
The Disease Model of Alcoholism Revisited:

Why People Drink

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Introduction

The view of alcoholism currently boasting wide acceptance is the disease model approach (Jellinek, 1960:8). This approach views alcohol as a disease of the individual. Juxtaposed with a view of alcoholism as moral degeneration or a personal weakness or failure, the disease perspective is a most desirable approach in that it provides both an impetus for treatment and sympathy of the alcoholic, and it removes or at least minimizes the guilt and reluctance to seek treatment which the alcoholic experiences.

Viewing alcoholism as a disease allows a professional group, namely the medical profession, to claim responsibility for its understanding and treatment and therefore affords the problem more exposure and respect (Jellinek, 1960:2). By labeling alcoholism a disease, it is put on par with other diseases and medical problems, thereby removing the stigma associated with the problem. We do not look down on people because they have diseases; in fact, we try to assist them in overcoming their problem by offering them such things as time off work, Medicare funding, and government or corporate funded rehabilitation programs. In the same way, defining alcoholism as a disease, should afford alcoholics this same degree of understanding, respect and sympathy. By giving the responsibility of research and study into alcoholism to the medical profession we effectively put the problem of alcohol addiction in a more favourable light than if it was considered the responsibility of psychologists, social workers, or clergypersons. A corollary of this observation is the fact that it is the medical profession which is expending the most effort and energy in supporting the disease model as the dominant way in viewing alcoholism (Milam & Ketcham, 1985).

Jellinek himself acknowledges the appropriation of alcoholism by the medical profession when he states that "a disease is what the medical profession recognizes as such" (1960:12). However, seeing alcoholism as a disease is not necessarily the only way to look at the problem; if the disease model were to lose its basis of consensus, many in the medical profession would be out of a job (Schneider, 1978). It can be argued that alcoholism is seen as a disease because it is profitable for the medical profession to see it as a disease.

Although the medical community has a vested interest in the disease perspective of alcoholism, this would be no reason to disregard the disease model if indeed the disease perspective is the best paradigm available. In this paper, a number of issues will be discussed. Initially a critique of the disease perspective will be offered. A more detailed and operational definition of alcoholism will be proposed and a new paradigm that incorporates the details and features of alcoholism ignored by the disease model will be suggested. This is an ambitious undertaking to be sure, but a necessary one.

A Critical Look at the Disease Model

In spite of its benefits, the disease model perspective is unable to adequately account for alcoholism and is itself beginning to fall victim to the same criticisms that

it levels at the moral degeneration perspective. In particular, our cultural milieu is increasingly coming to emphasize health, nutrition, and fitness as important life-style values. In such a milieu, disease is coming to be viewed as a personal failure in adequately caring for oneself. In this way, alcoholism, viewed as a disease, is becoming increasingly defined as a personal failure that creates guilt and a denial of a problem just as surely as the moral degeneration perspective does—a theory that the disease perspective was intended to counter.

More importantly, the disease model suffers from a number of logical and theoretical flaws that not only deny its validity as a scientific model, but also limit its practicality of researching, identifying, or treating alcoholism.

Jellinek, in his model, attempts to define alcoholism in such a way as to “establish its objective reality” (1960:34). In the process, he commits the error of “reification”; he defines alcoholism into reality instead of limiting it to serve as only a term. Reification of the term means that alcoholism, while originally a label referring to some objectively defined phenomena, has become an actual condition without the proof which is derived from empirical observation (Rohan, 1976:63). In effect, Jellinek has defined alcoholism into existence and alcoholism becomes a disease simply because it is defined as a disease. There appears to be no other reason for viewing it as pathology.

Disease model proponents are at a loss to explain why alcoholism is a disease except that they maintain there are a number of biological and genetic predisposing factors serving to “mark” a portion of the population as alcoholic. Arguably, this is analogous to defining that a portion of the population will succumb to the disease of being air traffic controllers, or doctors, or miners. Because “there are certain professions and occupations which by their nature carry a higher degree of risk (of alcoholism) than the population in general” (Keyton, 1973:32), there is no reason not to conclude that similar biological or genetic factors which constitute the disease marking the person an alcoholic should also constitute the disease which marks these people for such high risk careers.

