Monograph No. 6

Perspectives on Drug and Alcohol Dependence

Edited by

Janet Greeley
and
Nick Heather

National Drug and Alcohol Research Centre
University of New South Wales

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Proceedings from a symposium held during the XXIV International Congress of Psychology
Sydney, August 30, 1988

Edited by

Janet Greeley, Ph.D.
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Perspectives on Drug and Alcohol Dependence

Preface

Perspectives on Drug and Alcohol Dependence is the proceedings from a symposium held at the International Congress of Psychology, Sydney (28 August - 3 September, 1988). The symposium was organised and sponsored by the National Drug and Alcohol Research Centre (NDARC). Researchers, clinicians and commentators from a variety of disciplines were invited to present their views on current influential concepts and models in the field of drug and alcohol dependence.

Dr Loris Chahl, a pharmacologist, presented a physiological model of opiate dependence based on changes observed in the enteric nervous system (i.e., innervation of the gastrointestinal tract) as a function of treatment with various opioid agents and neuro-peptides.

A philosophical view of the usefulness of models of dependence was put forward by Dr Les Drew, a psychiatrist and well-known spokesman in the area of drug and alcohol dependence in Australia. Dr Drew argued for a harm-reduction approach in which knowledge about the natural history of drinking and drug use would play a greater role than specific models of dependence.

Professor Nick Heather, clinical psychologist and Director of NDARC, presented a revised view of the phenomenon of "loss of control". Professor Heather argued that loss of control, or impaired control, the term he prefers, is a useful and important concept in the study of dependence. He outlined current views on the subject and described several ways in which impaired control might be researched.

The editors wish to thank Ms Wendy Swift and Mr Brin Swain for their expert assistance in proof-reading and preparing the layout for this monograph.

Dr Stanton Peele, a social psychologist by training and well-known author on the subject of addiction, discussed his views on the role of psychology in the study of addictive behaviour. He argued that psychological theorists in this field had fallen prey to the reductionist views of addiction "which spring from biological and medical conceptions" (p. 25). He also discussed the moral considerations inherent in the treatment of addictive behaviours.

Dr Robin Room, a sociologist and Director of the Alcohol Research Group at the Medical Research Institute of San Francisco, also spoke on "loss of control". He considered the notion of a double loss of control as reflecting a cultural perspective on self-control, not only of one's drinking but also of one's life. Dr Room presented data from a survey of two samples - a group of adults from a general population and a group of clients in treatment for alcohol problems. He discussed the culture-boundedness of concepts of self-control and called for cross-cultural studies to examine the relevance of loss of control in the study of dependence in different countries.

Professor Shepard Siegel, an experimental psychologist whose work has had a great impact on current research in the study of dependence, presented a model of drug tolerance and withdrawal based on principles of Pavlovian conditioning. Professor Siegel outlined evidence from research with both animals and humans which supported this influential model. He also briefly considered the implications for treatment which this model prescribes.

The editors hope that this monograph will provide you with some ideas which may challenge or perhaps confirm some of your own views on drug and alcohol dependence.

J.G. and N.H.

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The Enteric Nervous System as a Model of the Central Nervous System for Studies of Opiate Dependence

Loris A. Chahl
Neuropharmacology Laboratory
Faculty of Medicine
University of Newcastle
Australia

Dependence and Tolerance

For the pharmacologist the phenomenon of "addiction" comprises physical dependence, tolerance and craving leading to drug seeking behaviour. The phenomenon is expressed most fully in all its aspects by the opiate drugs.

Physical dependence has been defined as an abnormal physiological condition induced by chronic administration of a drug, that remains covert as long as continuous drug exposure is maintained, but that on drug withdrawal leads to a series of pathophysiological events known as the abstinence or withdrawal syndrome (Villarreal & Castro, 1979). Dependence itself is inaccessible to direct measurement and its presence may only be expressed by the degree of the abstinence response. Ways of measuring physical dependence indirectly include measurement of the degree of the abstinence response following either drug withdrawal or administration of a narcotic antagonist, or less commonly, determination of the amount of drug required to suppress abstinence. Tolerance, on the other hand, is defined as a gradual decrease in responsiveness to a drug, taking days or weeks to develop. In pharmacological terms this may be measured by a shift in the opiate dose-response line to the right.

For many years, it was considered that tolerance and dependence were phenomena that reflected a common adaptive change in neurones, an idea which was sometimes called the unitary hypothesis. Recently, however, it is becoming increasingly clear that tolerance and dependence are initiated by different biochemical mechanisms. Tolerance on chronic administration of an opiate is now considered to be caused by uncoupling of the opiate receptor from intracellular systems, whereas dependence is apparently due to compensatory changes within an opiate-inhibited neuronal pathway. Thus, dependence, unlike tolerance, is displayed only by intact nervous circuits such as occur in two regions of the body, the central nervous system (CNS) and the enteric nervous system.

The original work of the author was supported by the National Health and Medical Research Council of Australia. The important contributions of higher degree students, Paul Brent and Peter Johnston and the expert technical assistance of Ann Lynch and Cynthia Kavanagh are gratefully acknowledged.

The Enteric Nervous System

The enteric nervous system is the intrinsic innervation of the gastrointestinal tract and is the only division of the peripheral nervous system which approaches the CNS in complexity. The enteric nervous system is capable of controlling reflex activity independent of CNS control and resembles the CNS in diversity of neuronal types and multiplicity of neurotransmitters. For the study of opiates and opioid peptides, the enteric nervous system has long been considered to offer unique advantages since the actions of these drugs and peptides appear to reflect their actions in the CNS.

The intestinal tissue which has been most widely used for the study of opiate mechanisms is the guinea-pig ileum. Preparations of 1.5-2 cm of isolated ileum are suspended in organ baths filled with oxygenated physiological saline (Tyrode) solution at 37 degrees centigrade. Contractions of the longitudinal muscle may be measured by attaching one end of the preparation to a force transducer connected to a polygraph. The muscle will respond to drugs added to the organ bath or to neurotransmitters released from the enteric nerves within the walls of the ileum.

It has been known for many years that the guinea-pig ileum may be made "dependent" on opiates either *in vivo* by pretreating guinea-pigs with morphine, or *in vitro* by incubating the ileum with morphine for several hours. Addition of the opiate antagonist naloxone to dependent ileum produces a contracture of the muscle which may be measured. More recently, it has been observed that dependence of the ileum on opiates and opioid peptides occurs very rapidly. Following 2 min contact of the opioid peptide (Met 5) enkephalin with the ileum, washout or addition of naloxone produced a contracture (Chahl, 1983). Likewise, naloxone precipitated a contracture following 2 min contact of the ileum with morphine and several other opioids (Chahl, 1986). Opiates and opioid peptides are not the only agents capable of producing dependence and therefore withdrawal contractures of the ileum. The antihypertensive agent, clonidine, which acts on presynaptic alpha adrenoceptors, and which has been used to inhibit some aspects of the opiate withdrawal response, rapidly produces dependence of the ileum as shown by contractures following addition of the alpha adrenoceptor antagonist, phentolamine (Chahl, 1985). The natural transmitter, noradrenaline, which also acts on alpha adrenoceptors produces similar responses to clonidine (Chahl, 1985).

The withdrawal responses of the guinea-pig ileum to opiates, opioid peptides, clonidine and noradrenaline were all inhibited by substance P antagonists (Chahl, 1983, 1985, 1986). The tachykinin (neurokinin) substance P is a peptide neurotransmitter, present in nociceptive primary afferent neurones and also in intrinsic neurones of the enteric nervous system. It is present in many neuronal pathways in the CNS and is particularly highly concentrated in the substantia nigra. Since substance P antagonists inhibited withdrawal responses it was proposed that substance P might be the major mediator of withdrawal in the enteric nervous system (Chahl, 1983). The release of substance P on withdrawal was suggested to occur because opiates, opioids, clonidine and noradrenaline all inhibit release of acetylcholine from cholinergic neurones which might result in disinhibition of inhibitory interneurones which in turn activate substance P neurones. It was proposed that release of substance P is suppressed by an action of the opiates and other drugs to inhibit substance P release. Following removal of the inhibitory drug from its receptors either by washout or addition of the appropriate antagonist, the activated substance P neurones release substance P onto both the smooth muscle directly and the

cholinergic neurones to cause acetylcholine release, and contracture of the muscle occurs. Although this proposal will, no doubt, prove to be an oversimplification and probably incorrect in many details, it is nevertheless a working hypothesis which may be experimentally tested in future experiments.

The Central Nervous System

If substance P mediates the opiate withdrawal response in the enteric nervous system, it was considered important to investigate whether it also mediates the opiate withdrawal response in the CNS. Therefore, experiments on the behaviour of conscious guinea-pigs during withdrawal from morphine were carried out. It was found that injection of naloxone hydrochloride, 15 mg/kg subcutaneously, 2 hours after injection of morphine sulphate, 15 mg/kg subcutaneously, induced marked locomotor activity, rearing, chewing, scratching, piloerection and clawing at the cage floor, as well as increased plasma catecholamines (Chahl & Thornton, 1987; Brent, Johnston & Chahl 1987). Substance P, 1 nmol injected intracerebroventricularly, mimicked this response since it also produced increased locomotor activity and other behaviours identical to those produced by morphine withdrawal including increased plasma catecholamines (Brent, Johnston & Chahl, 1988). However, substance P antagonists administered intracerebroventricularly do not appear to be effective antagonists of the central actions of substance P (Johnston & Chahl, unpublished observations). Thus, the similarities between the enteric and central nervous systems are striking. Withdrawal responses may be precipitated following a single dose of opiate, and although definitive evidence is still lacking for the CNS, these responses may be mediated by substance P or a related tachykinin in both systems.

In recent years interest has arisen in the mesolimbic system as a major pathway involved in opiate reward mechanisms (see Bozarth, 1987). The mesolimbic system includes the ventral tegmental area and nucleus accumbens, the nucleus accumbens possibly being the interface between motivation and behaviour. The role of substance P in this system is unknown at present although it is noteworthy that substance P injected into the ventral tegmental area produces increased locomotor activity in rats (Iversen, 1982). Elucidation of the role of substance P in opiate dependence and craving promises to provide insights into the complex relationships between motivation, reward and resultant behavioural responses.

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Will Considering Drug Dependence Reduce Drug Problems? Alcoholism as a Self-Limiting Disease Revisited; or There are Things We Don't Want to Know!

L.R.H. Drew
Consultant Psychiatrist
Canberra
Australia

Recently I was working on a paper on AIDS and IV drug use. A project officer assisting me drew up, on a white board, what our aims ought to be, documenting what I would like to see happen. She put a footnote at the bottom which said - "If you do what you always do you will get what you always get". I expect she is right. History has a habit of repeating itself. As a group, people don't learn. This seems also to be true of individuals. I find that the conclusions I thought I had reached over the last three years, in regard to both theory and policy about drug use were exactly the same as those I reached twenty five years ago.

In 1965, while in Oslo, I attended an international conference on alcohol problems. I had just spent six weeks studying the history, the literature and the then current practice in the alcohol field in Sweden, Finland and Norway as the guest of ANSVAR Insurance Company. At the conference many international experts spoke of their treatments and of their successes in terms of people giving up drinking. Finally the request came for comments or questions from the floor. I made the comment that no one had mentioned the natural history of drinking, a subject in which I had become interested as a result of what I had found in Scandinavia. I asked, "Did we know anything about this and should we take it into account in discussing treatment outcome?" I returned to my seat and the show went on. My comment and question were totally ignored. I felt like a brash young colonial who had blundered across sacred ground.

Later, I wrote an article documenting my findings (L.R.H. Drew, 1968). It took me over two years to get it published in the Quarterly Journal of Studies on Alcohol. I had to change the title because "Alcoholism as a self-limiting disease" was too presumptuous.

I expect that, seeing that I am still blundering across sacred ground, my paper today will evoke the same silence as my question did in 1965.

