Module 2:
Etiology and Natural History
Of Alcoholism

Etiology and Natural History of Alcoholism

Introduction

"Etiology" relates to the understanding of:

- the roots and origins of a specific problem
- the factors that shape its development, and
- the influences that might ameliorate its course

Understanding the natural history of a disease or disorder provides information critical for assessment and intervention, and for the development of effective prevention strategies. Alcohol use disorders represent a broad continuum, ranging from periodic problematic overuse to chronic, progressive alcohol dependence. Clinicians categorize individuals based on standardized criteria (i.e., abuse vs. dependence). Alcohol use disorders in the general population and different clinical settings range in frequency, severity, and symptoms.

The research presented is representative of a bio-psycho-social perspective on alcohol disorders. The content is also guided by a lifespan person-in-environment perspective, relating the interactive nature of individual and social context influences on the course of any disorder. Integral to this discussion of etiology is an awareness of the complexity contributed by human diversity. Developing an awareness of the natural history of alcohol use disorders helps social workers to identify persons at risk, why they are at risk, what might be done to alleviate risk, and what treatment strategies prove to be most effective.

In this module, "alcoholism" is applied to those persons whose symptoms are sufficiently severe to warrant the diagnosis of alcohol dependence according to standard criteria such as DSM-IV or ICD-10. Alcohol 'misuse' or 'abuse' is used to describe those who meet criteria for alcohol abuse by DSM-IV criteria.

Genetic Factors and Vulnerability to Alcohol Use Disorders

Alcoholism is a complex disorder with many pathways leading to its development. Genetic and other biological factors are likely involved in the emergence of alcohol dependence, along with cognitive, behavioral, temperament, psychological, and sociocultural factors. Alcohol use patterns, including alcohol abuse and alcohol dependence, are 'familial' in nature (cf. Heath et al., 1997; cf. Kendler, Heath, Neale, Kessler, & Eaves, 1992; Hesselbrock, 1995). That is, similar styles of alcohol use and the presence of alcoholism are often found within the same
family, running from parent to child and across multiple generations of biologically related individuals. However, many other traits or behaviors, such as religious or political affiliation, which have little or no biological basis and, therefore, cannot be under heavy genetic control, also run in families. While genetic and other biological factors cannot fully explain the presence or absence of alcohol use disorders, their contribution to susceptibility for developing alcohol use problems appears to be significant.

**Genetic Factors:**
Evidence for genetic influences on the emergence of alcoholism is derived from a variety of sources. While no single source of information definitively confirms a genetic hypothesis, the confluence of findings from (1) extended pedigree studies, (2) twin studies involving monozygotic (identical) and dizygotic (fraternal) twin pairs, and (3) studies of adopted individuals raised apart from their alcoholic parents, persuasively argues for a genetic component to the vulnerability continuum for developing alcoholism.

**Pedigree studies:** Family pedigree studies of alcoholic clients, compared to general population rates, typically show an increase in the lifetime prevalence of alcoholism across all classes of biological relatives. The increase in risk for developing alcoholism may be four- to seven-fold among first-degree relatives of an alcoholic compared to the general population (Cotton, 1979; Merikangas, 1990). First-degree relatives are those with the closest genetic ties-for example, parents and siblings.

**Twin studies:** Monozygotic twins are genetically identical, despite the appearance of some phenotypic differences—differences in how genotype is actually expressed. Dizygotic twins, on the other hand, are genetically like any other set of siblings-on average, they share about 50% of their genes. Twin studies examine the extent of concordance (degree and extent of similarity) between the two types of twins. If the degree of concordance is similar, a genetic basis cannot be conclusively identified. If, on the other hand, concordance is directly related to type of twin and monozygotic twins have greater concordance than dizygotic twins, there is a strong probability that a significant portion of the appearance of a feature is genetically driven. Monozygotic twins generally have a higher concordance rate of alcoholism compared to dizygotic twins. The concordance rate for dizygotic twins is the same rate as other non-twin sibling pairs (cf. Kendler et al., 1992; McGue, Pickens, & Svikis, 1992).

**Adoption Studies:** In an attempt to separate genetic from environmental effects in the susceptibility for developing alcoholism, studies of the offspring of alcoholic parents (typically an alcoholic father) adopted away at birth have been conducted (see Table 1). Studies conducted in Scandinavia and in the U.S. of adopted infants placed in nonalcoholic homes have typically found that the adopted children born of an alcoholic parent develop alcoholism as adults at a higher rate than do adopted children with neither biological parent affected with alcoholism (cf. Goodwin et al., 1974; Cloninger, Bohman, & Sigvardsson, 1981; Cadoret, Cain, & Grove, 1980).
Table 1: Adoptee Risk of Alcoholism by Alcoholism in Biological Parents

<table>
<thead>
<tr>
<th>Study</th>
<th>Positive</th>
<th>Negative</th>
<th>Relative Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Roe (1945)</td>
<td>0.0% (n=21)</td>
<td>0.0% (n=11)</td>
<td>1.0</td>
</tr>
<tr>
<td>Goodwin et al (1973)</td>
<td>18.0% (n=55)</td>
<td>5.0 (n=78)</td>
<td>3.6*</td>
</tr>
<tr>
<td>Cloninger et al (1981)</td>
<td>23.3% (n=291)</td>
<td>14.7% (n=571)</td>
<td>1.6*</td>
</tr>
<tr>
<td>Cadoret et al (1985)</td>
<td>31.1% (n=18)</td>
<td>23.9% (n=109)</td>
<td>2.6%</td>
</tr>
<tr>
<td>Cadoret et al (1987)</td>
<td>62.5% (n=8)</td>
<td>20.4% (n=152)</td>
<td>3.1%</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Roe (1945)</td>
<td>0.0% (n=11)</td>
<td>0.0% (n=14)</td>
<td>1.0</td>
</tr>
<tr>
<td>Goodwin et al (1977)</td>
<td>2.0% (n=49)</td>
<td>4.0% (n=47)</td>
<td>0.5</td>
</tr>
<tr>
<td>Bohman et al (1981)</td>
<td>4.5% (n=336)</td>
<td>2.8% (n=577)</td>
<td>1.6*</td>
</tr>
<tr>
<td>Cadoret et al (1985)</td>
<td>33.3% (n=12)</td>
<td>5.3% (n=75)</td>
<td>6.3*</td>
</tr>
</tbody>
</table>

