Insights

Eugene Bardach
Editor

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NOTICING THE MICRO-DISTRIBUTIONAL CONSEQUENCES OF CIGARETTE TAXATION AND ITS EQUIVALENTS

Mark A.R. Kleiman and Jonathan P. Caulkins

INTRODUCTION

While higher cigarette taxes (or the equivalent in forward-looking per-pack charges levied on cigarette producers through litigation) protect the public health by reducing smoking, they also impose financial burdens on those who continue to smoke. These burdens fall especially hard on low-income, heavily addicted, elderly smokers. Additionally, since the gains from not smoking decrease, and the difficulty of quitting increases, with age and years of smoking, the socially optimal price of a pack of cigarettes is not constant for all consumers. It would be possible to design policies to take at least some of these factors into account and thus mitigate the impact of higher prices on poor and elderly nicotine addicts, but there seems to be no sentiment for adopting them. In this essay we describe rough versions of such policies and then speculate on why there has been, and likely will continue to be, so little support for them.

WHO PAYS?

Both specific excise taxes on cigarettes and the recent settlement of tobacco liability litigation have the effect of raising the retail price of cigarettes. Any excise tax tends

to increase price; that the same is true of the settlement is the result of the details of the payment structure, which is forward-looking rather than backward-looking and thus results in a fixed additional cost to the companies per pack sold. A purely backward-looking settlement, one based on past rather than future sales, would come instead largely at the expense of shareholders and employees (Kleiman and Caulkins, 1997).

This reality is inconsistent with the script of the morality play in which the evil tobacco companies have at last been forced to pay for their nefarious deeds. Although higher prices may inflict some damage on the industry through lower sales, the hundreds of billions of dollars flowing to government coffers from the tobacco settlement and tobacco tax increases come overwhelmingly from the pockets of consumers, not producers.

THE SIMPLE WELFARE ECONOMICS OF TOBACCO TAXATION

Any excise tax distorts consumer choice by creating a wedge between resource costs and prices. In general, such a distortion is welfare-reducing. The exceptions are where the activity in question generates external costs in either production or consumption, in which case the excise can be seen as fixing a market failure (Pigouvian taxation), or where consumer choice itself is somehow distorted.

Despite all the fuss about "second-hand smoke," the main beneficiaries of increased cigarette taxation are those whose smoking behavior is changed by it. Price increases resulting from the settlement are predicted to save an aggregate of 250,000 life-years per year in the short run, with the gains escalating to 1 million life-years per year in the long run; the estimate of quality-adjusted life years, factoring in reductions in morbidity, would be substantially higher (Boyum, 1997).

This does not, by itself, suffice to show that the price increase represents a net improvement in social welfare. The gains in health status, savings in medical costs, avoidance of grief among smokers' intimates, etc. must be balanced against the pleasure, anxiety relief, and gains in capacity for work and endurance forgone along with cigarettes and against the unpleasantness of quitting for those whom higher prices have induced to quit smoking.

However, a simple thought-experiment suffices to show that, in fact, smoking as a whole generates net consumers' deficits. Using conventional valuations for lost life-years and estimates of the life-years lost as a result of smoking, one can calculate that the life expectancy lost as the result of smoking a pack of cigarettes would be valued by the average consumer at about $10 (Kleiman and Caulkins, 1997). Now imagine a health-risk-free cigarette, otherwise identical to the current product. It hardly seems plausible that many smokers would be willing to pay $12 per pack for the hypothetical healthy smokes. If they would not, then smoking is not a net beneficial activity from the viewpoint of (most) smokers, and we need theories of imperfect rationality (prospect theory, hyperbolic discounting, social influence, or addiction) to explain why actual behavior diverges from rational behavior.

THE MICRO-DISTRIBUTIONAL PROBLEM

While those induced to quit by higher prices at least enjoy some health benefits, those who neither quit nor substantially reduce their smoking are unambiguously damaged. All that happens to them is that they shell out more money for the same level of cigarette consumption.

The sums involved are not trivial, especially for those of limited means. For a pack-and-a-half-per-day smoker—roughly the median smoker—the calculated settlement-

induced price increase of $0.60 per pack multiplies out to $328.50 per year. (The Supplemental Security Income minimum payment is $6144.)