Very briefly, my point is that it is time for a major change of emphasis by those who profess to be expert about drug issues. To date, the

promotion of concepts such as 'drug dependence' has resulted in the community believing that drug use is a special problem, and that belief obstructs the development of a rational debate about drug policy. Similarly, concern about preventing or curing drug dependence has distracted attention from developing ways of reducing drug problems. I believe that we need to redress the problems that we have created. Models of drug dependence - or drug addiction - must be directed at normalising the subject of drug use. Only then will models of drug dependence be of value in reducing drug problems.

In my 1968 article I concluded that:

> The reported evidence demonstrates that alcoholism tends to disappear with increasing age.....a significant amount of this disappearance is probably due to spontaneous recovery.....Such conclusions are not new. As early as 1849 Magnus Huss pointed out that 'it is a rare exception to meet with an alcoholic who is over 60 years of age' and Amark, in 1949, deduced.....'that many alcoholics, at the age of 50 years, improve spontaneously....'. (p. 965)

I proposed then that:

> Increasing maturity and responsibility, decreasing drive, increasing social withdrawal, changing social pressures, reduced financial resources, and onset of psychiatric disturbance, are factors which often accompany ageing and which may contribute to this reduction of alcohol problems with increasing age. Other factors, such as social isolation or inadequacy and the early onset of severe complications of alcoholism, may inhibit natural recovery processes. However, no studies are available to support or refute these suggestions, as until now the significance of age, and of the natural history of alcoholism, have commanded relatively little interest among researchers. (pp. 963-965)

George Vaillant, in 1983, in his book *The Natural History of Alcoholism* proposed, quite correctly, that to state that natural recovery is spontaneous is to undervalue all of those factors I outlined and to dismiss important issues such as life events. He emphasised the need for "treatment" - if we wish to keep the term - to be directed at reinforcing the factors tending towards recovery, including value systems.

In my 1968 paper, I reviewed the literature, indicating the failure of treatment programs to influence drinking behaviour. However, I made reference to the fact that, "[i]n Scandinavia the approach to the treatment of alcoholism is to offer medical care and encourage continued social acceptance...[and, if necessary] custodial care. Attempts to accelerate recovery from the phase of excessive drinking take a very secondary place compared with efforts to reduce the complications encountered" (p. 965).

Today, as in 1965, the major demonstrable outcome of offering treatment to people with drinking problems is that more of them stay alive. We are still unable to demonstrate a significant long-term impact on drinking behaviour of treatment programs, regardless of their intensity of sophistication (Congress of the United States, 1983). Nevertheless, a problem centered approach remains uncommon. Rarely is there agreement that the first goal in treatment of persons with drinking problems should be to minimize harm (to tide the patient through their troublesome time) and that the secondary goal should be to facilitate natural processes tending towards recovery. Most people are still committed to active intervention and the search for a cure of the elusive disease of "alcoholism" - now called "ADS" (i.e., Alcohol Dependence Syndrome) by the sophisticated.

Since 1968 it has become fashionable, both amongst politicians and scientists, to give major consideration to drugs other than alcohol, particularly those used illegally by young people. Again, the fact that problematic use of such

drugs is time-limited, with natural renunciation of that pattern of drug use generally occurring by the age of forty years, has been documented but ignored. A search for cures has taken precedence over provision of care and assistance designed to minimize harm. The focus of attention has been on eradicating drug use, evolving models of drug dependence, and on dealing with this supposed disease or syndrome rather than efforts to reduce drug problems. Not surprisingly, rather than preventive education programs and law enforcement ushering in a new Utopia, with a generation of drug free responsible young adults, drug use and drug problems remain high and law enforcement action is still escalating. Also, not surprisingly, although the provision of treatment has been embarked upon with enthusiasm the results for drug users have been little better than well documented rates of natural change.

The approach of 1988 is little different to that of 1965. Although the number of persons who claim to have scientific expertise and who spend their time addressing drug issues has increased dramatically, their devotion to science seems to have been fairly fruitless. So far as outcome is concerned, one could hardly claim that the level of alcohol and other drug problems has been significantly reduced. So far as process is concerned, in 1988 as in 1965, most workers in the field are still preoccupied with the goal of attaining abstinence in their clients and they still avoid unpleasant realities. They invoke apparently scientific formulations about drug use - models of drug dependence - to substantiate their activities. There has been a constant stream of new treatment approaches and a regular succession of theoretical "fat words" (as Harry Levine describes them (Levine, 1984)) each pretending to advance our knowledge and our precision in describing reality. Mostly, (there are recent exceptions (Edwards, Arif, & Hodgson, 1981)) these words and formulations have been directed at re-clothing a disease concept of drug addiction and of ensuring that the subject of drug use remains the province of people with special expertise.

People who consider themselves to be scientists, but who fail to recognise the limits of science (Department of Health, 1985), promote the need for expert intervention and the concept of a disease (however, they may attempt to disguise it). The outcome of their activity is that drug use - an ordinary behaviour - has been obfuscated and the public has been confused. For instance, we now have a scientific definition of alcohol dependence. However, in fact, this scientific definition is really no different to the description of habitual drunkeness given two hundred years ago. Then, the habitual drunkard "loved to drink". Now, an alcoholic dependent person "gives too high a priority to drinking" (Drew, 1982).

Surely, it is now time to face the truth, to undo the havoc we have created, and to place harm reduction at the top of our priorities rather than being preoccupied with drug dependence and the goal of eliminating drug use.

The National Campaign Against Drug Abuse (NCADA), begun in Australia in April 1985, typifies the current situation. In spite of its aim being "to minimize the harm associated with drug use", the endorsed strategies are demand reduction (minimizing people's tendencies to use drugs) and control supply (minimizing people's access to drugs) - not a word about reducing harm (Department of Health, 1985).

The politicians considered that expanding treatment programs was a demand reduction strategy, presuming that the goal of treatment is to cure drug users. Therapists were expected to lead drug users to abandon drug use, thus reducing demand. These expectations of politicians were shared by - in fact probably arose from - treatment personnel. They believe treatment works and that drug use can be cured. Consistent with that

belief, many of them have opposed the use of methadone, even though it results in a reduction of harm and increased social integration, because it competes with drug free programs and it may perpetuate drug use. Similarly, many drug workers have refused to countenance involvement in supplying sterile needles and syringes to intravenous drug users because it would compromise them. If they gave out needles and syringes, or even educated their clients about how to sterilize used instruments, it would seem they were accepting the reality of drug use and the fact that it won't go away.

These attitudes seem odd when our goal is to help drug users and when AIDS and hepatitis - acquired by needle sharing - could kill a third or more of all current intravenous drug users within the next few years. It is particularly odd when all of those users will stop being drug users at some future date, with or without our help, provided they survive long enough without getting AIDS.

Perhaps it is a little unkind to say it but many drug workers seem to be like Nero, fiddling while Rome burns. Those of us who have claimed to offer leadership, particularly those of us who have contributed to the literature about models of drug dependence, have also indulged in irrelevancies. We have pursued an abstract scientific course rather than responding to existential needs. We have produced a psycho-bio-social model of drug dependence that excludes the essence of human existence - options, freedom to choose and the centrality of value systems (Edwards, Arif, & Hodgson, 1981). We have attempted to divorce ourselves from a moral stance and we have tried to appear scientific (Department of Health, 1985). We have produced a vast literature to try to prove that drug use and drug addiction are something special. However, we have paid little attention to developing scientific - or even simple - interventions which would reduce drug problems. For instance the mechanisms of drug-related deaths, an important issue to use as an example, is a vastly neglected subject. In 1982, I noted that lack of awareness by relatives and friends of the dangers of intoxication was a major issue, but that issue has still not been addressed (Drew, 1982).

Perhaps we fail to attend to the complications because to do so would be to admit that we aren't able either to prevent or to exorcise drug use. Perhaps we prefer to attempt to improve treatment techniques in the hope of one day being successful because we are afraid to stop. Perhaps the basic issue is our need to avoid facts about the limitations of human endeavour.

Having created our current situation we cannot, now, dismiss the subject of models of drug dependence. Not because they can be helpful in improving preventive programs or treatment approaches - but because models of drug dependence exert a major influence on the nature of the drug debate in the community. If the idea prevails that drug use - and more particularly drug addiction - is a special type of behaviour which is highly contagious, irreversible, inevitably leads to disaster, and is due to the special seductive properties of certain drugs, then our approach to reducing drug problems is not going to change. If, however, the ideas prevail that drug use is more similar to other behaviours than different from them and that there is little that is special about drug addiction compared with other addictions which are universally experienced, then the drug hysteria may abate and a rational approach to policies to reduce drug problems may be possible. It may then be accepted that, as with other human problems, there are no simple answers and few worthwhile short cuts.

We need a model of drug use so that we can promote two simple facts, and to undo the confusion that we have created. It must be known that:

(i) people get into trouble with drugs, in the same way that they do with many other things and behaviours (particularly behaviours giving short-term rewards);

(ii) people will find themselves in predicaments because of their drug use and may need help to get out - or simply need protection from harm until they are able to reorganise their lives.

However, rather than developing dramatic programs to eradicate drug use, we need to:

(i) promote the idea that human behaviour is always complex;

(ii) emphasise that there will always be a need for mutual concern (offering help to people in trouble);

(iii) highlight the need for self-governance (individuals maintaining a balance between short-term pleasures and long-term goals).

Experts in the drug field should make it clear that we have all failed in our efforts to understand and to deal with drug problems. For the future, we should direct our efforts so that, in another twenty years, we really will have changed in the way we deal with drug problems and have achieved a real reduction in their prevalence.

I commenced this paper by noting the cyclical nature of history. Later, I quoted the similarity of modern concepts to ancient concepts. I would like to end by drawing attention to the fact that most of the apparently new techniques of behavioural science are, in fact, resurrections of old, moralistic, disciplines. Here I refer to ideas and techniques such as self-efficacy, self-contracting, self-monitoring etc. Perhaps, if we had the humility to accept that we were not discovering something new and admitted the origins of these ideas we might be able to exploit the wealth of earlier wisdom with great advantage. As Solomon said, "There is nothing new under the sun".

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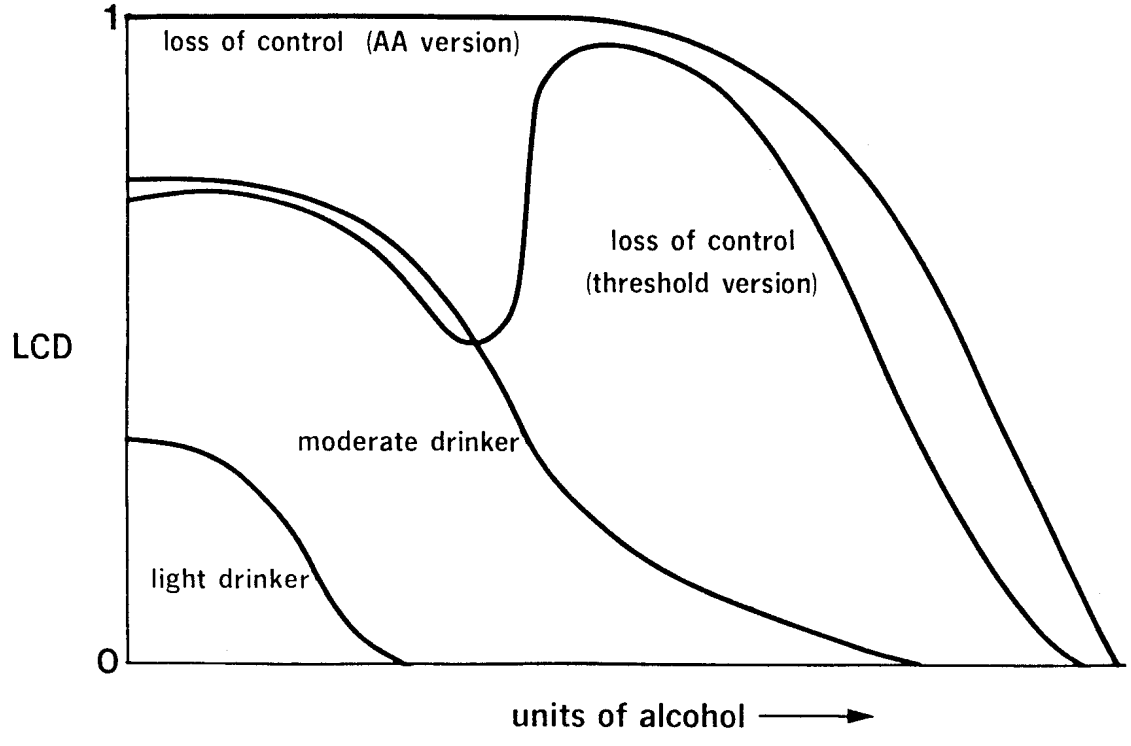
Figure 1
Likelihood of continued drinking for a hypothetical drinker (see text).

