Memory, Contagion, and Capture Rates: Characterizing the Types of Addictive Behavior that Are Prone to Repeated Epidemics

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Abstract: We extend the two-dimensional model of drug use derived in Behrens et al. (1999) by introducing an additional state representing memory of past years of drug abuse. This memory of past use dampens initiation and, hence, future use in a more subtle and plausible way than was possible in the two-state model. The resulting model reflects how a drug epidemic is spread as users "infect" non-users, and how drug epidemics might periodically re-occur when a new generation forgets the ill effects of drug abuse experienced by their forbears. Mathematical stability analysis of the three-dimensional non-linear model's dynamics suggests – among other observations – that drug prevention can temper drug prevalence and consumption, and it evidences the interplay between the deterrent power of negative experiences with drug abuse and the rate of forgetting. The conditions for a Hopf bifurcation's existence and its stability properties in the three-dimensional phase space allow a qualitative characterization of what types of drugs – in terms of the probability of escalating to heavy use and the length of a typical addiction career – are most prone to generate cyclic, i.e. recurrent, drug epidemics.

Keywords: Illicit drugs, chain of drug epidemics, memory, demand side control, dynamical systems, Hopf bifurcation, limit cycle

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1. Introduction

Epidemics of a behavior, such as drug use, can occur and reoccur if new (light) ”users” stimulate others to initiate the behavior in question and long-time (heavy) users suppress initiation. This dynamic can occur if the behavior is ”contagious”, but prolonged exposure to the activity creates problems that serve as ”negative advertisements” which discourage further initiation. Researchers (e.g., Musto, 1987 and Kleiman, 1992) have long described these dynamics in qualitative terms for addictive drugs. More recently Everingham and Rydell (1994) and Behrens et al. (1999) proposed a formal mathematical model of cocaine use that captures these behaviors. Despite the model's simplicity, it matches historical data on the US cocaine epidemic surprisingly well and yields a number of important policy insights. The present paper improves and extends the past work in two ways.

First, in the earlier model the deterrent effect on initiation was governed by the current number of heavy users. So when a heavy user exited the population, his or her contribution to deterring initiation went immediately to zero. I.e. there was absolutely no memory of past bad experiences with heavy use even though in reality not all knowledge of a heavy user disappears the moment the individual exits the population, particularly if the exit is by physical death from drug use (as opposed to ceasing use or moving out of the area). The current model avoids this problem by introducing a third state which reflects some sort of memory of drug abuse.

Second, we provide a complete, qualitative analysis of the three-dimensional model's stability behavior including conditions of a Hopf bifurcation's existence and its stability properties by performing the center manifold reduction. Hence, we are not confined to exploring the system's qualitative behavior for a particular set of parameter values (e.g., those for the US cocaine epidemic). Indeed, we can turn the question around and ask, ”For what sets of parameter values are certain behaviors, such as cycles, most likely to emerge?” By characterizing these parameter regions in terms of intuitively understandable characteristics of the behavior, such as the probability an individual escalates from light to heavy ”use”, we obtain quite general and powerful insights as to the types of behavior that are most likely to be difficult for authorities to control.

Many types of behavior are described as being ”contagious” and have distinctions drawn between low and high rate ”offenders” (e.g., vandalism vs. serious street crime and violence, workers bending the rules vs. workers constantly abusing the system, rude or inappropriate comments vs. more serious forms of sexual harassment, etc.). Hence, these qualitative insights do not necessarily pertain only to drug epidemics. Nevertheless, we retain the drug nomenclature here, speaking of ”light” and ”heavy users”, for simplicity and for consistency with past work.

2. Model Formulation

Everingham et al. (1995) introduced and parameterized a discrete-time, Markov model of cocaine demand in the US that differentiated between light ($L(t)$) and heavy ($H(t)$) users, with parameters *a*, *b*, and *g* representing the annual rate at which light users quit, light users escalate to heavy use, and heavy users quit, respectively.[1] The model was used to describe past trends and project a baseline counterfactual for purposes of policy analysis (Rydell and Everingham, 1994), but initiation was scripted, not modeled explicitly.

Behrens et al. (1999) converted this model of drug demand into continuous time and incorporated a feedback effect of the current prevalence, or level, of use on initiation into new use. In particular, they included an endogenous model of initiation in which some initiation was "spontaneous" (e.g., because of in migration) but most occurred through interactions with current light users. (Most people who start using drugs do so through contact with a friend or sibling who is already using. Indeed, the metaphor of a drug "epidemic" is commonly used precisely because of this tendency for current users to "recruit" new users.)

In keeping with Musto's (1987) argument that knowledge of the possible adverse effects of drug use acts as a deterrent or brake on initiation, Behrens et al. (1999) moderated the rate at which light users recruited new initiates by the negative "reputation" of the drug. The reputation was modeled as a negative exponential function of the relative number of current heavy and light users.[2] This model, which we will refer to here as the *LH model*, had interesting dynamics and generated a variety of policy insights, such as highlighting the benefit of varying the mix of interventions dynamically over the course of a drug epidemic (Behrens et al., 1999), but it had one odd and undesirable characteristic. According to this model, removing a heavy user (either naturally or through treatment) immediately and entirely erases all of that individual's contribution to the negative reputation of the drug. In reality, memories of drug problems can persist.

One way to avoid this problem is introducing a third state variable that explicitly represents knowledge of past use. Behrens et al. (2002a) took an initial step in this direction by using as a measure of the "history" of heavy use the

[1] Everingham et al. (1995) also had flow from heavy back to light use. As in Behrens et al. (1999), for both practical and theoretical reasons we eliminate this flow and increase the value of parameter *g*, the rate of desistance from heavy use, accordingly.

[2] Caulkins et al. (1999) evaluated sixty functional forms to find the most promising function for the reputation term. The ones used in Behrens et al. (1999) and this paper were among the subset that fit the data well. We cannot rule out other forms because it's not the case that no other form fits the data well. But these two are simple, fit as well as any of a large number of other functional forms, and – particularly the form used in this paper – have considerable intuitive appeal.

decaying memory of people who were ever heavy users, denoted by the state variable *E*. The term "history" is used to connote a cumulative process, with a reputation that is acquired over time. But bad reputations are not immortal, so they decay over time as well.

The major drawback of this approach, denoted here as the *LHE model*, is that three individuals who use heavily for one day, one year, and one decade, respectively, would all contribute the same amount to the memory of heavy use. Presumably in reality the longer an individual is addicted, the more problems he or she experiences, the greater the cost imposed on others, the more people who witness the behavior, etc. So an appealing alternative is to base the negative reputation not on some memory of the number of people who ever used heavily, but instead on memory of the number of heavy user years, i.e., the number of person years spent in heavy use.

Hence, in the present model we introduce a third state that represents the decaying memory of heavy user years (denoted $Y(t)$) and let this new state serve as the deterrent in the reputation function (Equation (2)). The flow into this state is the number of current heavy users ($H(t)$). The outflow is a simple exponential decay governed by a memory parameter (δ). When this parameter is small, memories of drug abuse are long-lived; when δ is large memory of heavy users' years of use and associated problems dissipates rapidly. As before, we assume a fixed flow of "innovators", τ, into the state of light use irrespective of current use, and interpret the parameter *s* as the rate at which light users "persuade" non-users to try an addictive substance, if there was no memory of the negative consequences of drug abuse. (Note that from now on we omit the notion of time for sake of simplicity.)

This leads to the following descriptive three-state model of light (*L*) and heavy (*H*) use and heavy user years (*Y*), which we refer to as the *LHY model* for obvious reasons,

$$\begin{aligned} \dot{L} &= I(L,Y) - (a+b)L, \quad & L(0) &= L_0, \\ \dot{H} &= bL - gH, & H(0) &= H_0, \\ \dot{Y} &= H - \delta Y, & Y(0) &= Y_0, \end{aligned} \tag{1}$$

where the endogenous initiation into light use is specified by function

$$I(L,Y) = \tau + sL\, exp\left(-q\,\tfrac{Y}{L}\right), \tag{2}$$

L = *number of light users,*
H = *number of heavy users,* and
Y = *decaying heavy user years*.

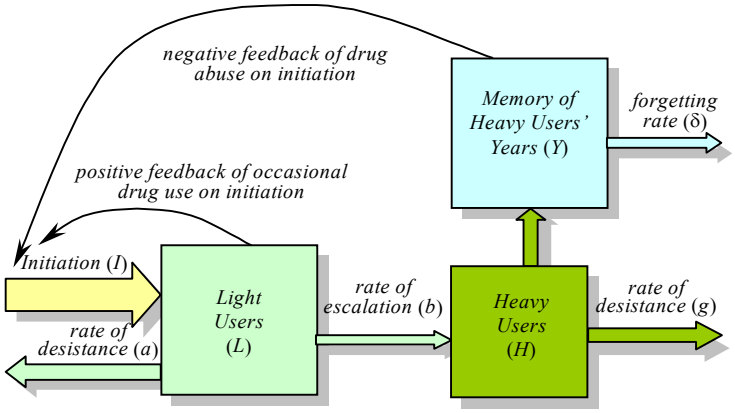

Figure 1. Flow chart of the *LHY* model

3. System Performance for the US Cocaine Problem

3.1 Shape and Robustness of the Modeled Cocaine Epidemic

To visualize the model's system behavior, show how well it fits data derived from an observed epidemic, and document its improvement over the previous models (Behrens et al., 1999, 2002a) we have to specify the parameter values a, b, g, δ, τ, s, and q. We do this for the cocaine epidemic currently observed in the United States, because of its magnitude and because data on that epidemic are reliable, as described in Appendix A.1 and summarized in Table 1.