This problem of the specific impoverishment of some current smokers is conceptually quite distinct from the issue of regressivity. A regressive tax disadvantages the poor by comparison with the wealthy. Cigarette taxation benefits non-smokers at the expense of smokers, for some of whom the added burden is substantial.

It is also the case that cigarette excises are regressive because smoking is concentrated toward the bottom of the income scale. However, since such taxes constitute only about 1 percent of total governmental revenues and expenditures, that regressivity could easily be offset by changes in a progressive direction either in revenue-raising (e.g., reducing the FICA rate while raising the annual contribution cap or raising the zero-tax-bracket level) or expenditure (e.g., expanding the EITC). But, since most people do not smoke, policies that are blind to smoking status are blunt instruments for undoing the specific distributional effects of cigarette price increases on poor habitual smokers.

Solving the Distributional Problem with a Lump-Sum Transfer

This distributional problem could be largely solved by combining a tax with a lump-sum transfer. Take the present value of the average payment stream to the public from the settlement—or from a tobacco excise tax increase—and distribute it as a lump sum to current smokers in proportion to their expected subsequent consumption. (Presumably that means as a decreasing function of age, since younger smokers will on average purchase more packs at future, tax- or settlement-enhanced prices than will older smokers.) That would leave in place the financial incentive to quit provided by higher prices, while on average fully offsetting the effect on smokers' purchasing power. Those who reduce their lifetime cigarette purchases more than average would come out somewhat ahead, while those whose reductions are zero or smaller than average would come out somewhat behind. The big losers would be those who started to smoke after the date of the price increase and rebate eligibility cutoff, but they would have no just complaint that they had been taken by surprise. A different argument—that policies should not explicitly reward law-breaking behavior—would suggest limiting eligibility for rebates to those currently of legal age to buy cigarettes.

Since smokers are already overtaxed in Pigouvian terms (Manning et al., 1989) (ignoring intra-family effects), the fact that a tax increase offset by such a lump-sum rebate would impose no new net fiscal burden on smokers constitutes an advantage, not an objection, except from the viewpoint of non-smoking taxpayers and budget-makers. Since it is the effect of tobacco taxes on the poorest smokers that is most troublesome, a possible compromise would phase the rebate down, and eventually out, with rising income.

The greatest administrative difficulty would be in defining and verifying the status of tens of millions of individuals as "current smokers," with sums at stake sufficient to motivate attempts by non-smokers to fraudulently represent themselves as smokers. The simplest proof for most people would be documenting that they had been paying smokers' rates for life insurance. (Likewise, the simplest disincentive to fraud would be making information about self-reported smoking status available to insurance companies.)

Those who had not declared their smoking status in buying life insurance would presumably need certification from a physician. For those with an existing relationship with a primary care provider, this determination could be made by consulting medical records. The cost of documenting one's smoking status would be greatest for those who have neither health nor life insurance, but the amounts at stake are great enough

(the present value of $328.50 per year over the expected number of future years of smoking) to justify the costs of the relevant medical tests even in that case. Likewise, the amounts in question are so large that setting 1 percent of the funds aside for auditing would be enough to hire one auditor for every 20 physicians.[1] That level of scrutiny, coupled with the high cost of losing one's medical license for fraudulent practice, ought to be sufficient to keep fraud associated with such rebates down to levels well below those tolerated in connection with other tax and benefit programs.

The Case for Price Discrimination

A lump-sum transfer solves the distributional problem without reducing the disincentive for cigarette consumption, because consumers still face higher prices. It thus protects the public health as well as taxation alone, without further impoverishing already-poor smokers. However, since the optimal price of cigarettes is not the same for every smoker, the uniform price increase created by taxation or its equivalent does not achieve a welfare maximum.

While it is possible that even those forced to quit by higher prices will, on average and on balance, come out ahead, it remains the case that their costs of not smoking are higher and their gains from not smoking are lower than for those who never started. Moreover, the benefits of quitting tend to decrease with age, due both to the partial irreversibility of the damage from smoking and the shorter life expectancy over which the benefits of quitting have to act. (In the extreme, those with terminal illness have little to gain from quitting.) This suggests that the socially optimal price of a pack of cigarettes is not a constant across the population, but rather varies with current smoking status and with age (cf. Moore, 1973).