Figure 2
LCD curves for different types of drinkers.



conception, loss of control was identified with physical dependence, as opposed to the psychological dependence characteristic of non-disease species of alcoholism. It was said to occur as a result of minor withdrawal symptoms in the presence of alcohol in the bloodstream, continued drinking being forced on the alcoholic by the need to abolish these unpleasant symptoms and the increased anxiety they provoke. Jellinek also states that loss of control does not emerge suddenly but rather progressively in the course of the alcoholic's drinking career and then only after many years of excessive drinking. Further, it does not occur every time the alcoholic takes a drink - a clear departure from the AA view. Finally, Jellinek chastises other students of the subject for failing to distinguish between loss of control and "uncontrolled" or "undisciplined" (sic, p. 42) drinking which involves the *deliberate* transgression of rules relating to amount, times, occasions and locales for drinking.

The type of loss of control applied by Jellinek to the *gamma* alcoholic referred to drinking to gross intoxication, unconsciousness or arrest on a single drinking occasion. He also recognized a further variety which described the drinker's inability to abstain from drinking for even a few days without the occurrence of withdrawal symptoms, while the ability to control intake on any given occasion remained unaffected. This was associated with the *delta* alcoholism found mainly in France and other wine-drinking countries. The type of loss of control applying to the so-called *gamma* alcoholic, and with which we are concerned in this paper, was simply termed by Marconi (1959) "inability to stop".

It is important to note that the centrality of loss of control, or at least of a refined version of it, has been retained in more modern disease views. In the increasingly influential Alcohol Dependence Syndrome, which many would regard as a quasi-disease formulation founded on the key assumptions of the disease theory (see, e.g., Shaw, 1985), the syndrome is defined as "a disability marked by impaired capacity to control alcohol intake" (Edwards, Gross, Keller, Moser, & Room, 1977, p. 17) and the leading symptom of the syndrome is said to be "impaired control over the drug ethyl alcohol"(p. 17).

Revisions of the Concept

The AA version of inability to stop was massively disconfirmed by a large body of findings based on controlled observations of alcoholics' drinking behaviour conducted mostly in the 1960s and early 1970s (e.g., Bigelow & Liebson, 1972; Cohen, Liebson, Faillace, & Speers, 1971; Cohen, Liebson, & Faillace, 1972; Gottheil, Corbett, Grasberger, & Cornelison, 1971; Gottheil, Murphy, Skoloda, & Corbett, 1972; Mello, 1972; Mello & Mendelson, 1965; Mendelson & Mello, 1966). These studies showed, in summary, that, when allowed to determine the volume and patterning of their own drinking, diagnosed alcoholics do not invariably, or even typically, drink to oblivion but do demonstrate positive sources of control over their drinking behaviour. Moreover, this research clearly showed that alcoholics' drinking behaviour was subject to the same classes of environmental contingency that shape the drinking behaviour of nonproblem drinkers. It was not, of course, denied that alcoholics drink in much larger quantities than other drinkers, but what was refuted was the idea that this difference could be explained, even in part, by a mechanical, "chain-reaction" type of loss of control of the kind favoured by Alcoholics Anonymous.

The crude "one drink, one drunk" hypothesis was also contradicted during roughly the same period by many observations from treatment outcome studies that diagnosed alcoholics could sometimes return to a "normal" pattern of drinking, a possibility inconceivable from the AA viewpoint (see Heather & Robertson, 1983). It must be stressed that this is a quite separate

issue from the continuing controversy over whether alcoholics should be encouraged or helped to achieve a controlled drinking goal. Even dedicated opponents of this treatment principle do not now deny that some alcoholics, showing the very highest levels of withdrawal symptomatology and measured alcohol dependence, have been able to achieve a stable pattern of nonproblem drinking, although the frequency with which this occurs is still subject to dispute (see Helzer et al., 1985). Nevertheless, the fact that it does occur, however infrequently, obviously poses considerable difficulties for an unrefined concept of loss of control.

One response to this evidence by those wishing to retain a disease view was a watering down of the concept of loss of control by the introduction of an element of unpredictability or uncertainty, a possibility first mentioned, as we have seen, by Jellinek (1960). For example, Keller (1972) asserted that the alcoholic had not lost control over his drinking but could never be *sure* that, once started, he would be able to stop. Ludwig and Wikler (1974) spoke of a *relative* inability to regulate alcohol consumption. And in the Alcohol Dependence Syndrome, control is viewed as "variably or intermittently impaired rather than 'lost'" (Edwards 1982, p. 29).

This new concept of an unpredictably impaired control, although based on extensive clinical experience, clearly poses formidable problems for theory and research. Since the conditions under which control will be observed and those under which it will not are unstated, the hypothesis that impaired control is the central attribute of alcoholism is, as it stands, unfalsifiable.

Another strand of evidence disturbing to the traditional view of loss of control is the results of "balanced-placebo" experiments using a priming dose of alcohol (Engle & Williams, 1972; Marlatt, Demming, & Reid, 1973). The initial conclusion from this work was that alcohol expectancies, based on whether or not the individual *believes* he has consumed alcohol, are more powerful determinants of subsequent drinking behaviour than the physiological effects of alcohol, based on whether or not alcohol was actually consumed. However, the experiments in question could only be regarded as analogue studies and, in any event, Stockwell, Hodgson, Rankin, and Taylor (1982) subsequently showed that, when subjects were distinguished in terms of level of alcohol dependence, the drinking of more severely dependent alcoholics was mainly a function of alcohol rather than expectancy effects. Nevertheless, there would now be general agreement that expectancies play some role in the explanation of impaired control, an example being the Abstinence Violation Effect described by Marlatt and Gordon (1985).

It was chiefly to account from within the disease framework for the results of priming dose experiments and observations of resumed normal drinking, that Glatt (1976) proposed the existence of a critical blood alcohol level beneath which loss of control is not elicited but above which it sets in. Unfortunately, such a threshold notion cannot account for some of the findings of the laboratory studies referred to earlier showing that, under appropriate circumstances, alcoholics are able to moderate their drinking even at very high BACs. Nevertheless, the idea of a threshold at which the alcoholic begins to "feel" his drink and at which control becomes much less likely is embodied in the clinical wisdom on the subject (see, e.g., Kjolstad, 1963) and may have some descriptive value in the earlier stages of a drinking episode.

Present Status of the Concept

To summarize the present status of the concept of loss of control, or impaired control, it is necessary to distinguish between its advancement as a description of alcoholic drinking and as an explanation of that drinking. As a description,

the crude version of loss of control which states that it will inevitably occur after only one drink has been clearly shown to be false; the later version of the concept which states that it occurs only above a certain BAC threshold conflicts to some extent with laboratory findings but may apply to the continuation of a drinking session at earlier stages; the revised concept of impaired control, which states that control is variably or intermittently impaired when the alcoholic takes a drink, is untestable.

As an explanation of alcoholic drinking, the concept has fared even worse. It has been frequently pointed out that, like many other purported explanations of behaviour used in psychiatric formulations, this explanatory concept is tautologous (see e.g., Schneider, 1978). The existence of a process known as loss of control which is supposed to cause excessive drinking is inferred from the observation of the same set of behaviours that it attempts to explain; it therefore has no independent status as an explanation and cannot specify the observable conditions under which loss of control will occur.

Although, as has been pointed out, loss or impairment of control still finds a place in the psychiatric account of alcoholic drinking, the above criticisms have led psychologists largely to ignore the issue in recent work in this area; since earlier formulations have been found to be either inaccurate or untestable, it has been concluded that there is nothing to explain. The position adopted in this paper is that this is a mistake. The alcoholic who drinks in a harmful fashion and *complains* he is doing so has clearly, in some sense, lost control of his drinking. More precisely, the alcoholic who frequently expresses a strong intention to drink moderately and persistently drinks to excess has, again in some sense, demonstrated impaired control over drinking. Moreover, many alcoholics seen in clinical practice report a strong, subjective sense of a compulsion to drink which they attempt to resist with little success (Edwards & Gross, 1976).

These commonplace observations cannot be ignored in an adequate account of alcoholic drinking and, indeed, lead to the conclusion that the issue of impaired control remains central to an understanding of alcohol dependence. The task confronting theory and research is to restate the problem in a way which can be made the subject of empirical enquiry.

Operationalizing Impaired Control

One way of thinking about a control which is said to be "variably and intermittently impaired" is as a statistical phenomenon in which we are concerned with the probability of whether or not a drinker will continue to drink under specified conditions. A way of operationalizing the concept in this fashion is suggested by a paper by Taylor (1979) on a method for describing variability in alcohol consumption levels.

Taylor is concerned to develop an alternative method for describing the distribution of alcohol consumption within a defined population. He proposes a recasting of the consumption distribution curve in the form of a "consumption containment rate" (CCR), using a method borrowed from the calculation of age specific mortality rates. Applied to the alcohol field, the CCR is definable as a population's tendency to contain its drinking within any given level. In practice, the CCR is obtained by calculating reverse-cumulated frequencies for each level of drinking (i.e., by calculating the number of individuals drinking as many or more than x units of alcohol per week).

All that needs to be done is to apply exactly the same method to a distribution of drinking quantities per occasion taken from a single individual over an extended period of time. Table 1 shows how this would be done in a hypothetical case.

By subtracting the CCR from unity we may arrive at what might be called the "likelihood of continued drinking" (LCD) (i.e., the probability that, having drunk x units, the individual will go to drink x + n units). By plotting the LCD against levels of consumption, we may see at a glance the degree to which, and the point at which, the individual will show "impaired control" over drinking. For the hypothetical data in Table 1, the resulting curve is shown in Figure 1.

In Figure 2 are shown different types of LCD curve for different types of drinker. At the top is the curve that would result from the AA version of loss of control; the LCD starts at "one" and only begins to trail off after sufficient drink has been taken to introduce the possibility of the alcoholic running out of money, being arrested etc.. In complete contrast to this, the curve for the "light drinker" begins low and quickly reaches zero. In between, there is the "moderate drinker" for whom the LCD commences at an intermediate level and takes longer to reach zero than for the light drinker. Perhaps most interesting is the curve that would result if the notion of a threshold BAC for loss of control were correct; the LCD for such a drinker might begin at a level close to the moderate drinker and would show an initial decrease; when the threshold level was reached, however, the LCD would rise rapidly and the curve would resemble that for the AA version of loss of control.

Table 1

Calculation of the Likelihood of Continued Drinking from a Hypothetical Distribution of Consumption Quantities Within an Individual.

(1) X (units of alcohol)	(2) F_X	(3) C_{+X}	(4) CCR	(5) LCD
1	32	231	0.139	0.861
2	53	199	0.266	0.734
3	41	146	0.281	0.719
4	28	105	0.267	0.733
5	21	77	0.273	0.727
6	22	56	0.393	0.607
7	17	34	0.500	0.500
8	8	17	0.471	0.526
9	5	9	0.556	0.444
10	4	4	1.000	0.000
	231			

(1) Consumption per occasion in units of alcohol.

(2) Frequency of occasions on which X units were consumed.

(3) Reverse cumulated frequencies, i.e. the number of occasions on which X or more units were consumed.

(4) Consumption Containment Rate (CCR) [(2)÷(3)], i.e. the probability that drinking will be contained at X units.