The findings from the three types of studies hold for both males and females, although the evidence for the importance of genetic factors in alcoholism risk currently appears to be stronger for males compared to females. These gender differences may reflect a real difference in male/female genetic risk, although to date, no sex-linked genes have been identified for alcoholism. More likely, the gender differences found are due to the moderating role of cultural and social factors that may limit females' exposure to heavy drinking. More recent epidemiological studies suggest that gender differences in the incidence and prevalence of alcoholism are declining among more recent birth cohorts.

It is also important to remember that all studies to date report only an increase in the statistical probability or risk for developing alcoholism among family members. While the statistical probability or risk of developing alcohol problems is higher among biological relatives of alcoholic than non-alcoholic biological relatives, in fact, most offspring of an alcoholic parent do not develop alcohol use problems or disorders in their lifetimes.

Finally, although great progress is being made in the search for the genetic bases of the susceptibility for developing alcoholism (NIAA, 2000; Hesselbrock et al., 2001), specific genes predisposing to alcohol use disorders have yet to be identified.

This is also true of specific genes that may provide protection from this predisposition. Therefore, it is probably most useful to consider that multiple genes in various locations contribute to the continuum of vulnerability. Genetic models that postulate a single gene are not supported by the research results.

Mechanisms of Heritability - Gene-Environment Interaction

While family pedigree studies, twin studies, and adoption studies implicate genetic factors in the development of alcoholism, their findings also indicate that the genetic factors are not deterministic. If certain genes actually predestined an individual to develop alcohol use problems, then all alcoholics would have a close relative with alcoholism, the concordance rates for monozygotic twins would approach 100%, and almost all offspring of an alcoholic parent-including adoptees-would eventually develop an alcohol use disorder. Since this clearly is not the case, environmental and other biological factors must also play an important role in alcoholism susceptibility (see Figure 1).
Many investigators have stressed the importance of the interaction between an inherited biological vulnerability and environmental risk factors for developing alcohol use disorders (cf. Kendler, 1995; Slutske et al., 1998; Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995). This gene-environment interaction (G x E) model assumes a synergy between genetic and environment factors that may contribute either to an increased susceptibility for developing an alcohol use disorder or attenuate possible genetic risk by producing a level of protection for vulnerable individuals (cf. Hesselbrock & Hesselbrock, 1990).

However, specific environmental factors that possibly affect the development of alcohol use disorders, such as those related to a family environment, social relationships, and parenting styles, have not been definitively identified.

Interestingly, cohort of birth and ethnicity are two major individual factors that also affect the susceptibility for developing alcohol dependence. Even though it is extremely unlikely that the human genome has changed significantly over the past 100 years, more recent birth cohorts have higher prevalence rates of alcoholism than birth cohorts from the earlier part of the twentieth century (Reich et al., 1988; Grant, 1997). The differences in prevalence rates are thought to be due to variations in the availability of beverage alcohol resulting from Prohibition, economic depression, or wartime shortages.

Differences in prevalence rates and the course of alcohol dependence have also been noted in relation to ethnicity (Hesselbrock, et al., 1998; NIAAA, 1998). For example, there appears to be little difference among ethnic groups with regard to first age of the early stages of alcoholism, but differences in first age begin to appear with later stages of the disorder.
The variability in apparent susceptibility to alcoholism is thought to be due to biological differences in alcohol metabolism, as well as social factors (e.g., involvement in religion, family relations). This suggests that, in addition and related to genetic factors, there exists a host of biological factors involved in alcoholism susceptibility. Considerable research is currently addressing the neurobiological pathways of alcohol sensitivity, alcohol craving, and addiction. It is expected that these studies will eventually lead to the development and testing of biological and/or pharmacological treatment alternatives. For example, specific medications typically used for treating depression, obsessive-compulsive disorders, or seizure disorders are being tested for their psychopharmaceutical effects on alcohol dependence.

**Cognitive Functioning and the Risk for Developing Alcoholism**

A growing number of studies have implicated heritable cognitive factors, including electrophysiological features related to central nervous system functioning, as being related to the vulnerability for developing alcohol and other substance use problems. Several studies have found poorer cognitive performance among alcoholics compared to controls on neuropsychological tests of memory, attention span, abstract thinking, verbal reasoning, and visual-spatial skills (DeObalia, Parsons & Yohman, 1983). Although specific cognitive deficits in persons at risk for developing alcoholism have not been consistently reported, tests measuring brain functioning in the frontal and temporal lobes among young adult males with a susceptibility for developing alcoholism were predictive of the age of taking their first drink and their frequency of drinking to get intoxicated (Hesselbrock, Hesselbrock, Bauer, & Gillen, 1991; Deckel, Bauer, and Hesselbrock, 1995).

