

Chapter 6

Causal Models of Alcohol and Other Drug Problems

Introduction

In Chapters 3, 4, and 5 we described some important sociocultural, psychological, and physiological factors which help determine the varied use patterns of alcohol and other drugs in various populations. We also sampled the kinds of problems which sometimes arise from that use. In this chapter, we ask: How can we relate all these facts to each other in a way that will allow us to better understand the causes of alcoholism and other drug abuse? In dealing with this question, we will describe common models the field has developed to aid in organizing all this information. We will also consider how we can use this knowledge about causes to (1) target high-risk subgroups for preventive intervention, and (2) reduce the likelihood of developing alcohol and other drug problems. Finally, in this chapter, we will also explore the ways in which things we have learned can help us provide treatment and other interventions for people who are already manifesting problems.

Etiological Considerations

The study of causes is called **etiology**. Investigating substance use problems in any group of people involves determining the important causal factors for that population. Specific policies and strategies are then developed to reduce the impact of those variables through prevention and treatment.

When the focus is on more than one individual, as is often the case for prevention, restricting the assessment to a group of people who are similar makes it easier to determine which causal factors are most critical. The more varied the population, the harder it is to pinpoint which of many factors is the most influential.

A recent comprehensive study about alcohol problems conducted by the Institute of Medicine¹ recommends a framework which can be used to analyze alcohol and other drug problems from a causal perspective:

- There is *no* likelihood that a *single* cause will be identified for all instances of alcohol (drug) problems.
- There is *every* likelihood that the range of causes which interact to produce alcohol (drug) problems can be identified.
- Alcohol (drug) problems will prove to be the result of *different* interactions of *different* etiological factors in *different* individuals, at *different* life stages, in *different* settings.

The Institute of Medicine study also quotes Harold Mulford (from the Encyclopedic Handbook of Alcoholism, 1982). In paraphrase: “This way of thinking views every drinker (user) as being at some stage of a dynamic, lifelong process influenced by a large number of weak, interacting social, psychological, and physical forces with no single factor except alcohol (the drug) being necessary, and none at all being sufficient to cause advancement in the process to the point of

being labeled ‘alcoholic’ or ‘problem drinker’ or ‘drug addicted.’”²

The Utilization of Models

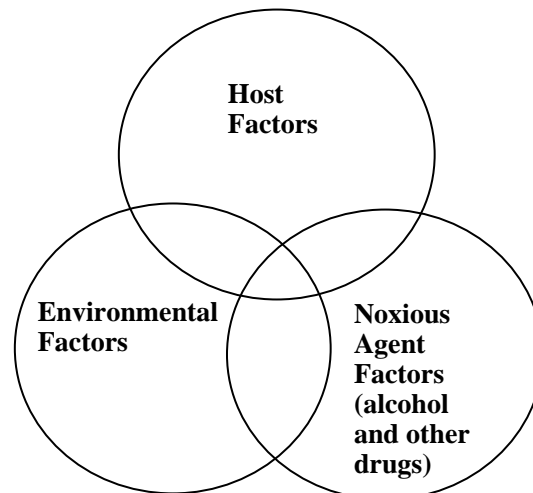
A **model** is an organizing framework, typically stated in shorthand form, which describes a system. Thus, an *etiologic* model would be a short statement, or diagram, which specified what ingredients were necessary, in what way, for drug abuse to occur. And a *conceptual* model is both a theoretical and philosophical construct, that specifies what is important and what is not, about a concept.

In the alcohol and drug area, the most familiar models are conceptual ones. Thus, medical-diagnostic labels—such as “alcoholism,” “alcohol dependence,” and “drug dependence” typically imply that a disease exists, with a distinct and specified set of signs and symptoms, and that it has its root cause(s) within the individual (rather than the environment, such as poverty). Some may use the term with the understanding that the drug itself is a root cause, rather than the individual. For example, it is said that cocaine is highly addictive, regardless of individual susceptibility. So when we designate a problem as “alcoholism,” or as a drug addiction, we are saying implicitly that the causes of this trouble are within the individual. This example is what is called the disease model or the disease concept of alcoholism.³ The disease model is also called a “medical model.” We typically are implying that the disease process is progressive, that it has a specified course, that it began with a common set of causal factors (common etiology), and that it is likely to be due to a set of disordered biochemical processes.