(5) Likelihood of Continued Drinking (LCD) [1-(4)], i.e. the probability that having consumed X units the individual will continue to drink.

What has been described is a method of avoiding the unfalsifiability of recent statements about impaired control. In principle, this method could be used to test predictions about the drinking behaviour of individuals in whom impaired control has been hypothesized to be present compared with those in whom it is regarded as absent. It would clearly take a large sample of accurate drinking data from each drinker to plot reliable LCD curves and this presents methodological problems. The "Timeline Method" described by Sobell, Maisto, Sobell, and Cooper (1979) might be a way of providing reliable data over a sufficiently long time period. This method has been shown to be reliable among a variety of drinking populations (see Sobell, Sobell, Leo, & Cancilla, 1988). It may also be possible to collect the required data prospectively, although this too is methodologically difficult.

A limitation of the use of the LCD is that it ignores the distinction made by Jellinek (1960) between drinkers who are unable to control their drinking and those who have never had any intention of controlling it. That this distinction is a real one is suggested by some preliminary data reported by Chick and Best (1978) who asked diagnosed alcoholics about their experiences of being unable to keep to a drinking limit. They found that 42% of 60 male inpatients denied recalling an occasion when they had set a limit or when they had set one and had not subsequently changed their minds. Similarly, when asked about difficultly in preventing getting drunk, 34% said that they had never tried consciously *not* to get drunk, mainly because they had never wished not to get drunk. It would appear, therefore, that for many alcoholics, the issue of impaired control has simply not arisen.

When in the Drinking History Does Impaired Control First Occur?

A recent development in our knowledge of impaired control concerns the stage in the drinking history when problems with control first become obvious. Jellinek's view, it will be recalled, was that it occurs only after many years of excessive drinking. Further, since loss of control is "pathognomic" of the crucial phase of alcohol dependence (Jellinek, 1952), it follows that it is, by definition, a relatively late occurrence in the development of dependence.

Research by Chick and Duffy (1979) casts doubt on this traditional view. These authors asked male alcoholics to rank order the age at which various "core symptoms" of alcohol dependence occurred and then developed a modal sequence for these items. (Previous work had shown that, while the negative consequences of excessive drinking may not occur in any predictable order, core aspects of alcohol dependence could be placed in a reliable, nonrandom order (Orford & Hawker, 1974)). The results of this investigation are shown in Table 2. It will be seen that an item reflecting impaired control ("Completely unable to keep to a limit") is accorded first place in the sequence, while two related items ("Difficulty preventing getting drunk" and "Difficulty cutting down") also appear early.

Chick and Duffy's (1979) findings are supported by those of Orford and Hawker (1974) who found that the item, "When you started drinking, you found you couldn't stop" clustered with early items which the authors grouped as describing "psychological dependence".

There are two important consequences of these findings. The first is that, if impaired control occurs early in the sequence and well before the onset of significant withdrawal symptoms, then Jellinek's (1960) explanation of loss of control in terms of the need to keep drinking to avoid or escape from minimal withdrawal is unlikely to be correct. Other kinds of explanation for impaired control must be examined.

Table 2
The "Typical" Developmental Sequence of 21 Sympotoms in Males admitted to an Alcohol Problems Clinic.

Completely Unable to Keep to a Limit
Needing more Than Companions
Difficulty Preventing Getting Drunk
Spending More Time Drinking
Missing Meals
Amnesias
Difficulty Cutting Down
Giving Up Interests
Restless Without
Change to Drinking Same on a Work Day as a Weekend
Organising Day to Ensure Supply
Tense on Waking
Passing Out While Drinking
Trembling
Can't Think of Anything Else
Retching
Sweating
Morning Drinks
Decreased Tolerance
Waking Feeling Frightened
Hallucinations

Source: Adapted from Chick and Duffy (1979). (All subjects do not have all symptoms, but if they have a given symptom, it is likely to have occurred in the position indicated.)

The second consequence is that many individuals who might be identified as showing impaired control could become prime candidates for early intervention strategies aimed at preventing the later stages of dependence, and the more harmful consequences of excessive drinking, from developing. Indeed, it may be that the early presence of impaired control can be used as a marker for distinguishing those relatively mild problem drinkers who are likely to go on to experience more severe problems from those who are not. This is obviously speculative but seems a reasonable hypothesis.

If impaired control can be identified and measured at early stages in the drinking career, a possible method of intervention readily suggests itself. This is the cue exposure with response prevention technique described by Rankin, Hodgson, and Stockwell (1983). Problem drinkers, who meet conventionally accepted criteria for allocation to a controlled drinking goal of treatment (e.g., a score of 30 or less on the Severity of Alcohol Dependence Questionnaire (Stockwell, Hodgson, Edwards, Taylor, & Rankin, 1979)), would be given amounts of their favourite beverage to a BAC at which impaired control had been shown to occur and then asked to actively resist further drinking. Subjective and physiological indices of desire for further alcohol would be monitored and the procedure repeated until craving responses had decreased to low levels. Practice at resisting further drinking in the laboratory would then be supplemented by supervised practice *in vivo*, using methods similar to those described by Blakey and Baker (1980), and then by unsupervised practice. This method could either be used on its own or added to self-management programmes aimed at controlled drinking. We intend to examine the feasibility of such a procedure at the National Drug and Alcohol Research Centre.

The Distribution of Impaired Control in the Drinking Population

The notion that impaired control first occurs early in the development of dependence suggests the possibility that it is by no means confined to those individuals who would be diagnosed as alcoholics or problem drinkers by conventional criteria but might, rather, be widely distributed among drinkers in the general population. Such a possibility would clearly depend on how impaired control was defined. In a frequently cited discussion paper, Storm and Cutler (1975) define loss of control as "drinking in a quantity and to a level of intoxication beyond what the drinker had anticipated or intended" (p. 152). According to this reinterpretation, loss of control is said to exist but not to discriminate between pathological and nonpathological drinkers and to result from processes more social psychological than physiological. Unfortunately, to our knowledge, Storm and Cutler's interesting suggestions have not been followed up in the published literature.

Storm and Cutler argue that the crucial questions concerning loss of control are, first, those concerning the determinants of the resolve to limit drinking in the first place; second, the frequency of occasions on which a need to exercise control is felt; and, third, the variety of contexts in which drinking is perceived as a possibility. Further questions then arise as to the determinants of failure of control once attempts at control have been instituted.

Specifically, they suggest that an important determinant of whether individuals will attempt to control their drinking is their anticipation of negative consequences. For example, more than a few drinks after work may interfere with activities planned for later in the evening. The frequency with which attempts are made to control drinking is likely to be associated with the severity and frequency with which negative consequences are experienced, suggesting that those with heavier drinking histories are more likely to both attempt control and to fear failure to do so. Determinants of these failures of control may involve a complex interaction of motivational and situational factors which are discussed at some length by the authors.

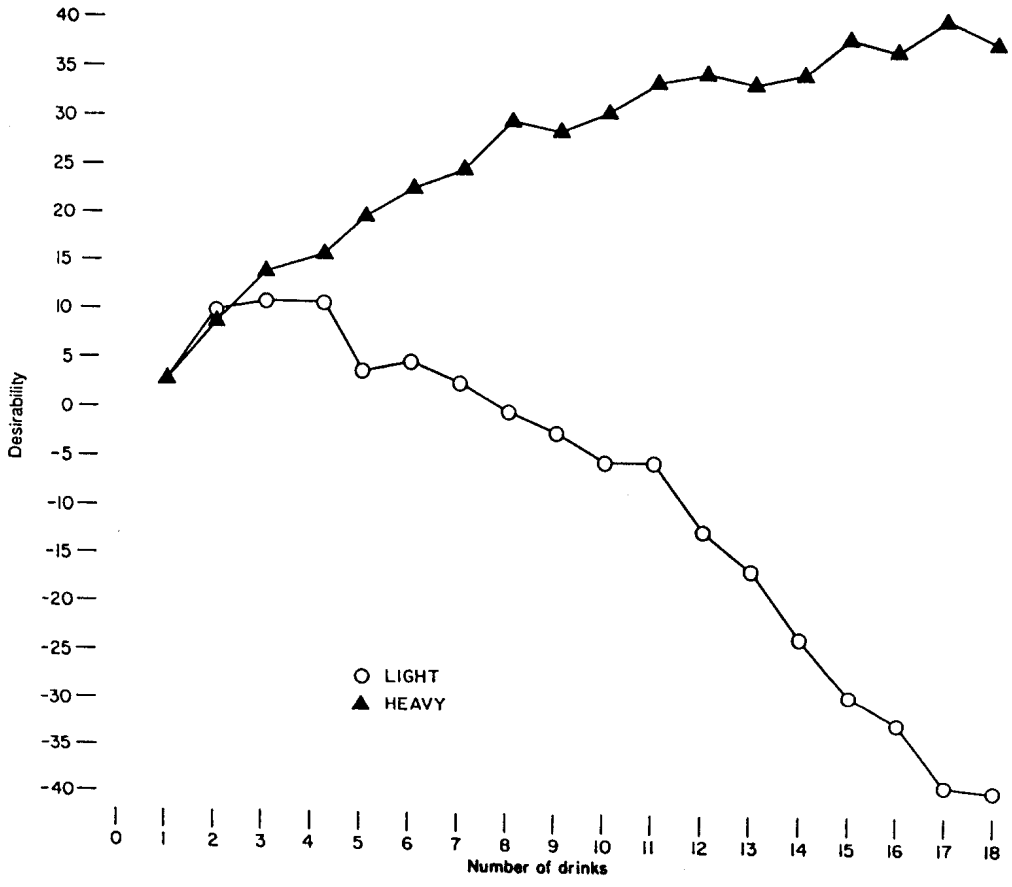
We are currently undertaking the task of converting Storm and Cutler's speculations into a form in which they can be used in a survey of impaired control in the general population, although this task presents some difficulties. We have so far conducted 10 unstructured interviews with individuals showing a range of drinking habits with a view to developing a semi-structured questionnaire.

Towards a Theory of Impaired Control

It has been implied earlier that, in order to provide an adequate explanation of impaired control, the concept must be related to specific hypothesized mechanisms that account for independently defined behaviours. Several explanations attempting to meet this criterion have been proposed (Ludwig, Bendfeldt, Wikler, & Cain, 1978; Marconi, Poblete, Palestini, Moya, & Bahimondes, 1970; Storm & Smart, 1965) but there will not be space to review this work here. Suffice it to say that the approach towards a satisfactory explanation is seen as involving research on several fronts - the development of a reliable and valid measuring instrument, surveys of impaired control and its determinants among general population samples, clinical research with clients for whom impaired control over consumption is the predominant problem and laboratory studies based on an analogue of impaired control.

With respect to the last type of research, a recently completed study by Poulos (1987) suggests an interesting possibility. Poulos studied 10 heavy drinkers and 10 light drinkers in a laboratory situation in which subjects consumed a drink containing about 4.2 ml pure ethanol every five minutes. Up to 18 such drinks were taken during a 90 minute experimental session. The main results of interest from this "forced drinking" experiment concern ratings of the desirability of each drink taken before it was consumed using a magnitude estimation

Figure 3
Desirability ratings for heavy and light drinkers in a forced drinking experiment (adapted from Poulos, 1987).



procedure. These results are shown in Figure 3. The remarkable feature of Figure 3 is that the mean desirability ratings of the heavy drinking group were all positive and continued to rise throughout the experimental session, whereas those for the light drinking group quickly became negative and showed a steep and consistent decrease. It might be concluded from this that Poulos has demonstrated "loss of control" among his heavy drinkers. However, although the desirability of drinking continued to increase, we cannot be sure that the heavy drinkers would have chosen to continue drinking if an alternative had been available or if there had been manifest costs attached to doing so. It is being suggested, in other words, that impaired control can be seen as involving a kind of successive cost-benefit analysis aimed at deciding between the alternatives of continuing and ceasing to drink. One could easily imagine an experimental situation in which the drinker is given a choice at each interval between drinking and not drinking (or, at least, choosing a nonalcoholic drink).