This model proceeds to claim that persons so “marked” are alcoholics even before they have their first drink and therefore, as soon as they do consume their first alcoholic beverage begin the journey down the path of alcoholism and are necessarily in need of redemption at some later point (Milam and Ketcham, 1985:34). A teleological error committed by proponents of the disease model is that they look at the effects of alcohol as proof of their theory. One of these effects is believed to be a loss of control over drinking behaviour (Jellinek, 1960:41). According the disease theorists, such loss of control is evidence of the physiological “need” for alcohol and therefore alcoholism is the product of a physiological craving for alcohol. Drinking behaviour is therefore not under volitional control and can be considered a disease, or a physiological and therefore medical problem.

However, new research is conjuring up strong opposition to this premise. In experiments manipulating the cognitions and expectations of alcoholics concerning their alcohol consumption, it has been found that the loss of control phenomenon cannot be attributed to any physiological reaction or adaptation to alcohol consumption but rather, is related to the expectations of what the person is drinking. If they are consuming alcohol but believe they are drinking a non-alcoholic beverage, the loss of control which is predicted according to the disease model is not exhibited (Marlatt, Demming and Reid, 1973).

While cross-cultural studies and twin-studies do suggest the presence of genetic factors in alcohol addiction (Goodwin, 1973; Bales, 1949; Petrakis, 1985), the claim to the existence of a “marked” population cannot be made until it can be sufficiently

demonstrated that these factors alone are not only necessary but sufficient explanations to account for the development of alcohol addiction in an individual. Just as the presence of Multiple Sclerosis in a family of origin and the subsequent genetic inheritance of a disposition to contract the disease is not a sufficient explanation to account for Multiple Sclerosis in a person, there is no proof that a genetic inheritance predisposing an individual to alcoholism is a sufficient explanation to account for "contracting" alcoholism. Rather, these predisposing factors may interact with personality, environmental, cultural, social learning, health and other factors to determine the probability of alcohol addiction, just as these other factors interact to account for the contraction of MS or other diseases. Until such time as proponents of the disease model can define what are these biological and genetic predisposing factors, propose a methodology for determining and measuring their existence, and lend tenability to the proposition that these factors constitute not only a necessary explanation, but also a sufficient explanation of alcoholism, the disease label which we have attached to alcoholism must not be considered a scientific label.

We must choose the label we use to describe alcoholism with care. The perspective from which we describe the problem will also determine which facts and components we look at and study in our attempt to better understand and explain the phenomenon:

Assuming an internal controlling condition is a preconception that interferes with the formation of an appropriate conceptual scheme. It prevents us from viewing alcohol misuse as a problem of action rather than condition, of doing rather than having, of agent rather than victim, as verb rather than noun. Such an understanding is important since our present conceptual scheme prevents us from focusing on the relevant variables in the control and modification of drinking. Our conceptual schemes influence what we select to observe, how we perceive what we select, and most importantly, how we respond. If we continue to see destructive drinking as a sign of an internal aberration, we will not only fail to assess drinking behavior and its characteristics, but we will not appreciate the relevance of external variables in the performance of drinking behavior (Rohan, 1976:64).

Additionally, alcoholism unlike genuine diseases exhibits symptoms specific to particular countries, cultures and economic classes. Whereas a particular type of cancer for instance affects the same organ in more or less the same way in all its victims, alcoholism manifests itself very differently in different cultures (Rohan, 1976). Jellinek himself admits this problem but dismisses it as unimportant (1960:13). Another criticism of disease theory is that it does not work. If penicillin was only 50% effective in controlling bacterial infections and if even in this 50% group, the infection was not cured, but only controlled or arrested, we would be reluctant to describe penicillin as a good antibiotic. In the same fashion, treatment methods using the disease perspective, such as AA exhibit a relapse rate of approximately 90% among those abstinent for one year or less and only around 40% for those abstinent for more than five years. In total, less than half the people who come to AA become "sober alcoholics" (Clinebell, 1978:144; Weisman and Robe, 1983). Surely, such a dismal figure demonstrates the need to spend some energy focusing on the other factors that contribute to the overall problem of alcoholism.