Initiation rates estimated with the *LHY* model fit official estimates (Johnson et al., 1996) reasonably well – slightly over-estimating initiation in early years and underestimating the peak (see Knoll and Zuba, 2000).[3] The *LHY* model's prevalence curves are very close to those that Everingham and Rydell (1994) estimated. (See Figures 2 and 3, respectively).

[3] This of course by no means validates the particular form of the initiation function (Equation (2)). As mentioned, other functional forms with about the same number of parameters and the same or similarly appealing properties produce comparably close fits to the historical data (Caulkins et al., 1999). True validation is very difficult, given the inability to collect further data.

Table 1: Base case parameter values for US cocaine epidemic

Parameters	Description
$a = 0.163$	*annual rate at which light users quit*
$b = 0.024$	*annual rate at which light users escalate to heavy use*
$g = 0.062$	*annual rate at which heavy users quit*
$\tau = 5\text{x}10^4$	*number of innovators per year*
$s = 0.610$	*annual rate at which light users attract non-users*
$q = 3.443$	*constant which measures the deterrent effect of heavy use*
$\delta = 0.291$	*forgetting rate*

Using Equation (A4) the base case parameter values listed in Table 1 induce the following equilibrium state,

$$\hat{\boldsymbol{X}} = \begin{pmatrix} \hat{L} \\ \hat{H} \\ \hat{Y} \end{pmatrix} = \begin{pmatrix} 276{,}365 \\ 108{,}503 \\ 372{,}477 \end{pmatrix},$$

which has the properties of a stable focus. The characterization of $\hat{\boldsymbol{X}}$'s stability behavior can be confirmed by calculating the eigenvalues of the linearized system around its equilibrium, $\lambda_1 \cong -0.305$, and $\lambda_{2,3} \cong -0.101 \pm i\,0.022$ as defined by Equation (A10) and (A11), respectively, in Appendix A.2. Note that modest changes in the size of some of the base case parameter values might easily transform the equilibrium into a stable node (see Table A1).

To visualize the global qualitative properties of the solutions of system (1) for the base case set of parameters, we project the entire phase space into the (*L,H*)-plane (Figure 3). Following the trajectory induced by system (1) in the (*L,H*)-plane shows the meaning of the "memory of drug abuse", *Y*. Emanating from a "no-memory"-status, people acquire a knowledge of the consequences of drug abuse over time, i.e. $\dot{Y} > 0$, which suppresses initiation. As a consequence the number of light users diminishes, i.e. $\dot{L} < 0$, and a few time steps later the number of heavy users does too, i.e. $\dot{H} < 0$. The memory of drug abuse, however, continues to accumulate knowledge until the number of heavy users declines to the value $H = \delta Y$. Then memories fade, i.e. $\dot{Y} < 0$, until they reach their equilibrium level.

Figure 3 not only shows the projection of the trajectories spiraling towards the unique stable equilibrium, it also shows Everingham and Rydell's (1994) estimates of the historical trajectory of the cocaine epidemic which to date has followed a track not so different from what the model predicts.

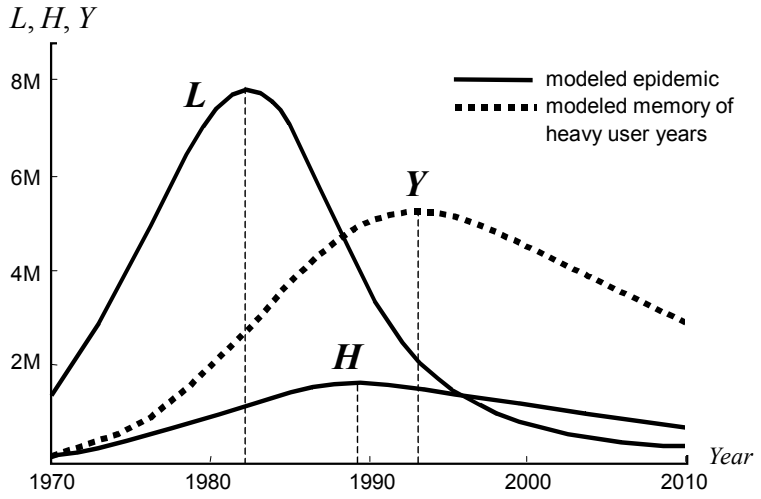

Figure 2. Plot of modeled numbers of users including the memory of abuse and "sequence of peaks" for the base case parameter-set as specified in Table 1. L_0=1,400,000, H_0=130,000, Y_0=110,000.

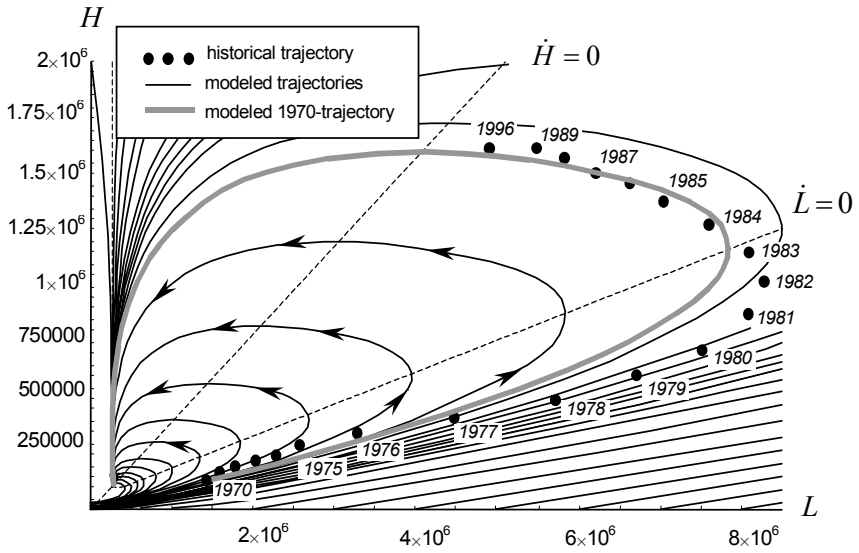

Figure 3. Projection of the phase portrait of model (1) for the base case parameter values (listed in Table 1) into the (*L,H*)-plane, including smoothed historical trajectory of the current US cocaine epidemic (Everingham & Rydell). Initial data for the 1970-trajectory: L_0=1,400,000, H_0=130,000, Y_0=110,000.

The modeled and historical data also move around their respective paths at nearly the same rate. The fit is not perfect entirely (the historical data reflect a slightly higher, sharper peak in light use), but it is remarkably good for the initial phase of the epidemic. The similarity is striking given that the actual epidemic has been subject to a varying set of drug control interventions over time that could be responsible for deviations from the model's uncontrolled path. Likewise, idiosyncratic historical events, such as Len Bias' death and the sharp increases in prices in late 1989, could account for some of the difference between historical and modeled data.

3.2 Can We Do Too Much Treatment?

So far the results found for the *LHY* model are similar to what Behrens et al. (1999) found for the *LH* model with one conspicuous exception. The exception pertains to the rate of desistance from heavy use, represented by parameter *g*. Behrens et al. (1999, 2000) found that in the *LH* model treating and removing heavy users can be counter-productive when there are relatively few heavy users because each heavy user contributes significantly to deterring initiation. If there are many heavy users relative to the number of light users, treating and removing some does not greatly jeopardize the negative reputation of the drug or the suppression of initiation. Hence, in the *LH* model, treating a large fraction of all heavy users throughout an epidemic, which raises the outflow from heavy use *g*, risks creating a never ending cycle of epidemics. With the current model and Rydell and Everingham's (1994) estimates of treatment efficacy, this is not a concern.[4] (See Appendix A.3 and A.4.) In particular given Rydell and Everingham's (1994) characterization of treatment's effectiveness, even if every heavy users received treatment every year (which Rydell and Everingham view as an upper bound on the rate at which people can be recruited into treatment), the parameter *g* would be increased only from 0.062 to 0.094. For the *LH* model, that placed *g* on the boundary between the range yielding damped oscillation (0.043 - 0.094) and the range yielding stable limit cycles (0.094 - 0.138). With this model, such values are comfortably within the region of damped oscillation. (See Figure 4 which shows how the qualitative system behavior depends on the interplay between the flow rate out of heavy use, *g*, and the forgetting rate whose base value for the cocaine epidemic is $\delta = 0.291$.)

[4] Rydell and Everingham (1994) modeled treatment as raising the outflow rate from 0.062 to 0.13 per year, and they considered treating every heavy user once per year as a practical upper bound on the rate at which people can be admitted to treatment.

