholism and the alcoholic lifestyle. Most likely it is both causal and consequential.

Closely related to impoverishment of self-concept is *low self-esteem*. This is also a consistent finding in a wide range of alcoholic populations. Low self-esteem in alcoholics has been found regardless of how self-esteem is measured.

Ego Weakness

Many personality studies have found evidence of ego weakness in alcoholics. This is true of research using objective measures and of research using projective instruments such as the Rorschach test. Ego weakness is manifested by impulsivity, the inability to delay gratification, low-affect tolerance, a propensity toward panic-level anxiety and prolonged depression, an unclear, confused sense of identity and lack of clear boundaries. Reality testing, an important ego function, is impaired in ego weakness.

The *Rorschach test* is an instrument used by psychologists to evaluate personality. It consists of a set of ten cards with ink blots on them. A subject is invited to say what he or she sees when shown the cards. Seven of the ten cards have color. The subject is assumed to project some aspect of self onto the cards in his or her responses; therefore, the Rorschach test is considered a *projective* test. The subject's responses are evaluated in terms of both their formal characteristics and their subject matter. Alcoholics show impaired reality testing on this test; that is, the things they see on the cards are not necessarily reasonable interpretations of what is there. Reality testing is measured as a percentage of *Good Form* (%F+) in the Rorschach protocol. Seventy to 80% F+ is considered optimal, higher percentages are seen as manifestations of rigidity and inability to regress in the service of the ego, (that is, to be playful and creative). According to this measure, alcoholics'

reality testing is not as low as that of psychotics, but it is not as high as that of nonalcoholics. This has important clinical implications. It confirms and complements the findings of cognitive deficit discussed in chapter 2, and it is additional evidence that poor information processing on both a neurological and a dynamic basis contributes to denial and resistance in clinical alcoholics.

Color on the Rorschach test elicits affective responses that are scored as %FC if integrated with form (for instance, "that is a pink rose") with form predominating; as %CF if integrated with form with color predominating (for example, "that's sand—it's kind of sand-colored there"); and as %C if unintegrated with form (for instance, "blood, it's red"). Alcoholics have difficulty putting form and color together in their responses. Their %FC is generally zero, they have few CFs, and if they respond to color at all, it is in C responses. So alcoholics' Rorschach protocols either have no color responses indicative of emotional blocking and repression, *or* they have one or more pure color responses indicative of an inability to contain feelings and/or being overwhelmed by them. *Lack of affect tolerance* means not being able to stay with feelings; instead, they are repressed, acted-out, or anesthetized (say, by alcohol). This is exactly the picture we get on clinical alcoholics' Rorschach protocols with their absence of all color responses or their presence of pure color (C) and absence of form color (FC) responses, a picture that is congruent with the lack of affect tolerance noted by clinicians and measured by objective tests of various sorts. Since color is an affective stimulus and

form a cognitive one, these results are interpreted as an inability to integrate feeling and thought. Both of the Rorschach findings of poor reality testing and lack of affect tolerance are indicative of ego weakness.

The Cattell *Sixteen Personality Factor Questionnaire* (16PF) is a psychometric instrument in widespread use in personality research. (Psychometric tests measure psychological traits.) Barnes (1979, 1983) reports that multiple studies consistently find that clinical alcoholics differ on up to 14 of the 16 scales on the 16PF. Of particular interest is the consistently low average scores of alcoholics on the C scale of the 16PF which measures ego strength and emotional maturity. Alcoholics also consistently score high on second order (derivative) measures of anxiety on the 16PF.

Another dimension of personality first elucidated and measured by Rotter (1966) is *locus of control*. Psychologists use a self-report to determine if a subject sees himself or herself as controlling or being controlled by a situation. Those who see themselves as in control of themselves and their actions are said to have an *internal* locus of control, those who see themselves as controlled are said to have an *external* locus of control. Externality is considered a sign of ego weakness. Almost all studies of alcoholics (Rohsenow, 1983) show them to have an external locus of control. Paradoxically, most studies of alcoholics early in sobriety who are patients in rehabilitation units show them to be moving toward greater externality as

they progress through rehabilitation. This is probably a result, however, of their relinquishing grandiosity, denial, and projective defenses. They are now more realistic in that they are aware that they are not in control of their drinking. Stable recovery results in a move toward internality.

The evidence is overwhelming that ego weakness is characteristic of clinical alcoholic populations; however, it is not known if it is characteristic of prealcoholic populations.

Antecedent Neurological Deficit

Tarter and Alterman (1989) have accumulated evidence from retrospective studies and studies of alcoholics' childhoods that learning difficulties and poor school performance are overrepresented in at least male alcoholic populations. They argue for a cluster of problems: hyperactivity, attention deficit disorder, and childhood conduct disorder being antecedent to some forms of alcoholism, and they hypothesized that both the childhood problems and the alcoholism are consequent upon an inherited neurological deficit. This would seem to be particularly the case with Cloninger's type 2 alcoholics. Other studies fail to confirm their findings on children of alcoholics and their theory has not found general acceptance. Most likely, they have identified the antecedents of one type of alcoholism; their dismissal of possible environmental causality, however, particularly given the fact that

most of these children grow up in alcoholic homes, is unconvincing. The most robust of their findings is the high prevalence of childhood *hyperactivity* in clinical alcoholic populations (established by retrospective study). Goodwin et al.'s (1973) Danish adoption study also found a correlation of hyperactivity and alcoholism, while Vaillant (see below) hedges on whether or not hyperactivity was a significant antecedent of alcoholism in his populations.

A possibly related and intriguing finding is the statistically significantly higher occurrence of *left-handedness* (London, 1990) in alcoholic populations. Left-handedness in alcoholics was highly correlated with alcoholism in the father and with Cloninger's type 2 male limited alcoholics. London believes that left-handedness is indicative of underlying difficulties with cerebral laterality (that is, differentiation of function between left and right hemispheres), possibly originating in abnormal intrauterine levels of hormones, especially testosterone. He hypothesizes that these factors may be operative in alcoholism, particularly type 2 alcoholism.

Confused Identity

There is considerable evidence of tendencies toward confused identity, including sexual identity, in alcoholics. Irgens-Jensen (1971) gave draftees into the Norwegian Navy the Draw-a-Person test. He found that those judged to be problem drinkers by a psychological interviewer or who became

problem drinkers during their tours of duty drew figures with many pathological features. These features can be interpreted as evidence of poorly differentiated, confused body images and insecure gender identity. Many studies, including those reviewed in this chapter, of clinical alcoholics using interviews, objective tests, and projective techniques support Irgen-Jensen's conclusions. Alcoholism counseling students, using the first edition of this text, who work as art therapists with alcoholics, have told me that the figure drawings that they see are strikingly similar to Irgen-Jenson's. Further, there is considerable clinical evidence that alcoholics suffer a great deal of sexual role conflict. Here the problem is not lack of gender identity but rather conflict over masculine strivings in female alcoholics and conflict over feminine characteristics in male alcoholics. These conflicts are, of course, endemic in our society during this period of changing role expectations. However, it is possible that those who suffer the most severe sex role conflicts turn to alcohol to attenuate the tension.

Stimulus Augmentation

The last important consistent finding in alcoholic populations is stimulus augmentation. The concept of *stimulus augmentation-stimulus attentuation*, discussed earlier in this chapter, was developed by Asenath Petrie (1978). She studied the way that subjects responded to the pressure of a wooden block pressed against their hands but out of their sight. It was

found that the perception of the size of the block and the intensity of the pressure was an individual difference that ranged along a continuum from those who perceived the block as greatly magnified and the pressure as highly intense (stimulus augmenters) to those who perceived the block as smaller and the pressure as less than it was (stimulus attenuators). Alcoholics are stimulus augmenters. Petrie's findings have been confirmed by EEG studies of evoke potentials. Since stimulus augmentation-stimulus attenuation is a relatively stable personal characteristic, there may be something either constitutional or acquired in alcoholics that leads them to experience stimuli in a particularly intense way. This would help explain their apparent lack of affect tolerance; the affects they experience may well be more intense. Although constitutional factors may play a role, from a psychoanalytic developmental standpoint stimulus augmentation can be seen as the consequence of failure to internalize the functions of the mother as a "stimulus barrier."

Cox (1987), reviewing the empirical psychological findings just presented and the longitudinal studies discussed below organizes these findings differently and concludes that there is compelling evidence of a prealcoholic (male) personality characterized by nonconformity, impulsivity, and reward-seeking characteristics, which would make for high scores on the MacAndrew primary alcoholism scale. There is even more compelling evidence that clinical alcoholics are characterized by negative affect

(depression and anxiety) and low self-esteem, as well as by a cognitive perceptual style that includes field dependence, external locus of control, and stimulus augmentation. The negative affect and low self-esteem are characteristic of MacAndrew's secondary alcoholics for whom they are antecedent. Cox points out that the proportion of male and female alcoholics in each group is quite different, with many more females being secondary alcoholics. He argues that negative affect and low self-esteem are consequences in primary alcoholics although they may be antecedents in secondary alcoholics. Citing Tarter and Alterman's (1989) studies, he speculates that the alcoholic perceptual style may be largely antecedent and be a manifestation of a specific inherited neural dysfunction. Other investigators see the alcoholic perceptual style as either a consequence or as importantly environmentally determined. In this chapter, my category of ego weakness cuts across Cox's organization of the data, but we are in agreement on the factual content of the empirical psychological research.