Differences in central nervous system functioning as measured by electroencephalographic (EEG) and event-related potential (ERP) methods have been found between alcoholic adults and control individuals (Begleiter, Porjesz, Bihari, & Kissin, 1984). Similar findings have been reported among individuals at risk for developing alcoholism and prior to the onset of heavy drinking (cf. Begleiter et al., 1984; Bauer & Hesselbrock, 1999). Typically, differences in EEG and ERP brainwave patterns are found in the frontal region of the brain, regions that are thought to be responsible for the cognitive skills of attention, planning, and foresight. Although these electro-physiological measures of brain activity do provide a 'marker' of risk for a poor adult outcome, including an increased risk for developing alcoholism, the exact relationship between electrophysiological measures and behavioral measures (i.e., neuropsychological) of cognitive functioning are not well established (cf. Hill, Shen, Lowers, & Locke, 2000; NIAAA, 1997, 2000). There exists some controversy as to the consistency concerning observations of EEG differences among children of alcoholic and of non-alcoholic parents (Sher, 1991).

**Psychopathology and Temperament as Risk Factors**

*Childhood behavior problems:*

Many studies over the past 40 years indicate that childhood problem behavior and aspects of a child's temperament may predict both behavior problems and problems with alcohol and substance abuse during adolescence and young adulthood. An association between behavioral problems (i.e., conduct problems, attention deficit disorder, and hyperactivity) occurring in childhood and adolescence and consequent poor adult outcomes, including alcoholism, has been repeatedly found in longitudinal studies (see Table 2). These associations appear in a
variety of samples, including child guidance clinic subjects (Robins, 1966), community samples (Jones, 1968), and among adopted individuals at risk for alcoholism (Cadoret et al., 1995).

### Table 2: Childhood Aggression, ASPD, and Alcoholism by Gender

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>None 5,308</th>
<th>ASP 86</th>
<th>Alcohol 3,161</th>
<th>Both 601</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vandalism</strong></td>
<td>N</td>
<td>15.7</td>
<td>69.7</td>
<td>30.3</td>
<td>68.9</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>4.6</td>
<td>55.0</td>
<td>17.2</td>
<td>57.3</td>
</tr>
<tr>
<td><strong>Physical fights</strong></td>
<td>M</td>
<td>8.1</td>
<td>57.6</td>
<td>22.8</td>
<td>58.6</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>3.5</td>
<td>40.0</td>
<td>15.6</td>
<td>55.3</td>
</tr>
<tr>
<td><strong>Use a weapon</strong></td>
<td>M</td>
<td>2.5</td>
<td>36.4</td>
<td>9.4</td>
<td>39.2</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.8</td>
<td>15.0</td>
<td>7.3</td>
<td>34.0</td>
</tr>
<tr>
<td><strong>Injured others</strong></td>
<td>M</td>
<td>2.7</td>
<td>28.8</td>
<td>6.0</td>
<td>23.7</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.3</td>
<td>20.0</td>
<td>4.2</td>
<td>19.4</td>
</tr>
<tr>
<td><strong>Bully others</strong></td>
<td>M</td>
<td>3.1</td>
<td>19.7</td>
<td>6.8</td>
<td>21.7</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>4.4</td>
<td>30.0</td>
<td>6.3</td>
<td>34.0</td>
</tr>
</tbody>
</table>

Source: Hesselbrock, et al., 2000

Many, but not all, of these individuals who develop alcoholism will go on to develop a severe form of the disorder (Hesselbrock & Hesselbrock, 1994). Even though problem behaviors typically begin during childhood for boys and adolescence for girls, the relationship to later alcohol and drug problems holds for both boys and girls, and across at least some ethnic minority groups (Hesselbrock, Segal, & Hesselbrock, 2000; Bucholz et al., 1996). It is important to note that recent studies indicate that the alcohol use disorders developed by conduct-disordered adolescents are not benign and do not necessarily resolve over time. For many, these alcohol use disorders persist into young adult life and possibly beyond (Rohde et al., 2001).

Conduct problems in childhood and adolescence are often accompanied by other externalizing behaviors such as attention deficit disorder, hyperactivity, and oppositional behaviors (American Psychiatric Association, 1994; Windle, 1996; Murphy & Barkley, 1996). Childhood hyperactivity and attention deficit disorder have also been linked to an increased risk for developing alcoholism, particularly among children of an alcoholic parent (Tarter, McBride, Buopane, & Schneider, 1977; DeObaldia et al., 1983). However, many of these studies fail to take into account the effect of co-occurring conduct problems or sample children with only hyperactivity or only attention deficit disorder. There is little evidence for the independent contribution of either hyperactivity or attention deficit disorder alone to the susceptibility for alcoholism (August & Stewart, 1983; Boyle et al., 1992).

**Temperament**

While considerable research has shown that a predisposition to alcoholism is partially due to genetic factors, several studies suggest that this genetic susceptibility may be expressed, in part, through an individual's temperament. Tarter and Vanyukov (1994), for example, propose a temperament model of alcoholism risk based on five temperament traits that increase an individual's liability for developing alcoholism.

There may be a host of biological factors involved in alcoholism susceptibility.

These traits include behavioral activity level, sociability, attention span/persistence, emotionality, and "soothability." Genetics influence each of these five traits, and an individual's liability is increased or decreased by the deviation of each trait from the population norm. Thus,
individuals whose personality traits are closer to the population norm are thought to have more control over their own behavior, including substance use. Individuals who have difficulties with behavioral and emotional regulation may be more prone to developing alcoholism in relation to environmental influences and stressors, including seeking environments conducive to alcohol and drug use. Indeed, each of these traits, or trait clusters, that constitute a “difficult” temperament relate to an increased risk for developing a problem with substance use and/or abuse (Ohannessian & Hesselbrock, 1995; Tarter, Kabene, Escallier, Laird, & Jacob, 1990). It should be noted, however, that prenatal, perinatal, and neonatal circumstances can have profound and persistent influences on temperament, as well (e.g., maternal stress and prenatal exposure to stress hormones; medications delivered during pregnancy and/or delivery; anoxia; hypoxia; birth trauma; child maltreatment; etc.). Etiological models (psychoanalytic, behavioral, cognitive, and social learning) have led to the development of different treatment methods for alcohol use disorders.