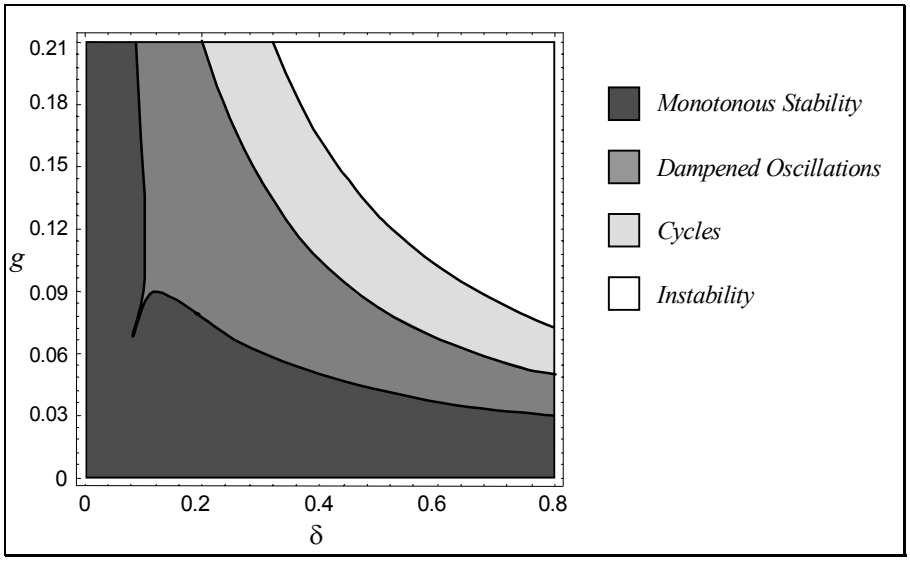

Figure 4. Stability regions (according to Table A1) for any combination of g and δ values for $a = 0.163$, $b = 0.024$, $s = 0.61$, and $q = 3.443$.

One final, interesting characteristic of this model is worth noting. Large values of Y_0 support (speed up) the burning out of the drug epidemic on its own accord. This might help to explain why Europe and some smaller US cities never had a cocaine epidemic as large as did cities where cocaine use spread before it acquired a bad reputation (e.g., New York, Los Angeles, and Miami). Perhaps the vicarious experience of the problems experienced by those cities served as a "protective factor" for cities that were exposed to cocaine later (see also Behrens et al., 2002b).

3.3 The Consequences of Forgetting Past Experiences and the Benefits of Prevention

Several parameters that might be influenced by prevention programs play an important role in determining the stability of the system and, hence, the likelihood the system will undergo repeated cycles or epidemic of drug use. Figure 5 shows how the qualitative system behavior depends on the interplay between these two parameters: (1) the parameter δ which governs how quickly heavy user years are "forgotten" and (2) the deterrence parameter, q. Large values of q imply that smaller ratios of memory (Y) to light users (L) are necessary to suppress initiation by a given proportion.

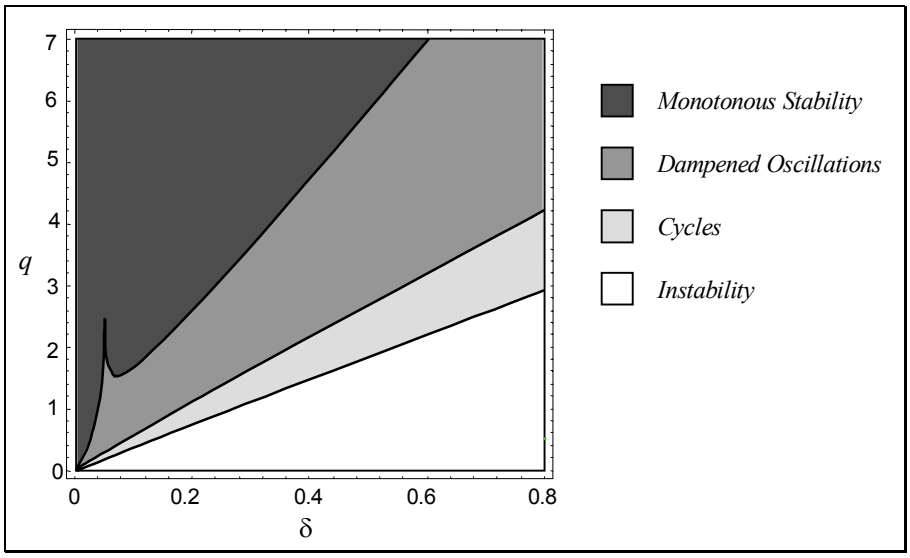

Figure 5. Stability regions (according to Table A1) for any combination of q and δ values for $a = 0.163$, $b = 0.024$, $g = 0.062$, and $s = 0.61$.

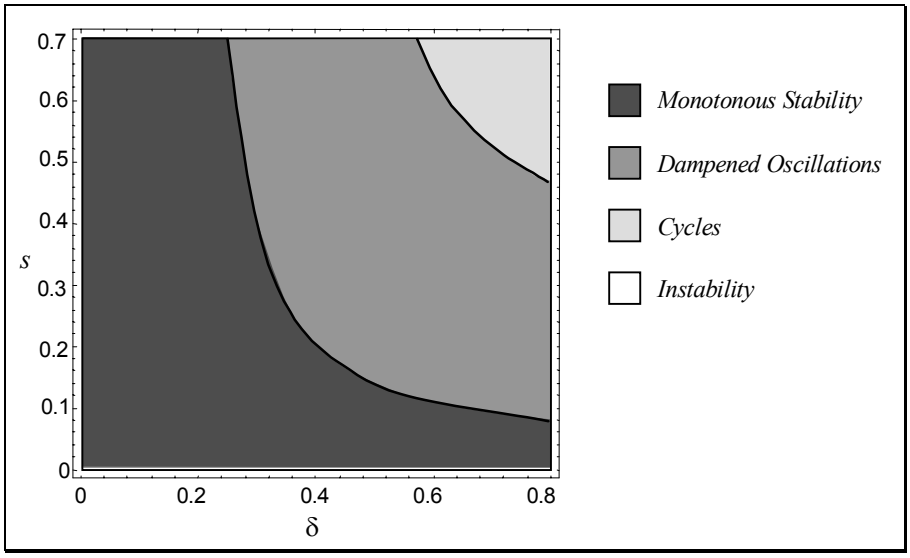

Figure 6. Stability regions (according to Table A1) for any combination of s and δ values for $a = 0.163$, $b = 0.024$, $g = 0.062$, and $q = 3.443$.

The upper left-hand portion of Figure 5 is the region of greatest stability. Moving towards the lower-right increases the risk of cycles. Thus, weakening the feedback effect through past memories either by having memories fade faster (δ larger) or be less poignant (q smaller) increases the likelihood of cycling. Conversely, prevention programs that accentuate or preserve such memories would reduce the risk of cycling. (For the conditions for a Hopf bifurcation's existence and its stability properties consult Appendix A.3 and A.4.)

A parallel figure for the level of contagiousness (s) and the forgetting rate (δ) is also interesting. (See Figure 6.) As determined in Appendix A.2, among others cycles may emerge when the parameter s is large, and prevention should help reduce s. Decreasing s, without adapting δ, removes the cycling system behavior and reduces prevalence, both in overall and in equilibrium terms. That is, moderating the contagious aspect of initiation, e.g., through prevention, reduces the likelihood of cycles and instability.

4. Analysis Across Types or Parameterizations of Epidemics

The last section applied the *LHY* model to a particular drug epidemic, namely the current cocaine epidemic in the United States, by selecting parameter values appropriate for that epidemic. This section, in contrast, strives to make general statements about any epidemic of delinquent behavior that obeys the general structure of the model outlined above. The goal is to identify which "types" of epidemics are most problematic, where the "type" of an epidemic is characterized by its parameter values.

Two key questions must be addressed in assessing how problematic an epidemic is likely to be. First, what is the qualitative structure of the epidemic behavior? Does it converge monotonically to an equilibrium, spiral into a single focus, cycle forever, or diverge? Second, if it does approach a single equilibrium, how prevalent is the endemic level of behavior in that equilibrium?

The first issue can be addressed through an investigation of the linearization of the *LHY* model (Equation (1)) around its equilibrium state. Not surprisingly, the expressions characterizing the 3-state, 7-parameter model are rather complex, so in addition to stating them in the next section, the subsequent section summarizes various graphical and numerical exercises that are conducted to help interpret the expressions in terms relevant to substantive experts.

4.1 Conditions Governing System Behavior

The conditions governing the system's qualitative behavior are derived in Appendix A.2. Since some of the conditions are rather complex, we state them in terms of the following aggregates:

$p := exp(-qb/g\delta)$ — p denotes the equilibrium reputation of the drug.