LONGITUDINAL STUDIES

Most of the findings so far discussed, except the Loper et al. MMPI studies, are about the clinical alcoholic personality. To determine what, if anything, is characteristic of the prealcoholic personality, longitudinal studies that follow a population sample from childhood through adulthood are needed. Such studies permit examination of the childhood characteristics of

people who later become alcoholic. There are few such studies. Besides the MMPI studies, there are essentially four: (1) the McCord and McCord (1960) study of Cambridge, Massachusetts, blue-collar boys; (2) Robbins, Bates, and O'Neill's (1962) study of child guidance clinic clients; (3) Jones's (1968, 1971) Oakland Growth Study, which is more middle-class and includes girls; and (4) Vaillant's (1983) study of Harvard graduates and Cambridge working-class men. The Danish, Swedish, and Iowa adoption studies discussed earlier are also longitudinal in design, but deal only with high risk subjects.

For the most part, these studies have shown that prealcoholics, those who later become alcoholic, were outwardly confident, nonconformist, rebellious, acting-out children and adolescents. Their personality profiles are similar to those of predelinquent or mildly delinquent youngsters. They tended to be restless, active (perhaps hyperactively so), and quite possibly angry. This is true of the Loper et al.'s (1973) psychopathically deviant, hypomanic middle-class college students, of McCord and McCord's working-class boys, of Jones's lower-middle and middle-middle-class junior high school boys and girls, of Robbins et al.'s child guidance clinic clients, and of Vaillant's blue-collar New England boys. In short, all of these prealcoholics resembled Blane's counterdependent alcoholics, Winokur's psychopathic alcoholics, and, to a lesser extent, Cloninger's male limited type 2, rather than the depressed, anxious, dependent clinical alcoholics lacking in self-esteem found in so many other studies. The picture also lends support to Tarter's

(1981) retrospective study that found a high correlation of childhood hyperactivity with adult alcoholism. What does all of this mean? Vaillant thought and vehemently argues that these results vitiated the dependency conflict theory (see McCord & McCord, below) of the etiology of alcoholism. Most of the other authors of these studies thought otherwise; they believed that the childhood profiles of their alcoholic subjects reflected a "reaction formation" against a deep-seated dependency conflict. In light of the open dependence and neediness of many adult alcoholics, this view is plausible. It is well known that mild and severe juvenile delinquency, as well as "acting out" in general, may be a symptom of masked depression. It may also be an expression of and a defense against underlying anxiety. The hyperactivity (Tarter, 1981), the hypomania (Loperet al., 1973), and the psychopathic deviance (Loper et al., 1973) found in alcoholics can be seen as manifestations of a "manic defense" against massive underlying depression. The hyperactive, hypomanic, unrestrained, shallow lifestyle these researchers found to be characteristic of prealcoholics is pathognomonic of a narcissistic personality disorder.

Seen in the light of the longitudinal data, Blane's (1968) counter dependent-openly dependent distinction is seen as a result of cross-sectional sampling. If Blane had followed his subjects longitudinally, he might have found many instances of early counterdependency as a manifestation of a manic defense against underlying depression, which then breaks down under

the psychophysiological assault on the integrity of the organism and its defenses by uncontrolled alcohol consumption. Eventually the defense fails; more general deterioration occurs; and depression, helplessness, and open dependence result. At least that is one way to explain the data. Let us look briefly at the individual studies.

McCord and McCord

McCord and McCord (1960) followed a population of white, predominantly Irish Catholic working-class boys who were at risk for delinquency from childhood into their 30s. They collected data from the boys' latency years in the 1930s through their early adulthoods in the 1950s. McCord and McCord (1962) described the childhood personalities of those who later became alcoholic as "outwardly self-confident, undisturbed by abnormal fears, indifferent toward their siblings, and disapproving of their mothers, . . . [They] evidenced unrestrained aggression, sadism, sexual anxiety, and activity rather than passivity" (p. 427). The McCords found that these active and aggressive children became dependent, passive, self-pitying, and grandiose after they developed alcoholism and that they also felt victimized by society. A combination of aggressivity and shyness made for the greatest risk of alcoholism. McCord and McCord theorized that this childhood pattern is a reaction formation to an intense unresolved *dependency conflict* resulting from inconsistent, erratic satisfaction of their childhood dependency

needs by their parents. Vaillant (1983) felt that the McCords explained away their actual findings on the basis of a theory. In his book *The Natural History of Alcoholism* Vaillant strongly argued that the McCords had no evidence of a dependency conflict. The point is important because it relates to the whole issue of the role of emotional factors in the etiology of alcoholism. I do not find Vaillant's criticisms convincing. The McCords did not have an antecedent theory into which they attempted to force their data, and they did have a great deal of highly specific data (gathered by "blind" interviewers, that is, interviewers who did not know the purpose of the study) on the home life of the children, which supported their interpretation of their findings.

Robbins, Bates, and O'Neil

Robbins, Bates, and O'Neil's (1962) study was based on a population of child guidance clinic clients, many of whom were referred for antisocial behavior. As such, it is a highly biased sample, and not surprisingly Robbins et al.'s findings on the childhood personalities of future alcoholics were very similar to those of the McCords. The boys who later became alcoholic were more active (or hyperactive), more acting out, and more aggressive than her population as a whole but less so than those who later became sociopathic. This was also true of the few girls in the study who later became alcoholic. Robbins et al. found that low family status, parental inadequacy, antisocial fathers, and antisocial childhoods all increased the likelihood of adult

alcoholism.

Jones

Jones (1968, 1971) followed Oakland, California, students from junior high school into adulthood. Hers is the only longitudinal study that includes a significant number of women. Although Jones's sample size was very small, it is probably no accident that her findings on the boys who later became alcoholic were virtually identical to those of the McCords and Robbins et al. The girls who later became heavy social drinkers were "expressive, attractive, . . . and buoyant" (p. 62); that is, they tended to have high levels of activity, but the girls who became alcoholic were "self-defeating, pessimistic, withdrawn, guilty, and depressive" (p. 62). Jones's findings are especially significant because her subjects were more middle class and they were not at risk for delinquency or in trouble at the time they were initially studied.

Vaillant

The most recent longitudinal study, which is based on the largest samples, is that of Vaillant (1983). Using *multiple regression research methodology* Vaillant sought to determine what antecedent variables determined what *percentage* of the variance in adult alcoholism. Percentage of variance is the square of the correlation between two variables, such as, disturbed childhood environment and alcoholism. Correlation is measured in

terms of a correlation coefficient that can vary from negative one (a perfect inverse correlation) to zero (no correlation) to positive one (a perfect correlation). The order in which variables are fed into the regression analysis influences the results since variables entered later can only account for a percentage of the remaining variance. That is, if all of the alcoholism is accounted for by the first four variables entered then nothing is left over to be accounted for by a fifth variable for even though it might have accounted for a percentage of the variance if entered first in the analysis. The researcher can also calculate a "regression equation," in which the coefficients, called *beta weights*, of each variable determine how much that variable contributes to the value of the dependent variable, in this case alcoholism. Beta weights do not depend on the order in which the variables enter the analysis. The point of all this statistical detail is that the methodological problems facing longitudinal researchers are formidable and that the data analysis chosen influences the results.

Vaillant reported on two relatively large research samples. One consisted of Harvard University students chosen for their mental health who were followed from their sophomore years into their 50s, and the other consisted of normal core-city working-class subjects who were followed from their childhoods into their 40s. Vaillant found that childhood and adolescent emotional problems and overtly disturbed childhoods did not predict (correlate with) adult alcoholism in either sample, although such disturbed

childhoods did predict adult mental illness. Vaillant found that *ethnicity* (Irish or northern European ancestry) and *parental alcoholism* did predict adult alcoholism. He argued that the clinical alcoholic personality is the result of drinking, not of premorbid personality factors. Vaillant also argued that adult alcoholics retrospectively falsify the degree of pathology in their childhood environments in order to rationalize their drinking. I have found that retrospective idealization of their childhoods is at least as characteristic of adult alcoholic patients as is retrospective devaluation or denial of whatever may have been positive in their childhoods; this, however, is a clinical not a research finding. Vaillant's data cannot be dismissed, but his interpretation of them is not entirely persuasive.

Zucker and Gomberg (1986) argue that methodological artifacts are responsible for Vaillant's conclusion that childhood experience and antecedent psychopathology play no role, or at least account for no variance, in adult alcoholism. They also suggest that he was looking for the wrong stuff, pathological dependency and negative affect, while if he had looked for Cox's stuff—nonconformity, impulsivity, and reward seeking at high enough levels to constitute an antisocial trend—he would have found it. In fact, they reanalyzed Vaillant's data (by changing the order of regression of his variables) and did find such childhood antecedents, including disturbed homes and psychopathology as they defined it, of alcoholism.

