Uses and Misuses of Epidemiology in Shaping and Assessing Drug Policy[1]

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Seven ways that epidemiology can be used in the analysis of drug policy are identified, as follows: to determine the causes of drug use and abuse; to monitor the levels of drug use and abuse in our communities; to identify trends over time in drug use and abuse; to complete the clinical picture of drug use and abuse; to identify syndromes of abuse; to identify target groups or individuals for preventive interventions; and to determine the effectiveness of interventions for the prevention or treatment of drug abuse. Common misuses of epidemiology are also identified.

I have argued previously that epidemiologic data should be the basic data by which we measure the impact of drug policies (Duncan, 1993). In this paper I will try to give a more extended discussion of the uses which should be made of epidemiologic data in the analysis of drug policies as well as a warning against some of the more common misuses of such data in the drug policy arena. Following in the footsteps of J. N. Morris (1955), I will describe seven uses of epidemiology:

First and foremost, *epidemiology is the science of causes*. There are numerous sciences which can contribute to our understanding of the causes of drug abuse but the ultimate test of any theory of drug abuse must be a test in a real human community—and the design and conduct of such

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tests is the specialty of epidemiology. No laboratory experiment or computer model can ever take the place of such a real world test.

Epidemiology has developed a more sophisticated concept of causation, models capable of reflecting the complexity of real causal relationships, and statistics specifically suited to the assessment of causality. Drug abuse research has increasingly adopted the epidemiologic strategy of studying risk factors in place of the search for Aristotlean causes, but the derived models are still very limited and the appropriate risk statistics are seldom used and poorly interpreted when they are used.

We need to be careful, however, not to confuse descriptive epidemiology of drug abuse with analytic epidemiology which can test causal hypotheses. Descriptive studies which find high rates of abuse in some populations or subpopulations and low rates in others can suggest hypotheses about causation. The accumulation of such findings lends support to a hypothesis but they cannot confirm it. Far too often, descriptive data is treated as if it was proof of a causal hypothesis. Only an experimental or cohort study which allows the calculation of relative and attributable risk can serve to confirm such a hypothesis. Proof, on the other hand, is a word little used in any science.

As Morris (1955, p. 399) stated, "the great advantage" of the epidemiologic approach to causal studies is "that it may be applicable in the early stages of our knowledge." We do not have to wait for our understanding of the causation of a drug problem to reach the stage of a fully-developed model before we can plan and implement effective prevention programming. Once we have determined that a particular variable is a risk factor for the problem, we can base preventive interventions on reducing that risk factor.

Second, epidemiology provides us with the means to monitor the levels of *drug use and abuse in our communities*. Such alternate measures as police or clinical statistics are likely to be misleading. Police statistics tell us more about the level of police effort and the priority given to drug law enforcement than they do about the levels of drug use or abuse in the community. Clinical statistics tell us something about how many abusers are seeking treatment, but even as a measure of desire for treatment they are distorted by the availability, accessibility and acceptability of treatment services in the community.

Epidemiology not only provides a measure of the levels of drug use and abuse in the community but can also provide a roadmap to guide us in navigating the drug problems of the community. This includes literal maps which can be constructed representing the varying prevalences or incidences of drug problems in the community—maps which can be valuable planning tools for locating services. Epidemiology can also tell us how drug

problems are distributed within any community over age groups, racial/ethnic groups or other social groupings of importance.

Third, by studying trends over time in drug use and abuse, we can predict the future of the drug scene and assess the impact of policy changes. Patterns are often seen to repeat themselves, with different drugs or in new populations, in ways that can be highly predictable. Past experience with policy impacts can be a valuable guide to future policy decisions.

Advocates of legalization of the currently illegal drugs, for instance, are often faced with questions about the impact that legalization would have on the prevalence of drug use. We cannot answer those questions with certainty but we can point to historical evidence of the kind I am referring to. We can point out that when a number of states decriminalized marijuana in the 1970's use of marijuana in those states did not increase any more than in neighboring states where marijuana possession continued to be a felony. That the only nation in Western Europe where hashish use has not increased since defacto decriminalization in the Netherlands is the Netherlands. That alcohol use in the United States did not increase during the first years after repeal of Prohibition.

Fourth, epidemiology can *complete the clinical picture* of drug use and abuse. This refers to the fact that clinicians who treat drug abusers inevitably gain a distorted view of drug use and abuse. For one thing, they obviously are exposed mainly to abusers rather than users and any generalization of their experiences to users is invalid. For another, most abusers never seek treatment (Anthony and Helzer, 1991) and those who do are not typical of the majority who don't. Finally, treatment settings place constraints on who they will serve which distort the kind of abuser they will see—if a treatment program requires that those they serve must admit that they are powerless to control their drug use, then all of the drug abusers seen at that program will self-report suffering from a loss of control. Epidemiology can give us a true picture of the condition by including the non-clinical majority along with the minority in treatment.