There are, of course, other alternative conceptual models in the area of alcohol and other drugs. Two prominent ones are:

- (1) The “problem use” model⁴ simply specifies the extent to which use of alcohol and other drugs occurs with other life difficulty or trouble; the more the trouble, the worse the problem. This model makes no assumptions about the degree to which trouble will be maintained over time, so it is a very different framework within which to view difficulty than a model which conceives of alcohol and other drug problems as having an irrevocably progressive course.
- (2) Related to the problem use model is the “epidemiologic model”: here, the location of trouble does not reside within the individual, but rather depends on the co-occurrence of multiple causal factors (see Figure 6.a). This view depicts difficulties related to alcohol and other drugs as emerging when there is an interplay of three types of factors: host factors (that is, within the individual user/consumer), environment factors which may enhance the likelihood of alcohol and other drug involvement (for example, living in a high stress, high poverty environment), and “noxious or infectious agent” factors, such as having exposure to the noxious agent (in this instance, exposure to alcohol and other drugs). Within the framework of this model, we can also see how the interplay of these factors can lead to more or less difficulty with alcohol and other drugs. If one is involved with a noxious agent which has a great capacity to give a strong and immediate high (for example, cocaine), then the likelihood of problems with alcohol and other drugs is greater because the agent is more potent. And, if one lives in a community (or country) where there are no laws restricting access to drug use, making the noxious agent more readily available, the likelihood of trouble is also greater.

Figure 6.a Epidemiologic Model



Within the framework of the epidemiologic model, disorder is a result of the interplay of three different sets of factors. Only when we have overlap among them (the shaded area of the diagram), which shows the operation of multiple influences, do we have problem outcomes as a result.

Thus, the models that we use have a *precise meaning* to us that shapes the way we use the terms. Some of these terms have alternative definitions, and not all people use the same definition. So it is absolutely essential for us to know how terms are being defined when we classify problems and apply these labels.

The choice of a particular model not only presumes a theoretical notion about the conditions that led an individual to become “alcoholic,” “chemically dependent,” or “addicted” (that is, the “cause”), it also embodies some view of what will take place after the condition is established. That is, there is a **course** to such a problem and a set of expected outcomes, or **prognosis**.

Taken together, these elements define a causal model of an alcohol and other drug problem. It is true that some models are not complete, and do not explicitly state each of these components. However, when complete, all have the following elements:

- A descriptive structure, or *concept*.
- A causal structure, or *etiology*.
- A developmental structure, or *course*.

Etiologic Models of Alcohol and Other Drug Problems

Causal models identify either a single-cause or multiple (usually interrelated) causes. Single-cause models enable one to focus narrowly on the problem (for example, “alcoholism” is a genetic disease; “heroin dependence” is an addictive disorder; the cause of “drug abuse” is poverty). Moral, medical, and spiritual models tend to be single-cause models, and most of them are incomplete insofar as they focus primarily on the descriptive structure of the problem, without much consideration of the other two components noted above.

Multicausal (or multifactorial) models are necessarily complex because they are based on the idea that several factors determine the problem. In addition, these factors interact with each other, and change in character over time; sometimes they add together, sometimes they work against one another. The way they interplay with each other may lead to a harmful outcome or a lessening of the problem over time. The application of such models to prevention planning presumes that if we understand the causes, we can modify the elements of the systems involved.

It is important to realize that a revolution is now taking place in the way that modern professionals understand and describe the complex processes that we used to call “addiction.” As we get better at tracking what goes into determining causes, it becomes increasingly more clear that some old views (that is, that *drugs* are addictive) are too simple-minded. By addiction, we mean “a pattern of behavior that involves overwhelming need (or craving), along with the development of physical dependence following on continued and heavy drug use.”⁵ The process is presumed to be compulsive, heavily driven by the pharmacological properties of the substance, and irrevocably leading to the development of tolerance and physical dependence. The reality of the situation is more complex than this; no one moves from nonuse to compulsive dependency without the intervention of other factors.