$\Omega := a + b - sp$ — Ω denotes the per capita net flow rate out of light use in equilibrium.[5]

$tr\hat{\mathfrak{J}} := \hat{I}_L - (g + \delta + a + b)$
$= -(g + \delta + sp\,ln\,p + \Omega)$ — $tr\hat{\mathfrak{J}}$ denotes the trace of the Jacobian evaluated at the equilibrium point which is calculated as the difference between the marginal change in incidence with respect to the number of light users and the sum of the flow rates a, b, and g and the forgetting rate δ.

$\Theta := 3g\delta - (\mathrm{tr}\hat{\mathfrak{J}})^2 -$
$- 3(g + \delta)(\mathrm{tr}\hat{\mathfrak{J}} + g + \delta)$ — Θ denotes the triple sum of the product of the outflow rates, g and δ, and the weighted[6] sum of these rates reduced by the square of the trace of the Jacobian.

$\Delta := \sqrt[3]{\frac{-\Xi + \sqrt{\Xi^2 - 4\Theta^3}}{2}}$ — Δ denotes a term that arises from solving the cubic characteristic equation where the term Ξ is determined by $\Xi := (\mathrm{tr}\hat{\mathfrak{J}})^3 + 3\Theta\mathrm{tr}\hat{\mathfrak{J}} + 27g\delta\Omega$.

$\rho := \frac{1}{3}\left(2\mathrm{tr}\hat{\mathfrak{J}} - \Delta + \frac{\Theta}{\Delta}\right)$ — ρ denotes the sum of the pair of conjugate complex eigenvalues which is uniquely determined for $\Xi^2 > 4\Theta^3$.

It is easy to see, that three of these aggregates constituting (multiples of) the coefficients of the characteristic polynomial (Equation (A8)) exhibit interesting features for sensibility analysis:

- The indicator $tr\hat{\mathfrak{J}}$ diminishes in size if a, b, or q increase, and grows if s increases. If $sp(ln\,p)^2 > g$ ($< g$) any expansion in g increases (reduces) $tr\hat{\mathfrak{J}}$. Increasing the value of δ enlarges (diminishes) $tr\hat{\mathfrak{J}}$ if $sp(lnp)^2 > \delta$ ($< \delta$).
- $\Theta + (tr\hat{\mathfrak{J}})^2$ diminishes if a, b or q decreases, and grows if s decreases. If $sp(ln\,p)^2 > g + (g/g+\delta)(\delta/3 - 2g - tr\hat{\mathfrak{J}})$ $(> g + (g/g+\delta)(\delta/3 - 2g - tr\hat{\mathfrak{J}}))$ any expansion in g increases (reduces) $\Theta + (tr\hat{\mathfrak{J}})^2$. Analogously, increasing the

[5] I.e., Ω denotes the outflow, $a + b$, minus the rate at which light users recruit initiates, sp.

[6] Note that the weight is determined by the marginal change in incidence with respect to the number of light users, reduced by the sum of the flow rates out of light use, a and b.

size of δ enlarges (diminishes) $\Theta + (tr\hat{\mathfrak{J}})^2$ if $sp(ln\, p)^2 < \delta + (\delta/g+\delta)(g/3-2\delta-tr\hat{\mathfrak{J}})$ ($> \delta + (\delta/g+\delta)(g/3-2\delta-tr\hat{\mathfrak{J}})$). Hence, in most cases $tr\hat{\mathfrak{J}}$ and $\Theta + (tr\hat{\mathfrak{J}})^2$ behave antagonistically.

- Ω diminishes if g, δ, or s increase, and grows if a, b, or q increase.

Finally, for the aggregates defined above (p, Ω, $tr\hat{\mathfrak{J}}$, Θ, Δ, ρ) following propositions summarize conditions for the four types of possible system behavior derived from Table A1 in Appendix A.2 and from the calculations performed in Appendix A.3 and Appendix A.4.

Proposition 1.

According to the system equations (1) prevalence always approaches a unique, low-level (but positive) equilibrium state (Equation (A4)) if

$$tr\hat{\mathfrak{J}} \le \rho \le min\left\{-\tfrac{1}{3}\left|\sqrt{3}i\left(\tfrac{\Theta}{\Delta}+\Delta\right)\right|, 0\right\}.^{7} \tag{3}$$

Proposition 2.

Oscillation occurs if and only if

$$\Xi^2 + 4\Theta^3 \ge 0\,. \tag{4}$$

If, additionally

$$tr\hat{\mathfrak{J}} < \rho < 0 \;\Leftrightarrow\; tr\hat{\mathfrak{J}} < \tfrac{\Theta}{\Delta} - \Delta < -2tr\hat{\mathfrak{J}} \tag{5}$$

holds then the trajectories spiral into the unique equilibrium (Equation (A4)).

Proposition 3.

For parameter values satisfying

$$\Xi^2 + 4\Theta^3 \ge 0 \;\wedge\; tr\hat{\mathfrak{J}} < \rho = 0\,, \tag{6}$$

a Hopf bifurcation exists as long as condition (A22) holds (see Appendix A.3 (ii)). The emerging periodic solutions are stable for all Hopf-critical-values satisfying (A39) (see Appendix A.4).

[7] This condition prevents prevalence from persistent oscillation or growth.

Proposition 4.

In equilibrium about $b/(b+g)*100\%$ *of users are heavy users. The number of steady state users is proportional to the number of innovators,* τ*, who initiate of their own accord, not at the urging of current users, although the way the steady state is reached does not depend on* τ*. Furthermore, the number of steady state users is indirectly proportional to the per capita net flow rate out of light use in equilibrium,* Ω *(as defined by Equation (A6)).*

4.2 Interpretation of Stability Conditions

Since we have achieved a complete, qualitative analysis of the stability behavior of the *LHY* model, it is possible to look at what "types" of (drug) epidemics are most prone to create certain features, e.g. to create cycles (Musto, 1987). The "type of epidemic" is defined by its parameter values. The model has seven parameters, but Proposition 4 above shows that one (τ) has no influence on the system's stability, so only six are of interest.

The three parameters pertaining to the initiation function are fairly easy to interpret. The parameter *s* governs how "contagious" the behavior is by determining the rate at which light users recruit new users. The reputation parameter *q* governs how effective memories of heavy use are in deterring initiation, and the parameter δ governs how quickly those memories fade. In contrast, the three flow parameters *a*, *b*, and *g*, do not correspond as closely to familiar characteristics of drug epidemics. To make the interpretation more intuitive, we transform them into more "tangible" attributes of an epidemic:

(1) the probability a new initiative will eventually escalate to heavy use, $\mathrm{P} := b/(a+b)$,
(2) average time from onset of heavy use to cessation, $G := 1/g$, and
(3) the average duration of use, $D := (1+b/g)/(a+b) = 1/(a+b) + G\,\mathrm{P}$.

The question of what types of epidemics manifest which types of stability behavior has now been reduced to describing a four-valued function of six variables:

Behavior = $f(s, q, \delta, \mathrm{P}, G, D)$

where the function *f*( ) takes one of the values: monotonous stability, damped oscillation, persistent oscillation, or instability.

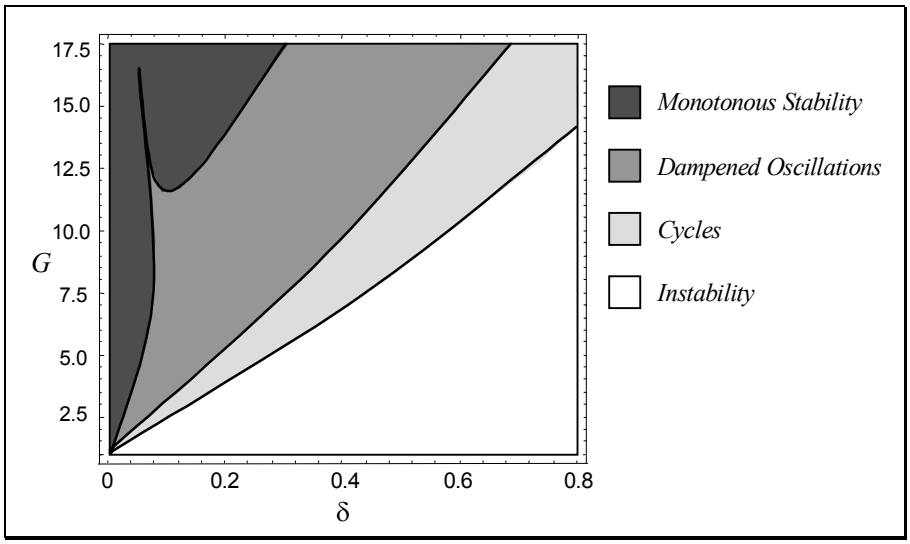

Figure 7. Stability regions (according to Table A1) for any combination of G and δ values for $a = 0.163$ and $b = 0.024$ (i.e. P = 0.128), $s = 0.61$, and $q = 3.443$.

