Age-Structured Single-State Drug Initiation Model — Drug Epidemics and Optimization of Prevention Programs *

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Abstract

This paper introduces a model for drug initiation that extends traditional dynamic models by considering explicitly the age distribution of the users. On the basis of a 2-groups model in which the population is split up in an user and a non-user group the advantage of a continuous age distribution is shown by considering more details and by yielding new results. Neglecting death rates allows to reduce the model to a single state (1-group) descriptive model which is capable to simulate the complex behavior of drug epidemics. Furthermore, prevention programs – especially school-based programs – can be targeted to certain age classes. So in order to discover how best to allocate resources to prevention programs over different age classes we formulate and solve also optimal control models.

Keywords: Nonlinear dynamic system, age-structured model, illicit drug, partial differential equation, optimal control.

AMS subject classifications: 35L60, 49J20, 65M20, 81T80.

1 Introduction

Often models of drug initiation and drug use are based on the same principles as epidemiological models, because drug use is clearly contagious in the sense that use by some individuals affects the probability that others will use through multiple mechanisms. In a very literal sense, most users are introduced to a drug by a friend or relative; the more drug users there are, the more likely an individual is to be offered the drug [1]. At a market level, the larger the market, the more diluted the enforcement risk, and the safer it is to try drugs [2]. At a reputational level, experiences of others can be instrumental in shaping perceptions of the riskiness of drugs, and those perceptions in turn influence initiation. Indeed, the very fact that the mechanism of transmission does not involve physical contact or interaction means that the dynamics of contagion can be more complex and more interesting.

Looking at drug use and the process of initiation in more detail, it is clear that the decision of a non-user to start consumption depends strongly not only on the individual's immediate, personal social environment, but also on the overall reputation of a drug in society, e.g., as portrayed in movies or news media. That means, an individual might want to use drugs even if none of the individual's associates encourages that desire. And, conversely, an individual may fear drugs even if no one he or she knows has suffered harm from them.

The consideration of age brings up a more realistic model, because it is certainly true that behavior can depend on age. E.g., people between 13 and 25 years of age are much more likely to start drug consumption than are people over 40. It is also true that not all people have the same influence on a person of a certain age. That influence can depend on their age and/or the age difference. Furthermore, prevention programs – especially school-based programs – can be targeted to certain age classes.

A simple way to introduce age is to split the population into different age groups. This leads to compartment models but with a large number of population groups [3]. An analysis of such large models is difficult, and it is necessary to increase the number of age groups in order to get a better approximation. Therefore, a more general method is to include age as a second parameter in addition to time. So a continuous age distribution of the population can be fully taken into consideration and the model description and analysis is independent of the number of age groups for which data are available. The analysis of such a model becomes in some way easier, because there are fewer groups to consider, but in some sense also more complicated, because this method leads to a system of partial differential equations – a further development of the so-called McKendrick equation [4].

In epidemiology, several papers on age-structured models are available. Murray [5, pp. 640–650] describes a simple S-I model with an age-dependent infectiousness, which can be solved analytically. Busenberg et al. [6] and Iannelli et al. [7] investigate the global behavior and threshold properties of age-structured S-I-S models, and Müller [8, 9] concentrates on optimal vaccination patterns for age-structured S-I-R models, but without time-dependence of the control.

Although the epidemiological models are very similar to the model described in this paper, there are some essential features in drug initiation, which do not allow to transfer the results to the initiation models.

The paper is organized as follows. We start with the formulation of the descriptive model in section 2, followed by a description of the input parameters and functions and the explanation of the numerical algorithm used to solve the equations in section 3. This section includes also some numerical results and their interpretation. In section 4 the formulation is extended to an optimal control model, and two different theoretical methods coming from literature are applied to slightly simplified versions of the model. Furthermore, an interpretation of the necessary conditions and some numerical results for one of the models are provided. The paper ends with some general conclusions and an outlook to future work in section 5.

2 Model Formulation

In the model we consider in this paper the population is divided into two different groups: the non-users and the users (or drug consumers). Neglecting any death rates or migration and assuming constant birth cohort size the sum of the non-user and the user populations is always constant, and so it is sufficient to consider only one state (e.g., the non-user population).

Furthermore, only the flow from the non-user state into the user state is taken into consideration, which means that once an individual starts consuming drugs, he will never stop being a consumer. So in that sense the user population does not represent the actual number of consumers, but the number of people, who have ever used drugs in the past.