What is a reasonable causal model of alcohol and other drug problems today? The one increasingly utilized by sophisticated practitioners and researchers is that all of these problems are multicausal. In other words, problems manifest themselves when a number of causal factors operate together.

In fact, we can see how these multifactorial dynamics may operate at all stages of problem development, from the earliest predisposing influences to dependence and even end-stage pathological consequences. The matrix in Figure 6.b—one representation of this complex situation for the development of alcohol and other drug dependence—might help you to visualize this process.

For simplicity, let’s consider three distinct areas of our lives that influence or determine whether problems develop: *biological*, *psychological*, and *sociocultural*. In the natural course of the process, events in each of these areas affect the vulnerability (*predisposition*), onset (*precipitation*), continuation (*perpetuation*), and negative consequences (*later pathology*). This diagram, showing a matrix of factors, implies that different combinations of these determinants occur under different circumstances, at different times in different people’s lives, to produce a wide range of different outcomes. It also suggests that forces in one area can be enhanced *or reduced* by forces in the others. A person who may have a genetic predisposition* (suggested by a parent or sibling with alcohol problems), may also have been exposed to powerful sociocultural influences that soften its effect, such as a circle of close friends who are highly valued and do not drink.

Figure 6.b Stages of Problem Development and Areas of Influence

Areas of Influence:	PREDISPOSING INFLUENCES	PRECIPITATING INFLUENCES	PERPETUATING INFLUENCES	LATER PATHWAYS
BIOMEDICAL/GENETIC				
PSYCHOLOGICAL/ BEHAVIORAL				
SOCIO/CULTURAL				

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Predisposing Factors

A closer look at each of the boxes in the matrix may help to clarify these points. Let’s talk about the *predisposing influences* first. Biomedical scientists have amassed a mountain of data over the past generation that largely supports the clinical observation of some hereditary vulnerability that can pass from parent to child. Sons of alcoholic fathers, for example, are more likely to require a higher dose of alcohol for intoxication—even before they have begun to drink on a regular basis. This does not seem to be due to alcohol metabolism, but rather to something that is unique in their brain function. Experiments in a number of different animals (which breed more frequently than people) have shown that, whatever the biological factors are for alcohol preference and sensitivity, they definitely can be inherited.

People often begin using alcohol or other drugs because they are predisposed by *psychological factors*. The purpose of drug use is to improve the way we feel about ourselves, perceive the world around us, and interact with others. Our moods can vary greatly and many people find that alcohol and other drugs can take the edge off uncomfortable feelings of anxiety or depression, at least momentarily. So those with a greater tendency in that direction are likely more predisposed, or vulnerable ahead of time. We also know that greater impulsivity as a psychological characteristic, is predisposing, perhaps because the impulsivity is more likely to drive the individual, when feeling down, to immediately search for a quick solution, rather than puzzle out what the underlying cause of the distress might be. People who have diagnosable mental illness are even more at risk for using drugs to manage their feelings, and we now know that a majority of mentally ill patients have a problem with alcohol and/or other drugs.⁷

The *sociocultural influences* that predispose people to alcohol and drug use run the gamut from subtle to direct. Some have attracted a great deal of notoriety—like the billboard marketing campaigns for various alcohol beverages that have been mounted on major thoroughfares and in minority communities of our larger cities. Perhaps less obvious, but just as influential, is the concentration of bars and package stores that sell alcohol in some neighborhoods. And even more subtle than this physical access to booze, is the social acceptability of heavy drinking and

drug use that exists in some communities.

Precipitating Factors

Now let's take a look at the *precipitating factors* in each of these domains. When we think of the *biological influences* that could trigger substance abuse, we often focus on such drugs as pain killers. In fact, problems can and do occur with narcotics (opiates) that are taken for severe pain conditions. But it is important to realize that they occur very infrequently with the bona fide pain patient.