### Psychological Models of Etiology

A number of models exist to explain the development of alcohol use disorders, including those grounded in the field of psychology. Each of these models involves different treatment methods.  

**Psychoanalytic models**

- Oral fixation
- Ego malfunction

Early psychoanalytic formulations concerning the etiology of alcoholism were based on the clinical experience of therapists trained in this tradition. In psychodynamic thought, alcoholism was viewed as a fixation on, or a regression to, the oral stage of development. More recently, though, malfunctions of the ego or an emphasis on the self have been proposed to explain alcoholism. Wurmser (1984–85), for example, views the use of alcohol or drugs as an attempt to escape from intense feelings of rage and fear arising from severe intra-psychic conflict due to an overly harsh superego. Khantzian, Halliday, and McAuliffe (1990), posited a self-deficit approach; that is, inadequacies of the ego underlie abuse of alcohol or drugs. Individuals choose a specific drug with particular properties to self-medicate their particular type of ego deficit. Other formulations indicate that alcoholism is a psychosomatic defense against psychic conflict (McDougall, 1989), or the result of disturbed object relations (Krystal, 1982). Although there is little empirical support for these etiological theories, many members of the larger treatment community continue to use these terms as clinical descriptors for their clients and to suggest a cause for the client's conditions. The interventions that emerge out of these models primarily include psychotherapeutic analysis approaches.

**Behavioral Models**

- Substance use is learned
- Substance use is maintained through conditioning mechanisms

Traditional behavioral models of alcohol and other substance use disorders have postulated that alcohol and other substance use behavior is learned and maintained either through classical or operant conditioning. In the typical classical conditioning paradigm, the development or "learning" of drinking behavior occurs through repeated pairings of: (1) a conditioned stimulus (CS), such as a particular person and an unconditioned stimulus (US), such as a particular
location or time of day with (2) alcohol consumption. After repeated pairings, a conditioned response (CR) develops where exposure to the CS or US results in the CR (drinking behavior). This model has been postulated to explain the initial development and maintenance of craving and conditioned tolerance (both conditioned responses), for alcohol as well as other drugs (Wikler, 1973; Siegel, 1983).

Operant conditioning principles have been applied to explain alcohol and other drug use as reinforcing. That is, an individual may drink in response to an antecedent stimulus such as bad mood, anger, social anxiety, physical pain, or even withdrawal symptoms. An association is then developed between the reinforcing effects (e.g., perceived better mood, anxiety reduction, lessening of the severity of the withdrawal symptoms or pain) with the antecedent stimulus. Thus, drinking is thought to increase as a result of either the positive (bringing a perceived reward) or negative (removing a perceived negative factor) reinforcing effects of alcohol. Recent 'self-medication' theories of alcoholism are based on the assumption that alcohol becomes a positive (or negative) reinforcement. One limitation of this model is the observation that the development of alcoholism in both clinical and non-clinical populations often precedes the development of a diagnosable affective or anxiety disorder (cf. Schuckit, Anthenelli, Bucholz, Hesselbrock, & Tipp, 1995; Schuckit & Hesselbrock, 1994).

In the learning paradigm, negative effects of drinking (e.g., hangover, social consequences, personal costs, legal problems) are viewed as being too far separated in time from the drinking behavior to seriously reduce its frequency-and the positive reinforcing effect is experienced first. This is the premise underlying the use of certain pharmacological interventions to curtail drinking (Gitlin, 1996). For example, drugs such as Antabuse (disulfiram) result in the relatively rapid onset of unpleasant physical symptoms after alcohol consumption (e.g., generalized malaise, flushing, sweating, headache, nausea, vomiting, palpitations and/or chest discomfort).

**Cognitive Models**

- Cognition and feelings direct behavior
- Alcohol use perceived as positive / negative reinforcement

Cognitive behavioral models of alcoholism emphasize the importance of cognitions (thoughts, understanding, beliefs) and feelings as preceding and directing behavior. In these models, the initial use of alcohol or other drugs is viewed as the result of several interacting factors (e.g., genes, temperament, and other psychological or social factors). These factors influence the individual's perception of alcohol use as either a positive or a negative reinforcer. As alcohol use increases, other coping mechanisms are used less frequently. Consequently, self-efficacy is reduced and positive expectancies increase as alcohol use increases. As high levels of use become more frequent, classical conditioning processes (e.g., conditioned craving, tolerance, and withdrawal) play an important role in the development and maintenance of heavy problem use (see Rotgers, 1996 for a review).

**Social Learning Models**

- Focus on cognitive constructs
- Expectancies
- Self-efficacy
- Attributions

Social learning theories focus on cognitive constructs such as expectancies, self-efficacy, and attributions to mediate the pathway from stimuli to alcohol use as a response. Expectancies of
the positive effects from using alcohol develop as conditioned cognitions from repeated classical or operant pairings of alcohol use with a positive experience (i.e., reinforcement). Self-efficacy refers to the expectation by individuals that they can successfully perform a particular coping behavior in certain situations and that the behavior will be reinforced. The Social Learning viewpoint describes alcoholism as a result of a failure to cope. The self-efficacy for coping without alcohol is low among alcoholic individuals, contributing to continued use and the eventual development of dependence. Petraitis, Flay, and Miller (1995) have postulated a social learning theory model of adolescent experimentation and the eventual problem use of alcohol and other drugs.