Finally, the disease theorist explains away the fact that some people who are abusive drinkers can be taught to drink in control. It argues that they are not alcoholic but only heavy drinkers. The theory thus differentiates between problem drinkers and alcoholics. Such a differentiation allows a convenient loophole for those alcoholics who wish to define themselves as merely problem drinkers (Milam and Ketcham, 1985:40). In this way, they can attempt to avoid treatment or counseling. Such a loophole does the theory no service at all. It only serves to limit the applicability of the

disease perspective in excluding definite cases of alcohol abuse by labelling these cases as non-alcoholic abuse. I suggest that if alcoholism is “any use of alcoholic beverages that causes any damage to the individual or society or both” (Jellinek, 1960:35), then for the disease model to exclude such cases of alcohol abuse demonstrates a strong limitation in the model.

We can see that there are serious limitations to the disease perspective yet this model is not without its own merit. In trying to develop a new perspective, we must be careful to try and incorporate the best of the old models. In particular, the disease perspective has an advantage in that it remove the blame from the alcoholic and puts it on other factors. This effectively reduces the problem of guilt and embarrassment which compounding the problems of alcoholism. The focus that the disease perspective puts on physiology should not be disregarded, yet such a single focus is perhaps less than fair to the other factors contributing to the overall problem of alcoholism. In searching for a better paradigm to explain alcoholism, we must attempt to be fair to these other factors.

Definitions

The starting point for a new look at the problem of why people drink is perhaps the reworking of our definitions. Defining alcoholism has always been a bit of an enigmatic problem. As Rohan points out, “one source of [this] conceptual ambiguity [is] in the use of ‘big fat words’” (1976:63). We must be careful to use the term “alcoholism” as a descriptor of what we mean when we see people's lives affected by alcohol and we must be careful not to allow the term to take on a life of its own. In order to be pragmatic, such a definition requires a high degree of preciseness and an emphasis on being empirically quantified. Definitions referring to some immeasurable internal state do us no good; neither do definitions that must be subjectively evaluated.

The definition proposed for this discussion satisfies these concerns. Alcoholism is defined as *a physical addiction or psychological dependence/habituation on ethanol that results in a prolonged and noticeable reduction in the person's ability to function psychologically, socially, or occupationally, or otherwise negatively affects the areas of the individual's family, health or financial security.*

This definition has a number of important components. Firstly, it does not matter whether the need for alcohol is physical or only psychological. The effect is the same: the person has a persistent or regular need for whatever alcohol provides for them. To argue that a person is not an alcoholic because their need is only psychological or they are only a problem drinker becomes a mute point.

Secondly, the effects of such addiction or dependence must be a result of ethanol consumption; they cannot simply coincide with ethanol consumption. For example, a person who has experienced profound personal trauma may experience a reduced level of personal functioning and may decide to increase their ethanol consumption. Such a scenario would rule out any assessment of alcoholism. If it can be determined that there were no profound personal stressors previous to the high level of ethanol consumption but that personal stressors were evident subsequent or as a result of ethanol consumption, then such a condition for the assessment of alcoholism has been determined. It is important to realize that some stressors (such as a driving while impaired offense) can be causally linked to drinking behaviour without too much difficulty, while other behaviour (such as marital strife or violence) have a linkage which is not as clear or may be only suspected. Where such behaviour cannot be causally linked to alcohol consumption, it should not be used as the sole incident in deciding an assessment of alcoholism.

Thirdly, the effect of such addiction or dependence must be prolonged. Weekend benders do not count, nor do occasional heavy drinking bouts during times of either celebration or personal distress. While such behaviour may not be particularly adaptive or beneficial, it is not classified as alcoholism. In the same way, a person prescribed Valium as a temporary measure to help them through a death in the family, or a marriage breakup, is not considered an addict to Valium.