Psychological and especially social factors seem to be of greater influence when it comes to onset of substance use. *Psychological influences* would include a greater tolerance of deviant activity, and *social influences* would include traveling with a peer group who themselves are already into alcohol and other drug use. Insofar as the transition from nondrug use to drug use requires a step into a "different world," the soon-to-be user has to be ready to leave the old world behind, and experiment with the new worlds out there. The more one has a curiosity about and interest in those other, seemingly more deviant worlds, the earlier one is likely to take that next step into drug use. Similarly, if one is already traveling with a peer network of users, it is an easier first step for oneself, and in fact, is likely to be encouraged by one's already using companions.

Perpetuating Factors

Continuation or *perpetuation* of drug use can also be attributed to factors in each of these areas. Here we see that the *biological influence* can be profound, for physical tolerance and dependence become independent driving forces. Not one person who begins using drugs believes—at the outset—that he or she will come under the drug's spell this way. Yet, it is far more likely to happen whenever the drug is taken frequently and in large amounts. *Psychological influences* at this stage of drug involvement sometimes reflect a loss of the ability to choose. The vicious cycle of drug seeking-using-recovering takes over, bolstered by an increased sense of denial, coupled with increasing problems in the rest of the drug user's life. The user now drinks and drugs because he needs to, not because he wants to. It is no wonder that we call the condition *addiction*, a term which literally means "enslaved." *Social factors* take on a special significance in this phase. Their nature changes radically from the context of predisposition or precipitation, for the user no longer enjoys wide social approval. Frequently, the individual has to engage in frankly antisocial behavior to sustain a lifestyle, and necessarily rejects disapproving family and friends. The friends retained are likely also to be users—and they each reinforce the other's behavior.

Factors in Later Pathology

Pathology—the negative consequence of disease—is typically manifested after years of drinking or drugging, though it can develop in a shorter timeframe. Although the term pathology is a medical one, it is just as applicable to the psychological and sociocultural outcomes of chronic substance abuse as it is to the biological. Of course, physical pathology such as cirrhosis, lung cancer, and AIDS is recognized by just about everyone as a likely outcome of drinking, smoking, and injecting drugs. Apparently, the fact that every chronic user does not experience these consequences leads some to conclude that using is worth the "crapshoot." They are willing to take the risk that it will not happen to them!

While we are not in a position to state with certainty yet that alcohol and other drug abuse can cause all other psychopathology (mental illness), the probability is very high that abuse will unmask and aggravate major mood disorders and schizophrenia. Further, the evidence is also quite good that major depression and sometimes increased antisocial behavior are a part of the pattern of sustained and severe alcohol and other drug involvement. Neurotic mood swings are common among chronic drug users of all kinds, but these conditions are usually tied to intoxication and/or withdrawal. At the very least, chronic abusers suffer with unstable and unpredictable mood states—leaving them, their families, and friends in a constant state of apprehension and fear.

Our society and culture suffer too as a direct result of substance abuse. The ravages of alcohol and other drug-related crime stand in grim testimony of this fact. Much domestic violence is associated with alcohol and stimulant abuse, and robbery is frequently committed in order to support a drug habit. Some might even argue that the institution of urine drug testing in the workplace is a detrimental change in our society, reflecting a disheartening loss of control over our value structure.

About Risk and the Development of Later Disorder

It would be a mistake, from this brief overview, to conclude that all of the factors we have discussed here always act in concert to push the individual to a problematic, pathologic outcome. At the same time, there is increasing evidence in the literature on causes, which allows us to fill in the first boxes of predisposing factors (in Figure 6.b). Signs of risk for later alcohol or other drug problems may be identified early in the life of some individuals. Insofar as this is so, it also implies that for individuals with a heavy-risk burden, the process of developing alcohol and other drug problems may already be well under way by adolescence. But a **developmental perspective** (see Chapter 4) also requires that we consider the (to some extent) random nature of the way risk unfolds. For instance, having a positive family history for alcoholism or other drug dependence, being male, being hyperactive, having a particular genetic makeup, are each separate attributes that may or may not co-exist and contribute to a pattern of later problems.

How then might these factors work together? A developmental perspective helps us to understand the process. Ongoing life events call upon us to act in ways that we have acted before. That is, they elicit behaviors that are easier for us because we have already learned them from prior experience. Thus, living in an environment where peers expect and encourage aggressive behavior is likely to lead an individual to develop a pattern of higher aggressiveness. To put the matter differently, risky behavior (or risky biology) is likely to be enhanced by life situations that encourage it to show itself. Habit formation starts to take place, and the process takes on a momentum of its own.