Differential pricing could be achieved by distributing coupons representing different levels of tax exemption to existing smokers, the level of exemption rising with age (and perhaps falling with income). The problems here are largely practical rather than conceptual. Determination of smoking status would have to be made on an ongoing basis, not just once. Moreover, either the exemption certificates or the cigarettes bought with them would become potential items of illicit commerce, especially since determining not merely who smokes but how much would probably be technically infeasible, requiring a uniform and therefore somewhat inaccurate issue of exemption coupons. Thus there would inevitably be persons holding certificates for more packs than they had any intention of smoking, each of them a potential source of cut-rate cigarettes to current non-smokers. The same would be true of those who quit smoking, who would have their entire supply of discounted cigarettes available for illicit resale.

Thus a price-discriminating tax may be infeasible in practice.

Why the Silence?

Price-discriminating taxation is attractive in principle but arguably unachievable in practice. Lump-sum payments, however, are an entirely feasible and useful response to the distributional problem. Why hasn't that option been pursued? Why, indeed, has the distributional issue been largely ignored?

The answer may lie in the politics of the problem. It is perhaps not surprising that the settlement ends up punishing the victims of the tobacco companies' actions, since the victims were not invited to the bargaining table. The three parties who were at

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[1]More precisely, 1 percent of the funds could pay the salary of one full-time auditor for each 20 physicians for one year. One year is presumably a sufficiently long "enrollment period" for this transfer program.

the table—the states, the tobacco companies, and the public health community—all got most of what they wanted.

The states got a budgetary windfall without having to appear to raise taxes (because most voters seem to believe that the money comes from the tobacco companies, which are seen as paying a penalty for past misconduct rather than a tax). In reality, the tobacco companies dodged (most of) that bullet, so they are happy with a settlement that immunizes them from lawsuits that could have hurt stockholders rather than smokers. Public health advocates want to stop smoking, seemingly regardless of the fiscal or other costs to smokers. They are pleased with the settlement because the resulting price increases reduce smoking and smoking-related deaths. As long as fewer smokers are dying of cigarette-related disease, the public health community has not complained about the remaining smokers' becoming somewhat poorer. (By contrast, in other contexts many within the public health field argue that poverty itself is a major public health problem and therefore count a less unequal distribution of income among their professional objectives.)

Both in the political process that ratified the negotiated settlement and in the more routine political processes that deliberate proposed increases in cigarette excise taxes, the group of losers—smokers—remains largely unorganized and somewhat stigmatized. Any attempt to mitigate their losses would come at the expense of other taxpayers or the beneficiaries of spending programs.

The lack of organization among smokers does not result from any neglect on the part of the cigarette industry, which has made vigorous efforts to organize "smokers' rights" groups: an instance of what has come to be called "Astroturf" lobbying (a reference to artificial grass roots). The industry's failure in this regard is instructive. While it is true that smokers as a group are being treated shabbily by their government, it is not the case that they regard their cigarette suppliers as their natural protectors. Surveys indicate that as many as 90 percent of them would like to quit smoking, and, at least in retrospect, regret having started. That makes them less willing to become political foot-soldiers in an industry-organized campaign.

There is a sharp contrast here with the case of alcohol. The costs of drinking, to drinkers and others, are comparable to those of smoking, and alcohol is, in Pigouvian terms, grossly under-taxed (Kleiman, 1992; Manning et al., 1989). But the costs of drinking are concentrated among a minority of problem (or unlucky) drinkers and third-party victims of crimes and accidents, leaving a very large group of satisfied customers. As a result, the beer industry has been far more successful in organizing its customer base to help resist tax increases, and otherwise protect the industry from the state, than has the tobacco industry. (The gun industry has been the most successful of all in this regard.)

This analysis, however, leaves to be explained why the concentrated costs imposed by tobacco taxation on poor smokers have not generated more concern among those ordinarily worried about distributional issues. Contrast the silence about smokers with the uproar of concern about the plight of poor single mothers (another largely unorganized group) under welfare "reform."

