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The Need for Dynamic Drug Policy

Jonathan P. Caulkins

Abstract:

Drug use in a population varies dramatically over time in no small measure due to nonlinear feedback among factors endogenous to the drug system. This suggests that drug policy ought likewise to be dynamic, varying the mix of strategies over time as drug use waxes and wanes. A growing literature that models drug "epidemics" mathematically supports this hypothesis and offers perspectives that may break policy logjams. For example, supply control may be most effective early, in the explosive growth stage of an epidemic. Conversely, treatment and measures to mitigate the consequences of dependent use and flagrant drug markets may have their comparative advantage later, in the endemic stage. Fully harnessing the power of dynamic drug policy will require more research and collection of new types of data, but the promise is worth the effort.

Keywords: Drug policy, epidemics, dynamic modelling, cycles

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Introduction

Drug researchers have long understood that there can be long-term waves of greater and lesser drug use (Musto, 1973) and that upswings can involve epidemic-like spread (Hunt and Chambers, 1976; Ferrence, 1994, 2001). These and other dynamics discussed below imply that policy ought to vary over the course of a drug use cycle, but drug policy debates have not yet internalized this perspective.

Variations in drug use and related problems can be both faster and greater in amplitude than are changes in risk factors such as the rates of poverty or single-parenting. Hence, although underlying "root causes" influence drug use, they cannot satisfactorily explain its dramatic variation. Much variation stems instead from feedback among factors that are internal to the drug "system". For example, new drugs can spread rapidly via a positive feedback loop generated by current users introducing the drug to their non-using friends. When use reaches critical levels, the markets supplying that use "tip" into more efficient and resilient forms (Tragler et al, 2001). Those denser markets achieve economies of scale that reduce prices (Caulkins, 1995; Jacobson, 2004). Lower prices in turn stimulate greater initiation and use (Chaloupka and Pacula, 2000; Grossman, 2004). Over time the ratio of longer-term users to recent initiates increases (Caulkins et al., 2004), which may lead the media and potential users to associate the drug with the problems of its chronic users. Musto (1973) and Kleiman (1992) describe and Behrens et al. (1999) model how this association can stifle initiation, which further skews the distribution of current users toward long-term users, triggering a negative feedback loop that can break an epidemic. Then, as users age, the nature of drug-related harms may shift away from violent crime and towards various health problems.

Since drug problems vary in these complex ways, it seems plausible that drug policy should vary over time as well. Yet it is rare to hear someone couch their drug policy recommendations in these terms. This dog not barking is striking and more than a bit troubling. It suggests that the mental models guiding policy discussions implicitly superimpose a static lens on an intrinsically dynamic phenomenon, akin to pop nostrums for get-rich-quick investing that never vary even as economic conditions change over the business cycle.

It is not clear why policy is not more often discussed in dynamic terms. Perhaps disciplinary boundaries and stove-piped bureaucracies create single-issue advocacy. Maybe both the health and criminal justice perspectives favour individual

level analyses. Whatever the reasons for their absence to date, dynamic perspectives on drug policy are in fact possible.

Models of Drug Epidemics

There is now a moderately large literature that applies dynamic systems methods to models of drug use. (See Caulkins (2005) for a review.) Even though there is no drug use pathogen, drug use can constructively be modelled as an epidemic in the same way that marketing models the spread of new product adoption as contagions (Bass, 1969; Mahajan et al., 2000). Marketers want to promote the spread of the latest gadget beyond the geek set. Policy makers want the opposite; they want to slow and contain the drugs' spread. Yet the underlying mathematics is the same in either case. Similar models have also been used to study the spread of fashions, fads, and even rumours (Noymer, 2001), which is a useful reminder that not all phenomena well-described by epidemic models are deadly threats that necessarily justify heroic control measures.

Not all epidemic models yield the same policy conclusions, and validation is a challenge. The unit of analysis is not the individual but the epidemic, meaning a drug-country or at least a drug-community combination. Hence, sample sizes are inherently limited, confounding variables are numerous, and randomized controlled trials are rare. Still, some general patterns merge.