Interestingly, although Vaillant didn't find a correlation between disruptive childhood environment and alcoholism, he did find a correlation between disruptive childhood environment and early onset and severity of alcoholism. On the surface, this looks like Cloninger's type 2 (male limited) alcoholism, while Vaillant's data suggest an environment etiology for the severity since he controlled for parental alcoholism. However, Vaillant's analysis didn't support the type 1-type 2 distinction. It is a puzzlement.

Vaillant basically argues that culture (as manifested by ethnicity in his study) and parental alcoholism (seen as primarily contributing to inherited biological vulnerability) are far more powerful determinants of (male) alcoholism than psychological, let alone antecedent psychopathological, factors. But since parental alcoholism is almost certainly a determinant of Vaillant's variables—boyhood competence (ego strength), child-hood environmental strengths, childhood emotional problems, and childhood environmental weaknesses—their effect is already factored in by the variable parental alcoholism and add no independent proportion of variance when they are entered into the regression analysis. Another way of saying this is to say that Vaillant's independent variables are not actually independent of one another, rather they manifest what statisticians call *collinearity*.

Vaillant comes to other important conclusions including support for the notion that alcoholism is a unitary phenomenon. He reaches this conclusion

because measures of alcoholism drawn from sociological models and measures of alcoholism drawn from medical-disease models are congruent. Noting that it is the number of alcohol-related problems, not their severity, that determines when problem drinking progresses into alcoholism, he concludes that Jellinek's model of progression poorly fits his and others' longitudinal data, but in spite of this, he thinks that alcoholism is usefully conceptualized as a disease. He compares it to hypertension, which also has a fluctuating course, is powerfully influenced by behavioral and situational variables, and is often treatable by wise self care. His data also show that problem drinking, particularly in adolescents and young adults, does not necessarily progress to alcoholism. In fact, many "problem drinkers" return to asymptomatic social drinking. However, for those who are most symptomatic there is no going back; they have the disease. In both his samples, alcoholism took a long time to develop, and his data do not support Cloninger's distinction between type 1 and type 2 alcoholics. Further, those who did manifest antisocial behavior had just as high a recovery rate, in fact at a younger average age, as the population as a whole.

One of Vaillant's most intriguing, albeit incidental, findings was that *heavy cigarette smoking* in adolescence was an excellent predictor of (that is, it had a high correlation with) adult alcoholism. This suggests some sort of nonspecific (to any particular drug) addictive factor, constitutional or acquired, in those "prealcoholics." Problem drinking tended to remit (if it did)

in the late twenties after marriage had changed the problem drinker's peer groups and self concepts ("Those wedding bells are breaking up that old gang of mine"), while alcoholism, if it went into remission, did so at a much later age. The rates of spontaneous remission were 2 to 3% per year. The most common reason alcoholics became abstinent was ill health. Looking at all the data available, Vaillant notes the early morbidity of alcoholics (average age 52) and concludes that half of alcoholics either die of complications of alcoholism or become severely socially impaired, while the rest either become abstinent or in a small percentage of cases return to asymptomatic, or at least "controlled," drinking. Although advocating it, he questions the effectiveness of professional treatment, seeing AA as the single most efficacious "treatment."

Vaillant is undoubtedly right in his most salient conclusion that significant portions of the clinical alcoholic personality are caused by the drinking and remit with sobriety. However, he notes that full psychosocial recovery is slow and must be measured in years, not months. Clinicians and AA members would agree.

SPECIAL POPULATIONS

There is a growing literature on *special alcoholic populations*: women, Blacks, Hispanics, Native American, ghetto dwellers, professionals, teenagers,

and the elderly. However, little is actually known about these groups and alcoholism. Several of my alcoholism counseling classes have criticized my use of the term "special populations," particularly as applied to women, who constitute fully a third of alcoholics, an enormous number of people. I considered dropping it, but the term is so firmly ensconced in the literature that I have retained it in this edition.

Alcoholism in Women

The largest body of literature on special populations concerns women. At one time alcoholism was considered an almost exclusively male disease; it is now known that this is certainly not the case. It is also known that alcoholic women are more likely to suffer from depression than are alcoholic men. That is, women are *more* likely than men to be Winokurian depressive secondary alcoholics, and they are also *less* likely than men to be Winokurian secondary sociopathic alcoholics. In other words, they are far more likely to be MacAndrew secondary alcoholics. Women are more likely to drink to alleviate intolerable feelings of worthlessness—that is, they suffer more than male alcoholics from devastating low levels of self-esteem. Blane (1968) thought that women drink to deal with feelings of inferiority and that men drink to deal with repressed dependency needs. The research data, although fragmentary, supports him. Wilsnack (1973) thought that women drink to alleviate sex role conflicts and to feel more feminine. There is considerable

research evidence (Wilsack, 1991) that female alcoholics have a higher than average rate of gynecological problems, but it is not clear if this increases sex role conflict, makes them feel less feminine, or is an etiological factor in their alcoholism. It is also known that, at least until recently, women were more likely to become iatrogenically cross-addicted to minor tranquilizers. Winokur's data on the relatively high incidence of depression in alcoholic women is compelling; the other findings are more questionable. Women do appear to have more difficulty maintaining self-esteem in our society; it is not known, however, whether this struggle is commonly self-medicated with alcohol or whether such self- medication is an important factor in the etiology of female alcoholism. Given Winokur's data, it may be. It has consistently been found that women suffer *more somatic damage from lower doses of alcohol consumed for shorter periods of time* than do men. They are particularly vulnerable to liver damage. There is no question that women suffer early and more severe physical, emotional, and social consequences of their drinking.

More women are being diagnosed and treated for alcoholism than in the past. Whether this means that female alcoholism is more common or that it is simply "coming out of the closet" is not clear. Young women are drinking more, and heavy drinking by women in their twenties is reported in the recent prevalence literature. Whether this will eventuate in an "epidemic" of female alcoholism remains to be seen.

Women more often than men report a precipitating event (such as loss of a loved one or failure to conceive a child) for their alcoholism. Similarly, they are more likely to report traumatic childhoods. Whether this actually reflects the prevalence of these events in the lives of alcoholic men and women, or whether it is an artifact of the greater social shame associated with alcoholism in women and their concomitant need to find a "reason" and of men's greater reluctance to self-disclose is not clear. However, there is evidence (Wilsnack & Beckman, 1984) of high rates of childhood incest (sexual abuse) in alcoholic women. They are more likely than male alcoholics to have an alcoholic spouse. Black women are far more likely to be abstinent than White women, although there are indications that Black women who do drink are at higher risk to develop problem drinking than White women who drink. There is fragmentary evidence that Lesbian women have high rates of problem drinking and alcoholism.

Wilsnack's (1973, 1984) research using the *Thematic Apperception Test* (TAT), a projective technique in which subjects are asked to make up stories in response to a picture on a stimulus card, demonstrated that women who drank heavily in a simulated social situation drank to feel more feminine, or at least their stories, which were assumed to be projections of their inner feelings, dealt with "feminine" material and themes. Wilsnack hypothesized that problem drinking in women is correlated with sex role conflict. Although there are other studies that tend to confirm her hypothesis, Wilsnack's data

are too fragmentary to permit any broad conclusions. One wonders if this finding would be replicated today.

Whether or not treatment of female alcoholism should be distinct from treatment of male alcoholism is a vexed question. I have had several female alcoholic patients reject AA, seeing it as a male oriented ideology. In particular, they objected to the notion of "surrender" and admitting they are "powerless" (see chapters 9 and 12), saying that while that may have been just the thing for male alcoholics, they had spent their lives in a state of powerlessness and were not about to surrender the power they had fought so hard to acquire (Nancy Roberts, personal communication, March, 1993). A recent issue of the *AA Grapevine* was largely devoted to letters for and against revising the AA "Big Book," *Alcoholics Anonymous* (Alcoholics Anonymous World Services, 1955), to remove sexist language and outlook.

Adolescent Problem Drinking

The data on adolescents show that problem drinking by youths is extremely common and that it is not predictive of adult alcoholism. Although this manifestation of rebellious acting out can have serious consequences, as when kids drive and drink, it does not in itself mean very much. However, those who have a family history of alcoholism *do* increase their risk of adult alcoholism if they drink heavily as teenagers. For this reason, alcohol

education can help prevent alcoholism in this population. On the other hand, diagnosing problem drinking adolescents as having a "disease" tends not to be helpful and is mainly untrue. Vaillant (1983) advocates teaching young people how to drink in a socially controlled and acceptable manner as the best prophylactic against problem drinking. Moderate drinking, rather than abstinence, is frequently the treatment goal with this population.

Alcoholism in Minorities

Plantation owners supplied their slaves with alcoholic beverages and encouraged holiday drinking as a cheap form of pacification. How, if at all, this influences contemporary Black drinking practices is unknown. Many black churches preach abstinence and this is reflected in the high abstinence rates among blacks.