This may provide a suitable analogue with which to study impaired control. In this experimental situation, the relative contingencies applying to stopping drinking as against continuing to drink could be manipulated. The effects on impaired control of social and other environmental variables could be investigated. Lastly, it might be possible to separate alcohol and expectancy effects on impaired control by applying a modification of the balanced-placebo design to this situation. These are all possibilities which will be explored at the National Drug and Alcohol Research Centre.

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New Jersey
USA

The study of addiction has more greatly influenced psychologists in the field than vice versa. Whereas the psychological enterprise emphasizes social causality, multi-determined responsiveness, the actor as causal agent, and the variability of human action, alcohol and drug addiction studies emphasize reductive causality, unidimensional responsiveness, the actor as passive respondent, and fixed patterns of action. Rather than combating these tendencies (which spring from biological and medical conceptions of addiction and alcoholism), most prominent psychologists in the field have fallen prey to them. One reflection of this trend is the effort to treat substance abusers by matching treatments with "objective" client characteristics, rather than asking respondents what treatment they prefer or would succeed better at. Indeed, the conception of addiction which today dominates the field reflects the triumph of objectivist conceptualization over observation of behaviour and belief in the psychological meaning of human conduct.

Introduction: The Dependence Shuffle

I violated my prescription in being invited to speak at this symposium on drug and alcohol dependence by using the term addiction (actually, "addictionology") in the title of my presentation. I *never* speak about dependence; I always speak about addiction. Addiction is what we are interested in. Dependence is an English word meaning that you rely on something or somebody. It has a rich traditional meaning which I respect, and it is a useful concept. But I am boycotting its use as a concept specifically related to drugs and alcohol. It tells us nothing *special* about these things. That it is used in connection with drugs and alcohol as though it conveyed some kind of specific scientific meaning is a strange hang-up having to do with our psychotic way of viewing drugs, a psychosis limited largely to the twentieth century. (I elaborate this view in a paper I have forthcoming in the *Annals of the New York Academy of Sciences*, entitled "Addiction as a Cultural Concept.")

As I pointed out in *The Meaning of Addiction*, the 1960s saw a rise in the classification of drugs according to their "dependence" liability. This was in conjunction with a move among World Health Organization pharmacologists — such as Isbell, Halbach, et al. — to discontinue relying on "addiction" to describe drug problems, since so many people took "addictive" drugs, like heroin, without becoming addicted and because there was such accumulated evidence that people became addicted to a host of nonnarcotic drugs. As a result, Halbach et al. wished to employ the terms "physical" and "psychic" dependence to convey their beliefs and feelings about drugs.

What they attempted to do in this endeavour was to paper over the truism that addiction is not a pharmacological concept by substituting different words for addiction. Psychic dependence was thus used to label drugs like marijuana, amphetamines, and cocaine which they didn't want people to use, but which they were reluctant to call addictive, while physical dependence was the term they applied to the narcotics, which they wanted to think of as addictive despite evidence that reactions to these substances were as variable, subjective and culturally and situationally determined as were reactions to all psychoactive substances.

Thus, they created psychotic tables of illicit substances (and one legal drug, alcohol) which said they all produced psychic dependence, while another column claimed narcotics (and probably alcohol too) caused physical dependence. Of course, where would legal substances like caffeine and nicotine go? Or, what differentiated the psychic dependence produced by marijuana from that produced by watching television? Or why, if we could no longer confidently distinguish narcotics as producing addiction could we blithely list them as physical-dependence producing?

The answer to this man-made dilemma, as I made clear in *The Meaning of Addiction*, is to eliminate the distinction between psychic and physical dependence and, especially, to stop trying to make dependence a characteristic of drugs rather than of activities and human action and reaction. In addition, since addiction was a great, useful word before it was commandeered by pharmacologists and physicians, all we had to do was to return to its standard English meaning — which was not only evocative, but scientifically accurate in describing addiction in behavioural terms and as resulting from an interaction between personality and activity or substance use.

The proof of the pudding (or of the psychosis) in our terminology has been the amusing reaction in the scientific community to cocaine. Through the 1970s, pharmacologists working busily in their laboratories at Michigan and elsewhere established that cocaine did not produce physical dependence. Then, with the outburst of cocaine use and abuse in the 1980s, spokespeople for psychosis, like Ron Siegel, began telling us that cocaine "produced a psychological addiction so potent that it was indistinguishable from physical addiction". Aha, here is science so subtle that it can not be observed by the human eye or comprehended by the human brain — it looks, acts, and feels like addiction but, because of our alchemic categories, it can't possibly be addictive! Naturally, when public and political outcries against cocaine became sufficiently strident, this hang-up was overcome and we declared cocaine *was*, after all, addictive.

Meanwhile, in a field where people rush to explain what they don't know by reference to the action of molecules and neurotransmitters, the distinguished"psycho"pharmacologist Sidney Cohen intoned:

> Under conditions of access to large amounts of cocaine the human response remarkably resembles that of the laboratory animal. Cocaine-dependent humans....exhibit behaviours markedly different from their precocaine lifestyle. Cocaine-driven humans will relegate all other drives and pleasures to a minor role in their lives. The drive for cocaine will compel them to perform unusual acts in comparison to former standards of conduct. (Cohen, 1981)

They act this way, according to Cohen, because: "If we were to design deliberately a chemical that would lock people into perpetual usage, it would probably resemble the neuropsychological properties of cocaine".

Meanwhile, the epidemiology of cocaine use tells us quite a different story. Ronald Siegel followed a group of cocaine users from

the time they began use in college. Eighty percent maintained social or recreational use in the decade they were followed up. A more recent study — *The Steel Drug* (an unfortunately lurid title) — was published by a distinguished group of Canadian researchers at the Addiction Research Foundation. To compensate for the overemphasis on the small minority of cocaine users in treatment, this study chose middle-class users through newspaper ads and by referrals from colleagues. Again, most cocaine users in the ARF study did not report negative consequences, and most negative consequences were not of the addictive variety. However, twenty percent of subjects did report uncontrollable urges to continue use. Yet, in the case descriptions of the worst problem users in this book, nearly all had quit or cut back without actually receiving treatment for cocaine addiction! In place of an image of the incurable, irresistible addictiveness of cocaine, we have here an epidemiological profile which more or less resembles any habitual activity, from watching TV to attending church.

But my point is not to establish some semblance of reality or reason in regard to cocaine use. It is to observe the way that cocaine was "discovered" to be addictive. For now, the niceties of Siegel's and others' bizarre descriptions of "psychological addictiveness" indistinguishable from "physical addictiveness" have given way to unqualified descriptions of cocaine as inherently addictive. Of course, I do not mean to indicate that cocaine is not addictive because laboratory scientists were following a different myth before 1980 than that which is now in vogue. What the story of cocaine — as well as the entire drug dependence classification fiasco — proves is that we declare to be addictive what we don't like, are frightened of, and that people continue to do anyhow. This does not mean addiction is not real, but only that it does not comprise the pharmacological, Platonically idealized category researchers swear it is.

Cultural Attitudes Toward Controlled Drinking: The More "Scientific", the More Intolerant?

It is a truism that fundamental conceptions of reality affect the methods we use to study that reality and the results of that study. I have recently published a work in which I found that in different eras, different types of professional working in different countries have discovered wholly different rates of controlled drinking among alcoholics, from practically no such drinking to rates of remission due to controlled drinking equal to or greater than those obtained through abstinence (Peele, 1987). These differences were partly the result of definition. For example, is any alcoholic relapse tolerated in a definition of successful remissions? Vaillant (1983) actually classified abstaining alcoholics who had up to a week's worth of abandoned drinking as being in remission while calling controlled drinkers who showed a single slip of binge behaviour as actively alcoholic. At the other extreme, genetic researchers Goodwin, Crane and Guze (1971) called alcoholics (these were ex-felons) who still drank and some of whom regularly got drunk - but who eliminated criminal and other antisocial behaviour - to be in remission.

On the other hand, we cannot discount that different researchers with different expectations actually find different behaviours at different times. Orford and Keddie (1986) have concluded that persuasion that one type of goal (controlled drinking or abstinence) is achievable or not will make it relatively more likely that this goal will be achieved. Obviously, researchers (especially clinician/researchers) who accept controlled drinking outcomes will be more likely to find these, all other circumstances being equal. I believe this holds even for investigators who *claim* they are either abstinence-oriented but

find controlled drinking outcomes (such as D.L. Davies (1962) did in his highly publicized study) or who claim to be controlled drinking-oriented but conclude that controlled drinking rarely or never appears. Of course, the entire point of my earlier paper was that cultural and historical climates change toward topics such as controlled drinking, and researcher expectations and findings follow suit.

In that earlier article, I categorized countries in terms of how readily their nationals accepted and discovered controlled drinking results. In doing so, in every country that had an active alcoholism research and treatment program, I found great pressures against controlled drinking — even though there were clearly large differences in the tolerance (even encouragement) of controlled drinking outcomes and treatment programs across national boundaries. Yet, I continually heard rumors that in other countries out of the mainstream of international alcoholism research — countries in Southern and Eastern Europe — controlled drinking was the typical therapy and outcome used with drinking problems. Harry Levine told me about attending an international conference in an Eastern European country in which he found most of those outside North America and Northern Europe were unfamiliar with — one might say uncomprehending of — disease notions of alcoholism. Martha Sanchez-Craig told me that in Spain she found community alcoholism workers simply assumed control of drinking was the goal to pursue. Another woman told me that in Russia, when a worker was brought before the work group to discuss his alcoholism, the session *began* with a glass of vodka.

It occurred to me that even my attempt to broaden American horizons about the notion of alcoholism was quite limited, and that if I explored folk notions — folk notions perhaps broadcast as national policies or attitudes in different countries — I would discover even greater variation in conceptions of and approaches to drinking problems. In particular, I began to conceive that countries arranged on a continuum of their "advances" in alcoholism research might equally well reflect less acceptance of controlled drinking. That is, the more theoretical reflection on the nature of alcoholism and the more research on outcomes conducted in the country, the more pressure to eliminate controlled drinking categories in favour of abstinence and to structure conceptions about alcoholism in disease-type terms.

Might there be something inherent in the objectification of addiction and contemporary scientific approaches to it that *demands* disease-like conceptions and practices? For example, in my article on cultural variations in controlled drinking outcomes, I created a table ordering various professions in terms of their frequency of discovering controlled drinking vs. abstinence outcomes. I rated behaviourists toward the abstinence end, along with physicians, in contrast with psychodynamic psychologists and sociologists. That is, behaviourists — in their efforts to catalogue controlled drinking outcomes — created highly objective criteria that specified amounts drunk on particular days and that often had the effect of eliminating patients who *ever* became drunk from moderate drinking categories. This methodological narrowing of the controlled drinking range was paralleled by a theoretical narrowing. That is, even as behaviourists rejected disease conceptions of alcoholism, by formalizing concepts like alcohol dependence they came to see an entity that had particular contours and rules. For example, Goodwin and his colleagues could only find remission in former alcoholics who still got drunk because they had a flexible notion of alcoholism that was subsidiary to their views of subjects' overall functioning. As soon as scientists' gazes shifted from the background to the figure (if that is a fair description in this

case), they saw a distinct outline and ignored the fuzziness or gestalt characteristics of the phenomenon that Goodwin et al. perceived.

In other words, the idea that there is something real called "alcoholism", which the individual is in danger of relapsing to, is one accepted by the major advocates of a non-disease approach to alcoholism, principally behavioural psychologists. But if we discarded entirely the concept of alcoholism, and simply observed people's drinking in relation to their subjective states of mind and feeling, along with their environmental and situational conditions, including their life-stage, we would tend to make statements such as, "As a young adult recently abandoned by his lover, and exposed to a heavy drinking crowd of single, working-class men, Jack drank extremely heavily. When he got married, he gradually cut back his drinking (although not without conflict with his wife, who wanted him to be more abstemious than he was) until, when his first daughter was born, he ceased getting drunk entirely".