First, most epidemic analyses credit drug control programs with a less central role in driving drug use and related problems than is implicit in many policy discussions. To caricature debates in Washington, DC, if use or some other drug indicator goes up, policy has failed. If use goes down, successful policy must be the reason. Such simplistic "score carding" overlooks the magnitude of natural or "uncontrolled" dynamic variation.

Second, most models agree that person to person spread ("viral marketing") is a key dynamic. Analyses that supplement purely epidemiological interactions with market models often suggest that market effects (e.g., economies of scale or learning by doing) and/or market-enforcement interactions (e.g., Kleiman's (1993) notion of enforcement swamping) are important accelerants. With or without explicit inclusion of market factors, the models identify relatively brief periods of explosive, nonlinear growth during which effects – both good and bad – can be amplified. Some models

suggest that if it were possible to go back in time and intervene in those critical years, the peaks in use could not only be delayed but also made noticeably less severe.

In technical terms these various models are consistent with rapid growth, overshoot, peak, and partial decline, some with and some without subsequent undershoot (i.e., with or without some degree of damped oscillation).

There are two primary schools of thought concerning the post-peak decline. One suggests that the peak is an ephemeral flash created by an initially large pool of susceptibles that the drug must more or less inevitably burn through. Most consume the drug only briefly, so use soon settles down among a small subset that progressed to compulsive use and among new birth cohorts just reaching the age when experimentation with drugs is common.

The second suggests that negative feedbacks from use to initiation emerge with a lag. These reduce the "infectivity" of current users even if the population susceptible to initiation is not truly exhausted. This could happen because the drug acquires a negative reputation as the (delayed) dangers of use manifest or because use is no longer concentrated among trend-setting ages, economic classes, or sub-cultures.

There is nothing incompatible about these two schools of thought. The real world may represent a blend, with the mix skewed toward limits on susceptibility for some drugs and populations (perhaps heroin in Italy, cf. Rossi 1999, 2001) and toward reputational feedback in others (e.g., US cocaine, cf. Behrens et al., 2000).

Policy Implications

Different analyses can reach different conclusions about the absolute efficiency of various interventions, but there is less disagreement about how the effectiveness of a given intervention varies over an epidemic. The majority of relevant models support the following broad generalizations at least for expensive, dependence-inducing drugs such as cocaine, heroin, and methamphetamine. (Similar models have not been analyzed for other drugs, including marijuana or LSD.)

Preventing an initiation in the early stages of an epidemic is tremendously valuable because it short circuits a chain reaction that would have involved many people (Winkler et al., 2004). (In technical terms, the reproductive rate at that point was large.) However, primary prevention cannot be timed to react to a burgeoning epidemic because of intrinsic lags (Caulkins et al., 2002). For example, the median age of cocaine initiation in the US is 21, but students in school-based prevention

programs are younger, often only 13. So if school-based prevention interventions were to have any hope of dramatically affecting cocaine initiation, the ideal time to have run them would have been in the early 1970s, 8 years before the peak in initiation. However, no one knew in 1970 that there was a cocaine epidemic brewing. Conversely, the vast majority of cocaine consumed from 1985 – 2005 was consumed by people who were already older than 13 in 1985. For instance, over 85% of people the Treatment Episode Data Set (TEDS) records as receiving treatment for cocaine between 1992 – 2003 were born before 1973. So prevention programs initiated around the time the cocaine epidemic became salient could not possibly have had a dramatic effect on use over the next generation, regardless of how effective they were.

Treatment also has limited ability to stave off a burgeoning epidemic because early in the epidemic, most users do not have a treatable medical condition. Precise estimates are not available because population-level estimates of treatment need exist only for recent years, but need for treatment is correlated with average duration of use. In 2003, 39% of respondents reporting past-year cocaine use to the US Household Survey had been using for 10 or more years. In 1979, the peak year for cocaine initiation, that proportion was just 3%. For drugs that are not injected, the role of harm reduction strategies is similarly limited when most users are not experiencing significant harms with their use.