The following equation formally describes the non-users' dynamics in terms of $P(t,a)$ – the number of non-users aged a at time t – and $\mu(t,a)$ – the initiation rate:

$$P_t + P_a = -\mu(t,a)P(t,a) \tag{2.1}$$

with initial and boundary conditions

$$\begin{aligned} P(0,a) &= P_0(a), \\ P(t,0) &= k, \end{aligned} \tag{2.2}$$

for $t \in [0,T]$ and $a \in [0,\omega]$, where $P_0(a)$ is the initial population distribution at $t = 0$, and k is a fixed birth cohort size. (Choosing $k = 1$ allows to interpret $P(t,a)$ as the proportion of non-users.)

The rate of initiation $\mu(t,a)$ is assumed to depend on three different factors:

1. a basic, age-specific initiation rate $\bar{\mu}(a)$ representing the probability that a non-user starts drug consumption without any influence from others;

2. the influence $\Phi(R(t,a))$ of the reputation of the drug $R(t,a)$ on the initiation of a non-user of age a; and

3. a prevention factor $\Psi(w(t,a))$ incorporating the effects of age-specific prevention programs $w(t,a)$ on the initiation rate.

Summarizing, we have

$$\mu(t,a) = \bar{\mu}(a)\Phi\big(R(t,a)\big)\Psi\big(w(t,a)\big). \tag{2.3}$$

The function $R(t,a)$ represents the reputation of the drug at time t acting on a non-user of age a. It is useful to distinguish between a *global* (or social-wide) reputation $R_{\text{glob}}(t,a)$ of the drug, as portrayed in news media and movies, and a *local* (or personal) reputation $R_{\text{loc}}(t,a)$, representing the influence of the social environment of an individual. The global and local reputations are assumed to be

$$R_{\text{glob}}(t,a) = \quad m_1(a) \int_0^\omega m_2(a') \left(i_u \left(1 - \tfrac{P(t,a')}{k} \right) - (1 - i_u) \tfrac{P(t,a')}{k} \right) da', \tag{2.4}$$

$$R_{\text{loc}}(t,a) = \quad \int_0^\omega m_3(a,a') \left(i_u \left(1 - \tfrac{P(t,a')}{k} \right) - (1 - i_u) \tfrac{P(t,a')}{k} \right) da', \tag{2.5}$$

where the functions and constants have the following meaning:

$m_1(a)$ measures the degree of influence of the overall reputation of the drug on a non-user of age a. Usually it is assumed that it decreases with age, which means that older people have a fixed opinion and they do not accept a *popular* opinion as easily as younger people do.

$m_2(a')$ describes how important the users and non-users of age a' are for the overall reputation of the drug. It should be large for age groups which dominate the public opinion (representing the group of opinion leaders).

$m_3(a, a')$ indicates the influence of users and non-users of age a' on a non-user of age a. This influence depends primarily on the age difference. Especially for young non-users (who have the highest underlying proclivity to initiate into drug use) it is assumed, that persons who are of the same age or a little bit older set examples, therefore their influence is very high. On the other hand, persons who belong to their parents' generation may evoke a contrarian response or at least have only a small impact.

i_u measures the relative influences of the numbers users i_u and non-users $(1-i_u)$ on the reputation. It is necessary to have $i_u \in [0, 1]$ in order to avoid a reverse effect of users or non-users on the reputation. So choosing $i_u = 1$, the public opinion is dominated by the user group, setting $i_u = 0$, only the non-user group is responsible for the reputation, and for $i_u = 0.5$, the influence of one user on the initiation is compensated by one non-user.

The total reputation is a combination of the local and the global reputations which leads to

$$R(t, a) = \int_0^\omega m(a, a') \left(i_u - \frac{P(t, a')}{k} \right) da' \tag{2.6}$$

with

$$m(a, a') = i_{\text{loc}} m_3(a, a') + (1 - i_{\text{loc}})\, m_1(a) m_2(a'), \tag{2.7}$$

where $i_{\text{loc}} \in [0, 1]$ measures the influence of the local reputation and $(1 - i_{\text{loc}})$ that of the global reputation.