The explanation may be that no one really has much sympathy for smokers as such. Either they are regarded as having made a voluntary choice whose consequences they should be prepared to accept, or they are thought of as a species of drug addict. Two decades of concentrated national effort around stigmatizing drug users may have had some spillover effect on attitudes toward those trapped in the use of a legal, non-intoxicating substance. It is also possible that years of resenting second-hand smoke has made non-smokers resent smokers as well. Whatever the explanation, smokers' decisions to damage their own health are widely seen as adequate justification for making them pay what is, in effect, a punitive tax.

The result is a policy very hard to defend in either ethical or policy-analytic terms. Noticing that fact logically precedes inventing ways to amend it.

MARK A. R. KLEIMAN is Professor of Policy Studies at the University of California, Los Angeles.

JONATHAN P. CAULKINS is Professor of Operations Research and Public Policy at Carnegie Mellon University's Heinz School of Public Policy and Management and Director of RAND's Pittsburgh office.

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CAN WE PROTECT DRUG USERS FROM HEPATITIS C?

Harold A. Pollack

INTRODUCTION

Syringe exchange programs (SEP) are among the most prominent and controversial interventions to prevent blood-borne disease among injection drug users (IDUs). Many studies establish that SEP is effective for HIV prevention. Yet SEP appears strikingly ineffective in preventing hepatitis C (HCV), the other prominent epidemic among IDUs.

The mixed success of SEP is important for public health policy. The story is also of broader interest because it illustrates two insights that apply to other policy domains.

First, SEP provides a useful warning against sweeping defense or condemnation of generic policy approaches based upon the limited history of a single intervention.

In this respect, SEP bears some similarity to many economic policy debates in which formal or informal models play a large role. Is privatization superior to public production? Rather than answer this question in the abstract, investigators such as John Donahue offer particular analysis to identify the proper domain of application for this plausible but generic strategy (Donahue, 1989). Privatization improves production efficiency when public decision makers can easily monitor private

performance, when there is a competitive market to supply the desired services, and when the improved efficiency of private production outweighs the transaction costs required to establish and maintain such contractual arrangements.

Second, tractable analytic models can provide useful insight regarding the environments, outcomes, and populations for which a generic strategy is most likely to prove effective. What is the likely effect of tobacco excise taxes on lung cancer mortality? Given standard life tables that show lung cancer mortality rates among smokers, non-smokers, and ex-smokers, a simple economic model indicates that the price-elasticity of demand is the critical, often unknown, parameter that distinguishes the result of this intervention across populations.

In the case of SEP, analytic models of disease transmission are less familiar, but prove equally helpful in identifying critical factors. When such causal models are available, policy analysts who are prepared to use them can make an important contribution, often by deflating the overly generalized skepticism or enthusiasm that surrounds many controversial interventions.

SYRINGE EXCHANGE

Policymakers and clinicians have struggled to control infectious disease among IDUs. Existing U.S. policies have yielded, at best, mixed results. High HIV prevalence in many northeastern cities stands in marked contrast to much of Australia and Western Europe where HIV is well-controlled among IDUs (Des Jarlais et al., 1995; IOM, 2000).

One cause of this policy failure is federal policymakers' continued reticence to embrace effective programs that provide IDUs with sterile injection equipment. Although SEP has not been scrutinized through prospective randomized trials, an impressive body of evidence supports the effectiveness of these interventions (DHHS, 1998; IOM, 1995, 2000). Edward Kaplan and Robert Heimer's "circulation model" provides the most widely cited and ingenious analytic argument that SEP reduces the rate of new HIV infections (Kaplan, 1995). Linking a formal model of short-term changes in the rate of new infections to lab data from distributed needles, these authors estimated that the New Haven SEP reduced HIV incidence by one-third.

SEP has attracted vigorous, sometimes bitter, debate, particularly over whether SEP implicitly condones drug use. Yet the utility of SEP in addressing new public health problems has received less careful attention. This gap in policy discourse becomes especially pronounced when one considers the poor track record of SEP and other efforts in preventing hepatitis C.

Like HIV, HCV is a blood-borne virus spread through contaminated injection equipment among IDUs (Crofts et al., 1994). Also like HIV, HCV is a slow-acting, chronic condition that had gone largely undetected until it was widespread among IDUs.