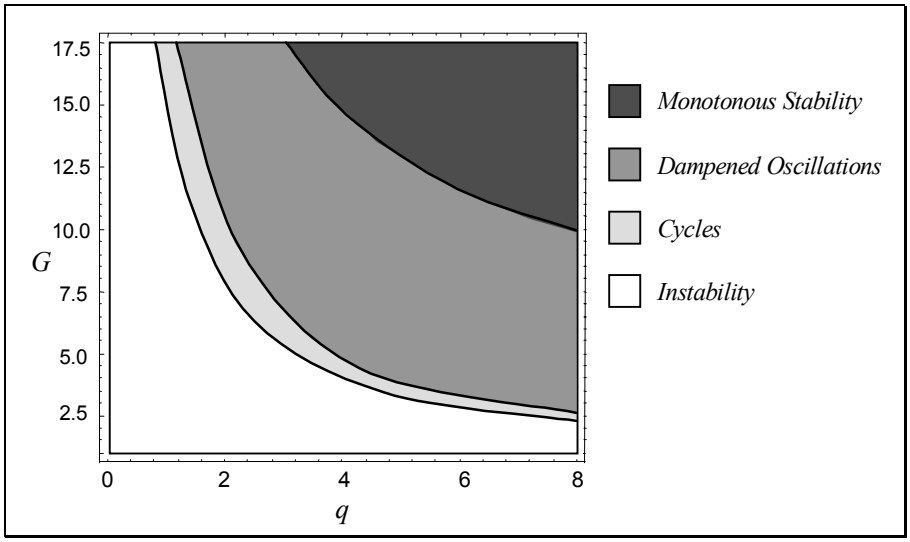

Figure 8. Stability regions (according to Table A1) for any combination of G and q values for $a = 0.163$ and $b = 0.024$ (i.e. P = 0.128), $s = 0.61$, and $\delta = 0.291$.

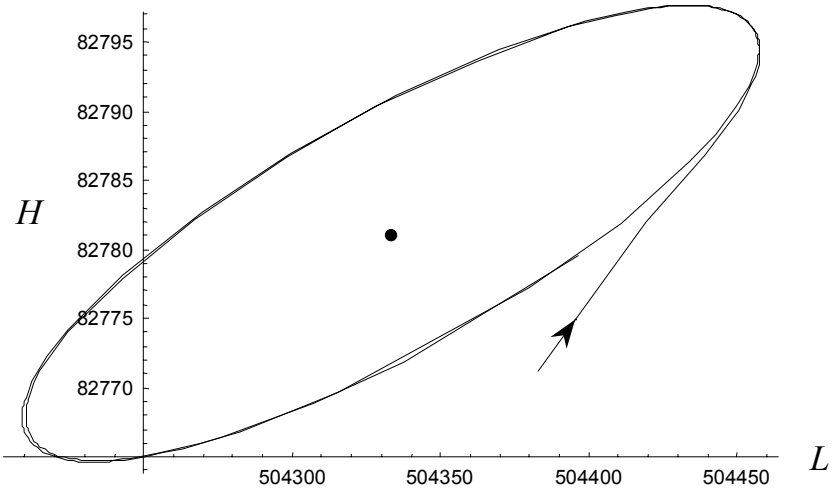

Figure 9. Stable limit cycle for $a = 0.163$ and $b = 0.024$ (i.e. P = 0.128), $G_c \cong 6.757$, $s = 0.61$, $\delta = 0.291$, and $q = 3.443$.

One useful way to get insight into this function is to look at its dependence on two parameters at a time. Figures 5 and 6 did this for q and δ and for s and δ, respectively. A variety of other such figures can be created, e.g. the interplay between the average length of the "addiction career" and the persistence of the memory of drug problems, as Figure 7 and 8 illustrate for US cocaine. Any drug control intervention targeted at a reduction of the average time from onset of heavy use to cessation, G, (and, consequently, on the reduction of the average duration of use) has to be combined with intensified media campaigns to keep up the memory of abuse and/or to increase its deterrent effect. Otherwise cyclic bahavior might occur (see Figure 9).

In addition to Figures 5–8, exhaustive, but basic, numerical calculations allow the following general statement:

Conditions for cycling. Cycling will occur for epidemics with the following characteristics:

- highly contagious (large s),
- knowledge of the problems of heavy use is discounted (small q) and/or transient (large δ),
- long average durations of use (large D) and addiction (large G) relative to the persistence and deterrence of memory of drug problems.

Conversely, we can exclude cycling for drugs that have a "well-balanced relationship" between the average duration of use, the addiction career length, and the decay of memories of past problems (see Figures 7 and 8). I.e. drug epidemics exhibiting this property will decay on their own accord.

4.3 Size of the Endemic Problem

When the epidemic approaches a single, stable equilibrium the level of use at that equilibrium can be called the "endemic" level of use. The memory of past use is not of direct concern: it affects societal costs only indirectly, through its influence on initiation. Rather it is the number of users and the proportion of users who are heavy users that is of greatest interest. Both quantities are defined by Equation (A4) and can be more intuitively expressed by means of the terms P, G, and D (which are defined on page 13). This leads to a simple, closed form expression for the total number of users,

$$\#\text{ of users} = \frac{\tau D}{1 + sp(G\mathrm{P} - D)}, \tag{7}$$

where the equilibrium reputations is defined as $p := \exp(qG(G\mathrm{P} - D)/\delta\mathrm{P})$. The proportion of users who are heavy users is equal to $G\mathrm{P}/D$ and, hence,

$$\#\text{ of heavy users} = \frac{\tau G\mathrm{P}}{1 + sp(G\mathrm{P} - D)}. \tag{8}$$

The first thing to notice is that the magnitude of the endemic problem scales linearly in τ. In the long run, the size of the problem depends directly on the rate of initiation by people whose determination to use cannot be swayed by the negative experiences of others. Influencing this rate through prevention efforts, providing competing activities that are more appealing, or other measures may be an important way to address an endemic problem.

The second observation is that increasing the parameter q and decreasing the parameters s or δ are unambiguously beneficial since those changes reduce the number of users without altering the proportion who are heavy users. All three of those parameters may be thought of as the targets of various types of prevention interventions. Which is the most effective leverage point, depends on the other parameter values. Additionally, all these forms of prevention do not only have the desired effect on the endemic level of use, but also on the entire epidemic. I.e. in absence of these interventions the peak level of use was much greater than the initial or equilibrium level of use.

Decreasing the average time from the onset of heavy use to cessation (G), e.g. by expanding treatment, is unambiguously good – *as long as the adverse consequences of abuse are remembered* – because it reduces the number of users and it reduces the proportion of users who are heavy users. The consequences of altering the probability of escalation to heavy use (P) are less clear-cut. Increasing P always reduces the endemic level of light users, but unless $\mathrm{P} < (1 + spD)/(1 + spG(1 - q/\delta))$ it increases the number of heavy users. Whether that is beneficial overall depends on the relative per capita costs associated with light and heavy users. Increasing both $(D–G\mathrm{P})/\mathrm{P}$ and G by the same proportion reduces the endemic number of users without altering the proportion of users who are heavy users. Over the course of the epidemic increasing P or G raises the peak level of heavy use compared to the initial or equilibrium level of use while it reduces the peak level of light use.

The parameters governing the negative feedback process do not affect the character of the endemic level in terms of proportion of users who are heavy users. Thus, for the most part, the recipe for a serious problem is the same whether one is worried about an unstable transient or a high level of endemic use. Behaviors for which initiation is contagious (high s) and impervious to past experience (low q, high δ) and use is persistent (high P, high G) are likely to be the most difficult to control. A steady stream of initiates who are blind to the danger of heavy use (large τ) increase the size of the problem but doesn't threaten the stability of the epidemic.

4.4 Implications for Common Policy Interventions

Combining these insights about the dependence of a behavior's stability, endemic levels, and numerical investigations, we can draw some inferences about the likely effects of several types of interventions.

(1) **Prevention**. For any epidemic, regardless of its special features, it is always useful to reduce the drug's contagiousness (e.g., through peer resistance based prevention programs), but this is particularly so at the onset of a drug epidemic (when both the number of heavy users and the memory of the years of abuse are small in size). Doing so helps to decrease consumption significantly and to avoid cycles.

(2) **Treatment**. For epidemics with a low forgetting rate it is always useful to help heavy users quit use, e.g., through treatment. However, for epidemics

with high forgetting rates, intensive treatment pursued throughout the epidemic can induce cycling in the system behavior.

(3) **Relative Timing of Prevention and Treatment**. With this model the extreme finding from Behrens et al. (2000) that one should never pursue school-based prevention and treatment at the same time is not obtained. However, it remains useful to intensify prevention in the beginning and treatment later in the epidemic.

(4) **Enforcement**. For epidemics with a high probability of users escalating to heavy use, it may be useful to pursue measures that discourage escalation. Using enforcement to keep prices high may be one such intervention. Note that these measures have to be planned in a very careful and well-balanced way, because a low probability of escalation from light to heavy use might cause cycling behavior (especially when appearing together with a fast forgetting process).

5. Conclusions

The analysis performed in this paper yields several conclusions. First, when using parameter values based on the past US cocaine epidemic, model (1) generally shows the same behavior as the model presented and investigated by Behrens et al. (1999), where the entire analysis was done with the simple, two-state *LH* model. For example, prevention programs that soften the contagious character of drug initiation lower the threat of recurrent epidemics (cycles), as would programs that raise the deterrent effect spread by a single ever-heavy user. Since the two-state *LH* model is simpler, for most purposes reaches the same conclusions, and reproduces historical data almost as well, the power of that parsimonious model is affirmed.