Enforcement's effectiveness at suppressing drug use declines markedly as the size of a drug market grows (Kleiman, 1993; Tragler et al., 2001). However, enforcement has unique ability to focus its effects in both space and time. If a crack house opened next door, neither funding school-based prevention nor additional treatment slots would bring rapid relief. Parking a patrol car in front of the crack house would at least displace the activity. Similarly, assume treatment was five times more cost-effective than incarceration at reducing drug use. Incarceration could still be twice as cost-effective at reducing drug use this year – because incarceration's effects on drug use are concentrated in the present whereas treatment's effects may be spread over a decade or more (Rydell et al., 1996). Hence, these models suggest that supply control programs may have a unique capacity to disrupt contagious spread of a new drug, but limited ability to eradicate established markets. (Enforcement may also be able to displace established markets into less destructive forms, such as forcing visible street dealing to convert to covert beeper sales.)

Harm reduction offers particular advantages later in the epidemic cycle, when use has stabilized at high, endemic levels. For injectable drugs in countries with low violence and few street markets, harm reduction may focus on syringe exchange programs, safe injection rooms, and training ambulance crews to treat overdose (Ritter and Cameron, 2005). For drugs that are not injected and which are supplied through violent street markets, harm reduction may focus instead on using enforcement to target the minority of dealers who cause the greatest social harm (Caulkins, 2002). In either case the premise is that with or without the harm reduction, the flow of new people into problem drug use will be modest, so reducing harmfulness of drug use has few drawbacks (MacCoun, et al., 1996; MacCoun, 1998; Stratton et al., 2001). That may not be a safe premise early in an epidemic, when there are feedbacks that can amplify small shocks to the system into dramatic effects on its trajectory.

Even if one accepts these generalizations, there is great need for more research on various topics, including interactions among epidemics for different drugs, interactions among markets supporting drugs at different stages in the epidemic cycle, and interactions between drug policy and other policy domains. We also need more quantitative histories of epidemics of use of various drugs in various countries.

Practical Considerations

Although much work remains to be done, there is cause for optimism that adopting a dynamic perspective on drug policy will bear fruit. Among other things, it can bring people together, whereas static framings are often divisive, pitting special interest groups against each other. With most dynamic models studied to date, essentially every mode of intervention has a valuable role to play at one point in the drug use cycle or another. Treatment providers have a lead role late in an epidemic, when the focus is on consequences not prevalence. Enforcement officials can take heart in their unique capacity to effect change early on, during the crucial early stages. Some are also delighted to be absolved of responsibility for stamping out the use of established drugs, freeing them to focus on particularly noxious dealers without being crushed under a perceived obligation to process vast numbers of low-level dealers. Prevention proponents can gain from standing back from tactical issues of timing particular drug epidemics and instead be viewed as background investment in the

human capital of our youth, as well as a form of cheap insurance against the worst ravages of a possible but as of yet undetected future epidemic.

Implementing dynamic policy does pose challenges not presented by static policies that offer budgetary stability or, better yet, scaling up all efforts proportionately. Since numbers of dependent users rise and ebb over years not months, the problem would be most acute for law enforcement. Fortunately, most drug enforcement is not done by agencies that specialize only in drug enforcement, but rather by general purpose law enforcement agencies, so increasing and reducing drug enforcement means shifting detectives back and forth between functional areas (e.g., from working crimes against property to drug cases and back), not hiring and firing people. In single purpose agencies such as the U.S. Drug Enforcement Administration, the dynamics might for practical reasons have to be shifting a stable pool of agents from focusing on one drug to another. That still poses challenges. Process productivity measures such as arrests made per agent per year could be hurt by focusing on emerging rather than established markets, but such challenges are not in some sense unique to dynamic drug policy. They are raised by many tough-minded reforms that seek to allocate resources based on measures of bottom-line effectiveness.

In sum, adopting a dynamic perspective on macro-level drug policy may be practically challenging but politically useful, as well as being intellectually compelling.

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