The data on Blacks show that Black males have a lower rate of alcoholism than do White males, but that Black women, if they drink, have higher rates of alcoholism than do White women. Since many Black women have been forced to be the breadwinners in single-parent homes, this finding is consistent with Wilsnack's hypothesis. It is possible that Black women forced into traditionally male roles drink to feel more feminine. There can be no doubt that alcoholism among poor urban Blacks and demoralized populations such as Native Americans is connected with feelings of

hopelessness and helplessness. It is anomic drinking. Alcoholism serves as a passive-aggressive expression of rage and as a means of anesthetizing that rage.

Blacks suffer more than Whites from the medical complications of alcoholism. This is especially true of cirrhosis. Whether this difference is genetic-biological or economic-social is not known, but the differential rate is well established (Lex, 1985). Middle-class Blacks are often "top shelf' drinkers and the social status so accrued may contribute to denial if they develop alcoholism. Hispanic males, who have high rates of alcoholism, are postulated to have a unique drinking pattern related to machismo, but there is little research evidence for this, so the degree of truth, if any, in this stereotype is not known. Hispanic women tend to abstain.

Alcoholism in the Elderly

The most important finding about alcoholism in the elderly is that it exists. Most elderly alcoholics are survivors of a lifelong career of alcohol abuse, but some are newly recruited to the ranks of the alcoholic because of their inability to handle the losses of later life and the narcissistic blow inflicted by retirement. Thirty percent of the residents of one Florida retirement community drank daily, but it was not clear if this was detrimental, or to how many it was detrimental. Since relatively small

quantities of alcohol can cause serious physical damage in the elderly, it is especially important that this syndrome be recognized and treated. I once treated a retired woman librarian in her 70s who had developed late-onset alcoholism when she could not adjust to retirement. She was a classic "old maid," prim, proper, and rather supercilious. She responded to treatment, became sober and joined AA, where she met a hell-raising retired sailor who had been in more beds, in more ports, than she had books in her library. They fell in love, married, and lived happily for eight years until he died of a heart attack. She is still sober and active in AA.

Alcoholism in the Disabled

Another population that has been underserved both in terms of treatment and in terms of investigation by researchers is the disabled. Although the scientific evidence is lacking, it would appear from the reports of clinicians and administrators of chronic care facilities and rehabilitation agencies that the prevalence of alcoholism among the disabled is high. The alcoholic disabled population is bimodal, consisting of those whose disabilities have been caused by or are related to their drinking and those who turned to alcohol in a futile attempt to cope with their disabilities. Recently, this population has finally begun to receive alcoholism treatment, as witnessed by the New York University Department of Rehabilitation Counseling's initiating a combined master's degree in rehabilitation and

alcoholism counseling.

Alcoholism in the Mentally Ill

In recent years there has been much emphasis on treatment of *dual diagnosis patients*, those who suffer from both alcoholism and mental illness. The prevalence rates of alcoholism in those suffering from major psychiatric illness has been estimated as high as 50 percent (Richards, 1993). Undoubtedly, part of this is attributable to unwise, poorly managed deinstitutionalization of psychiatric patients who lack the inner resources to cope with life on the "outside" and turn to alcohol and other drugs as both self-medication and as *prostheses* to provide what they cannot provide for themselves (such as self-esteem, inner calm, the ability to be aggressive) because of some inner, perhaps developmental, perhaps biochemical, deficiency. The drinking fails as both self-medication and as prosthesis and their condition worsens. The causal vector between mental illness and alcoholism may go in either direction, or in both directions, or the two conditions may be independent diseases. In any case, treatment must address *both* disorders or the prognosis is poor.

Although it is an advance that the coexistence of alcoholism and major mental illnesses such as schizophrenia is now widely recognized and addressed, I wonder if the high prevalence of dual-diagnosis disorder is not

partly an artifact of face saving by an alcoholism treatment community that, in response to a long history of ineffectual treatment based on resolution of underlying psychological conflict, had come to deny, or at least minimize, antecedent psychopathology in alcoholics. Having a formidable array of research as well as a clinical rationale on their side, they are reluctant to give up their hard won insight that "it's the booze that does it, stupid," and as we have seen, there is much truth in that viewpoint. However, the current focus on dual diagnosis now allows us to speak of anxiety disorders, obsessive-compulsive neurosis, depression, and personality disorders co-morbid with, and even perhaps antecedent to, alcoholism, without altering a basic, and tenuously held, conceptual framework.

We are appallingly ignorant about female, gay-Lesbian, Hispanic, and African American drinking practices and about alcoholism in these populations. The literature is scant and not very illuminating. Unfortunately, it also is not very helpful clinically. Its main contribution has been to make alcoholism counselors and mental health workers aware that alcoholism is not exclusively a disease of middle-aged, red-nosed, Irish men and of social outcasts. Sensitivity to culture, gender, age, environmental stress, and the effects of economic deprivation and racial discrimination is essential if a counselor or psychotherapist is to be effective. This is part of empathic listening.

APPENDIX 6A

MMPI Psychopathetic Deviate (Pd) Scale (50 Items)

	Response	Item
8	F	My daily life is full of things that keep me interested.
16	T	I am sure I get a raw deal from life.
20	F	My sex life is satisfactory.
21	T	At times I have very much wanted to leave home.
24	T	No one seems to understand me.
32	T	I find it hard to keep my mind on a task or job.
33	T	I have had very peculiar and strange experiences.
35	T	If people had not had it in for me, I would have been much more successful.
37	F	I have never been in trouble because of my sex behavior.
38	T	During one period when I was a youngster, I engaged in petty thievery.
42	T	My family does not like the work I have chosen (or the work I tend to choose for my life work).
61	T	I have not lived the right kind of life.
67	T	I wish I could be as happy as others seem to be.
82	F	I am easily downed in an argument.
84	T	These days I find it hard not to give up hope of amounting to something.
91	F	I do not mind being made fun of.
94	T	I do many things which I regret afterwards (I regret things more and

		more often than others seem to).
96	F	I have very few quarrels with members of my family.
102	T	My hardest battles are with myself.
106	T	Much of the time I feel as if I have done something wrong or evil.
107	F	I am happy most of the time.
110	T	Someone has it in for me.
118	T	In school I was sometimes sent to the principal for cutting up.
127	T	I know who is responsible for most of my troubles.
134	F	At times my thoughts have raced ahead faster that I could speak them.
137	F	I believe that my home life is as pleasant as that of most people I know.
141	F	My conduct is largely controlled by the customs of those about me.
155	F	I am neither gaining nor losing weight.
170	F	What others think of me does not bother me.
171	F	It makes me uncomfortable to put on a stunt at a party even when others are doing the same sort of things.
173	F	I liked school.
180	F	I find it hard to make talk when I meet new people.
183	F	I am against giving money to beggars.
201	F	I wish I were not so shy.
215	T	I have used alcohol excessively.
216	T	There is very little love and companionship in my family as compared to other homes.
224	T	My parents have often objected to the kind of people I went around with.
231	F	I like to talk about sex.

235	F	I have been quite independent and free from family rule.
237	F	My relatives are nearly all in sympathy with me.
239	T	I have been disappointed in love.
244	T	My way of doing things is apt to be misunderstood by others.
245	T	My parents and family find more fault with me than they should.
248	F	Sometimes without any reason or even when things are going wrong I feel excitedly happy, "on top of the world."
267	F	When in a group of people I have trouble thinking of the right thing to talk about.
284	T	I am sure I am being talked about.
287	F	I have very few fears compared to my friends.
289	F	I am always disgusted with the law when a criminal is freed through the arguments of a smart lawyer.
294	F	I have never been in trouble with the law.
296	F	I have periods in which I feel unusually cheerful without any special reason.

APPENDIX 6B

MacAndrew Alcoholism Scale (MAC)

	Response	Item
1	T	I have used alcohol excessively.*
2	F	I have used alcohol moderately (or not at all).*
3	T	I have had periods in which I carried on activities without knowing later what I had been doing.
4	F	I have never been in trouble with the law.
5	T	I have not lived the right kind of life.
6	T	I like to cook.
7	T	I sweat very easily even on cool days.
8	T	My parents have often objected to the kind of people I went around with.
9	T	I played hooky from school quite often as a youngster.
10	T	I would like to wear expensive clothes.
11	T	As a youngster I was suspended from school one or more times for cutting up.
12	T	While in trains, buses, etc., I often talk to strangers.
13	T	I pray several times every week.
14	T	I deserve severe punishment for my sins.
15	T	I have had blank spells in which my activities were interrupted and I did not know what was going on around me.
16	T	I have a cough most of the time.
17	F	I do not like to see women smoke.