On the other hand, behavioral or biological patterns can be “diluted” if the individual is fortunate enough to encounter situations and people which inhibit or reduce high-risk behavior. This is the theoretical basis for suggestions that problem drug involvement can be substantially reduced in a population simply by delaying the onset of drug use. In so doing, the initial use pattern never becomes part of the habit set. By moving the age of first use into a life period where external circumstances do not encourage alcohol or other drug involvement, the likelihood that the individual’s actions will become habits is reduced.

The opinion that fate is inevitable, and that causes are predetermined is imposed on facts after an individual has become troubled, alcoholic, or drug dependent. But a developmental perspective invites us to appreciate how risks combine with each other. It requires that we understand how other people and circumstances aid or inhibit what takes place over time. Evidence suggests that, even after adolescence, the patterns of risk for problem alcohol and other drug use exist to a greater degree for some individuals than others, and vice versa. Therefore it may be just as likely, or more so, that the risks are reduced through life experience rather than that they may be increased, and that fate will pursue its unchangeable course. This leads us to be optimistic about prevention and early intervention as the knowledge base from research continues to expand.^{8,9}

The Continuum of Severity

In addition to the specifics of *who* is using *what* drugs, *when*, *where*, and *how*, we also have to think of these problems in terms of how severe they are; that is, how much do they impact the abuser's life and the lives of those around him? When we think of "severity" this way, we conceive of it as a continuum, ranging from no trouble, to a great deal of trouble. Within this framework, the commonly used terms "alcoholic" or "addict" are simply shorthand labels for the observation that an individual is far to the severe end of the continuum.

When we classify difficulties this way, we can think more easily about **causes** as a process where multiple factors of risk add together. If one has a lot of risk factors, then one is more likely to have problems of greater seriousness. If one has only a small amount of trouble, it is likely that the amount of risk which preceded the symptoms will also have been less. Within this framework, we can see that risk is a fluid characteristic that may even change at different times during the life cycle, as circumstances change. Also, it is important to remember that riskiness as curiosity is not always dangerous; at some times it may actually be helpful. Thus, to get into some trouble with drunkenness as a teen-ager may not be all bad, *if* one discovers the destructiveness of that experience and is careful about drinking in the future. On the other hand, one who gets severely intoxicated repetitively, and ignores this, may be in major trouble even though young.

Prevention programs for high-risk populations are usually targeted at groups that are not yet in obvious trouble, but have a high likelihood of developing problems. The assumption in this kind of work is that we are able to understand the causes even before problems develop, and if we understand them, we can control them.

Typologies and Understanding the Causes of Chronic Difficulty

Even though we may hope to limit or prevent problem outcomes, there will probably always be some people who are unfortunate enough to have moved up the ladder all the way from risk into major and long-term trouble. It is this latter group of people, with a chronic disorder, who are most often found in treatment agencies, prisons, and inpatient facilities. These individuals have moved beyond just "riskiness," and appear to have long-standing difficulties. Yet even here, all people with alcohol and other drug problems are not the same in background, consequences, and prognosis.

Typologies are systems for classifying these differences. They have been created with the

expectation that we will be able to better describe subgroups of individuals with common characteristics and similar needs. No typological system yet constructed is totally comprehensive for all alcohol or other drug problems, and a given system is usually relevant only to a subset of individuals. For example, we do not expect a model which explains chronic alcoholism to be useful in explaining how one got drunk the first time. Similarly, a model which explains the “causes” of heroin dependence may not be helpful in explaining how one initiates use of the drug. Nonetheless, within bounds, typological models can be employed as a kind of shorthand that keys us to certain symptoms, and that suggests the kind of assessment needed for a comprehensive picture of an individual’s functioning. We will now discuss the typological distinctions that have emerged as important subclassifications.

Severe Alcohol Problems With and Without Antisocial Behavior

One distinction for severe alcohol problems that has proven useful in describing differences in background and response to treatment is that between severe alcohol problems with and without antisocial behavior. Table 6.1 describes a number of the differences observed for these two types (and there may be others) that clearly have relevance for the kind of intervention one would carry out.