**Alcohol expectancies:**

Positive expectancies of effects of alcohol  
Social facilitation  
Enhanced sexual performance  
Increased personal power  
Social assertiveness/relaxation

Many experimental laboratory studies of alcohol intake assess the ability of alcohol to influence certain behaviors such as the induction of aggression, increased sexual arousal, or tension reduction. These approaches all assume a cognitive influence surrounding alcohol use. An underlying assumption of such studies is that individuals have certain positive expectations of the effects of alcohol related to the behavior being studied. Consequently, a number of specific alcohol-related expectancies have been identified. These include social facilitation, enhanced sexual performance and pleasure, increased personal power and aggression, social assertiveness, relaxation and tension reduction, as well as a general positive outcome that may result from drinking.

Several instruments are available to assess the expectancies of alcohol's effects (cf. Brown et al., 1980; Southwick et al., 1981; Christiansen et al., 1982). These expectancies probably reflect not only a person's own experience with alcohol, but may also result from exposure to beverage alcohol advertising and from observing the behavior of others when they are drinking (both real life and media models).

Exposure to these modeling events can begin early in life, even during childhood. Miller et al. (1990) examined the alcohol expectancies of elementary school children across the first through fifth grades. They found that the positive expectancies of the effects of alcohol increased with age, most notably among 8-10 year olds. Importantly, a variety of studies have shown that positive expectancies of alcohol's effects predict initiation of drinking, intention to drink, and drinking rates among middle school (Christiansen et al., 1989) and college students (Stacy et al., 1990).
Although originally linked to attitudes and beliefs about the reinforcing properties of alcohol, expectancies are more recently believed to be related to memory processes. Thus, positive expectancies of alcohol use may be encoded in close association with usual drinking practices and be easily retrieved from memory in future drinking situations. On the other hand, negative expectancies arising from unpleasant drinking experiences are probably less likely to be associated with usual drinking practices, but may be more closely tied to heavy drinking episodes. Consequently, among light-to-moderate drinkers, negative expectancies of alcohol’s effects are less likely to be activated or to play an inhibitory role in most drinking situations.

It is important to recognize that each of these etiological models (psychoanalytic, behavioral, cognitive, and social learning) have led to the development of different treatment methods for alcohol use disorders. To date, however, no single particular psychological treatment method has consistently been demonstrated to be superior to another across groups. Furthermore, newer pharmacological agents, such as naltrexone and acamprosate, appear to be effective in reducing drinking in some clients, particularly when used in conjunction with psychotherapy or other behavioral intervention approaches. Together, these findings also point to the importance of integrating biological and psychological factors in the treatment of alcoholism, and in understanding its etiology.

**SOCIOCULTURAL MODELS OF ETIOLOGY**

Some cautions are urged when reviewing possible environmental risk factors for alcohol use disorders (Sher, 1991). The most significant caution is to recognize that, although certain environmental conditions and risk factors are associated with alcoholic families, these conditions are not necessarily related to the later development of alcohol problems or alcohol use disorders in the offspring. Variations in the environmental conditions expressed in alcoholic families, such as poor parenting or disorganized family life, need to be empirically related to specific outcomes to have etiologic significance. Second, even when the environmental factor of interest has been empirically associated with a particular outcome, its role as a cause cannot be assumed—causality cannot be inferred from correlation alone.

For example, it may be that the factor does indeed cause the outcome. But, an equally plausible explanation may be that the second variable actually causes the first, or that the two variables are interrelated in a dynamic mutual influence pattern. Still another possibility is that a third variable, such as a co-morbid psychiatric condition in either parents or children, may falsely lead to the appearance of causation when the effect is almost entirely due to this third unmeasured variable influencing both the first and second.
An example of this is the association between parental alcohol abuse and the presence of oppositional behavior disorders in children. On one hand, the oppositional behavior might be blamed on the parental drinking pattern. On the other hand, Lang, Pelham, Johnston, and Gelerrnter (1989) observed that interactions with oppositional children may result in increased alcohol consumption among the adults providing their care.

**Family violence:**
Alcohol use is present in a substantial proportion of domestic violence incidents reported in the general population. An estimated 67% of persons who victimize an intimate partner (e.g., spouse, boy/girlfriend, ex-partner) have used alcohol acutely or chronically at the time of the arrest, compared to 38% who victimized an acquaintance or 31% who victimized a stranger (NIAAA, 2000). Because both alcoholism and family violence have some demonstrated family "transmission" patterns, social workers may assume that there is a strong relationship between family of origin alcoholism and present family violence. However, a careful and critical reading of the research literature does not provide clear and consistent evidence. Many published studies are based upon small samples, do not adequately separate different types of partner abuse, sample from populations or agencies that are likely to have high rates of both alcoholism and violence, and/or do not use adequate comparison groups. While alcohol use and violence are clearly associated, the causal relationships between parental alcoholism, family violence, and alcohol problems among the offspring have not been firmly established.

It is also possible that some aspects of family violence, including sexual abuse of children and adolescents, may differentially affect girls and boys. These differences may appear as differences in susceptibility to poor adult outcomes, including the development of alcohol use disorders. Several studies indicate that the prevalence of early sexual abuse is much higher among female alcoholic clients than that found in the general population of adult females.

**Family interaction:**
Implicit in the discussion to this point is the notion of alcohol (and other drug) use disorders as being multiply determined by a complex association of genetic, environmental, personality, and other factors. Frequently, more than one member of the nuclear or extended family experiences a substance dependency. This complicates the identification of specific influences that family environment, child-rearing practices, or inter-parental interaction may play in the development of alcoholism. Three general contemporary models of family influences can be identified: a family disease model, a family systems model, and a behavioral family approach (McCrady & Epstein, 1996; McCrady, Kahler, & Epstein, 1998).