Unlike HIV, HCV is highly infectious and is efficiently transmitted through limited or a single exposure (CDC, 1998). HCV is now endemic among IDUs. HCV-caused hepatic failure among IDUs is the most prevalent diagnosis requiring liver transplantation in the United States. Measured HCV prevalence exceeds 85 percent among Baltimore IDUs, with a reported incidence of 0.16 infections per person per year among young susceptible IDUs (Garfein et al., 1996, 1998). Recent data from Tacoma, Washington indicate 86 percent prevalence, and even higher rates of new infections (Hagan et al., 1999).

Although some analysts express hope that SEP might slow HCV spread (Hagan et al., 1995; Lurie et al., 1993), no prevention intervention has been consistently documented to accomplish this task (Coutinho, 1998). Most discouraging, many

populations exhibit high prevalence despite well-implemented prevention interventions. In Victoria, Australia, 4.5 percent of IDUs tested positive for HIV-while 68 percent tested positive for HCV (Crofts et al., 1994). Hagan et al. (1995), authors of the most hopeful earlier study, report no SEP protective effects in a later cohort study (Hagan et al., 1999).

EPIDEMIOLOGICAL MODEL

Why is HCV so difficult to control? A bare-bones transmission model illustrates the fundamental prevention challenge in high-risk populations.

Disease transmission is shaped by several biological and social processes that policy intervention might change. The mixing patterns through which infected IDUs share needles with susceptible individuals will strongly influence disease spread. Biological properties of the virus and of susceptible and infected persons also matter. These factors—along with specific behaviors when needles are shared—influence the probability of actual transmission after a person has been exposed. The duration of active drug use among infected and susceptible IDUs is another important variable.

A growing literature examines these factors within the population of IDUs. Random-mixing provides the workhorse model for HIV prevention policy analysis (Kaplan, 1995) and so provides a revealing framework to compare the two diseases. When IDUs randomly share needles, the probability that a susceptible person will share needles with an HCV-infected IDU is the same as overall HCV prevalence within the drug-using population. In the most graphic form, random-mixing might occur in a shooting gallery where an IDU obtains an unsterilized syringe from a stranger, injects himself, and then returns the syringe for use by the next IDU.

This model has clear limitations. More careful analysis of heterogeneity offers more hopeful results regarding low-risk IDUs, and indicates that random-mixing fails to capture the dynamics of low-risk groups. Unfortunately, a burgeoning epidemiological literature suggests that random-mixing provides an all-too-accurate description of disease spread through frequent high-risk behavior (Kaplan and Lee, 1990).

More formally, susceptible IDUs have constant probability κ of contracting HCV from a single exposure. An IDU shares needles with some other (randomly chosen) person λ times per week, and IDUs have drug use "careers" that last an average of D weeks.

Given these assumptions, disease prevalence over time follows a classic logistic curve. The rate of new infections initially rises, as infected persons share with many susceptible persons and as few infected IDUs die, quit injecting, or otherwise exit the active population. Over time, however, the susceptible pool will shrink, and the rate of new infections will slow. Eventually, prevalence will level off at some steady-state value π^* (possibly zero). At this prevalence, the number of infected IDUs who leave the active drug-using population exactly balances the number of new infections. Put differently, each infected person creates, on average, exactly one new infection before she leaves the IDU population.

When does this happen? If some fraction π of the population is currently infected, random-mixing implies that an infected person will, on average, infect some fraction $\kappa[1-\pi]$ per needle-sharing episode and, on average, $\kappa\lambda[1-\pi]$ other people every week. She will infect an average of $D\lambda\kappa[1-\pi]$ other IDUs over her entire drug-using career before exiting the population.

When $D\kappa\lambda<1$, there is no epidemic. When $D\kappa\lambda$ exceeds 1, prevalence will change over time until, on average, each infected person creates exactly one new infection before leaving the population. If there is a positive steady-state, then, $D\kappa\lambda[1-\pi^*]=1$. Thus:

$$\pi^* = \max \{0, 1 - \frac{1}{D\kappa\lambda} \} \tag{1}$$

In this framework, SEP prevents disease spread by effectively reducing infectivity κ to some fraction [1-e] κ. (To highlight harm reduction, we presume that the SEP does not alter the frequency or duration of injection drug use.) For a slowly changing epidemic, SEP has little short-term effect on disease prevalence. Thus, if current prevalence is some level π, the immediate effect of SEP is that each infected IDU will infect only [1-e] κλ[1-π] others every week. Short-term analysis such as the circulation model captures precisely this effect.