One striking exception, however, pertains to the issue of whether treating a constant fraction of all heavy users throughout the epidemic is likely to induce cycling. The two-state *LH* model said yes. This three state *LHY* model, like Behrens et al.'s (2002a) three-state *LHE* model says no. Finding this with the *LHY* model is more persuasive than finding it with *LHE* model because treatment had no effect on the drug's reputation in the *LHE* model. In some sense, the *LH*- and *LHE* model took two extreme positions about the persistence of memories of heavy use, and the *LHY* model takes a more nuanced view. Yet in this important issue, the *LHY* model reaches the same qualitative conclusion as the *LHE* model.

The main substantive contribution of this paper stems not from the refined understanding of the past US cocaine epidemic, but rather from its ability to make qualitative statements about what attributes of a behavior are likely to lead to ”epidemics“ in two senses of the word: (1) that the peak level of use is much greater than the initial or equilibrium level and that (2) the epidemics could recur. No other model in this class – i.e. one that includes a memory of the adverse consequences of an intense form of delinquency – has admitted complete analytical solution of its stability, so comments could only be made about drugs characterized by specific sets of parameter values (e.g., those estimated from the US cocaine epidemic). Note: This generality actually extends to behaviors other than drug use. The conclusions apply to any form of delinquency which involves initiation to low intensity activity, escalation to more intensive states of activity, desistance, and a feedback from current and remembered activity levels to initiation of the sort described here.

In brief the finding is that epidemics exhibiting insufficient deterrent power of memories and/or a short retention of memories of the consequences of a “heavy form” of delinquency are most prone to reoccur, as highly “contagious” epidemics, and those with long average cure durations of delinquent behavior (especially when combined with short “intensified delinquency” careers).

Among the findings with respect to control are that it is always useful to apply prevention programs, but that this is particularly so at the onset of an epidemic of delinquent behavior. For epidemics with a low forgetting rate it is always useful to help “heavy offenders” to desist, e.g., through “treatment”. However for epidemics with high forgetting rates, intensive treatment can induce cycling in the system behavior. For epidemics with a high probability of offenders escalating to a “heavy” stage of delinquency, it may be useful to pursue measures that discourage escalation, but such measures should be carefully planned, because a low probability of escalation from a light to a heavy form of delinquency might cause cycling behavior (especially when appearing together with a fast forgetting process).

Appendix

A.1 Specification of Parameter Values for the US Cocaine Epidemic

The flow rates *a* and *b* in Model (1) are computed as the time-continuous equivalents of the Everingham-Rydell estimates (1994, p.43). For example, they estimate that 15% of light users quit use each year and 2.4% escalate to heavy use, so we set $a = -ln(1-0.15) \cong 0.163$ and $b = -ln(1-0.0242) \cong 0.024$, to three decimal places. Additionally, Everingham and Rydell (1994, p.42) gave ranges of parameters for which they found good fits to historical data reported by the National Institute on Drug Abuse (1991), which are $a \in (0.157, 0.168)$ and $b \in (0.02, 0.03)$. For reasons described in Behrens et al. (1999), we choose $g = -ln(1-0.06) \cong 0.062$. [8]

In Model (1) initiation depends on light users and decaying cumulative heavy-user-years. Hence, we have to estimate values for the "initiation-parameters" τ, *s* and *q*. For the determination of the parameters *s* and τ we follow Behrens et al. (1999) yielding the values $s = 0.61$ and $\tau = 50{,}000$. The corresponding size of parameter *s* can be interpreted as indicating that approximately two light users would "persuade" one non-user per year to try cocaine, if there was no memory of years of abuse giving cocaine a bad reputation. The constant *q* measures the deterrence caused by the memory of heavy user years[9] and is, consequently, correlated with the "forgetting rate", δ.[10] The values for δ and *q*, respectively, are found by minimization of the sum of the squared difference between the modeled and observed annual cocaine initiation rates from 1971 to the year *x*, where *x* varies from 1980–1989 (with initial numbers of users set equal to the number Everingham and Rydell (1994) estimate were present in 1970, where each of the 10 estimation-runs was performed for (a) $Y_0 = H_0$, and (b) $Y_0 = 0.5*H_0/\delta$). For the tables including the appropriate pairs of parameter estimates, the LS values of deviation, and a visualization of the results in graphical form see Knoll and Zuba (2000).[11]

[8] We acknowledge that the current cocaine epidemic seems to be over and the next epidemic may be governed by different flow parameters, but we stick with the Everingham and Rydell flow parameters because we do not have data to estimate parameters for any other epidemic and some parameter values are necessary to ground the numerical analysis.

[9] Note that the *LHY* model is consistent with the one presented by Behrens et al. (1999): The parameter-value *q* changes compared to the *LH* model because the initiation function is augmented with a new state variable.

[10] Note that – at least for cocaine – the relationship between the values of *q* and δ appears to be approximately linear, i.e. $q=f(\delta)=c\delta$ (*c*...constant).

[11] Following Everingham and Rydell (1994), we estimate historical initiation rates by taking the average of so-called "difference" and "retrospective" estimates (Gfroerer and Brodsky (1992)) but substitute the newer retrospective estimates provided by Johnson et al. (1996) for those used by Everingham and Rydell. Estimates of rates of light and heavy use are taken from Everingham and Rydell (1994).

A.2 Local Stability Analysis of System (1)

Since the initial conditions are always positive ($L_0, H_0, Y_0 > 0$) and the first quadrant is an invariant set we restrict our analysis to the positive quadrant of the (L,H,Y)-space. First we derive the equations for the isoclines:

$$\dot{L} = 0: \quad Y(L) = -\tfrac{L}{q}\, ln\left(\tfrac{a+b}{s} - \tfrac{\tau}{sL}\right), \text{ for } \tfrac{\tau}{s+b} < L, \tag{A1}$$

$$\dot{H} = 0: \quad H(L) = \tfrac{b}{g} L\ , \tag{A2}$$

$$\dot{Y} = 0: \quad Y(L) = \tfrac{1}{\delta} L\,. \tag{A3}$$

Since $\tau > 0$, the intersection of the isoclines uniquely defines the equilibrium state,

$$\hat{\boldsymbol{X}} := \left(\hat{L} \quad \hat{H} \quad \hat{Y}\right)^T = \tfrac{\tau}{\Omega}\left(1 \quad \tfrac{b}{g} \quad \tfrac{b}{g\delta}\ \right)^T, \tag{A4}$$

in the positive section of the phase space where

$$\Omega := a + b - sp\ > 0\,, \text{ and} \tag{A5}$$

$$p := exp\left(-\,q\, b/g\delta\right) > 0\,. \tag{A6}$$

Equation (A4) also reflects the equilibrium ratio $L : H = 1 : b/g$ which leads to an equilibrium fraction of heavy users of $b/(b+g)$. Using the Jacobian matrix evaluated at the stationary point,

$$\hat{\mathfrak{J}} = \begin{pmatrix} -sp\ln p - \Omega & 0 & -spq \\ b & -g & 0 \\ 0 & 1 & -\delta \end{pmatrix}, \tag{A7}$$

the local stability behavior of system (1) can be determined in the usual manner by linearization of the system around the equilibrium state (determined by Equation (A4)). The corresponding characteristic polynomial,

$$P(\lambda) = \lambda^3 - \mathrm{tr}\hat{\mathfrak{J}}\lambda^2 + \tfrac{1}{3}\left(\Theta + \left(\mathrm{tr}\hat{\mathfrak{J}}\right)^2\right)\lambda + g\delta\Omega = 0\,, \tag{A8}$$

for

$$\Theta := -\left(\mathrm{tr}\hat{\mathfrak{J}}\right)^2 - 3(g+\delta)\left(\mathrm{tr}\hat{\mathfrak{J}} + g + \delta\right) + 3g\delta \in \Re\,, \tag{A9}$$

may be solved analytically yielding the spectrum of eigenvalues for $\Delta \neq 0$ (see Equation (A14)),

$$\lambda_1 = \mathrm{tr}\hat{\mathfrak{J}} - \rho\,, \tag{A10}$$

$$\lambda_{2,3} = \tfrac{1}{2}\rho \pm i\omega\,, \tag{A11}$$

where the term $\mathrm{tr}\hat{\mathfrak{J}}$ denotes the trace of the Jacobian matrix (A7) evaluated at the stationary point and where

$$\rho := \tfrac{1}{3}\left(2\mathrm{tr}\hat{\mathfrak{J}} - \Delta + \tfrac{\Theta}{\Delta}\right), \tag{A12}$$

$$\omega := -\tfrac{\sqrt{3}}{3}\left(\mathrm{tr}\hat{\mathfrak{J}} - \Delta\right) + \tfrac{\sqrt{3}}{2}\rho\,, \tag{A13}$$

$$\Delta := \sqrt[3]{\tfrac{-\Xi + \sqrt{\Phi}}{2}}\,, \tag{A14}$$

$$\Phi := \Xi^2 + 4\Theta^3\,, \tag{A15}$$

$$\Xi := \left(\mathrm{tr}\hat{\mathfrak{J}}\right)^3 + 3\Theta\mathrm{tr}\hat{\mathfrak{J}} + 27g\delta\Omega \in \Re\,. \tag{A16}$$

Hence, we can describe the stability behavior of system (1) linearized around its equilibrium state as listed in Table A1.