The function $\Phi(.)$ is a non-negative monotonically increasing function satisfying

$$\Phi(R) = \begin{cases} d \in [0, 1] & \text{for } R \to -\infty \\ 1 & \text{for } R = 0 \\ e \in [1, \infty) & \text{for } R \to \infty \end{cases} \tag{2.8}$$

meaning that zero reputation has no impact on the initiation, a very negative reputation reduces the initiation by $(1 - d) \cdot 100\%$, while a very positive reputation increases the rate by $(e - 1) \cdot 100\%$.

The function $\Psi(.)$ describing the influence of prevention on initiation is assumed to be of the form

$$\Psi(w(t,a)) = (1-c)e^{-\varepsilon w(t,a)} + c, \tag{2.9}$$

where $(1-c) \in (0,1)$ measures the maximal proportion of reduction of the initiation rate and ε is the efficiency rate of prevention.

Due to these complex equations the model is far from being solved analytically. So in the following section a numerical algorithm is developed, which allows to run simulation experiments on different data sets and to investigate the behavior of the system in different cases.

3 Numerical Simulation

Before starting with simulation experiments some functions have to be specified.

- $\bar{\mu}(a)$: Three different types of basic initiation are used to investigate different features of the model. The first type is constant, the second one is a step function with a constant positive initiation below the age of 25 years and zero initiation for older people. The third version is chosen according to data for marijuana initiation in the US. It has a sharp peak around the age of 16 years and is nearly zero outside the interval $(10, 25)$ (see figure 1).

- $m(a, a')$ describes the overall influence of the users of age a' on the non-users of age a. For $m_3(a, a')$ a form is used where the influence of people who are 2 years older is maximal, while it decreases to a negative value for users, who are more than 12 years older or more than 8 years younger. With increasing age difference the influence stays negative, but converges to zero. Also with increasing age of the non-user the absolute value $|m_3(a, a')|$ decreases. For the global part of the influence, $m_2(a')$ is chosen so that the age range from 20 to 30 years is the *leading group* with the largest influence on the overall reputation. $m_1(a)$ is monotonically decreasing in order to represent the fading influence on older people.

- $\Phi(.)$ measures the influence of the reputation term on the initiation rate. To satisfy equation (2.8) it is set to

$$\Phi(R) = d_1 + \frac{1}{d_2\pi}\arctan(d_3 * R), \tag{3.1}$$

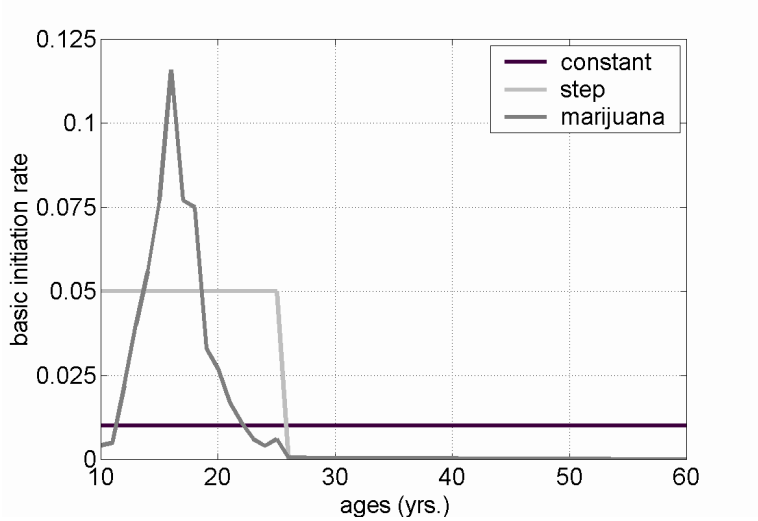

Figure 1: Different types of initiation rates $\bar{\mu}(a)$ used in the simulation experiments.