These analyses do not consider what happens later, as prevalence approaches steady-state. Applying the same calculations that produced Equation 1, SEP reduces steady-state prevalence to

$$\max[0, 1 - \frac{1}{[1 - e]D\kappa\lambda}] = \max[0, \frac{\pi^* - e}{1 - e}] \qquad (2)$$

Combining Equations 1 and 2, the SEP-related decline in steady-state prevalence is given by

$$\Delta\pi^* = \frac{e(1 - \pi^*)}{1 - e} \qquad (3)$$

A sufficiently effective SEP (e > π*) will eradicate the disease. Unfortunately, SEP has little long-term effect when π* is close to 1. For highly infectious agents, SEP reduces the immediate rate of new infections, but has little long-run effect; SEP delays, but rarely prevents, blood-borne infection.

The basic problem is that steady-state disease prevalence is a strongly concave function of the transmission probability κ. Because of this concavity, modest interventions that bring small reductions in infectivity are highly effective against hard-to-transmit diseases such as HIV. Steady-state prevalence is far less responsive to the same intervention given a more-infectious agent.

Figure 1 illustrates these effects based on New Haven data. Other SEP, including those in San Francisco and Tacoma, distribute twice as many syringes per local IDU and so may offer superior best-practice models (Lurie et al., 1993). New Haven nonetheless provides a well-evaluated baseline to explore how a reasonable SEP might reduce disease spread. The straight line shows the estimated post-SEP prevalence in steady-state, plotted against π*, what prevalence would have been in the absence of SEP. Kaplan and Heimer estimate an approxamite value of e = 1/3, which is drawn in the figure.

In the arena of HIV prevention, Figure 1 replicates Kaplan and Heimer's finding that SEP can significantly reduce disease spread. Pre-SEP, approximately 65 percent of New Haven IDUs were HIV-infected (Kaplan, 1995). At this value of π*, reducing short-term incidence by one-third is predicted to reduce steady-state prevalence by almost 20 percentage points, to 47.5 percent.

The same graph yields much more discouraging results as one moves into the plausible prevalence range for HCV. Measured HCV prevalence is 85 percent among Baltimore and Tacoma IDUs. Setting π*=0.85, reducing short-term incidence by one-third reduces steady-state prevalence by less than one-tenth, to 78 percent. If, as often presumed, SEP serve high-risk subgroups (Hagan et al., 1999), the predicted prevalence reduction is likely to be even smaller.

DISCUSSION

In a politicized debate, one is tempted to offer sweeping defense or condemnation of SEP. The wiser course is to evaluate SEP's ability to improve specific outcomes in specific settings for particular groups of IDUs. It then becomes apparent that SEP's
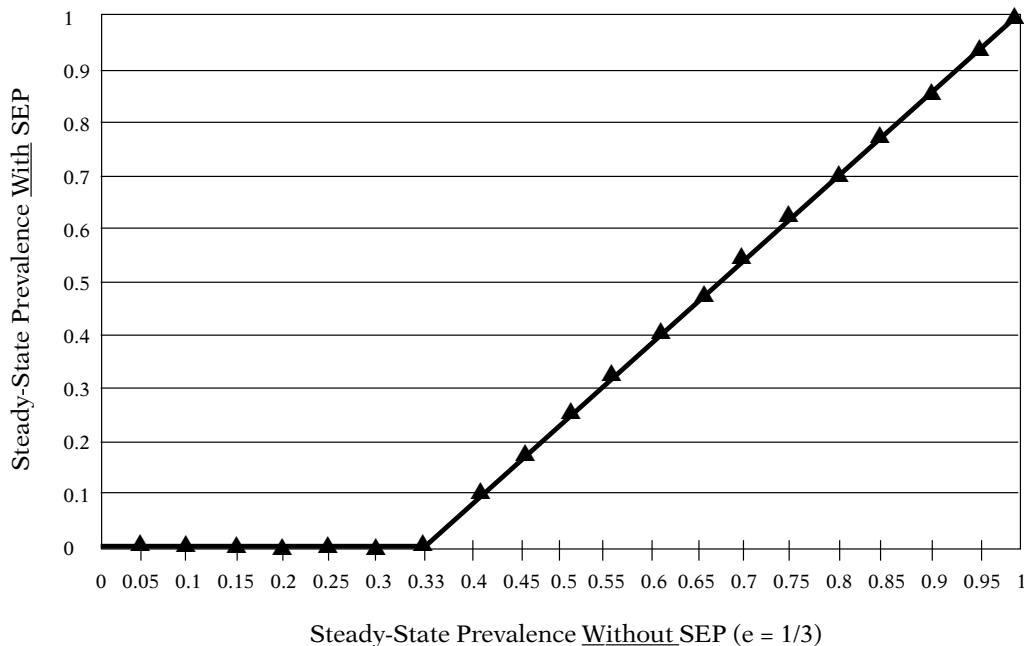
Figure 1. Impact of Syringe Exchange Programs (SEP) on Steady-State Disease Prevalence.