The family disease model is based on an assumption that all family members suffer from some degree of either alcoholism or codependency. Further, alcoholism and codependency are interrelated in such a manner as to enable (perpetuate) the alcohol problem. Although in this model the specific etiology is regarded as biological, alcoholism is being maintained by the family disease (Sheehan & Owen, 1999).

In the family systems model, the etiology of alcoholism and substance abuse is focused on the behavior of family members around drinking, with particular attention paid to the family of origin and the role of the spouse/partner (O'Farrell & Fals-Stewart, 1998; Steinglass, Bennett, Wolin, &
Reiss, 1987; Steinglass, Weiner, & Mendelson, 1971). The model assumes that, over time, alcohol use stabilizes the family system and that the family organizes their interactions and structure around alcohol use to achieve and preserve system 'homeostasis.' In other words, the family maintains the alcohol problem despite the associated problems because it is requires less effort than changing or because it allows the family to avoid changing a more disturbing problem (e.g., sexual abuse).

The behavioral family approach focuses on the family members' behaviors (especially those of the spouse/partner), as both antecedents to and reinforcers of, alcohol or substance use. These responses are thought to help develop and maintain the drinking problem. Bennett and Wolin (1990) found that continuing interaction between adult offspring and their alcoholic parents is associated with increased rates of alcoholism, at least among the male offspring. On the other hand, certain family rituals, such as eating dinner together or celebrating holidays together, may serve to protect offspring against the development of alcoholism (cf. Bennet et al., 1987). It is important to note that family member behavior can influence the alcoholic individual to consider change, act to change, maintain the change, or relapse to drinking (Walitzer, 1999).

**Peer influence:**
Adolescents often cite an increased ability to socialize with friends, reducing tension and anxiety (especially in mixed gender situations), reducing boredom, and/or getting high as reasons for their alcohol and other substance use. As indicated above, expectations of alcohol's effects in these areas are associated with both initiation of alcohol use and drinking rates, particularly among adolescents. Peer influences are consistently cited as risk factors for initiating alcohol, tobacco, and other drug use among children and adolescents (cf. Kandel & Yamaguhi, 1999; Wills, Vaccaro, & McNamara, 1992; Averna & Hesselbrock, 2001). Peers influence adolescents' values, behaviors, attitudes, and choice of other friends. However, the closeness of the specific peer relationship is an important determinant of the strength of peer influences on drinking behavior. Alcohol use by an adolescent's best friend is more predictive of alcohol use and maintenance of drinking behavior than reports of use by other friends. Characteristics of peers may also be relevant. Reports of use by same-age peers do not appear to be related to either initiation or maintenance of drinking during adolescence (Morgan & Grube, 1991; NIAAA, 1994). Associating with deviant friends tends to promote the acceptance of deviant behaviors, including the use of alcohol and other drugs (Loeber, Stouthamer-Loeber, Van Kammen, & Farrington, 1991; Robins & McEvoy, 1990). However, it is not clear if associating with deviant peers is a risk factor for, or the result of, maladaptive behaviors. Deviant peer group involvement co-occurs with several other risk factors such as family problems, other mental health problems, low self-esteem, stress, and alcohol availability. Deviant peer group involvement is typically higher among alcohol-using adolescent boys than girls. Peer and friend relations (Ohannessian & Hesselbrock, 1993) probably exert less influence on drinking behavior in adulthood than during adolescence. At least among adult persons living together in a stable relationship, social support from close family members is more predictive of drinking behavior than social support from friends.
Social environments:
A variety of social and environmental factors may affect a person's risk for developing alcohol use disorders. Both social learning and social control theories consider that social environments provide a wider context for biological, psychological, and personality factors to interact in determining a person's susceptibility for developing alcohol abuse problems. Peer influences to initiate or maintain use, stressful and negative life events, and family environment (including poor parenting styles) appear further to enhance the likelihood of developing alcohol or drug-related problems among adolescents and young adults who are at high risk due to a family history of alcoholism. However, an adolescent's exposure to alcohol tends to be more limited in the presence of good relations with non-using peers (particularly best friends), family rituals that actively seek to prevent alcohol use, and consistent parental supervision and discipline. Reduced exposure to alcohol use, in turn, limits the opportunity for expression of genetic, psychological, and personality susceptibility risk factors for developing alcoholism (see Hesselbrock & Hesselbrock, review, 1990).

Social Policy Issues
Social policy, even though often ignored in etiological formulations, can have a wide influence on the risk for developing problems with alcohol abuse and its concomitants (e.g., illness, injury, etc.). Social policy can influence the availability of beverage alcohol and provide punitive measures for violation of purchase and consumption regulations. To some extent, limited access to alcohol serves to protect against the development of alcohol problems and abuse or dependence (Holder, 1999). Over the years, local, state, and federal governments have used a variety of measures to restrict the availability of beverage alcohol. Prohibition, local option, and minimum legal age for purchase have had both short- and long-term effects in restricting the availability of alcohol. Lowering the legal age for purchase in the early 1970s led to increases in both alcohol consumption and auto injuries and fatalities among adolescents. These trends were reversed when the minimum legal drinking age was again raised. The direct effect of changes in legal drinking age on other alcohol-related behaviors (e.g., assaults, teen pregnancies, sexually transmitted diseases, and accidental drowning) is more difficult to assess because the minimum drinking age and reporting practices have varied considerably from state to state.