Table A1: Equilibrium stability properties of linearized system (1).

Inequalities	Stability properties
$\Phi < 0 \ \wedge\ tr\hat{\mathfrak{J}} \le \rho \le -\tfrac{1}{3}\left\lvert \sqrt{3}i\left(3\rho - 2\left(\mathrm{tr}\hat{\mathfrak{J}} - \Delta\right)\right)\right\rvert$	Stable node
$\Phi \ge 0 \ \wedge\ tr\hat{\mathfrak{J}} < \rho < 0$	Stable focus
$\Phi \ge 0 \ \wedge\ tr\hat{\mathfrak{J}} < \rho = 0$	Center
$\Phi \ge 0 \ \wedge\ \rho > max\{0,\ tr\hat{\mathfrak{J}}\}$	Unstable focus
$(tr\hat{\mathfrak{J}} > \rho)$ or $(\Phi < 0 \ \wedge\ \rho > -\tfrac{1}{3}\left\lvert \sqrt{3}i\left(3\rho - 2\left(\mathrm{tr}\hat{\mathfrak{J}} - \Delta\right)\right)\right\rvert)$	Saddle/Unstable Equilibrium

Note, that any parameter constellation yielding $\mathrm{tr}\hat{\mathfrak{J}} > \rho$ implies the existence of (at least) one unstable direction in the 3D phase space. (Therefore, the only cases we are interested in imply that $\mathrm{tr}\hat{\mathfrak{J}} \le \rho$, where we observe a transcritical bifurcation for $\mathrm{tr}\hat{\mathfrak{J}} = \rho$.) Note that transcritical bifurcations also occur for $\rho = \pm 2i\omega$ given that $\Phi < 0$.

A.3 Conditions for the Existence of a Poincaré-Andronov-Hopf Bifurcation

The (Poincaré-Andronov-)Hopf theorem (see, e.g., Guckenheimer and Holmes, 1983, p.151; Hassard et al., 1981, p.14ff.; Wiggins, 1990, p.276) states that limit cycles exist if (i) two purely imaginary eigenvalues exist for a critical parameter value, such that (ii) the imaginary axis is crossed with non-zero velocity.

(i) For $\Delta \neq 0$ (see Equation (A14)) condition

$$\rho = 0 \quad \Leftrightarrow \quad \mathrm{tr}\hat{\mathfrak{J}} = \tfrac{\Delta^2 - \Theta}{2\Delta} \tag{A17}$$

defines a neutral stability surface in the six-dimensional parameter space where the linearization of system (1) evaluated at the equilibrium point, $\hat{\boldsymbol{X}}(\rho = 0)$ (see Equation (A4)), possesses two purely imaginary eigenvalues if and only if

$$\mathrm{tr}\hat{\mathfrak{J}} < 0 \quad \wedge \quad \left(\left(\mathrm{tr}\hat{\mathfrak{J}}\right)^3 + 3\Theta\mathrm{tr}\hat{\mathfrak{J}} + 27g\delta\Omega\right)^2 + 4\Theta^3 \geq 0 \tag{A18}$$

and

$$\left(\left(\mathrm{tr}\hat{\mathfrak{J}}\right)^3 + 3\Theta\mathrm{tr}\hat{\mathfrak{J}} + 27g\delta\Omega\right)^2 + 4\Theta^3 \geq 0\,, \tag{A19}$$

where Ω and Θ are given by Equations (A5) and (A9), respectively—since the third eigenvalue $\lambda_1(\rho = 0) \neq 0$ (see Equation (A10)) differs from zero according to condition (A18).

(ii) Moreover, to guarantee the existence of a (representative) center manifold, *C*, passing through the bifurcation point $(\hat{\boldsymbol{X}}_c, \mu_c)$ in $\Re^3 \times \Re$ (and containing all local recurrent behavior of system (1), (2)), we will have to assure that

$$\Lambda(\mu_c) := \left.\frac{d\left(Re\,\lambda_{2,3}(\mu)\right)}{d\mu}\right|_{\mu=\mu_c} = \tfrac{1}{2}\left.\frac{d\rho(\mu)}{d\mu}\right|_{\mu=\mu_c} \neq 0\,, \tag{A20}$$

(in addition to (i)) where μ_c denotes the Hopf-critical-value of an arbitrary system parameter $\mu \in \{a, b, g, s, q, \delta\}$.

Note that for the special case $\Delta^2 = -\Theta$ condition (A20) is always satisfied for $\mu_c \in \{a_c, b_c, s_c, q_c\}$ which proves the existence of a persistent cycle for any of these (critical) parameter(s values). If, furthermore,

$$g_c^3\delta^2 \neq spq^2b^2 \;\text{ and }\; g^2\delta_c^3 \neq spq^2b^2 \tag{A21}$$

condition (A20) also holds for $\mu_c \in \{g_c, \delta_c\}$.

But if $\Delta^2 \neq \Theta$ the condition which has to be satisfied in addition to (A17)-(A19) to accomplish the existence of a Hopf bifurcation becomes more complex:

$$\Lambda(\mu_c) = \begin{cases} -\Psi + \frac{3g\delta(\Delta^2+\Theta)}{\Delta\sqrt{\Phi}} \neq 0 & \text{for } \mu_c = a \\ -\Psi\left(1+\frac{spq^2b}{g^2\delta^2}\right) + \frac{3(g\delta+spq)(\Delta^2+\Theta)}{\Delta\sqrt{\Phi}} \neq 0 & \text{for } \mu_c = b \\ -\Psi\left(1-\frac{spq^2b^2}{g^2\delta^2}\right) - \frac{3spqb(\Delta^2+\Theta)}{g\Delta\sqrt{\Phi}} + \Sigma_g \neq 0 & \text{for } \mu_c = g \\ \Psi p\left(1+\frac{qb}{g\delta}\right) - \frac{3g\delta p(\Delta^2+\Theta)}{\Delta\sqrt{\Phi}} \neq 0 & \text{for } \mu_c = s \\ -\Psi\frac{spqb^2}{g^2\delta^2} + \frac{3spb(\Delta^2+\Theta)}{\Delta\sqrt{\Phi}} \neq 0 & \text{for } \mu_c = q \\ -\Psi\left(1-\frac{spq^2b^2}{g^2\delta^3}\right) - \frac{3spqb(\Delta^2+\Theta)}{\delta\Delta\sqrt{\Phi}} + \Sigma_\delta \neq 0 & \text{for } \mu_c = \delta \end{cases} \tag{A22}$$

where

$$\Psi := -\frac{2}{3\Delta}\left(\mathrm{tr}\hat{\mathfrak{J}} - \Delta + \frac{3}{2}(g+\delta)\right) + \frac{\Theta(\Delta^2+\Theta)}{3\Delta^3\sqrt{\Phi}}\left(\Delta^2 + \frac{\Theta}{\Delta}\mathrm{tr}\hat{\mathfrak{J}}\right) + \\ + \frac{\Delta^2+\Theta}{3\Delta^2\sqrt{\Phi}}\left(\frac{\Theta^2}{\Delta^2} - \Delta\mathrm{tr}\hat{\mathfrak{J}}\right)\left(\mathrm{tr}\hat{\mathfrak{J}} + 3(g+\delta)\right), \tag{A23}$$

$$\Sigma_g := -\frac{1}{\Delta}\left(\mathrm{tr}\hat{\mathfrak{J}} + 2g + \delta\right) + \frac{3\delta(\Delta^2+\Theta)}{\Delta\sqrt{\Phi}}\Omega + \frac{\Delta^2+\Theta}{\Delta^2\sqrt{\Phi}}\left(\frac{\Theta^2}{\Delta^2} - \Delta\mathrm{tr}\hat{\mathfrak{J}}\right)\left(\mathrm{tr}\hat{\mathfrak{J}} + 2g + \delta\right), \tag{A24}$$

$$\Sigma_\delta := -\frac{1}{\Delta}\left(\mathrm{tr}\hat{\mathfrak{J}} + g + 2\delta\right) + \frac{3g(\Delta^2+\Theta)}{\Delta\sqrt{\Phi}}\Omega + \frac{(\Delta^2+\Theta)}{\Delta^2\sqrt{\Phi}}\left(\frac{\Theta^2}{\Delta^2} - \Delta\mathrm{tr}\hat{\mathfrak{J}}\right)\left(\mathrm{tr}\hat{\mathfrak{J}} + g + 2\delta\right), \tag{A25}$$

and where the term $\mathrm{tr}\hat{\mathfrak{J}}$ denotes the trace of the Jacobian matrix (A7) evaluated at the stationary point, Ω is defined by (A5), p is defined by (A6), Θ is defined by (A9), Δ is defined by (A14), and Φ is defined by (A15).