This last description, rich in idiography, is more akin to the biographer's enterprise. Yet, it represents a far more accurate characterization of Jack's drinking than saying Jack was an alcoholic (or, since he moderated his drinking, only a problem drinker). Thus, as a more accurate cataloguing of the available data, this becomes a better scientific description of Jack's drinking. Generalizing from the categories that predicted Jack's drinking problems (young, male, single, working-class, heavy drinking cohort), we can also create exceptionally strong predictive models of the type of drinking someone matched to Jack in all these regards is likely to do. Here, we see, we can link objectifiable, scientific categories to individual behaviour, and thus fulfil scientific goals, certainly better than we can by throwing around like manhole covers terms such as "alcoholic" and "problem drinker".

One Practical Outcome

The American psychologist William Miller (1983) has written convincingly about the role of motivation to achieve a drinking goal — whether abstinence or moderation — as a primary determinant of successful outcome from alcoholism treatment. Essentially, this amounts to asking the therapy client what goal he or she prefers and feels better able to attain. Simultaneously, Miller has written about creating objective classifications of alcoholics' characteristics for the purpose of *assigning* them to the proper treatment or treatment goal. These two approaches clearly may lead to conflicting aims in treatment. How do we respond then? To deny the client's stated goal is quite a long step toward both infantilizing and repressing the person. That is, on what grounds can we legally and morally say that we discount people's desires about their own bodies and souls? Of course, this is quite reminiscent of the all-purpose disease-view-of-alcoholism term, "denial", which means that when a client doesn't think he has the disease of alcoholism and you do, that you are right. Increasingly, in the United States, this also means you may coerce the person, using legal or job-related means, to enforce your perception over his.

We certainly have come a long way in psychology to the goal, described meticulously in George Orwell's *1984*, of completely removing individual responsibility from the species, only here we do it in the name of psychology, therapy, and a concern for humanity. Even Big Brother's forces didn't have the gall to make this claim — they imposed their will through a right-wing, militaristic mentality that emphasized an ever-present, ruthless, and heavily armed enemy. Consider that, in the United States, we have created the largest peace-time blockade in history, as well as substantial overseas imperialism, devoted strictly to the aim of preventing

Americans from consuming drugs. Either we are wrong that Americans hunger for these drugs or that they are unable to make sensible decisions about what they put in their bodies, or else we have reached a state where we feel everyone must be regulated for their own good.

In this regard, I think of a future technology which allows us to apply sensors to people so that we can instantaneously know if they are consuming an illicit drug. We won't need urine-testing then, and we will still have an infallible detector of drug use of which we disapprove, so that we can then arrest or remand *every single* drug user for treatment. Will this kind of control, as the kind of ideal version of drug-testing, lead us to a better world composed of better people? I think not, and I certainly do not look forward to it.

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Self-Control Ideology and the Experience of Addiction in General and in Clinical Populations

Robin Room
Alcohol Research Group
Medical Research Institute of San Francisco
California
USA

In popular conceptions, loss of control over drinking involves a double loss of control, over one's life as well as over one's drinking. A measure of "control worries", concerning the experience of difficulties in controlling one's life, is developed and interrelated with loss of control over drinking and with heavy drinking behaviour in samples both of the general population and of alcohol treatment clients in a California county. Loss of control over drinking is often but not always conditioned on general control worries, and control worries add modestly to the extent heavy drinking accounts for loss of control over drinking.

The Double Loss of Control

"We admitted that we were powerless over alcohol — that our lives had become unmanageable". This famous First Step of Alcoholics Anonymous clearly contains within it the "loss of control" over drinking which Jellinek (1952) identified as the "pathognomic symptom" of the disease concept of alcoholism. But the AA's First Step extends beyond Jellinek's symptom; it refers to the experience of a double loss of control — not only over one's drinking, but also over one's life. The two losses of control are conceived of as linked. In an AA interpretation, the causal arrow points from the loss of control over drinking to the loss of control over one's life. At an experiential level, the sequence may often be reversed: the feeling that one's life is out of control may precipitate the conclusion that one's drinking is out of control. Either way, in the long run, the experiences are seen as conditionally related: one can lose control of one's life in other ways than by losing control of one's drinking, but loss of control over one's drinking is presumed to carry with it loss of control over one's life.

In his well-known intepretation of "the discovery of addiction" Harry Levine (1978) makes a related formulation at a cultural-historical level. The idea of addiction to alcohol, Levine argues, arose at a time and in the context of a shift in American culture towards a heightened expectation of individual self-control. Addiction to alcohol became a culturally-

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understood explanation of failure of self-control and of failures in life. Drawing on this and other work, it has more recently been argued (Room, 1985) that alcoholism can be viewed as a "culture-bound syndrome": only where there is a cultural expectation of individual self-control can one experience a loss of control either over one's life or over one's drinking. Testing such a proposal, of course, requires cross-cultural studies.

These formulations raise the question of the empirical relationship between experiences of the two kinds of loss of control. Is the experience of loss of control of one's drinking preconditioned on the experience that one's whole life is out of control? To what extent do generalized worries about control explain the relation between heavy drinking and the experience of a loss of control? This paper explores these issues in two samples, one of the general adult population and the other of clients entering alcohol treatment agencies.

Measuring Loss of Life-Control

In the social and clinical psychological literature on alcohol, the topic of life-control has primarily been addressed by studies using Rotter's Internal-External (I-E) Locus of Control Scale. Reviews of this literature note that matched-control studies using the I-E scale usually find that alcoholics have higher external-control scores than nonalcoholics, and there are also some findings of a greater external-control orientation among heavier drinkers than among social drinkers (Barnes, 1983; Rohsenow, 1983; Cox, 1987). This literature has to some extent been undercut by the developing critiques of the meaning and internal structure of the I-E scale. Analyses by Gurin, Gurin and Morrison (1978) divided a set of I-E scale items into five factors, differentiating a factor of "personal control" — five items phrased in the first person about the respondent's perception of control of what happens to him — from factors of "control ideology", "success mobility", "political control" and "interpersonal control". As this analysis underlines, much of the content of the I-E scale reflects judgements about how the world works — whether people get ahead by hard work or by luck, whether voters can control politicians — rather than statements about a personal sense of control or loss of control over one's own actions and life. Even the items in the "personal control" dimension are conceptually somewhat distinct from self-control or loss of control. Gurin et al. note that the "personal control" factor, composed of items primarily attuned to a sense of mastery over one's own fate, correlates quite highly (.52) with a separate measure of "sense of personal efficacy". It should be noted that Alcoholics Anonymous-attuned therapeutic programmes tend to be quite critical of an inflated sense of personal mastery over fate, emphasizing "turning it over" to a higher power, rolling with the punches and taking "one day at a time". As Rohsenow (1983, p.47) puts it, "alcoholics who believe that they have the ability to control their own lives may not be liked by treatment staff, because this belief is contrary to traditional treatment philosophies". It is quite possible that the high scores on "external control" of clinical samples are heavily influenced by the ideology of their treatment setting and experiences.

One response to the reconsiderations of Rotter's I-E scale has been to "shift from assessment of global control orientation to more specific expectancies of control" (Rohsenow, 1983, p.47), as in Donovan and O'Leary's (1978) Drinking-Related Locus of Control Scale. To do this, however, is to lose sight of the broader territory of experience represented by AA's First Step, and reported by many in our culture who have experienced problems with their drinking. As we pondered how to measure a generalized self-control or loss of control dimension, we recognized that a wide terrain was involved. As noted above, the emphasis in the tradition epitomized by Rotter's scale is on expectancies about mastery versus fatalism. But self-control

as an everyday conscious experience and practice might well be quite separate from expectancies. A convinced fatalist can still believe in a duty of self-control (as Weber argued about Calvinism), while an overreaching expectation of mastery may contribute to a feeling of lack of self-control. Levine's analysis would point us not towards expectancies, but toward a more immediate sense of control: to an emphasis on the moment-to-momentness of maintaining self-control, to a continuing guard against "slipping" from grace. Limited in the availability of questionnaire space, we decided to include 6 items oriented to this existential, moment-to-moment facet of self-control, in questionnaires which already included items oriented to loss of control over drinking. The questions were asked both of a random one-third of the respondents in a survey of the general

Table 1
*Self-Control Ideologies and Concerns in the General Population and in Alcohol Treatment Clients**

"Please tell me, in general, whether you strongly disagree, disagree, agree or strongly agree with the following":

	N	PERCENT "AGREE" OR "STRONGLY AGREE" GENERAL POPULATION (652)	TREATMENT CLIENTS (381)
a. It's important to feel in control of yourself every waking moment.		91%	92%
b. You've got to relax and let your guard down every now and then if you want to enjoy life.		75	70
c. I worry about keeping control of my behavior.		22	77
d. I think of myself as quite self-controlled.		95	49
e. I feel unable to control my own life.		6	59
f. My life would go much better if I could just get control of myself.		11	87
CONTROL WORRIES SCALE - ITEMS c,d,e,f: (4 points for "strongly agree", 1 for "strongly disagree", etc.,except reversed on d.)			
Low control worries	4- 6	35%	1%
	7- 8	33	8
	9-11	21	42
High control worries	12+	1	49
Correlations of Control worries scale			
with item a		-.17	.03
with item b		.06	.03
of item a with item b		.01	-.12

*In this and the following tables, percentages are based on weighted data, while N's shown are unweighted. The general population data are weighted for household composition, the treatment clients to compensate for oversampling of women.

adult household population of a California county, and of a sample of the clients of the county's in-patient public alcohol treatment agencies (roughly evenly split between detoxification centres and recovery homes). Alcohol treatment clients were interviewed in the first days of their entry into treatment, to minimize the effects of treatment ideologies on their responses (although many, of course, had had previous contact with treatment agencies). The data from both samples were weighted for representativeness, by household composition in the general population and to compensate for an oversampling of women in the treatment population. The general design of the series of studies of which these two data-sets are a part is described elsewhere (Room and Weisner, 1988).

Table 1 shows the distributions of responses to the six new items on self-control ideologies and

Table 2
Factor Analyses of Self-Control Ideologies and Concerns, in the General Population and in Alcohol Treatment Clients

"Please tell me, in general, whether you strongly disagree, disagree, agree, or strongly agree with the following":

	PERCENT "AGREE" OR "STRONGLY AGREE"					
	GENERAL POPULATION			TREATMENT CLIENTS		
	I	II	III	I	II	III
a) It's important to feel in control of yourself every waking moment.	-.37	.79	.05	.05	.79	.44
b) You've got to relax and let your guard down every now and then if you want to enjoy life.	.11	.06	.99	.09	-.66	.64
c) I worry about keeping control of my behaviour.	.59	.44	.05	.59	.18	.39
d) I think of myself as quite self-controlled.	-.63	.45	-.09	-.65	-.10	.48
e) I feel unable to control my own life.	.74	.05	-.06	.72	-.20	-.23
f) My life would go much better if I could just get control of myself.	.73	.37	-.24	.74	-.00	.23
Eigenvalue	1.98	1.16	1.00	1.83	1.15	1.10

concerns in the two samples. On the face content of the first two items, it would seem that responses to them reflect the respondent's agreement or disagreement with them as general principles of conduct. It will be seen that a strong majority of respondents in both samples agree with both statements, although the statements appear somewhat contradictory. We could reconcile these responses by concluding that most respondents do not "want to enjoy life". A more likely conclusion is that we are probing an area where the culture has built-in contradictions: there is an official norm of invariable utmost attention and self-control, but alongside it there is also a slightly subterranean normative system concerning "letting go" of the official standard in appropriate circumstances. The distributions of responses of these general principles of conduct are very similar in the two samples.

In contrast to the first two items, items c, d, e and f in Table 1 are phrased in the first person: they refer not to opinions on a general principle of conduct but to the respondent's concerns or struggles about his or her own behavior. In the general population, an overwhelming majority think of themselves "as quite self-controlled", and correspondingly few feel that their life is out of control. But almost one-quarter report that they "worry about keeping control" of their behaviour. For a substantial minority, then, self-control is not a given, something that is automatic and unconscious.