Fourthly, the reduced ability to function in the many different areas of a person's life as a direct result of ethanol consumption is indicative of alcoholism. We do not go through the Twelve Steps with the person who drinks six cups of coffee before leaving for work in the morning, nor do we join some version of Al-Anon if we have a spouse who is a chain smoker. Similarly, people addicted to alcohol but who do not show any "prolonged and noticeable reduction in the person's ability to function" may have an addiction to ethanol, but are not considered alcoholics. People have a tacit right in our society to participate in any vice they wish so long as (a) they do not injure any other person or cause a problem for society as a whole and (b) the activity is not illegal. Related to the current discussion, this means that people have a right to consume alcohol if they wish so long as it does not impair their personal functioning or that of another person, or negatively affect their lives.

This emphasis on a noticeable reduction in functioning is what gives the definition an objective criteria and an evaluative base for assessing a person as an alcoholic. This base is important not only for statistical purposes but also for carrying out a personal inventory with the suspected alcoholic with the intent of having them accept the definition for themselves. If it can be shown to them that they are experiencing the detrimental effects of alcohol, then it should be easier for them to accept an assessment of alcoholism.

Finally, a reduction in the person's ability to function is not necessarily exhibited in all avenues of a person's life. A noticeable reduction in only a few areas, or even a single area is sufficient for an assessment of alcoholism.

The use of the term "assessment" is also very important. The medical model would prefer the term "diagnosis." Assessment is the preferred term because that is exactly what it is: a value-free, and evaluative decision regarding the problem in a person's life, which in this case is alcoholism. We diagnose diseases, and we assess problems. Alcoholism is not a disease; it is a problem.

Alcoholics, or more appropriately people suffering from alcoholism, are like the rest of us; they suffer from problems. Their problems may be different from ours but the differences are often only cosmetic and not necessarily qualitative. Their problems may include a different genetic makeup, a particular economic or family background, historical or biographical factors, different attitudes, and different demands from their peer groups, financial difficulties, or unemployment. They differ from us only in that their attempt to find a solution to their problems led them to alcoholism and in the process, they added one more problem to their list: alcoholism.

Whatever brought these people to become alcoholics, they are not inferior to those of us who are not plagued by alcoholism. We have our own vices and solutions for dealing with our problems, some good, some bad, but by assessing a person as an alcoholic, and more importantly helping them to assess themselves as an alcoholic, we begin the first step in helping the person achieve a viable and lasting solution to some of their problems.

It might be concluded based on the preceding argument, that what is being suggested is a cognitive approach to alcoholism. Certainly, a cognitive approach is one valuable perspective to the problem. But it is also recognized that alcoholism

comes in many different colours; therefore medical knowledge, classical conditioning paradigms (Siegel and Hinson, 1983; Kesner and Cook, 1983), Transactional Analysis (Berne, 1972), and other approaches all have their places in the overall program to study and assist alcoholism and the alcoholic.

The theoretical model being suggested is one which attempts to address the problem of alcoholism in an inclusive approach rather than the exclusive one of the disease perspective.

In conclusion, we wish to propose a theory which can explain the full range of alcohol addiction, even if such an explanation currently lacks specificity due to insufficient research, rather than proposing a specific but myopic approach. In this way, one of the purposes of scientific theories is satisfied, namely, to give a direction to further empirical research. As well, the theory remains open-ended as a way of permitting the inclusion both of further research as it becomes available and other treatment variables as they become recognized (Chafetz, 1978). This stands in stark contrast with the limitations of the disease model discussed above.