18	F	My table manners are not quite as good at home as when I am out in company.
19	T	I have few or no pains.
20	T	I do many things which I regret afterwards (I regret things more or more often than others seem to).
21	T	I like to read newspaper articles on crime.
22	F	I am worried about sex matters.
23	T	My soul sometimes leaves my body.
24	T	Christ performed miracles such as changing water into wine.
25	T	I know who is responsible for most of my troubles.
26	T	The sight of blood neither frightens me nor makes me sick.
27	F	I cannot keep my mind on one thing.
28	T	In school I was sometimes sent to the principal for cutting up.
29	T	The one to whom I was most attached and whom I most admired as a child was a woman (mother, sister, aunt, or other woman).
30	F	I have more trouble concentrating than others seem to have.
31	T	I am a good mixer.
32	T	I enjoy a race or game better when I bet on it.
33	T	I enjoy gambling for small stakes.
34	T	I frequently notice my hand shakes when I try to do something.
35	T	Everything is turning out just like the prophets of the Bible said it would.
36	T	If I were in trouble with several friends who were equally to blame, I would rather take the whole blame than to give them away.
37	T	I was fond of excitement when I was young (or in childhood).
38	T	I have at times had to be rough with people who were rude or annoying.
39	T	If I were a reporter I would very much like to report sporting news.

40	F	I am certainly lacking in self-confidence.
41	T	I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off mistakes onto those under them.
42	T	I readily become one hundred percent sold on a good idea.
43	T	I think I would like the work a forest ranger does.
44	T	Evil spirits possess me at times.
45	F	Many of my dreams are about sex matters.
46	F	I liked school.
47	T	I have been quite independent and free from family rule.
48	F	I have often felt that strangers were looking at me critically.
49	F	I used to keep a diary.
50	T	I seem to make friends about as quickly as others do.
51	F	I have never vomited blood or coughed up blood.

* Not included in the MacAndrew Alcoholism Scale

Reprinted by permission from "The differentiation of male alcoholic outpatients from non-alcoholic psychiatric out-patients by means of the MMPI," by C. MacAndrew, 1965, *Quarterly Journal of Studies on Alcohol 26,* pp. 238-246.

About the Author

Jerome D. Levin, Ph.D., has treated addictions for over thirty years. He is the author of eleven previous books and has taught at Suffolk Community College, Marymount Manhattan College, St. Joseph's College, and the New School for Social Research, where he directed a program to train addiction counselors for over twenty-five years. He practices psychotherapy in Manhattan and Suffolk County, New York. You can contact Dr. Levin at jeromedlevin@gmail.com or (212) 989-3976.

References

Abraham, K. (1979). The psychological relations between sexuality and alcoholism. In *Selected papers on psychoanalysis* (pp. 80-90). New York: Brunner/Mazel. (Original work published 1908)

Alcoholics Anonymous World Services. (1952). *Twelve steps and twelve traditions*. New York: Author.

Alcoholics Anonymous World Services. (1976). *Alcoholics anonymous* (3rd ed.). New York: Author.

Alexopoulos, G. S., Lieberman, K. W., & Frances, R. J. (1983). Platelet MOA activity in alcoholic patients and their first-degree relatives. *American Journal of Psychiatry, 140*(11), 1501-1503.

Amark, C. (1951). A study in alcoholism: Clinical, social, psychiatric, and genetic investigations. *Acta Psychiatrica Neurologica Scandinavica, 70*, 1-283.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed. rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Babor, T. F., Wolfson, A., Boivan, D., Padouco-Thomas, S., & Clark, W. (1992). Alcoholism, culture, and psychopathology: A comparative study of French, French- Canadian, and American alcoholics. In J. E. Helzer & G. J. Canino (Eds.), *Alcoholism in North America, Europe, and Asia* (pp. 182-195). New York: Oxford University Press.

Bales, R. F. (1959). Cultural differences in rates of alcoholism. In G. McCarthy (Ed.), *Drinking and*

intoxication. Glencoe, IL: Free Press.

Balint, M. (1968). *The basic fault*. London: Tavistock.

Bandura, A., & Walters, R. H. (1963). *Social learning and personality development.* New York: Holt, Rinehart & Winston.

Barnes, G. E. (1979). The alcoholic personality: A reanalysis of the literature. *Journal of Studies on Alcohol, 40*, 571-634.

Barnes, G. E. (1983). Clinical and prealcoholic personality characteristics, In B. Kissin & H. Begleiter (Eds.), *The biology of alcoholism, Vol. 6. The pathogenesis of alcoholism: Psychosocial factors* (pp. 113-196). New York: Plenum.

Bateson, G. (1971). The cybernetics of "self': A theory of alcoholism. *Psychiatry, 34*, 1-18.

Bateson, G., & Ruesch, J. (1951). *Communication: The social matrix of psychiatry*. New York: W. W. Norton.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* New York: International Universities Press.

Begleiter, H., Porjesz, B., Bihari, B., & Kissen, B. (1984). Event-related brain potentials in boys at risk for alcoholism. *Science, 225*, 1493-1495.

Bentham, J. (1939). An introduction to the principles of morals and legislation. In E. A. Burtt (Ed.), *The English Philosophers from Bacon to Mill* (pp. 791-852). New York: The Modem Library. (Original work published 1789)

Berglas, S. (1985). Self-handicapping and self-handicappers: A cognitive/attributional model of interpersonal self-protective behavior. In R. Hogan and W. H. Jones (Eds.), *Perspectives in personality theory: Theory, measurement and interpersonal dynamics*. Greenwich, CT: JAI Press.

Blane, H. T. (1968). *The personality of the alcoholic: Guises of dependency.* New York: Harper & Row.

Bleuler, M. (1955). Familial and personal background of chronic alcoholics. In O. Drethelm (Ed.), *Etiology of chronic alcoholism* (pp. 110-166). Springfield, IL: Charles C. Thomas.

Bohman, M. (1978). Some genetic aspects of alcoholism and criminality: A population of adoptees. *Archives of General Psychiatry, 35*, 269-276.

Bradshaw, J. (1988). *Healing the shame that binds you*. Deerfield Beach, FL: Health Communications.

Bunzel, A. (1940). The role of alcoholism in two Central American cultures. *Psychiatry, 3*, 361-387.

Butzel, R. (1982). Intoxication and withdrawal. In N.J. Estes & M. E. Heinemann (Eds.), *Alcoholism: Developments, consequences, and interventions* (2nd ed., pp. 102-108). St. Louis: Mosby.

Cadoret, R. J., O'Gorman, T. W., Troughton, E., & Heywood, L. (1984). Alcoholism and antisocial personality: Interrelationshps, genetic and environmental factors. *Archives of General Psychiatry, 42*, 161-167.

Cahalan, D., Cisin, H., & Crossley, H. (1969). *American drinking practices: A national survey of behavior and attitudes* (Monograph 6). New Brunswick, NJ: Rutgers Center for Alcohol Studies.

Cappell, H. & Herman, C. P. (1972). Alcohol and tension reduction: A review, *Journal of Studies on Alcohol, 33*, 33-64.

Child, I, Bacon, M., & Barry, H. (1965). A cross-cultural study of drinking. *Quarterly Journal of Studies on Alcohol* (Suppl. 3), 5-96.

Clark, W. B. & Cahalan, D. (1976). Changes in problem drinking over a four-year span. *Addictive Behaviors, 1*, 251-259.

Clark, W. B., & Midanik, L. (1982). Alcohol use and alcohol problems among U.S. adults. Results of the 1979 national survey. In *Alcohol consumption and related problems* (Alcohol and Health Monograph No. 1, DHHS Publication No. ADM 82-1190). Washington,

DC: U.S. Government Printing Office.

Cloninger, C. R. (1983). Genetic and environmental factors in the development of alcoholism. *Journal of Psychiatric Treatment and Evaluation, 5*, 487—496.

Cloninger, C. R. (1987a). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry, 44*, 573-588.

Cloninger, C. R. (1987b). Neurogenetic adaptive mechanisms in alcoholism. *Science, 236*, 410-416.

Cloninger, C. R., Bohman, M., & Sigvardsson, S. (1981). Cross-fostering analysis of adopted men. *Archives of General Psychiatry, 36*, 861-868.

Cloninger, C. R., Sigvardsson, S., & Bohman, M. (1988). Childhood personality predicts alcohol abuse in young adults. *Alcoholism: Clinical and Experimental Research, 12*, 494-505.

Conger, J. J. (1956). Reinforcement and the dynamics of alcoholism, *Quarterly Journal of Studies on Alcohol, 13*, 296-305.

Conner, R. (1962). The self-concepts of alcoholics. In D. Pittman & C. Snyder (Eds.), *Society, culture and drinking patterns* (pp. 455-467). Carbondale: Southern Illinois University Press.

Cox, W. M. (1985). Personality correlatives of substance abuse. In M. Galizio & S. A. Mausto (Eds.), *Determinants of substance abuse: Biological, psychological, and environmental factors* (pp. 209-246). New York: Plenum.

Cox, W. M. (1987). Personality theory and research. In H. Blane and K. Leonard (Eds.), *Psychological theories of drinking and alcoholism* (pp. 55-89). New York: Guilford Press.