On each of these four items, the alcohol treatment clients show a dramatically different pattern of response from the general population sample. While almost half think of themselves "as quite self-controlled", substantial majorities report not only worrying about their self-control but also feeling out of control.

The factor structure of the six items in Table 1 was examined separately in each sample in principal components factor analyses (Table 2). Although the distributions on the items were very different in the two samples, the structure of interrelations among responses to the items were strikingly similar in the two populations. The last four items all loaded most strongly on the first factor, while each of the first two items loaded highly on its own separate factor (in the treatment sample, item b also loaded highly negatively on item a's factor). On this basis, an additive "control worries scale" was constructed from items c, d, e and f, ranging from a low of 4 to a high of 16 points. In the bottom half of Table 1, summary distributions are shown for the two samples on this scale; as implied by the distributions on the component items, the scale discriminates quite strongly between those entering alcohol treatment and the adult general population. The correlations at the bottom of Table 1 show that there is very little pairwise relationship between the scale, and either of the first two items. One's control ideology, in other words, seems to have little relationship with one's own experience of concern about self-control.

Control worries seem to be fairly evenly spread by gender and age (table not shown). The distributions of scores by gender are very similar; there is some tendency for high control worries to decline with increasing age, particularly among men. In light of the great differences in control worries between the general population and those entering treatment, we examined control worries among those in the general population who reported that they had ever had experience of alcohol treatment (including going to Alcoholics Anonymous). The growth of the alcohol treatment system and the effects of its outreach efforts can be seen in the quite substantial proportion of adults in the county who have experience of alcohol treatment: altogether 11% reported such experience at some time in their life. The distribution of those with experience of treatment on the control worries scale did not differ greatly from the distribution

Table 3.
Indicators of Loss of Control over Drinking in the General Population and in Alcohol Treatment Clients

A. Have you ever....?

B. Did this happen in the last 12 months?

C. How often did this happen to you in the last 6 months — At least 5 times a wk, 3-4 times wk, 1-2 times wk, 1-3 times mo, at least once, never in past 6 mos?

		PERCENT OCCURRED IN LAST 12 MONTHS	
		GENERAL POPULATION	TREATMENT CLIENTS
	(N)	(651)	(381)
a. Found that once you started drinking it was often impossible to stop drinking until you became intoxicated.		2%	64%
b. Promised yourself not to drink alone but were not able to keep that promise.		1	28
c. Felt that you should cut down on your drinking or stop altogether.		8	87
d. Tried to cut down or control your drinking but were unable to do so.		1	65
e. Gotten drunk even when there was an important reason to stay sober.		1	64
Drinking control loss scales: (3 points if weekly +, 2 points if 1-3 times a month, 1 point if in last year,0 points if not in last year)			
Low drinking control loss-	0	91%	9%
	1	5	4
	2-6	3	31
High drinking control loss-	7-15 pts.	0	57

of other general-population respondents: 20% of the ever-treated, and 22% of the never-treated reported high control worries -- as against 91% of those in the current treatment sample. Ever-treated respondents were somewhat less likely to report low control worries than the never-treated (22% vs 37%), but still contrasted strongly with the current treatment sample (1%). This suggests that the very high control worries of the current treatment sample at least partly reflect the immediate situation in which they find themselves, thrust into the treatment system often in the immediate wake of distressing life events (Weisner, forthcoming). We should, accordingly, be cautious about interpreting a high control worries score as a continuing personality characteristic of the respondent. (See Rohsenow's parallel argument (1983, pp. 39, 44) about treatment samples' high I-E scores.)

Table 4.
Control Worries, Drinking Control Loss and Heavy Drinking: Correlations and Contingencies

General population above the diagonal

	1	2	3	4	5	6
1. Control worries		.14	.16	-.13	.01	-.07
2. Drinking control loss	.21		.62	-.05	.13	-.10
3. Frequency 12+ drinks	.05	.50		.11	.27	-.17
4. Years of education	-.01	.00	-.09		.07	-.10
5. Gender (male)	-.09	.05	.16	.07		-.04
6. Age summary (high)	-.05	.11	.10	.12	.10	

Treatment clients below the diagonal

	Drank 12+ drinks at least once in 12 months			
	NO		YES	
Control worries (low=4-8)	low	high	low	high
General population sample: (N)	(455)	(109)	(49)	(36)
proportion 2+ on loss of control drinking --	1%	3%	11%	37%
Treatment clients sample: (N)	(7)	(65)	(24)	(280)
proportion 2+ on loss of control drinking --	5%	50%	90%	95%

Empirical Relations Between the Two Losses of Control

Table 3 shows the 12-month prevalence in the two samples of items related to the other dimension of the "double loss of control" — of indicators of an experience of loss of control of drinking. As would be expected from the composition, the two samples differ quite radically on these items; a "drinking control loss" scale, summing scores assigned for the frequency of each item's recent occurrence, discriminates very strongly between the two samples.

Table 4 shows the interrelations of the two types of control loss and their relations with demographic variables -- educational level, gender and age -- and with a measure of frequent heavy drinking -- how often the respondent drinks 12 or more drinks on an occasion. Inspection of the correlations shows that, in both samples, the relation between the two sides of the "double loss of control" is fairly modest; the relationship is somewhat stronger in the treatment sample than in the general population sample. Control worries have a positive but modest correlation with frequent heavy drinking in the general population. Drinking control loss, on the other hand, is strongly related to frequent heavy drinking in both samples. While the relations of the heavy drinking measures with demographic dimensions

are in the expected directions (those involved in the general population are younger, maler, and less educated), the correlations are mostly fairly low. In most instances, heavy drinking is more strongly related to demographic differentiations than either control worries or drinking control loss.

Clearly, in these results, the strongest predictor of the experience of loss of control over drinking in each sample is the frequency of drinking 12 or more drinks. Does the respondent's experience of generalized control worries add to the prediction of loss of control over drinking? Multiple regression results (not shown) suggest that the answer is a tenuous "yes", particularly among those entering treatment: the beta weights for the frequency of 12+ drinks and for control worries are respectively .58 and .05 in the general population, and .49 and .18 in the treatment clients. Adding gender, age and educational level to the regressions affects these beta weights very little.

As envisioned in AA's First Step and other conceptualizations, however, the relation between the two control losses would not be symmetrical, so that Pearson's correlations may be an insensitive measure of the association. Instead, we might expect the two concepts to be related hierarchically. Cross-tabulations suggest that this is largely true in the general population: 76% report a low score on both measures, 20% a low score on drinking control loss but a high score on control worries, and 3% report a high score on both, while only 1% report the "deviating pattern" of high drinking control loss and low control worries. Among those entering treatment, the answer is more equivocal: the percentages are, respectively, 1%, 11% and 81% for the three "ordered" categories, but 7% for the "deviating pattern" -- not very much smaller than the 11% for the other cell of discordance. For a minority of those entering treatment, the experience of loss of control over drinking does not seem to presuppose experiencing a loss of control over one's life.

The second part of Table 4 shows that the respondent's score on control worries does to some extent interpret the relationship between heavy drinking and the experience of drinking control loss. In this mode of analysis, contol worries appear to play a stronger role in the general population sample than among treatment clients, specifying that frequent drinkers are much more likely to report loss of control over drinking if they have high general control worries.

Discussion

Assessing the results of our exploration of the "double loss of control" in two U.S. samples, then, we have found that those entering treatment differ greatly from the general population on both dimensions: not only are they much more likely to report indicators of losing control of their drinking; they are also much more likely to feel that their life as a whole is out of control. If we consider the two samples together, there is a clear and strong association of a high score on each of the dimensions. But the association appears to be largely specified by the situation of entering alcohol treatment: the within-sample association of the two dimensions is only moderate, particularly in the general population sample. While respondents show a strong commitment overall to an ideology of vigilant self-control (though interspersed with "time-outs"), commitment to this ideology has virtually no relationship with the experience of personal life-control loss or concerns. While the strongest predictor of the experience of losing control of one's drinking is frequent heavy drinking, the experience of life-control loss or concerns to some extent mediates or specifies this relationship.

The analysis results thus modestly support the usefulness of paying attention to the second

dimension of the experience of the "double loss of control" described in the First Step of Alcoholics Anonymous. Those entering treatment are set apart from members of the general population by high scores on both dimensions. But our analysis leaves some puzzles for further work. It is not always true that the experience of a generalized loss of control is a precondition of the experience of loss of control over drinking. That life-control loss may be so particularly associated with entering alcohol treatment suggests the therapeutic usefulness of a greater attention in research and in practice to the events and circumstances under which clients come to treatment (Weisner, 1987), and to their possible therapeutic indications.

The implicativeness of the study would be greatly increased if parallel studies were undertaken in other cultural contexts. Would as many as 90% of the respondents in other cultural contexts agree that "it is important to feel in control of yourself every waking moment"? If not, what are the implications for expectancies and attributions about drinking and drunkenness? Is life-control loss so closely associated with entry into alcohol treatment in other cultural settings — or is there less "identity stripping" in other cultures' handling of alcohol-related problems? Can we find cultures without a strong ideology of self-control, and does the experience of loss of control over drinking occur in such cultural situations?

At a more general level, perhaps it is time for cross-disciplinary dialogue and work on issues of control and self-control in alcohol and related studies. There can be few terms used in the alcohol field with more different denotations than "control" (Room, 1984, pp. 296-7). The denotations overlap without coinciding: "controlled drinking", "self-control", and "loss of control" all have something to do with each other, but the relationships are far from clear, and each phrase is a favourite with a different constituency. Each phrase covers a wide span of behaviour and cognition, as Orford's discussion of "change as self-control" (1985, pp. 285-291) elucidates. In this dialogue and work, there is a need to transcend not only the discipline-boundedness, but also the culture-boundedness, which has often been characteristic of previous work in the field.

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Pavlovian Conditioning and Environmental Control of Tolerance and Withdrawal Symptoms

Shepard Siegel
Department of Psychology
McMaster University
Canada

The major drug abuse treatment problem is relapse following completion of the withdrawal crisis. Understanding this problem requires an appreciation of the role of drug-predictive signals in dependence.

Environmental Cues and Withdrawal Distress

Both experimental studies (with animals) and clinical and epidemiological studies (with humans) provide evidence that drug-predictive signals contribute to withdrawal distress.

Animal Studies

Experiments by Hinson, Poulos, Thomas, and Cappell (1986) and Thompson and Ostlund (1965) were designed to evaluate the contribution of drug-associated cues to relapse to morphine self-administration in rats. In these experiments, rats were made dependent upon morphine, and then withdrawn from the drug. During a final readdiction phase, they were given the opportunity to consume a morphine solution. During readdiction, rats displayed greater avidity for the opiate when it was presented in the original addiction environment than when it was presented in an alternative environment.

Human Studies

Relapse in human addicts, like relapse in experimentally-addicted animals, is influenced by drug-associated cues. Several clinicians have noted that the physical environment in which drugs had previously been used frequently elicit withdrawal symptoms (and relapse to drug use) in the individuals who had been drug free for a considerable period of time (see Siegel, 1988). Not only is relapse in humans related to the presence of drug-associated cues, but successful abstinence is related to the absence of these cues.

Evidence in support of the salutary effect of protection from drug-associated cues is provided by follow-up studies of returning Vietnam veterans who were addicted to heroin while in Vietnam. It was expected that this new population of heroin addicts would substantially add to the indigenous civilian addict population. However, unlike most civilian addicts, these Vietnam addicts returned to an environment very different from that in which they used drugs. They also

evidenced much less relapse than civilian addicts (O'Brien, Nace, Mintz, Meyers, & Ream, 1980; Robins, Helzer, & Davis, 1975). Others have reported that environmental alteration favours long-term abstinence in treated civilian addict populations in studies conducted in Detroit, Michigan (Ross, 1973), San Antonio, Texas (Maddux & Desmond, 1982), and Sweden (Frykholm, 1979).

Environmental Cues, Pavlovian Conditioning, Drug Tolerance, and Drug Dependence

Findings that environmental cues importantly contribute to relapse are accommodated in a model of drug tolerance and dependence that emphasizes learning principles. The model will first be discussed with respect to tolerance, and then the relevance of this analysis of tolerance to dependence will be discussed.