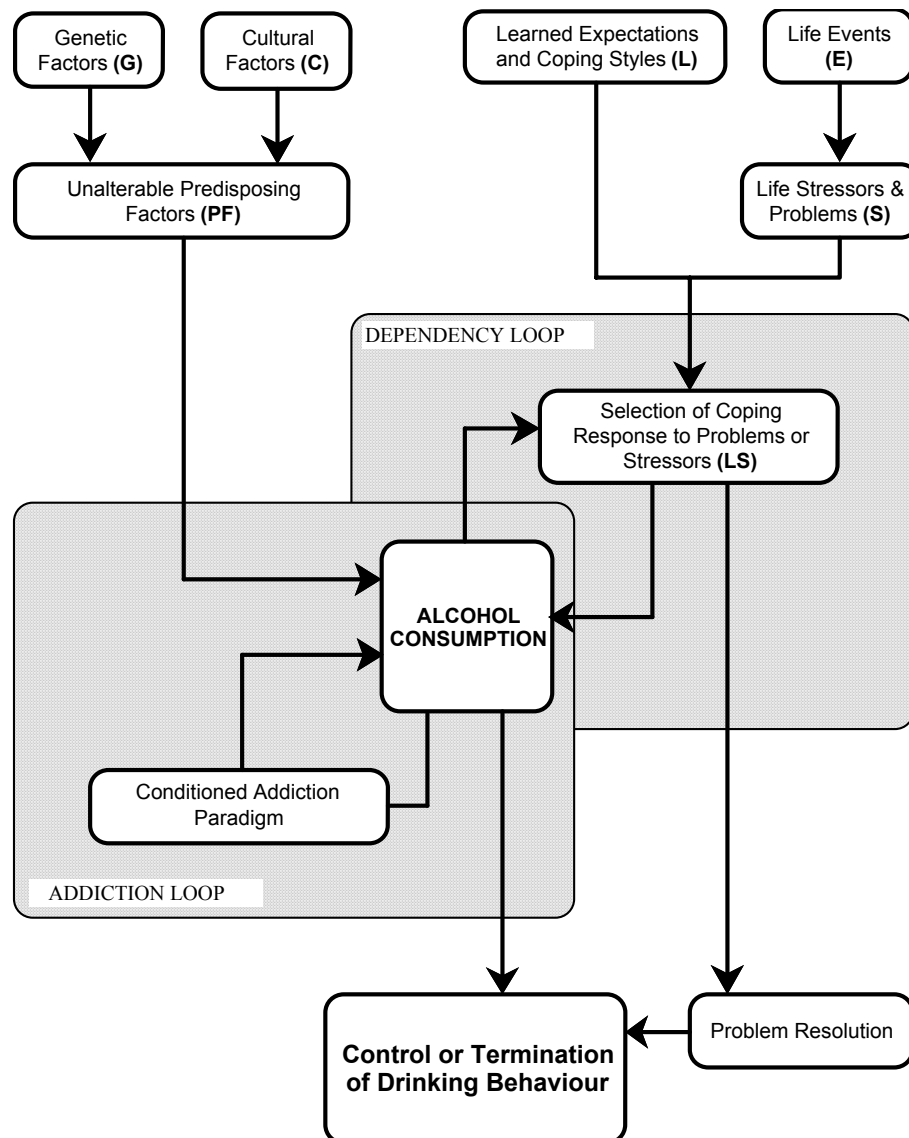
A New Theoretical Perspective

The schematic of this new and more inclusive theory is found in the accompanying diagram. It is a model that tries to explain the etymology of alcoholism using the current research available and suggesting hypotheses for further research. Like all models, this one will inevitably be modified over time, and perhaps ultimately discarded. Its main advantage is that it serves as a useful and effective tool to drive both research and treatment of the problem at hand: alcoholism.

The existence of *Genetic Factors* (abbreviated as the **G-factor**), which helps account for alcoholism rates, has long been recognized. Petrakis (1985), in his excellent survey of the research into the genetic factors contributing to alcoholism, makes this point obvious. Yet, genetic markers are not the sole determinant of whether a person will become an alcoholic. Petrakis points out that there are two types of genetic predispositions to alcoholism: milieu-limited and male-limited (1985:8). While the male-limited variety does seem to be unaffected by the post-natal environment, it accounts for less than 25% of all male alcoholics and around 4% of all general population males. This effectively accounts for less than 2% of the population. Milieu-limited alcoholism "is called milieu-limited because its occurrence and severity in susceptible offspring are influenced by the postnatal environment" (1985:8). Predispositions to alcoholism in females are attributed to this milieu-limited model. Unlike the heavy drinking male-limited alcoholic, milieu-limited alcoholism is characterized by mild to moderate alcohol abuse and as such, may not even be assessed as alcoholism by experts.