For example, let us take the case of phencyclidine. Originally introduced as a tranquilizer, under the brand name Sernyl^r, it soon reached the streets deceptively being sold as THC and continues to be sold as PCP or angel dust. About ten years after it became a common street drug the media suddenly became aware of PCP and it was soon labeled a "killer drug" and "the most dangerous drug on Earth." In the three trials of the Rodney King case, the defense argued that the police officers who beat Rodney King were justified in doing so because they believed he was under the influence of PCP and was therefore a deadly menace who could only be controlled in the way they did.

The clinical literature on PCP describes such effects as panic, paranoia, violence, respiratory distress, coma and death. You might wonder why anyone would want to take a drug that scared them into a panic, made it hard for them to breath and might kill them. The reality, of course, is that these are effects of PCP but they all are rare effects. They are the effects, however, that are most likely to bring a PCP user into an emergency room or crisis center and thus to become part of the clinical lore and literature. As to the super strength, uncontrollability and invulnerability to small calibre bullets, that is just plain urban myth.

Fifth, epidemiology will allow us to identify syndromes of *abuse*. Diagnostic classifications in the past have been based primarily on theory and on drug-specificity. The editors of DSM-IIIR have made a real effort to base their nosology on scientific, and especially epidemiologic, evidence and the forthcoming DSM-IV promises to be even more imperically-based.

In DSM-III (A.P.A., 1987) the category of Substance Use Disorders is divided between abuse and dependence—distinctions most of us are comfortable with but whose empirical reality has not been clearly established. DSM-III then divides the Substance Use Disorder along the lines of drug categories—alcohol, barbiturates, opiates, cocaine, amphetamines, phencyclidine, hallucinogens, cannabis, tobacco and "other, mixed, or unspecified"—despite the fact that most substance use and abuse is "mixed." In my experience, most contemporary heroin addicts use cocaine about as frequently as they do heroin. Abusers of cocaine are likely to use barbiturates or similar acting sedatives "to take the edge off" their habit.

Sixth, we can use epidemiologic data to identify target groups or individuals for preventive interventions. Groups which show the highest relative and attributable risk for onset of a problem are the appropriate targets for primary prevention programs. Too often target groups are chosen, instead, on the basis of political clout or of untested assumptions. It is for these reasons that we find primary prevention efforts targeted on populations with near zero incidence or populations where the prevalence has already reached a high level with few new cases occurring. I have seen such programs trying to prevent crack use among suburban adolescents or discouraging the onset of marijuana use among college students.

This sixth use presents a serious risk of misuse. Not only is the application of group probabilities to individuals a statistically questionable practice, but there are also ethical questions raised by such a practice. The risk of stigmatizing the individuals so targeted and thus creating a self-fulfilling hypothesis is a danger that must not be overlooked (Duncan and Gold, 1982, p. 185).

Seventh, epidemiologic methods permit us to determine the effectiveness of interventions for the prevention or treatment of drug abuse. While

progress has been made, and is being made, in this area, we still know very little about which treatments work for drug abuse and even less about which abusers they work best for.

Epidemiologic evidence clearly shows that most drug abusers recover without treatment (Anthony and Helzer, 1991). Unfortunately, most treatment research makes pre and post comparisons of patients or comparisons between groups who receive different treatments. What is needed if we are ever to have an answer to the question, "does treatment work?" are studies which compare treatment to no-treatment. The epidemiologic research design known as cohort studies provide our best prospect for resolving questions about the effectiveness of treatment while escaping the ethical concerns of an untreated control group in an experimental study.

MISUSES

If I were to ask you how many "crack babies" are born each year in the U.S., I am sure that the figure 375,000 would at least occur to most readers of this paper, whether you offered it up as an answer or not. William Bennett and other drug warriors have made much of this figure in justifying increased funding for their war. Newspaper columnists Jack Anderson and A. M. Rosenthal have written about the problem of 300,000 to 400,000 "crack-addicted" newborns annually. If this figure was accurate, then one in every ten newborns in America would have to be born to a crack-addicted mother. I hope it is obvious to all of you that this just isn't so.