Cruz-Coke, R., & Varela, A. (1966). Inheritance of alcoholism: Its association with colour-blindness. *Lancet, ii,* 1282-1284.

DeLuca, J., & Wallace, J. (1981). *The fourth special report to the U.S. Congress on alcohol and health* [DHHS Publication No. (ADM)81-1291], Washington, DC: U.S. Government Printing

Office.

Deutsch, H. (1965). Some forms of emotional disturbance and their relationship to schizophrenia. In *Neuroses and character types* (pp. 262-281). New York: International Universities Press.

Durkheim, E. (1987). *Suicide.* Glencoe, IL: Free Press.

Ellis, A. (1962). *Reason and emotion in psychotherapy*. Seacaucus, NJ: Citadel.

Ellis, A. (1988). *Rational-emotive therapy with alcoholics and substance abusers*. Oxford: Pergamon Press.

Fenichel, O. (1945). *The psychoanalytic theory of the neurosis.* New York: W. W. Norton.

Field, P. (1962). A new cross-cultural study of drunkenness. In D. Pittman & C. Snyder (Eds.), *Society, culture, and drinking patterns* (pp. 48-74). Carbondale: Southern Illinois University Press.

Fingarette, H. (1988). *Heavy drinking: The myth of alcoholism as a disease*. Berkeley, CA: University of California Press.

Freud, S. (1953). Interpretation of dreams. In J. Strachey (Ed. and Trans.), Standard edition of the complete psychological works of Sigmund Freud (Vols. 4 & 5, pp. 1- 628). London: Hogarth Press (original work published 1900).

Freud, S. (1955). Beyond the pleasure principle. In J. Strachey (Ed. and Trans.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. 18, pp.1-64). London: Hogarth Press (original work published 1920).

Freud, S. (1956). The ego and the id. In J. Strachey (Ed. and Trans.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. 19, pp. 1-66). London: Hogarth Press (original work published 1923).

Freud, S. (1957). On narcissism: an introduction. In J. Strachey (Ed. and Trans.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. 14, pp. 67-104). London:

Hogarth Press (original work published 1914).

Freud, S. (1961). Dostoevsky and parricide. In J. Strachey (Ed. and Trans.), *Standard edition of the complete psychological works of Sigmund Freud* (Vol. 21, pp. 173-194). London: Hogarth Press (original work published 1928).

Freud, S. (1974). *Cocaine papers* (B. Byck, Ed.). New York: New American Library.

Freud, S. (1985). *The complete letters of Sigmund Freud to Wilhelm Fleiss* (J. M. Mason, Ed. and Trans.) Cambridge, MA: Harvard University Press. (Originally written 1897).

Fromm, E. (1941). *Escape from freedom.* New York: Rinehart.

Fromm, E. (1964). *The heart of man.* New York: Harper & Row.

Galanter, M. (1993). *Network therapy for alcohol and drug abuse: A new approach in practice.* New York: Basic Books.

Gay, P. (1988). *Freud: A life for our time.* New York: W. W. Norton.

Gilman, A. G., Goodman, L. S., & Gilman, A. (Eds.). (1985). *Goodman and Gilman's The pharmacological bases of therapeutics* (7th ed.). New York: Macmillan.

Glover, E. (1928). The etiology of alcoholism. *Proceedings of the Royal Society of Medicine, 21.* 1351-1355.

Goldstein, G. (1976). Perceptual and cognitive deficit in alcoholics. In G. Goldstein & C. Neuringer (Eds.), *Empirical studies of alcoholism* (pp. 115-152). Cambridge, MA: Bollinger.

Goodwin, D. W. (1988). *Is alcoholism hereditary?* New York: Ballantine Books.

Goodwin, D.W., Schulsinger, F., Hermansen, L., Guze, S. B., & Winokur, G. (1973). Alcohol problems in adoptees raised apart from alcoholic biological parents. *Archives of General Psychiatry, 28,* 283-343.

Hall, T. (1989, March 15). A new temperance is taking root in America. *The New York Times*, pp.

Al, A6.

Harris, L., and Associates, Inc. (1971). *American attitudes toward alcohol and alcoholism* (Study no. 2188). A survey of public opinion prepared for the National Insti¬tute on Alcohol Abuse and Alcoholism. New York: Louis Harris.

Hartocollis, P., & Hartocollis, P. (1980). Alcoholism, borderline and narcissistic disorders: A psychoanalytic overview. In W. Fann, I. Karacon, A. Pokomy, & R. Williams (Eds.), *Phenomenology and treatment of alcoholism* (pp. 93-110). New York: Spectrum.

Hartocollis, P. (1968). A dynamic view of alcoholism: drinking in the service of denial. *Dynamic Psychiatry*, 2, 173-182.

Heath, D. B. (1958). Drinking patterns of the Bolivian Camba. *Quarterly Journal of Studies on Alcohol, 19*, 491-508.

Heath, D.B. (1991). Continuity and change in drinking patterns of the Bolivian Camba. In D. Pittman & H. A. White (Eds.), *Society, culture, and drinking patterns reexamined* (pp. 78-86). New Brunswick, NJ: Rutgers Center of Alcohol Studies.

Hewitt, C. C. (1943). A personality study of alcohol addiction. *Quarterly Journal of Studies on Alcohol, 4,* 368-386.

Horton, D. (1943). The functions of alcohol in primitive societies: A cross-cultural study. *Quarterly Journal of Studies on Alcohol, 4,* 199-320.

Hull, J. G. (1981). A self-awareness model of the causes and effects of alcohol consumption. *Journal of Abnormal Psychology, 90,* 586-600.

Huxley, A. (1954). *The doors of perception.* New York: Harper & Row.

Irgens-Jensen, O. (1971). *Problem drinking and personality: A study based on the Draw- a-Person Test.* New Brunswick, NJ: Rutgers Center for Alcohol Studies.

Jacobson, E. (1938). *Progressive relaxation.* Chicago: University of Chicago Press.

James, W. (1902). *Varieties of religious experience.* New York: Longmans.

Jellinek, E. M. (1952). Phases of alcohol addiction. *Quarterly Journal of Studies on Alcohol. 13*, 673-684.

Jellinek, E. M. (1960). *The disease concept of alcoholism.* New Haven, CT: College and University Press.

Jellinek, E. M. (1962). Cultural differences in the meaning of alcoholism. In D. J. Pittman and C. R. Snyder (Eds.), *Society, culture, and drinking patterns* (pp. 382-388). Carbondale: Southern Illinois University Press.

Jellinek, G. (1994). Heredity and premature weaning: A discussion of the work of Thomas Trotter, British Naval Physician. In J. Levin & R. Weiss (Eds.), *The dynamics and treatment of alcoholism: Essential papers* (pp. 28-34). Northvale, NJ: Jason Aronson. (Trotter's original work published 1804, Jellinek's article, 1943)

Jessor, R. (1987). Problem-behavior theory, psychosocial development, and adolescent problem drinking. *British Journal of Addictions, 82*, 331-342.

Jessor, R., Graves, T. D., Hanson, R. C. & Jessor, S. L. (1968). *Society, personality, behavior: A study of a tri-ethnic community.* New York: Hold, Rinehart & Winston.

Jones, K. L., Smith, D. W., Ulleland, C. N., & Streissguth, A. P. (1973). Pattern of malformation in offspring in chronic alcoholic women. *Lancet, i*, 1267-1271.

Jones, M. C. (1968). Personal correlates of antecedents of drinking patterns in adult males, *Journal of Consulting and Clinical Psychology, 32,* 2-12.

Jones, M. C. (1971). Personality antecedents and correlates of drinking patterns in women, *Journal of Consulting and Clinical Psychology, 36,* 61-69.

Julien, R. M. (1991). *A primer of drug action* (6th ed.). San Francisco: W. H. Freeman.

Jung, C. G. (1973). In G. Adler (Ed.), *C.G. Jung: Letters, Vol. 11, 1951-1961* (pp. 623- 625). Princeton, NJ: Princeton University Press. (Originally written 1961)

Kaij, L. (1960). *Alcoholism in twins: Studies on the etiology and sequels of abuse of alcohol.* Stockholm: Almquist & Wiksell.

Kaminer, W. (1993). *I'm dysfunctional, you're dysfunctional: The recovery movement and other self-help fashions.* New York: Vintage.

Kandel, D. B. (1975). Stages in adolescent involvement in drug use. *Science, 190,* 912- 914.

Kandel, D. B., Yamaguchi, K., & Chen, K. (1992). Stages of progression in drug involvement from adolescence to adulthood: Further evidence for the gateway theory. *Journal of Studies on Alcohol, 53,* 447-457.

Kant, I. (1929). *The critique of pure reason* (2nd Ed., M. K. Smith, trans.). London: MacMillan. (Original work published 1787)

Kazin, A. (1976, March). 'The giant killer': Drink and the american writer. *Commentary, 61(3),* 44-50.

Keller, M. (1958). Alcoholism: Nature and extent of the problem. *Annals of the American Academy of Political and Social Science, 315,* 1-11.