The fact that the postnatal environment affects the occurrence of this second type of alcoholism suggests that genetic markers alone are not sufficient predictors of alcoholism occurrence in an individual. For this reason, *Cultural Factors* (or **C-factor**) and learned expectations are also important. This importance is perhaps most salient in discussing the difference between Jellinek's concepts of gamma and delta alcoholism (1960:38). Gamma alcoholism is found in North America and is characterized, among other things, by a loss of control but not necessarily an inability to abstain. Delta alcoholism common to Europe is characterized by an inability to abstain but seldom involves a loss of personal control over one's drinking. The cultural influences affecting drinking behaviour and thus alcoholism rates can be found not only in cross-cultural studies, but also in intra-cultural research (Gusfield, 1962; Lawrence and Maxwell, 1962; Bales, 1962). It is proposed that these cultural

factors mediate the rate of alcoholism in three ways: first, they determine the level of acceptable exposure to alcohol and therefore the quantity, frequency and regularity of alcohol consumption. Second, cultural factors affect the general attitudes which members of the culture, or social class have towards alcohol consumption. In this way, the relative approval or disapproval of alcohol use is culturally determined. In the same way, the culture has much to do in influencing individuals to look upon alcohol consumption in a positive or negative light. Finally, cultural factors as more broadly represented in the way political and economic forces determine the price of alcohol and thus, influence alcohol consumption (Popham and Schmidt, 1962:26).



Both these factors, genetic markers and cultural influences, serve as *Unalterable Predisposing Factors* (called the **PF-factor**) in the development of alcoholism. These predisposing factors are important elements in determining the potential of the person to become an alcoholic. In this way they may well be necessary elements in the development of alcoholism in an individual, and while it is possible that they may be sufficient factors in some cases, such as male-limited alcoholism, they are usually only contributing factors in the development of alcoholism. They are called "Unalterable" because they are given factors and are not easily altered by therapeutic interference or preventative counseling. It is possible that some people

may become alcoholic simply because of their G-factors, C-factors or combined PF-factors, but the experiences of many alcoholics cannot be accounted for simply by reference to PF-factors. Other factors need to be investigated and accounted for in the overall attempt to explain alcoholism.

Learned Expectations and Coping Styles (or **L-factor**) are similar to cultural factors except that they refer to the specific values and expectations found in the family of origin and the individual's peer group and can therefore be quite distinct from the cultural values of the society as a whole (Boothroyd, 1980). These expectations affect drinking behaviour in a number of different ways. The perceived effects of alcohol consumption and whether these effects are seen in a positive or negative light have been found to display a strong correlation with eventual alcoholism.

Presumably, these attitudes to alcohol are acquired from the family of origin and play a major part in determining how the individual will come to evaluate alcohol consumption and whether or not the person will use or abuse alcohol or abstain altogether (Christiansen and Brown, 1985; Christiansen, Goldman and Brown, 1985; Christiansen and Goldman, 1983). It is argued that if people come to see alcohol in a positive light and feel that alcohol improves social behaviour, increases arousal, and decreases tension, they will be more likely to use alcohol in an attempt to achieve these ends (Christiansen, Goldman and Brown, 1985). Similarly, they will be more likely to turn to alcohol to enhance recreational enjoyment as well as coping with life stress and anxiety and, it is hypothesized, they are more likely to abuse alcohol and become alcoholics.