Taxation has also been viewed as a method for controlling the availability of alcohol, since higher taxes on alcohol typically lead to higher prices. For some individuals, raising taxes on beverage alcohol has been associated with decreased drinking. However, light and heavy drinkers appear to be less responsive to increased prices than are moderate drinkers (Manning, Keeler, Newhouse, Sloss, & Wasserman., 1991). In addition, Kenkel (1996) found that drinkers who are better informed about the risks of excessive alcohol use showed greater reductions in drinking due to price increases than did less informed drinkers. The use of taxes to increase the cost of obtaining alcohol-containing beverages is not straightforward. Even though federal taxes are applied uniformly to all units of beverage alcohol produced, manufacturers and retailers operating in different locales and competitive markets may choose to differentially pass along
the cost to the purchaser. Further, the cost of a unit of beverage alcohol can vary considerably by beverage type, geographic region, bottling type, and type of establishment where the beverage is purchased.

**Clinical Heterogeneity**

The above sections have provided a general overview of different factors thought to be related to the etiology and natural history of alcohol. Two additional factors must also be considered: clinical heterogeneity and gender. The many pathways to heavy drinking and alcohol dependence can often be reflected in the clinical heterogeneity or subtypes of alcoholism observed by the clinician. Differences in the development of the disorder can also be traced to factors more typically found in one gender versus the other.

**Clinical heterogeneity / multivariate subtypes:**

While alcoholics share many attributes related to their disorder, clinicians have also noted many individual differences in symptom patterns, drinking patterns, comorbid psychiatric problems, pathways to alcoholism, and personality characteristics. Bowman and Jellinek (1941) were among the first to propose a set of conceptual alcoholism typologies that were hierarchical and based on drinking patterns and personality factors.

The schema resulted in a representation of 17 subtypes. Jellinek revised this scheme in 1960 to include the Alpha, Beta, Gamma, and Delta types that are more widely known today. The Alpha and Beta types were not considered to be severe forms of alcoholism (see Table 3). The Alpha type was thought to have psychological dependence, while the Beta type could also experience physical/medical problems due to poor nutrition and health rather than directly from drinking. Neither type was thought to experience loss of control over drinking, suffer from an inability to abstain, or experience withdrawal symptoms.

**Table 3: Alcohol-Related Features of Type A and Type B Alcoholics**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Type A</td>
<td>Type B</td>
</tr>
<tr>
<td>Onset of alcohol symptoms (years)</td>
<td>17.9 (5.5)**</td>
<td>15.5 (2.9)</td>
</tr>
<tr>
<td>Onset of regular drinking (years)</td>
<td>17.3 (4.6)**</td>
<td>15.5 (3.7)</td>
</tr>
<tr>
<td>Onset of problem clustering (years)</td>
<td>25.1 (9.2)**</td>
<td>20.6 (6.3)</td>
</tr>
<tr>
<td>Longest abstinence (months)</td>
<td>21.7 (43.4)</td>
<td>20.0 (24.1)</td>
</tr>
<tr>
<td>% alcohol treatment (any)</td>
<td>58.9**</td>
<td>96</td>
</tr>
<tr>
<td>% Inpatient treatment</td>
<td>47.6**</td>
<td>86.7</td>
</tr>
<tr>
<td>% currently abstinent (6 months)</td>
<td>24.8*</td>
<td>38.7</td>
</tr>
</tbody>
</table>

*p< .05, **p< .01 (Source: Schuckit et al, 1995)
Neither of the Alpha or Beta forms of alcoholism was considered to lie within the disease concept of alcoholism, however, both the Gamma and Delta forms were considered in this manner (Jellinek, 1960). The Gamma type of alcoholism was thought to be the predominant form of alcoholism and was characterized by physical dependence, craving for alcohol, and withdrawal following cessation of a drinking episode. Even though loss of control over the amount consumed is absent among Delta alcoholics, they are unable to totally abstain from drinking. The physical consequences of drinking are typically less severe among Delta alcoholics than among Gamma alcoholics. Another type, Epsilon, was proposed to denote a special type of alcoholism based on binge drinking. Jellinek invoked heredity and genetics only when he was unable to explain the process of addiction in relation to a psychological or sociological etiology. However, Jellinek considered the Gamma type of alcoholism to be relatively homogeneous and did not adequately address either the heterogeneity or the multidimensional nature of alcoholism often found within this category.

Over the past twenty years, a number of clinicians and researchers have made attempts to identify more homogeneous subtypes of alcoholism. More recently, multivariate statistical methods such as cluster analysis (Babor et al., 1992), discriminant function analysis (Cloninger et al., 1981), and latent class analysis (Bucholz et al., 1996) among others (Hesselbrock, M.N., 1995) have been applied to clinical data to identify homogeneous subtypes of alcoholism that may eventually prove to be differentially responsive to particular treatment modalities. Most prominent among these are the Type 1 / Type 2 alcoholisms proposed by Cloninger and associates (1981) and the Type A / Type B forms of alcoholism proposed by Babor et al., (1992). The two formulations are remarkably similar, even though the Cloninger et al. sample was derived from a Swedish community-based adoption study sample, while the Babor et al. typology was based upon an inpatient sample. Both samples contained males and females. Both Type 1 and Type A were considered to be milder forms of alcoholism, were more prevalent among females, had a later onset of the disorder, and typically had significant anxiety and depressive symptoms. Type 2 and Type B alcoholism were characterized as having an earlier onset, the presence of childhood problem behavior risk factors, adult anti-sociality, familial alcoholism, and a more chronic treatment history.