Thus, we can conclude that a (Poincaré-Andronov-)Hopf bifurcation exists, as long as condition (A22) is satisfied for the Hopf critical values (defined by $\rho = 0$ if and only if conditions (A18) and (A19) are satisfied as well) where all other parameters are fixed at their base case values.

A.4 Deriving Stability Conditions for the Poincaré-Andronov-Hopf Bifurcation

We have given conditions for the existence of periodic solutions in Appendix A.3, but have not said a word about the cycle's stability behavior. To do this we have to determine the orbit structure near the fixed point – defined by the vector field induced by system (1) – restricted to the center manifold, *C*, as described below.

In performing the center manifold reduction, we first have to transform the equilibrium (A4) to the origin, and then perform a linear transformation of the coordinates,

$$\begin{pmatrix} L \\ H \\ Y \end{pmatrix} \rightarrow \boldsymbol{T}\begin{pmatrix} L-\hat{L} \\ H-\hat{H} \\ Y-\hat{Y} \end{pmatrix} = \boldsymbol{T}\begin{pmatrix} l \\ h \\ y \end{pmatrix}, \tag{A26}$$

where

$$\boldsymbol{T} := \begin{pmatrix} \boldsymbol{e}_1 & i(\boldsymbol{e}_2 - \boldsymbol{e}_3) & i(\boldsymbol{e}_2 + \boldsymbol{e}_3) \end{pmatrix} \tag{A27}$$

denotes the matrix[12] of (complex) eigenvectors,

$$\boldsymbol{e}_j = \begin{pmatrix} e_{1j} \\ e_{2j} \\ e_{3j} \end{pmatrix} := \begin{pmatrix} \frac{1}{b}(g+\lambda_j)(\delta+\lambda_j) \\ \delta+\lambda_j \\ 1 \end{pmatrix}, \quad j = 1,2,3, \tag{A28}$$

associated with the (complex) eigenvalues, λ_j, $j = 1,2,3$, determined by Equations (A10) and (A11). Evaluated at the Hopf-bifurcation point ($\rho = 0$) we can re-write system (1) as follows [13]

$$\dot{l} = tr\hat{\mathfrak{J}}\, l - 2\omega\varphi(l,h,y) \tag{A29}$$

$$\begin{pmatrix} \dot{h} \\ \dot{y} \end{pmatrix} = \begin{pmatrix} 0 & \omega \\ -\omega & 0 \end{pmatrix} \circ \begin{pmatrix} h \\ y \end{pmatrix} + \begin{pmatrix} tr\hat{\mathfrak{J}} \\ \omega \end{pmatrix} \varphi(l,h,y) \tag{A30}$$

where ω represents the imaginary part of the conjugate complex pair of eigenvalues (see Equation (A13)), $tr\hat{\mathfrak{J}}$ indicates the trace of the Jacobian matrix (A7) evaluated at the equilibirum point (A4), and where

$$\varphi(l,h,y) = \overline{m}\left(pq(l+2y) + \tfrac{\tau}{\Omega}\left(\nu(l,h,y) - p\right) + \varsigma(l,h,y)\left(\nu(l,h,y) - p(1 - ln\, p)\right)\right), \tag{A31}$$

---

[12] Note that the matrix $\boldsymbol{T}$ corresponds to the real Jordan normal form, $\boldsymbol{J} \equiv \boldsymbol{T}^{-1}\hat{\mathfrak{J}}\boldsymbol{T}$.

[13] From now on ^{T} indicates transpose and $\circ$ indicates matrix multiplication.

$$\overline{m} := -\frac{sb}{2\omega\left(tr\hat{\mathfrak{J}}^2 + \omega^2\right)}, \tag{A32}$$

$$\nu(l,h,y) := Exp\left[-q\frac{l+2y+\frac{b\tau}{\delta g\Omega}}{\varsigma(l,h,y)+\frac{\tau}{\Omega}}\right], \tag{A33}$$

$$\varsigma(l,h,y) := \tfrac{1}{b}\left(\left(g+tr\hat{\mathfrak{J}}\right)\left(\delta+tr\hat{\mathfrak{J}}\right)l - 2\omega(g+\delta)h - 2\left(\omega^2 - g\delta\right)y\right). \tag{A34}$$

and where Ω is defined by Equation (A5), and p is defined by Equation (A6). Thus, a little algebra yields

$$\varphi(0,0,0) = 0\ ,\ \varphi_l(0,0,0) = 0\ ,\varphi_h(0,0,0) = 0\ ,\text{ and }\ \varphi_y(0,0,0) = 0\,. \tag{A35}$$

Hence, the (invariant) center manifold, C, can be locally represented by

$$C = \left\{\begin{pmatrix} l & \begin{pmatrix} h \\ y \end{pmatrix} \end{pmatrix}^T \in \Re\times\Re^2 \;\middle|\; l = \psi(h,y),\ \left|\begin{pmatrix} h \\ y \end{pmatrix}\right| < \varepsilon,\ \psi(0,0) = 0,\ D\psi(0,0) = 0\right\}, \tag{A36}$$

where $\psi(h,y)$ is approximated by a power series of order three,

$$\begin{aligned} &\psi(h,y) = \alpha_1 h^3 + \alpha_2 h^2 y + \alpha_3 h y^2 + \alpha_4 y^3 + \alpha_5 h^2 + \alpha_6 h y + \alpha_7 y^2 \\ &\psi(0,0) = 0,\quad \psi_h(0,0) = 0,\quad \psi_y(0,0) = 0. \end{aligned} \tag{A37}$$

According to Wiggins (1990), for instance, any point on C must satisfy the condition

$$\begin{aligned} D\psi(h,y)\left[\begin{pmatrix} 0 & \omega \\ -\omega & 0 \end{pmatrix}\circ\begin{pmatrix} h \\ y \end{pmatrix} + \begin{pmatrix} tr\hat{\mathfrak{J}} \\ \omega \end{pmatrix}\varphi(\psi(h,y),h,y)\right] \\ = tr\hat{\mathfrak{J}}\,\psi(h,y) - 2\varphi(\psi(h,y),h,y), \end{aligned} \tag{A38}$$

where φ is determined by (A31). Thus Condition (A38) allows to *numerically* determine the coefficients of the power series (A37) by comparison. Let us finally regard the cubic coefficient for higher-dimensional systems[14] determined by

$$\begin{aligned} \kappa := -\tfrac{sb}{32\omega^2\left(tr\hat{\mathfrak{J}}^2+\omega^2\right)}\Big(tr\hat{\mathfrak{J}}\omega\left(\overline{\varphi}_{hhh} + \overline{\varphi}_{hyy} + \overline{\varphi}_{yy}^2 - \overline{\varphi}_{hh}^2\right) + \omega^2\left(\overline{\varphi}_{hhy} + \overline{\varphi}_{yyy}\right)\Big) - \\ - \tfrac{sb\left(tr\hat{\mathfrak{J}}^2-\omega^2\right)}{32\omega^2\left(tr\hat{\mathfrak{J}}^2+\omega^2\right)}\overline{\varphi}_{hy}\left(\overline{\varphi}_{hh} + \overline{\varphi}_{yy}\right) < 0\,, \end{aligned} \tag{A39}$$

[14] The definition of κ for higher-dimensional systems is given by, e.g., Guckenheimer and Holmes (1983, p.152ff.)

where

$$\begin{aligned}\overline{\varphi}(h,y) := {} & 2pqy + \tfrac{\tau}{\Omega}\left(\overline{\nu}(h,y) - p\right) + \\ & + \left(pq + \tfrac{1}{b}\left(g + tr\hat{\Im}\right)\left(\delta + tr\hat{\Im}\right)\left(\overline{\nu}(h,y) - p(1 - ln\, p)\right)\right)\psi(h,y) + \\ & - 2\tfrac{1}{b}\left(\omega(g+\delta)h + \left(\omega^2 - g\delta\right)y\right)\left(\overline{\nu}(h,y) - p(1 - ln\, p)\right),\end{aligned} \tag{A40}$$

$$\overline{\nu}(h,y) := Exp\left[-q\,\frac{\psi(h,y) + 2y + \frac{b\tau}{\delta g\Omega}}{\frac{1}{b}\left(\left(g + tr\hat{\Im}\right)\left(\delta + tr\hat{\Im}\right)\psi(h,y) - 2\omega(g+\delta)h - 2\left(\omega^2 - g\delta\right)y\right) + \frac{\tau}{\Omega}}\right], \tag{A41}$$

and where the term $tr\hat{\Im}$ denotes the trace of the Jacobian matrix (A7) evaluated at the stationary point, Ω is defined by (A5), p is defined by (A6), ω is defined by (A13), and $\psi(h,y)$ is defined by (A35) according to (A36).

As long as conditions (A22) and (A39) are satisfied for the Hopf critical values (defined by $\rho = 0$ if and only if conditions (A18) and (A19) are satisfied as well) where all other parameters are fixed at their base case values, the emerging cycles are stable and we observe persistent oscillation.

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