Learned expectations concerning alcohol also affect alcohol consumption directly by teaching the individual when it is appropriate to consume alcohol. Furthermore, these expectations affect alcohol consumption indirectly by providing the individual with a set of coping or problem solving strategies to use in resolving *Life Stressors and Problems* (or the **S-factor**). The interaction of these life stressors and the person's coping abilities leads to a *Selection of Coping Response to Problems or Stressors*, called the **LS-factor**, and plays an important role in whether the person will drink, and why. If this repertoire of strategies is too narrow or lacks effectiveness, or if the repertoire suggests an acceptability in using drugs or alcohol in order to relieve tension or stress, then it is more probable that the individual will turn to drug and alcohol use to cope with life stress and anxiety and perhaps become an alcoholic. For example, a young man whose father, whenever under life stress came home to beat the mother, has just recently moved in with a new girlfriend himself. Having learned a coping strategy from his own father, namely wife beating, as a way of dealing with stress, he comes to respond in kind. To alleviate the guilt that emerges from this ineffective strategy, he may well turn to alcohol. In this way, alcoholism becomes a secondary product of the learned responses acquired from the family of origin.

From the schematic, we can see that individuals consume alcohol for a combination of two reasons: as part of the coping strategy in response to life stressors, and for purely social, cultural and hence recreational purposes. The interaction of these two components, the PF-factor and the LS-factor, will determine if the person's drinking is explained as a response either to predisposing factors, or life stressors, or some combination of the two. It is more able to account for reasons why a person drinks, and is better suited to looking for the causes, and consequently the remedies of drinking.

This model also accounts for why some people can voluntarily control their own excessive drinking without external assistance. If their drinking is in response to life stressors and they have not developed into an addiction or dependency, they have a great deal of control over their decision to drink. If the life stressor contributing to the

problem is resolved, the incentive to drink is lessened. As well, the person may decide on their own to control or terminate their drinking behaviour.

The person becomes an alcoholic only when they become physically addicted, psychologically dependent or both addicted and dependent to alcohol. The *Dependency Loop* demonstrates how alcohol use can become its own problem. By turning to alcohol consumption as a problem solving strategy, the reasons for the stress are not dealt with and will usually build up thus generating even more stress and psychic tension, which must be relieved, once again by turning to alcohol. In addition, behaviour while intoxicated, such as assault, breaking furniture or drunken driving can add its own problems to the total S-factor. If alcohol consumption is personally viewed in a positive light and if it becomes a prime coping strategy, then there is a high probability that the individual will become psychologically dependent on alcohol.

It must be cautioned that psychological dependency infers much more than a lack of "will-power". A person is psychologically dependent not because they lack the personal discipline to control their drinking, but because the psychological "payoffs" in drinking far outweigh the "debts". With such a high degree of payoffs to drinking, will power has very little to do with a person's dependency on alcohol. Of course, there may come a time when the debts outweigh the payoffs. At this point, the person may either stop drinking if they have not become addicted to alcohol, or else continue to drink in order to avoid the physical debts of addiction which will ensue should consumption be stopped.

The *Addiction Loop* represents both a pharmacological response and a classically (or Pavlovian) conditioned response to alcohol consumption. More research needs to be done before the actual nature of physical addiction is understood and as this research becomes available, it is probable that the exposition of the Addiction Loop may be significantly modified. Current research is suggesting that what has traditionally been called pharmacological addiction is better understood by using a Pavlovian conditioning paradigm.

As Hinson and Siegel (1983), Kesner and Raymond (1983) have discovered in studying the effects of morphine addiction, regular and conscious drug usage instills an unconscious expectation in the individual for future drug usage. In anticipation of continued regular and persistent drug use, the body attempts to counteract the expected effects of the drug by enhancing the body's reaction to the drug's effects and in this way, rapidly returns the animal's physiology to a state of equilibrium. For example, if the animal has come to expect regular doses of morphine, a drug that is both a sedative and an anesthetic, the body compensates for the expected effects of morphine injections by increasing the animal's level of hyperactivity and sensitivity to pain. If by past experience, the drug comes to be expected on a regular and predictable schedule and the drug is not forthcoming, the physiology will still respond in anticipation of the drug and the animal will be overly hyperactive and sensitive. In effect, the body will be out of equilibrium because the expected drug was not forthcoming. Support for this view with particular reference to alcohol studies shows that if an alcoholic is given alcoholic beverages to consume but is told they are not receiving any alcohol, they do not exhibit a loss of control (Marlatt and Rohsenow, 1980).