Environmental-Specificity of Tolerance

Results of a number of studies indicate that tolerance is not the inevitable result of repeated drug administration. Rather, the drug-experienced organism may or may not display the hyporesponsivity to the drug that characterizes tolerance, depending on whether the drug is administered in the usual drug administration environment or an alternative environment.

Early studies of the environmental-specificity of tolerance demonstrated that rats displayed the expected analgesia-tolerant response to the last of a series of morphine injections only if this final injection occurred in the same environment as the prior injections in the series (e.g., Adams, Yeh, Woods, & Mitchell, 1969). Results of many subsequent experiments have confirmed and extended these observations, in several species (including both humans and snails), using a range of morphine doses and various procedural modifications (Siegel & MacRae, 1984). The environmental-specificity of tolerance has also been demonstrated with many nonopiate drugs: ethanol, pentobarbital, amphetamine, scopolomine, haloperidol, and a variety of benzodiazepines (see review by Siegel, in press). Such findings have inspired analyses of tolerance that emphasize Pavlovian conditioning principles.

Pavlovian Conditioning and Tolerance

Pharmacological conditional responses (CRs) may be seen by administering an inert substance in the presence of the usual drug-signaling cues. The form of the pharmacological CR depends very much on the nature and mechanism of the drug effect (see Eikelboom & Stewart, 1982). For many effects of many drugs, the CR is an anticipatory compensation: drug-associated environmental cues elicit responses that are opposite to the drug effect. For example, the subject with a history of ethanol administration (and its hypothermic consequences) displays a CR of hyperthermia (see Siegel, 1987). Similar drug compensatory CRs have been reported with respect to a variety of effects of morphine (analgesia, temperature, locomotor activity, and gastrointestinal transit time), as well as many other drugs (Siegel, in press; Siegel & MacRae, 1984).

Drug-compensatory CRs would be expected to attenuate the drug effect. As the association between the environmental CS and the pharmacological UCS is strengthened by repeated pairings, the effect of the drug becomes increasingly attenuated. Such a progressively diminished response to a drug over the course of repeated administrations defines tolerance.

Evidence for Conditioning Analysis of Tolerance

There is a considerable amount of evidence supporting the conditioning analysis of tolerance. Much of these findings have been reviewed elsewhere (Siegel, in press) and are only briefly summarized here.

Environmental Specificity of Tolerance . The observation that there often is pronounced environmental specificity of tolerance is readily interpretable by an analysis of tolerance that incorporates Pavlovian conditioning principles. If the repeatedly-drugged organism receives the drug in the context of the usual predrug cues, the compensatory CR partially cancels the drug effect, thus tolerance is observed. On the other hand, if this drug-experienced organism receives the drug in the context of cues not previously associated with the drug, there would be no pharmacological CR cancelling the drug effect, and the tolerance attributable to such a CR would not be observed.

Extinction of Tolerance. Following CR acquisition, presentation of the CS without the UCS causes a decrease in CR strength (i.e., "extinction"). If drug tolerance is partially mediated by drug-compensatory CRs, extinction of these CRs should attenuate tolerance. That is, established tolerance should be reversed by placebo administrations. Such extinction has been demonstrated with respect to tolerance to many effects of many drugs (see review by Siegel, in press).

Retardation of Tolerance. A variety of nonpharmacological procedures retard the acquisition of CRs. According to the conditioning interpretation of tolerance, similar procedures should retard the development of tolerance. One technique for attenuating the strength of an association is to repeatedly present the CS alone prior to pairing it with the UCS. The deleterious effect of such preconditioning exposure to the CS has been termed "latent inhibition" (Mackintosh, 1974). If drug tolerance is mediated, at least in part, by an association between predrug cues and the drug, it would be expected that rats with extensive experience with the administration cues prior to the time that these cues are paired with the drug should be relatively retarded in the acquisition of tolerance (compared to rats with minimal pre-exposure to these cues). This should occur despite the fact that the groups do not differ with respect to their histories of drug administration. Such latent inhibition of tolerance to several drugs has been reported (see Siegel, in press).

Another procedure for decreasing the strength of a CS-UCS association is partial (as compared to consistent) reinforcement. That is, if only a portion of the presentations of the CS are paired with the UCS, CR acquisition is retarded (compared to the situation in which all presentations of the CS are paired with the UCS; see Mackintosh, 1974). It would be expected, then, that a group in which only a portion of the presentations of the drug administration cues is actually followed by morphine (i.e., a partial reinforcement group) should be slower to acquire tolerance than a group that never has exposure to environmental cues signaling the drug without actually receiving the drug (i.e., a continuous reinforcement group), even when the two groups are equated with respect to all pharmacological parameters. Such a finding has been reported with respect to tolerance to the analgesic, thermic, and anorexigenic effects of morphine (Krank, Hinson, & Siegel, 1984; Siegel, 1977, 1978).

Other Evidence for the Conditioning Analysis of Tolerance. In addition to the research summarized above, results of many other experiments have provided further evidence that Pavlovian conditioning contributes to tolerance to many drugs. These experiments demonstrate

that nonpharmacological manipulations of predrug environmental cues affect both CR acquisition and tolerance in a similar manner. For example, tolerance to both morphine and pentobarbital is subject to inhibitory learning, sensory preconditioning, and disruption by an arbitrary novel stimulus. Furthermore, morphine tolerance can be manipulated by compound conditioning phenomena such as "blocking" and "overshadowing." A full discussion of these findings is beyond the scope of this review, but it should be emphasized that a variety of additional experiments support the conditioning analysis of tolerance (see Siegel, in press).

Pavlovian Conditioning and "Withdrawal Symptoms"

It is likely that drug-compensatory CRs contribute to withdrawal symptoms, especially those seen long after detoxification. That is, some drug "withdrawal symptoms" are, more acutely, drug "preparation symptoms":

> Consider the situation in which the addict expects a drug, but does not receive it; that is, no drug is available, but the addict is in an environment where he or she has frequently used drugs in the past, or it is the time of day when the drug is typically administered, or any of a variety of drug-associated stimuli occur. Research with animals demonstrates that presentation of cues previously associated with drug administration, but now not followed by the drug, results in the occurrence of drug-compensatory CRs... In the situation in which the drug addict expects but does not receive the drug, it would be expected that drug-compensatory CRs would also occur. These CRs normally counter the pharmacological disruption of functioning which occurs when the anticipated drug is administered. However, since the expected drug is not forthcoming, the CRs may achieve expression as overt physiological reactions, e.g., yawning, running nose, watery eyes, sweating... or form the basis for the subjective experience of withdrawal sickness and craving. (Hinson & Siegel, 1982, p.499).

Many investigators have noted that environmental cues affect the display of the symptoms of withdrawal from a variety of drugs.

Observations of Addicts. One way to evaluate the role of environmental cues in withdrawal distress is simply to ask addicts to recall the circumstances in which they suffer such distress. Several investigators have done just this, and have noted that both opiate addicts and alcoholics report that such distress is especially pronounced in the presence of drug-associated cues (see review by Siegel, 1988). Several clinicians have reported that opiate withdrawal symptoms are displayed when, during behavior therapy (even with long-detoxified former addicts), drugs are discussed or the paraphernalia of addiction (syringe and tourniquet) are viewed (see review by Siegel, 1988). Several anecdotal reports of environmentally-elicited withdrawal-symptoms and craving are provided by Biernacki (1986), Teasdale (1973), and Wikler (1977).

In the case of orally ingested drugs, such as alcohol and tobacco, an especially effective cue for the drug's systemic effects should be the flavor of the drug. It has been reported that cigarette smokers will display nicotine-withdrawal symptoms if they experience the taste of the cigarette without the usual accompanying nicotine administration (i.e., they puff on a cigarette containing much less than the usual amount of nicotine (Schachter, 1977)). It is well known that alcoholics find the taste of alcohol a potent elicitor of craving and have difficulty in refraining from drinking if they sample an alcoholic beverage. This "loss of control" is apparently elicited by the taste cue, since if the taste of the alcoholic beverage is masked, a sip does not elicit such craving (see review by Siegel, 1987).

Experiments Concerning Environmental-Elicitation of Withdrawal Distress. There are several laboratory demonstrations of the ability

of drug-associated cues to elicit withdrawal distress. For example, it has been noted that former addicts display physiological signs of narcotic withdrawal when they performed the "cooking up" ritual while being monitored by a polygraph (O'Brien, Testa, O'Brien, & Greenstein, 1976). Teasdale (1973) showed addicts slides of both opiate-related material (e.g., inserting a syringe into a vein) and nonopiate-related material (e.g., a hand holding a cup of coffee). On the basis of a variety of psychometric measures, Teasdale (1973) concluded that the opiate-related slides induced more emotional responding and evidence of withdrawal distress than the nondrug-related slides. Sideroff and Jarvik (1980) also reported that drug-associated cues elicited symptoms of withdrawal. They presented a videotape depicting scenes of heroin preparation and administration to groups of both heroin addict-patients and nonaddicts. They found that the videotape elicited evidence of withdrawal (changes in heart rate and galvanic skin response, and subjective ratings of anxiety and craving) in only the addict group.

Similar findings have been reported with respect to alcohol. Ludwig and colleagues (e.g., Ludwig, Wikler, & Stark, 1974) have presented results of experiments demonstrating that alcoholics, in the presence of laboratory reconstructed alcohol-associated cues (e.g., a mock barroom, or the odour of bourbon) display withdrawal sickness, subjective reports of alcohol craving, and (if liquor is available) relapse to drinking.

Implications for Treatment

According to the conditioning model, those drug-compensatory CRs which contribute to tolerance (when the anticipated drug is administered) contribute to withdrawal symptoms (when the anticipated drug is not administered). It follows that treatment techniques should address the crucial contribution of environment-drug associations to dependence (see Poulos, Hinson, & Siegel, 1981).

As described previously, when treatment consists primarily of a period of "detoxification" in an insulated treatment environment and the released patient is returned to the original addiction environment, treatment success is poor — the vast majority of the treated addicts quickly relapse following reexposure to predrug cues. The conditioning analysis suggests several factors which should be considered in a treatment program to minimize such relapse.

Environmental Change and Treatment Effectiveness

Transfer of an addict to an environment not associated with drug use should promote recovery. This is what happened with soldiers addicted while in Vietnam (e.g., Robins et al., 1975), and with experimentally addicted rats (e.g., Hinson et al., 1986; Thompson & Ostlund, 1965). As discussed in the beginning of this chapter, results of several epidemiological studies suggest that environmental change is frequently associated with long-lasting abstinence.

Of course, environmental change may be a good prescription, but it is not one that can readily be implemented. Since such changes usually do not occur, a function of treatment might be the extinction of the pharmacological associations that contribute to relapse.

Extinction of Responses to Drug-Associated Cues

The primary treatment implication of the conditioning analysis of withdrawal is that the usual predrug cues must be subjected to extinction. There are reports of the effectiveness of extinction-like procedures in eliminating the ability of predrug cues to elicit craving and withdrawal distress (see Siegel, 1988).

Summary and Conclusions

Results of much research demonstrate that tolerance is not the inevitable consequence of repeated drug exposure: often the drug-experienced organism demonstrates tolerance when the drug is administered in the context of the usual predrug cues, but not in the context of alternative cues. Such findings are incorporated in a model of tolerance that emphasizes the Pavlovian conditioning of an association between predrug cues and the systemic effect of the drug. Such an association results in drug-compensatory CRs. These pharmacological CRs may be displayed as "withdrawal symptoms" and craving when the organism with a history of drug administration is confronted with the usual predrug cues without the usual pharmacological consequences.

An implication of the conditioning analysis is that successful treatment of drug addiction should acknowledge not only pharmacodynamic and pharmacokinetic principles, but also the powerful evocative effects of drug-predictive environmental cues. Permanent abstinence is most likely if the treated addict is either protected from reexposure to these predrug cues or by a treatment protocol which incorporates extinction of the association between these cues and the drug.

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