Table 6.1 Differences Between Antisocial and Nonantisocial Alcoholics¹⁰

CHARACTERISTIC	ANTISOCIAL ALCOHOLICS	NONANTISOCIAL ALCOHOLICS
Severity of alcohol problems	Greater	Lesser
Onset	Adolescence	Adulthood
Family history of alcoholism	Greater	Lesser
Other drug involvement	More likely	Less likely
Marital violence	More likely	Less likely
Marital history	More likely divorced or headed in that direction	More likely still married
Paternal antisocial history	More likely	Less likely
Occupational functioning	Poorer	Better

Interestingly, this same distinction has also proven useful in categorizing persons with drug involvement other than alcohol. A difference in heritability for these two subtypes has not been established yet for drugs other than alcohol, but the typology is quite similar in other ways.

Let us consider the value of classification further. Does the fact that antisocial alcoholism shows greater evidence of family history mean that antisocial alcoholism is simply inherited? By no means; the answer is far more complex. We need to consider the contextual framework within which this problem evolves. People with these characteristics are also noted to have grown up in

families where many more alcoholics were around them. Thus, they had greater opportunity to observe others using alcohol as a way of coping with life's problems. They also had greater opportunity to observe adults using aggressive means to resolve conflict, and probably had early contact with peers who came from similar backgrounds. They would have been more likely to be introduced to the use of alcohol earlier than would other children of their age group. Remember also that to become alcoholic, one also must grow up in a society where alcohol is available. So genetic variation is probably only part of the story, and a biopsychosocial explanation means that these different kinds of causes combine with each other as the individual grows and develops.

Severe Alcohol Problems, Other Drug Involvement, and Co-Morbidity

We have noted earlier that alcohol and other drug involvement does not occur alone, but frequently shows itself in the presence of other kinds of emotional and behavioral difficulty. Speaking in the language of medical diagnosis, the presence of co-existing problems is called **co-morbidity** (see also Chapter 2). What the term means is that other disorders occur along with the alcohol and other drug problems. When such co-morbidity occurs, the nature of the problem that the individual and intervention staff have to deal with becomes different. To use the example just described, alcoholism with antisocial symptomatology is very different than alcoholism without the antisocial "co-morbidity." Similarly, drug abuse with depressive disorder is a different clinical picture than drug abuse by itself. In fact, an issue of active debate in the field concerns how many different alcoholisms there may be. The answer is currently far from clear, but the important point to remember is that there is diversity of process operating here, such that not all individuals will show the same pattern of difficulties, *and not all individuals can be expected to have the same outcomes.*

Greater detail about co-morbidity is beyond the scope of this text, but it is important to know that the co-morbidities most often found for those with both alcohol and other drug abuse/dependence are those of antisocial personality disorder, depression, mania, and anxiety.^{11,12} It is also important to emphasize that co-morbidity is often a *result* of severe substance abuse. This is especially true of depression.

Some of this co-occurrence may be a direct physiological result of having taken alcohol or other drugs too heavily, for too long. Or, the depression may be a biochemical result of chemical abuse. Other existing problems are best explained in life terms. When someone is severely involved in alcohol or other drug abuse for long periods of time, they are likely to be less able to cope with life difficulties, more involved in conflict with others, and generally do a poorer job of dealing with life's stresses. Under these circumstances, it is not surprising that they get depressed—as would anyone who was doing so poorly at the job of living.

The last point, and perhaps the most important from the standpoint of our interest in causal models, is that many co-morbidities exist with alcohol and other drug problems. Therefore, it may be inferred that alcohol and other drug problems rarely occur by themselves. For each individual, the problems are embedded in a different set of life tasks and troubles. Co-morbidity is an example of these differences, but also a clue to appropriate management of the problem, which must take *all* issues into consideration. It is generally agreed now that we must confront and deal concurrently with the alcohol, other drugs, and other problems.