success in HIV prevention partly reflects unusual opportunities afforded by a difficult-to-transmit infectious agent. The same analysis shows how a simple but explicit model can expose, and therefore correct, implicit assumptions behind unguided policy intuition.

Some stated assumptions of these models highlight important limitations of the random-mixing model. Indeed these limitations themselves may highlight possible avenues for intervention. For example, one might assume that interventions aimed at prevention are most effective when designed to help the IDUs at greatest risk. Because, however, HCV appears so difficult to control among high-risk IDUs, an expanded model of behavioral heterogeneity suggests that low-risk subgroups might actually derive greater benefit from targeted intervention.

The current model does not explore how long it takes to approach steady-state prevalence. Short-run analysis is often appropriate to control slowly changing epidemics, especially when interventions have uncertain long-term effects. (Full analysis of transient effects is beyond the current paper.) Consistent with the observed high incidence among young IDUs, mathematical simulations indicate rapid convergence to steady-state.

Notwithstanding these technical concerns, the present results tell a discomfortingly simple story that matches available data (Hagan et al., 1999). Ironically, present findings indicate that HIV, though deadly, is more responsive to modest prevention interventions than are other prevalent threats to IDUs. These interventions work

because HIV is near an epidemiological "tipping point" where small behavior changes can have large population effects.

The search for accessible tipping points becomes less helpful when one confronts more infectious ailments. To be either effective or cost-effective in preventing HCV, prevention interventions must achieve high compliance by active IDUs. Given a baseline prevalence of 85 percent, an SEP that reduces needle-sharing by one-third reduces steady-state prevalence by only 7 percentage points. An SEP that reduced needle-sharing by twice as much would bring more than four times the predicted reduction in HCV prevalence.

Experience suggests that the most promising strategy to create such effective programs is to improve and expand current programs that already serve the IDUs at greatest risk of disease. Some SEPs confront this challenge by establishing linkages with drug treatment, prevention case management, and other wrap-around services that go beyond syringe distribution (Heimer, 1998; Thomson et al., 1998). Strengthening these linkages would also remind skeptical citizens that SEP seek to reduce and, ultimately, to halt clients' injection drug use. Unfortunately, the continuing ban on federal funding for SEP undermines precisely the kind of evaluation needed to design enhanced interventions.

Can we successfully control HCV? No one really knows. Like many other public health researchers, I believe that a combination of SEP and related services offers the most promising and humane, if easily oversold, strategy to help fellow citizens who abuse illicit drugs. Promising models have been proposed within the correctional system, drug treatment, and other pertinent settings. Experience with hepatitis B suggests that such models would become especially critical if an effective HCV vaccine became available.

Analytic models leave open the possibility that well-implemented interventions may still fail. It is heartbreaking that elected policymakers appear so unwilling to run this test.

HAROLD POLLACK is Assistant Professor of Health Management and Policy at the University of Michigan School of Public Health, Ann Arbor.

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