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The personality traits (reward dependence, harm avoidance, novelty seeking) so prominent in Cloninger’s conceptualization do not appear to be sufficient to define Type 1 / Type 2 (Hesselbrock, M.N., 1995). Although Type 1 and Type A alcoholism are very common among women, a significant number of female alcoholics can be classified as having Type 2 / Type B alcoholism. A number of studies have attempted to replicate the Type 1 / Type 2 typologies in other samples (clinical and non-clinical) with limited success (Glenn & Nixon, 1991; Schuckit, Irwin, & Mahler, 1990). Type 1 / Type 2 do not appear to be heritable (Gilligan, Reich, & Cloninger., 1988).

Schuckit et al. (1995a) were able to identify the Type A / Type B subtypes of alcoholism in a large sample derived from a family study of alcoholism. Mezzich and colleagues (1993) found Type A / Type B among adolescents with alcohol dependence. The heritability of Type A / Type B has not yet been examined. Other subtypes of alcoholism have been identified through the application of multivariate statistical methods to a variety of clinical assessments, such as the MMPI (Blashfield, 1984) and clinical interview data (Bucholz et al., 1996). (A review of typological classifications of alcoholism can be found in volume 20, issue 1 of Alcohol Health & Research World (NIAAA, 1996); see also Hesselbrock, 1995.)

**Gender differences:**

Gender differences in drinking patterns, the metabolism of alcohol, the course of developing alcohol problems, the point and lifetime prevalence of alcohol problems (including alcohol abuse and dependence), co-morbid psychiatric conditions, the physical consequences of chronic alcohol use, and treatment utilization have been noted in the literature (see review in Wilsnack & Wilsnack, 1997). Typically, women are less likely to consume alcohol than men, and when they do, women typically drink smaller amounts per occasion. Mortality rates among women are higher than among men who drink heavily, due to a variety of causes such as accidents, violence, suicide, and medical illnesses (NIAAA, 2000).

Still, there are many similarities among male and female alcoholics. Schuckit et al (1995a) reported a high level of correspondence in the temporal sequencing of the occurrence of 19 major alcohol-related life problems among male and female alcoholics, as well as among drinking but non-alcoholic individuals (see Table 4). Although the age of first appearance of the different symptoms examined may vary by gender, the order of appearance was very similar between males and females. Further, once alcohol dependence develops, the clinical manifestations of the disorder show few gender differences at either the low or high end of severity (Hesselbrock, M.N., 1991a; Del Boca & Hesselbrock, 1996; Hesselbrock, Segal, & Hesselbrock, 2000).

At an intermediate level of severity of alcohol dependence, women often manifest more anxiety and depressive symptoms but lower levels of antisocial behaviors compared to men. An antisocial type of alcoholism predominates at the more severe end of the alcohol dependence spectrum and few gender differences are noted (Bucholz et al., 1996; Hesselbrock et al., 2000). However, women tend to do as well as men following treatment for alcoholism, and in some settings, they may have a better outcome (Gomberg, 1999).
Table 4: Sequence of Development of Alcohol-Related Life Experiences for Men & Women

<table>
<thead>
<tr>
<th>Item #</th>
<th>Life Experience</th>
<th>Men (n=317)</th>
<th>Women (n=161)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Physical fights while intoxicated</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Rank</td>
<td>Age</td>
<td>Rank</td>
</tr>
<tr>
<td>2</td>
<td>Use in hazardous situations</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>Hit others (non-fight)</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>Arguments while drinking</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>Started when not want to</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>Drink more than intended</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>Problems at school/work</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>8</td>
<td>Hit/threw things while drinking</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>9</td>
<td>Lost friends due to drinking</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>10</td>
<td>Blackouts</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>41</td>
<td>Liver, ulcer, pancreatitis</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>42</td>
<td>3rd abstinence of 3+ months</td>
<td>43</td>
<td>43</td>
</tr>
<tr>
<td>43</td>
<td>Convulsions following abstinence</td>
<td>36</td>
<td>36</td>
</tr>
<tr>
<td>44</td>
<td>4th abstinence of 3+ months</td>
<td>44</td>
<td>44</td>
</tr>
</tbody>
</table>

Overrall rho = .84, p< .01 (Source: Schuckit et al, 1995)

Concluding Thoughts About What We Know

1. To date, the scientific evidence clearly shows that drinking behavior, including alcohol dependence, is highly heritable. Biological offspring, both sons and daughters, of an alcoholic parent are at increased risk for developing alcoholism compared to offspring of non-alcoholic parents. Offspring of an alcoholic parent have a 2-4 fold increased risk for developing alcoholism themselves.

2. There is no evidence that a specific genes or genes "predetermine" alcoholism for an individual. It is likely that a variety of genes, each making a small contribution, increase a person's susceptibility for alcohol dependence. Transmission is most likely polygenic, with many susceptibility loci, each with small/medium effects and low penetrance. Most likely, there are a number of genes that predispose an individual to psychopathology. Some may increase the likelihood of developing alcohol dependence. Regions on several chromosomes seem to be promising.

3. Social, psychological, and environmental factors interact along with genetic susceptibility to influence a person's overall risk for developing alcohol problems. Environmental factors likely play a significant role, as is true with other common diseases and disorders (e.g., heart disease, cancer, diabetes, asthma)

4. Current findings indicate that individual variations in alcohol's effects on the central nervous system (alcohol sensitivity) and differences in alcohol metabolism (affective tolerance) are likely to be important determinants of the risk for developing alcohol problems.
5. Genetic studies may help us understand the biological basis of alcohol dependence. For example, is individual variation rooted in the genetic basis for differences in ethanol metabolism (e.g., absorption and elimination rates)? Or, does it lie in differential central nervous system (CNS) effects of ethanol (e.g., neurotransmitter and receptor mechanisms)? In fact, "protective" genes may exist, as well. Identification of susceptibility and protective genes may lead to the development of targeted prevention and intervention strategies.

6. Treatment works for those with a family history of alcoholism.
References