Alcoholism As a Genetic Disorder

It is not uncommon in this era to read or hear it said that “alcoholism is a genetic disorder.” Unfortunately, a whole set of other implications are frequently assumed as part of this message. One implication is that, if the disorder is genetic, it means that there are not other learned, or culturally produced aspects of the disorder. A second frequent assumption is that the primary influences—or most important causes—are *biological* in nature, and under genetic control. Third, it implies that if we can discover which individuals carry the “alcoholic gene,” we can warn them about risk, and that all others, who do not have it, are free of risk. Fourth, it implies that the primary intervention will be biochemical, either by way of a drug, or perhaps, in some distant time, by way of genetic engineering.

All of these implications are incorrect, and potentially dangerous. No genetics researcher would subscribe to them today. As we have already noted in both Chapter 5 and in the section on typologies above, the evidence for specific genetic effects linked to *some forms* of alcoholism appears now to be solid, but it *does not exist for all forms*. In fact, those forms that have the strongest genetic ties are actually the less common subtypes found in the population. And it must be noted that some people who have strong genetic predisposition may never develop problems since they may never be exposed to the circumstances or to life problems that are necessary to move from genetic disposition (**genotype**) to the full-blown expression of a problem (**phenotype**).

Finally, it needs to be stressed that the state of knowledge at the moment is very incomplete, and the extent to which “genetic vulnerability” is specific to alcoholism, rather than to other co-morbidity, is not yet known. So we need to be careful, in the degree to which we attempt to use “single-cause” explanations to account for this complex set of problems.

Implications of Current Models of Etiology for Assessment and Intervention

Assessment needs to be an integral part of any well-conceived intervention for prevention or treatment. In many ways, assessment can be thought of as the *first stage* of the intervention, rather than a separate process that is disconnected from whatever intervention takes place later. Thus, the information we receive in a carefully designed assessment is likely to inform us about the alcohol and other drug disorder we are dealing with, as well as suggest details about the kind of intervention we should conduct. It is also likely to provide information that will be useful in gauging how long the intervention effort may take. Perhaps most importantly, it will help to establish appropriate outcome objectives and highlight the signs we need to look for to judge that the intervention is truly working.

There are three major implications to be gathered from this discussion of etiology:

- (1) If the level of risk is important in determining the nature and severity of a problem (how difficult it may be to manage or resolve), then *assessment* of risk factors is important in any comprehensive evaluation of alcohol and other drug problems. In the case of treatment, it would be important to collect information about an individual’s earlier functioning. For example, learning how well they have done with their education and job, the extent to which their family has experienced alcohol and other drug problems, and the kinds of other health problems (co-morbidity) they have had. We would want to assess the influences that could be supportive of positive change. Do social relationships exist or not? To what extent is the individual’s social network involved with alcohol and other drug use?
- (2) The distinction between antisocial and non-antisocial alcoholism has major implications in terms of

how severe the alcoholic problems are likely to be for men, and how difficult they are to treat. There appears to be similar relationships for women, with and without depression. On these grounds, assessments that inquire about the degree to which there is antisocial or depressive behavior along with the alcoholism should be more useful than assessments that only evaluate the alcohol problem.

- (3) From a long series of studies done at the Philadelphia VA Medical Center and NIDA Treatment Research Center,¹³ it is apparent that the antisocial distinction is an especially important one when it comes to the treatment of men with drug dependence. The kind of treatment that is likely to be effective for one versus the other subtype appears to be quite different. Those without an antisocial history are more likely to benefit from treatment that focuses both on their drug involvement *and* helps enhance social relationships within the family or on the job.

Persons with an antisocial history, on the other hand, appear to respond more to behavioral treatment alone, with a heavy focus on their drug-related difficulties. Related to this point, these two subtypes appear to have a different history of recovery. Non-antisocial persons with drug involvement seem to have faster recoveries, perhaps because their problems have tended to start later. Drug dependence with antisociality appears to require much more sustained work for recovery.

Given the earlier onset of alcohol and other drug problems that occur with antisocial behavior, and the greater difficulty in treating them once they are established, it is likely that early identification and prevention activities are especially important for this subtype.¹⁴ In the years to come, it is likely that prevention professionals and clinicians will focus increasingly on early assessment and intervention. If we can start earlier, we may very well discover that those with problems are rapidly responsive to properly focused intervention efforts.

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