Clearly, the imbalance or "debt" which we call addiction is psychological and conscious in origin although it does manifest both psychological and physical distress if the expected chemical is not forthcoming. This distress is called "withdrawal". Because there is a strong desire by the individual to return to the expected equilibrium, the person will develop a strong craving for the drug in order to return their physiology to a normal state or in other words, to avoid withdrawal. The role of

expectations regarding drinking behaviour explains why a conditioned addiction to alcohol is often present in the chronic alcoholic (Milam and Ketcham, 1985). It is easily seen that, contrary to the disease model understanding that sees alcoholism as a maladaptive and destructive response, an addiction approach sees the development of alcoholism as a natural and healthy bodily response to the persistent introduction of a noxious substance. Like acquiring a suntan in the continued presence of ultraviolet radiation, the body's ability to respond to the presence of alcohol is an adaptive rather than maladaptive one. If the body were unable to respond, the imbibitor would soon die, just as the sun-tanner would soon shrivel up with skin cancer. It is these two loops, the addiction loop and the dependency loop which separate alcoholism from the non-alcoholic drinker. It is recognized that an alcoholic can be either dependent or addicted to alcohol or even both dependent and addicted simultaneously. However, more often than not, a person progresses from a psychological dependency to a physical addiction. This switch from dependency to addiction accounts for the development or stages of alcoholism that the disease theorists describe (Milam & Ketcham).

Conclusion

According to a factor theory, concurrent with the other aspects of alcohol addiction counseling, the LS-factor can be reduced in order to minimize the chances of a return to alcoholism. It is also important to recognize that successful assistance for the alcoholic in resolving their problems often involves the teaching and practising of new life-style and coping strategies. This is of course what AA tries to do in many of their twelve steps (Maxwell, 1984).

Furthermore, this theory recognizes that some people who are alcoholics using the proposed definition may be able to return to social drinking (Mills, Sobell, and Schaefer, 1971; Schaefer, 1972; Marlatt and Rohsenow, 1980). While there is a contemporary controversy over whether the effects of a physical (or conditioned) addiction can ever be removed or extinguished, and therefore if a formerly addicted individual can ever socially drink again (Kalant and Hawkins, 1969:32), there is considerable evidence that social drinking is entirely possible for the person who was only dependent upon alcohol (Larkin, 1978:20). Indeed, should social drinking be possible, and the person is informed that social drinking is possible, there is a strong likelihood that the person will return to moderate drinking. An unanswered question to date concerns when abstinence should be encouraged, and when moderation is possible.

A factor theory of alcoholism goes a long way towards encompassing all the cases of alcohol abuse. It also provides a broader basis of contributing factors for alcoholic behaviour. In this way, the researcher has a new theoretical tool in the search for facts and relationships at work in alcoholism. At the same time, the alcoholic counselor has a broader understanding of the components making up alcoholism and can not only help the alcoholic come to a better understanding of their problem, but is better equipped to recognize the signs and complicating factors which hinder the person from resolving their alcoholism.

A factor theory also has the advantage of including more professionals under the umbrella. There is still an important role for medical professionals to play in the assessment and resolution of alcoholism, but this theory also invites the participation of other types of professionals (as well as the alcoholics themselves) in the resolution and prevention of alcoholism. Legislators have a responsibility to modify the cultural factors contributing to alcoholism. Teachers, social workers, industrial consultants and clergypersons have an opportunity to help modify social systems so they can be made more supportive and in this way, minimize the stress which people must cope with (Christiansen, Goldman, and Inn, 1982:343). In this way, a factor theory puts the

responsibility for alcoholism prevention and resolution squarely where it belongs: on the shoulders of all of us.

There are some limitations to a factors approach as well. Because it has a broader perspective, it lacks a high degree of specificity and while it tries to explain which relationships are important in the development of alcoholism, it cannot attach any sort of numerical preciseness to alcoholism. This is perhaps an unavoidable feature of any theory that tries to explain human behaviour. Nevertheless, I believe that a factors approach lends much to the understanding and treatment of alcoholism. It can explain everything that a disease perspective is able to, and yet it can account for much more. Time alone will be able to tell what such a model is worth.

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