Kernberg, O. (1975). *Borderline conditions and pathological narcissism.* New York: Jason Aronson.

Khantzian, E.J. (1981). Some treatment implications of ego and self-disturbances in alcoholism. In M. H. Bean & N. E. Zinberg (Eds.), *Dynamic approaches to the understanding and treatment of alcoholism* (pp. 163-188). New York: Free Press.

Kierkegaard, S. (1944). *The concept of dread* (W. Lowrie, Trans.). Princeton, NJ: Princeton University Press. (Original work published 1849)

Kirkpatrick, J. (1977). *Turnabout: Help for a new life.* New York: Doubleday.

Klausner, S. (1964). Sacred and profane meanings of blood and alcohol. *The Journal of Social Psychology, 64,* 27-43.

Knight, R. P. (1937). The dynamics and treatment of chronic alcohol addiction. *Bulletin of the*

Menninger Clinic, 1, 233-250.

Knight, R.P. (1938). The psychoanalytic treatment in a sanatarium of chronic addiction to alcohol. *Journal of the American Medical Association, 111,* 1443-1448.

Kohut, H. (1971). *The analysis of the self: A systematic approach to the psychoanalytic treatment of narcissistic personality disorders.* New York: International Universities Press.

Kohut, H. (1977a). *The restoration of the self.* New York: International Universities Press.

Kohut, H. (1977b). *Psychodynamics of drug dependence* (National Institute on Drug Abuse Monograph 12, pp. vii-ix). Washington, DC: U.S. Government Printing Office.

Krystal, H., & Raskin, H. (1970). *Drug dependence: Aspects of ego function.* Detroit: Wayne State University Press.

Levin, J.D. (1981). A study of social role conflict in chronic alcoholic men afflicted with Alcoholics Anonymous (Doctoral dissertation, New York University, 1981). *Dissertation Abstracts International, 42-12B* 4970. (University Microfilms No. 8210924)

Levin, J.D. (1987). *Treatment of alcoholism and other addictions: A self-psychology approach.* Northvale, NJ: Jason Aronson.

Levin, J.D. (1991). *Recovery from alcoholism: Beyond your wildest dreams.* Northvale, NJ: Jason Aronson.

Levin, J.D. (1993). *Slings and arrows: narcissistic injury and its treatment.* Northvale, NJ: Jason Aronson.

Levin, J.D. & Weiss, R. H. (Eds.) (1994). *The dynamics and treatment of alcoholism: Essential papers.* Northvale, NJ: Jason Aronson.

Lex, B. (1985). Alcohol problems in special populations. In J.H. Mendelson & N. K. Mello (Eds.), *The diagnosis and treatment of alcoholism* (2nd ed., pp. 89-188). New York: McGraw-Hill.

Lipscomb, T. R., Carpenter, J. A., & Nathan, P. E. (1980). Static ataxia: A predictor of alcoholism? *British Journal of Addictions, 74,* 289-294.

London, W. P. (1990). Left-handedness and alcoholism. In S. Coren (Ed.), *Left-handed-ness: Behavioral implications and anomalies,* pp. 457-484. New York: Elsevier Science.

Loper, R. G., Kammeier, M. L., & Hoffman, H. (1973). MMPI characteristics of college freshmen males who later became alcoholics. *Journal of Abnormal Psychology, 82,* 159-162.

MacAndrew, C. (1965). The differentiation of male alcoholic outpatients from nonalcoholic psychiatric outpatients by means of the MMPI. *Quarterly Journal of Studies on Alcohol, 26,* 238-246.

MacAndrew, C. & Edgerton, R. B. (1969). *Drunken comportment.* Chicago: Aldine.

MacAndrew, C., & Geertsma, R.H. (1963). Analysis of responses of alcoholics to Scale 4 of the MMPI. *Quarterly Journal of Studies on Alcohol, 26,* 23-38.

Maccoby, M. (1977). Alcoholism in a Mexican village. In McClelland, D., Davis, W. N., Kalin, R., & Wanner, E. (Eds.). *The drinking man,* pp. 232-260. New York: Free Press.

Mahler, M., Pine F., & Bergman, A. (1975). *>The psychological birth of the human infant: Symbiosis and individuation.* New York: Basic Books.

Marlatt, G. A. & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors.* New York: Guilford Press.

Masserman, J., & Yum, K. (1946). An analysis of the influence of alcohol in experimental neurosis in cats. *Psychosomatic Medicine, 8,* 36-52.

Masters, W. H., & Johnson, V. E. (1970). *Human sexual inadequacy.* Boston: Little, Brown.

McClelland, D. C., Davis, W., Kalin, R., & Wanner, E. (1972). *The drinking man: Alcohol and human motivation.* New York: Free Press.

McCord, W., & McCord, J., with Gudeman, J. (1960). *Origins of Alcoholism.* Stanford, CA: Stanford

University Press.

McCord, W., & McCord, J. (1962). A longitudinal study of the personality of alcoholics. In D. J. Pittman and C. R. Synder (Eds.), *Society, culture and drinking patterns* (pp. 413-430). Carbondale, Ill.: Southern Illinois University Press.

Mello, N.K., & Mendelson, J.H. (1970). Experimentally induced intoxication in alcoholics: A comparison between programmed and spontaneous drinking. *Journal of Pharmacology and Experimental Therapeutics, 173,* 101-116.

Mello, N. K. & Mendelson, J. H. (1972). Drinking patterns during work-contingent and noncontingent alcohol acquisition. *Psychosomatic Medicine, 34,* 139-164.

Menninger, K. (1938). *Man against himself.* New York: Harcourt Brace.

Meyerson, A. (1940). Alcohol: A study of social ambivalence. *Quarterly Journal of Studies on Alcohol, I,* 13-20.

Murray, H. A. (1938). *Explorations in personality.* New York: Oxford University Press.

National Institute on Alcohol Abuse and Alcoholism (1983). *Fifth special report to the U.S. Congress on Alcohol and Health from the Secretary of Health and Human Services* (DHHS Publication No. 84-1291).Rockville, MD: Author.

National Institute on Alcohol Abuse and Alcoholism (NIAAA) (1988). *Sixth special report to the U.S. Congress on alcohol and health from the Secretary of Health and Human Services* (DHHS Publication No. 85-0009).Rockville, MD: Author.

National Institute on Alcohol Abuse and Alcoholism. (1990). *Seventh Special Report to the U.S. Congress on Alcohol and Health from the Secretary of Health and Human Services* (DHHS Publication No. 90-1656). Rockville, MD: Author.

National Institute of Alcohol Abuse and Alcoholism (1994). *Eighth special report to the U.S. Congress on alcohol and health from the Secretary of Health and Human Services* (NIH Publication No. 94-3699). Rockville, MD: Author.

Park, P. (1973). Developmental ordering of experiences in alcoholism. *Quarterly Journal of Studies on Alcohol, 34,* 473-488.

Pavlov, I. (1927). *Conditioned reflexes.* Oxford, England: Oxford University Press.

Peninston, E. G. & Kulkosky, P. J. (1989). Brainwave training and B-endorphin levels in alcoholics. *>Alcoholism: Clinical and Experimental Research, 13,* 271-279.

Peninston, E. G., & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy, 3,* 37-55.

Petrie, A. (1978). *Individuality in pain and suffering* (2nd ed.). Chicago: University of Chicago Press.

Pitts, F. N., Jr., & Winokur, G. (1966). Affective disorder—VII: Alcoholism and affective disorder. *Journal of Psychiatric Research, 4,* 37-50.

Polich, J., Armor, D., & Brainer, H. (1981). *The course of alcoholism.* New York: Wiley.

Polich, J., & Bloom, F. E. (1987). P300 from normals and adult children of alcoholics. *Alcohol, 4,* 301-307.

Pollock, V. E., Volavka, J., Goodwin, D. W., Mednick, S. A., Gabrielli, W. F., Knop, J., & Schulsinger, F. (1983). The EEG after alcohol administration in men at risk of alcoholism. *Archives of General Psychiatry, 40,* 857-861.

Porjesz, B., & Begleiter, H. (Eds.). (1983). *The biology of alcoholism, vol. VII: The pathogenesis of alcoholism, biological factors* (pp. 415-483). New York: Plenum Press.

Prochaska, J., & DiClemente, C. C. (1984). *The transtheoretical approach: Crossing the traditional boundaries of therapy.* Homewood, IL: Dow-Jones/Irwin.

Prochaska, J. V., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change: Applications to addictive behaviors. *American Psychologist, 47,* 1102-1114.

Propping, P., Kruger, J., & Mark, N. (1981). Genetic disposition to alcoholism: An EEG study in

alcoholics and their relatives. *Human Genetics, 35,* 51-59.

Rado, S. (1933). The psychoanalysis of pharmacothymia. *Psychoanalytic Quarterly, 2,* 2-23.

Richards, H. J. (1993). *Therapy of the substance abuse syndromes.* Northvale, NJ: Jason Aronson.