The origin of this statistic is a 1987 survey of 36 hospitals by Ira Chasnoff (1989), founder/Director of the National Association for Perinatal Addiction Research and Education. He found reported rates of maternal exposure to drugs that ranged from 0.4% to 27%, with a mean of 11%. Despite the fact that the surveyed hospitals were a non-representative sample of U.S. hospitals, with major urban areas over-represented, Dr. Chasnoff applied that rate—rounded off to 10%—to the annual U.S. birth rate, thus concluding that 375,000 infants are born each year who had been exposed prenatally to some illicit drug—not just to crack. Not only is the number exposed to crack much smaller than the widely quoted figure, but the number negatively affected by maternal crack use is certainly very much smaller still. This is an example of two of the most common misuses of epidemiology—*generalizing from a nonrepresentative sample* and *measuring one thing and generalizing to another.*

The misuse of generalizing from nonrepresentative samples is made even worse, in many instances, when researchers generalize from clinical or jail samples. Vaillant (1987), for instance, cites unpublished (and pre-

sumably rounded-off) data on 19,000 consecutive admissions to an alcohol detoxification unit over six-and-one-half years time. Only 5,000 individuals accounted for the 19,000 admissions, with half having been admitted only once. On the other hand, 25 of the patients were admitted 50 to 200 times each during the study period. A researcher collecting 100 consecutive admissions would sample only 12 of the 50% of patients who never came back, but would also sample 12 of the 0.5% who averaged 100 admissions apiece. Thus the sample would greatly over-represent the most serious cases, even if we were only interested in persons who enter treatment.

The most common instance of measuring one thing and generalizing to another is *confusing the epidemiology of drug use with the epidemiology of drug abuse*. Prevalence of drug use is a meaningful measure for a policy which is predicated on the assumption that all use of certain specified drugs is harmful. The reality is that harmfulness cannot consistently be equated with any one drug. Citing Norman Zinberg (1984), Waldorf, Reinarman, and Murphy (1991, p. 37) note that drug-related harm "almost always depends on the user's psychological set, the social setting of use, as well as dose, chronicity of use, health of user, and a variety of other factors." The failure to differentiate between drug use and drug abuse was identified by Duncan and Gold (1981) as the critical flaw in most prevention efforts.

For drug use surveys to be useful in assessing the effectiveness of a harm reduction policy, they would have to be more focused on abusive, harmful use rather than use in general. The Epidemiologic Catchment Area Study (Anthony and Helzer, 1991) has demonstrated the practicality of studying the prevalence of substance abuse disorder in a community populations. Simpler epidemiologic surveys may be most effective if they measure the prevalence of specific drug-related harms or problems—such as driving under the influence or drug-related illnesses, as in Duncan and Martin (1987) or Duncan (1991).

Another common misuse is *reliance on retrospective reports* to study causal sequences. Human memory is not a completely reliable instrument. Self-reports of personal history must be carefully checked for reliability and validity before they are relied upon. Furthermore, we must be on guard against confounding of other variables with self-report of past experiences.

In recent years a growing number of prevention curricula for our schools have stated the objective of "delaying the onset" or "postponing the onset" of tobacco, alcohol or other drug use. Delaying or postponing, rather than "stopping" seems to be a surprising retreat in the war on drugs. These objectives are based on the hypothesis that the earlier young people begin using a drug, the more likely they are to become heavy users. This hypothesis has been supported by a number of surveys of alcohol use among public school (Barnes and Welte, 1986) and college (Friedman and

Humphrey, 1985; Gonzalez, 1983) students. These surveys had found that students who reported being younger when they first drank alcohol were more likely to currently be heavy drinkers. In violation of one of the most basic principles of research, a causal hypothesis is being taken as confirmed by evidence that is associational.

Worse still, the hypothesis is being confirmed through the association between current self-report and retrospective self-report. The vagaries of self-reported drug use are well known but those for memory of drug use are even greater. In a cohort study of a thousand children born in New Zealand in 1972 and 1973, Casswell et al. (1990), surveyed the children regarding their alcohol consumption at ages 9, 11, 13, and 15. They found that level of usage at any one age was only weakly associated with level of usage two years later. "Of those in the heaviest drinking top 10%, about one in five were still in the top 10% two years later." Four years later, only one in seven were. Even more important, the older the students became, the younger they reported themselves to have been when they began using alcohol.

The Casswell et al. study is one of a number which show that the older juvenile respondents are, the younger they report having been when they first began to drink. If older subjects report younger onset of drinking and if heavy drinking is more common among older adolescents, then heavy drinking will inevitably be associated with self-reported earlier initiation of alcohol use, for those reasons alone. The only longitudinal study of other drug use that I know of is an unpublished study conducted by the Irish Institute of Education in Limerick, which found that the younger children were when they first tried smoking tobacco, the less likely they were to become regular smokers.

These are not the only misuses of epidemiologic data but they are among the most major. Other common misuses might include drawing causal conclusions from prevalence statistics, assuming causation from association, and screening minority populations in the search for cases. So long as we remain alert to misuses, epidemiology can provide us with a powerful set of tools for the analysis of policy-relevant data.

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