Robbins, L. N., Bates, W. N., & O'Neal, P. (1962). Adult drinking patterns of former problem children. In D. J. Pittman & C. R. Snyder (Eds.), *Society, culture and drinking patterns* (pp. 395-412). Carbondale: Southern Illinois University Press.

Robbins, L. R., & Smith, E. M. (1980). Longitudinal studies of alcohol and drug problems: sex differences. In O. J. Kalant (Ed.), *Alcohol and drug problems in women.* New York: Plenum Press.

Roe, A. (1945). The adult adjustment of children of alcoholic parents raised in foster homes. *Quarterly Journal of Studies on Alcohol, 5,* 378-393.

Rohsenow, D. J. (1983). Alcoholics' perceptions of control. In W. M. Cox (Ed.), *Identifying and measuring alcoholic personality characteristics* (pp. 37-48). San Francisco: Jossey-Bass.

Rooney, J. F. (1991). Patterns of alcohol use in Spanish society. In. D. Pittman & H. R. White (Eds.), *Society, culture, and drinking patterns reexamined* (pp. 78-86). New Brunswick, NJ: Rutgers Center of Alcohol Studies.

Rotter, J. B. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs, 80.*

Rush, B. (1994). An inquiry into the effects of ardent spirits upon the human body and mind. In J. Levin & R. Weiss (Eds.), *The dynamics and treatment of alcoholism: Essential papers* (pp. 11-27). Northvale, NJ: Jason Aronson. (Original work published 1785)

Sandor, S. (1968). The influence of ethyl alcohol on the development of the chick embryo.*Revue du-Embryologie et de-Cytologie, Serie Embryologie, 5,* 51-76.

Schuckit, M. A., & Gold, F. O. (1988). A simultaneous evaluation of multiple markers of

ethanol/placebo challenges in sons of alcoholics and controls. *Archives of General Psychiatry, 45,* 211-216.

Shaffer, H. J. (1992). The psychology of stage change: The transition from addiction to recovery. In J. H. Lowinson, P. Ruiz, & R. B. Millman (Eds.), *Comprehensive textbook of substance abuse* (2nd ed., pp. 100-105). Baltimore: Williams & Wilkins.

Shaffer, H. J. (1994). Denial, ambivalence, and countertransferential hate. In J. D. Levin & R. H. Weiss (Eds.), *The dynamics and treatment of alcoholism: Essential papers,* pp. 421-437. Northvale, NJ: Jason Aronson.

Sher, K. J. (1987). Stress response dampening. In H. T. Blane & K. E. Leonard (Eds.), *Psychological theories of drinking and alcoholism* (pp. 227-271). New York: Guilford Press.

Shipley, T. E., Jr. (1982). Alcohol withdrawal and its treatment: Some conjectures in the context of the opponent-process theory. *Journal of Studies on Alcohol, 43,* 548-569.

Simmel, E. (1948). Alcoholism and addiction. *Psychoanalytic Quarterly, 17,* 6-31.

Skinner, B. F. (1938). *The behavior of organisms.* New York: Appleton-Century-Crofts.

Smith, M. O. (1989). *The Lincoln Hospital acupuncture drug treatment program.* Testimony presented to the Select Committee on Narcotics of the U.S. House of Representatives, July 25, 1989.

Sobell, M. B. & Sobell, L. C. (1978). *Behavioral treatment of alcohol problems: Individualized therapy and controlled drinking.* New York: Plenum Press.

Sobell, M. B., & Sobell, L. C. (1993). *Problem drinkers: Guided self-change treatment.* New York: Guilford Press.

Solomon, R. L. (1977). An opponent process theory of acquired motivation: The affective dynamics of addiction. In J. D. Maser & M.E.P. Seligman (Eds.), *Psychopathology: Experimental Models* (pp. 66-103). San Francisco: W. H. Freeman.

Spitz, H. I., & Rosecan, J. S. (1987). *Cocaine abuse.* New York: Brunner/Mazel.

Szasz, T. (1958). The role of the counterphobic mechanism in addiction. *Journal of the American Psychoanalytic Association, 6*, 309-325.

Tabakoff, B., Hoffman, P. L., Lee, J. M., Saito, T., Willard, B., & Deleon-Jones, F. (1988). Differences in platelet enzyme activity between alcoholics and nonalcoholics. *New England Journal of Medicine, 318,* 134-139.

Tarter, R. E. (1981). Minimal brain dysfunction as an etiological disposition to alcoholism. In R. E. Meyer (Eds.), *Evaluation of the alcoholic: Implications for research, theory, and treatment* (NIAAA Monograph Series). Washington, DC: National Institute on Alcohol Abuse and Alcoholism.

Tarter, R. E. & Alterman, A. I. (1989). Neurobehavioral theory of alcoholism etiology. In C. D. Choudron & D. A. Wilkinson (Eds.), *Theories of alcoholism.* Toronto: Addiction Research Foundation.

Tillich, P. (1952). *The courage to be.* New Haven, CT: Yale University Press.

Trimpey, J. (1989). *Rational recovery from alcoholism: The small book* (3rd ed.). Lotus, CA: Lotus Press.

Vaillant, G. E. (1983). *The natural history of alcoholism: Causes, patterns and paths to recovery.* Cambridge, MA: Harvard University Press.

Varela, A., Rivera, L., Mardones, J., & Cruz-Coke, R. (1969). Color vision defects in non-alcoholic relatives of alcoholic patients. *British Journal of the Addictions, 64,*67-71.

von Knorring, A. L., Bohman, M., von Knorring, L., & Oreland, L. (1985). Platelet MAO activity as a biological marker in subgroups of alcoholism. *>Acta Psychiatrica Scandinavica, 72,* 51-58.

Williams, G. D. & DeBakey, S. F. (1992). Changes in levels of alcohol consumption: United States 1983 to 1988. *British Journal of Addictions, 87 (4),* 643-648.

Wilsnack, S. C. (1973). Sex role identity in female alcoholism. *Journal of Abnormal Psychology, 82,* 253-261.

Wilsnack, S. C. (1984).Drinking, sexuality, and sexual dysfunction in women. In S. C. Wilsnack & L. J. Beckman (Eds.), *Alcohol problems in women: Antecedents, consequences, and interventions,* pp. 189-228. New York: Guilford Press.

Wilsnack, S. C. & Beckman, L. J. (Eds.) (1984). *Alcohol problems in women: Antecedents, consequences, and intervention.* >New York: Guilford Press.

Wilsnack, S.C. (1991). Sexuality and women's drinking: Findings from a U.S. national study. *Alcohol and Health Research World, 15(2),* 147-150.

Wilson, G., & Lawson, D. (1978). Expectancies, alcohol, and sexual arousal in women. *Abnormal Psychology, 85,* 358-367.

Winnicott, D. W. (1958). The capacity to be alone. In *The maturational processes and the facilitating environment* (pp. 29-36). New York: International Universities Press, 1965.

Winnicott, D. W. (1960). Ego distortion in terms of true and false self. In *The maturational processes and the facilitating environment* (pp. 140-152). New York: International Universities Press, 1965.

Winokur, G. (1974). The division of depressive illness into depressive-spectrum disease and pure depressive disease. *International Pharmaco-psychiatry, 9,* 5-13.

Winokur, G., Rimmer, J., & Reich, T. (1971). Alcoholism IV: Is there more than one type of alcoholism? *British Journal of Psychiatry, 18,* 525-531.

Winson, J. (1985). *Brain and Psyche: The Biology of the Unconscious.* New York: Anchor Press, Doubleday.

Winson, J. (1990). The Meaning of Dreams. *Scientific American,* November 1990. 86-96.

Wise, R. A. (1987). Psychomotor stimulant properties of addictive drugs. *Pharmacological Therapy, 35,* 227-263.

Witkin, H. A., & Oltman, P. K. (1967). Cognitive style. *International Journal of Neurology, 6,* 119-

137.

Witkin, H. A., Karp, S. A., & Goodenough, D. R. (1959). Dependence in alcoholics. *Quarterly Journal of Studies on Alcohol, 20,* 493-504.

Wurmser, L. (1978). *The hidden dimension: Psychodynamics in compulsive drug use.* New York: Jason Aronson.

Zucker, R. A. & Gomberg, E. S. L. (1986). >Etiology of alcoholism reconsidered: The case for a biopsychosocial process. *American Psychologist, 41,* 783-793.

Zuckerman, M. (1979). *Sensation seeking: Beyond the optimal level of arousal.* New York: Wiley.

Notes

[6] Statisticians speak of significant and nonsignificant differences. A significant difference is one that is sufficiently unlikely (usually meaning the odds are less than 1 in 20) to be a chance or accidental finding, about which one can be confident that the difference is due to the properties of what is being compared—in this case alcoholism and its absence. Statistical significance is a statement about how certain one is of a conclusion, not of how important it is. The degree of certainty does not indicate how large the difference or correlation between groups is; this is expressed differently, usually by a correlation coefficient. Statisticians speak of significance levels, such as the .05 level, which means there is a 5-in-100 or 1-in-20 chance that the finding is random.