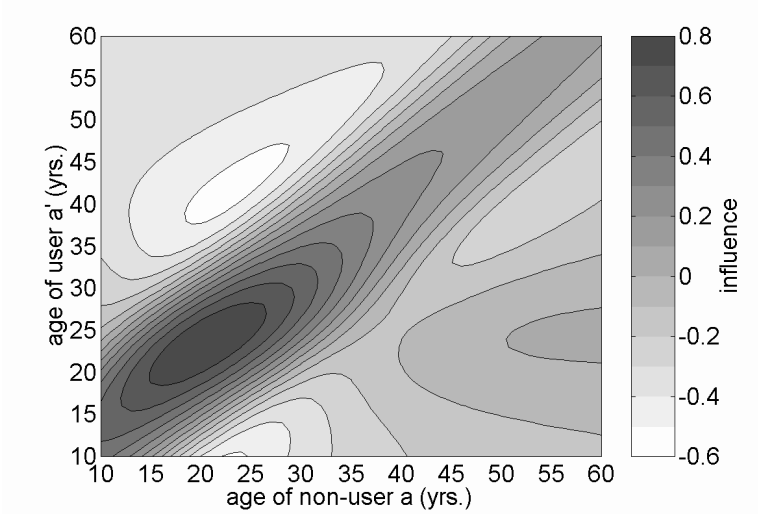

Figure 2: Influence $m(a, a')$ of the user group on the non-user group ($i_{\text{loc}} = 0.75$).

where d_1 (default value: 1) and d_2 (default value: 1) determine the decrease and increase of the initiation rate due to the drug's reputation, and d_3 represents the rate of response to changes of the reputation.

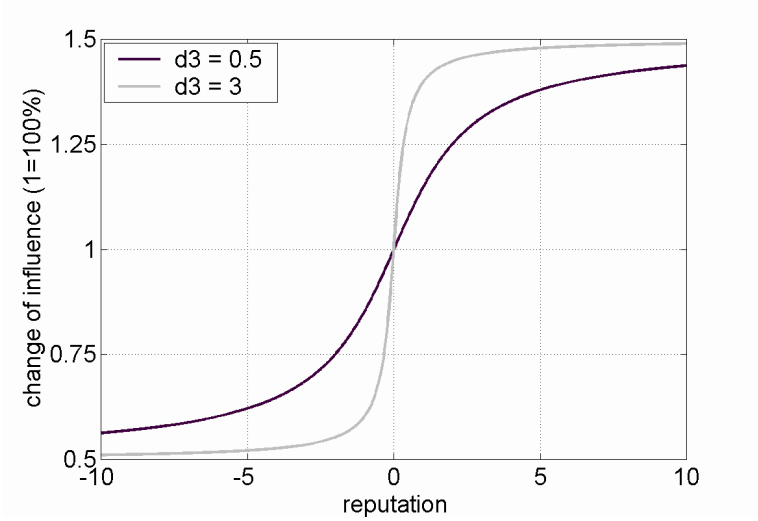

Figure 3: Change of the initiation rate depending on the reputation ($d_1 = 1$, $d_2 = 1$, $d_3 = 0.5, 3$).

In order to get results it is necessary to apply some numerical transformations. Using the method of lines [10] the partial differential equation (2.1) is rewritten as a system of ordinary differential equations by discretizing the age and substituting the partial derivatives w.r.t. a by finite approximations. Hence, instead of a continuous age variable a we have now a finite sequence of age classes $(a_i)_{i=0..N_a}$.

Remark. *The usual way to solve these equations would be the method of characteristics by reducing the PDEs to ODEs along the family $t = a + c$ of lines. This would avoid discretizing errors in this first step. But the calculation of integrals w.r.t. age — as they occur in the reputation term — require an interpolation between the grid points or a synchronous integration of the ODE equations with a certain shift depending on the starting point.*

So equation (2.1) becomes

$$\begin{aligned} &P_{a_0}(t) = k, \\ &P'_{a_i}(t) = -\boldsymbol{\Delta_a}\big[P_{a_i}(t)\big] - \mu_{a_i}(t) P_{a_i}(t) \quad i = 1, \ldots, N_a, \end{aligned} \tag{3.2}$$

where $\boldsymbol{\Delta_a}$ denotes an appropriate finite difference operator w.r.t. a.

The initial condition of equation (2.2) leads to initial conditions for the ODEs:

$$P_{a_i}(0) = P_0(a_i) \quad i = 1, \ldots, N - a. \tag{3.3}$$

The initiation rate is transformed analogously to individual initiation rates for each age class a_i

$$\mu_{a_i}(t) = \bar{\mu_{a_i}} \Phi \left(\mathbf{\Omega_a} \left[m_{a_i,a_j} \left(i_u - \frac{P_{a_j}(t)}{k} \right) \right] \right) \Psi \left(w_{a_0}(t), \ldots, w_{a_{N_a}}(t) \right), \quad (3.4)$$

where $\mathbf{\Omega_a}$ is the approximation function for the integral w.r.t. age. The *Newton-Cotes* formula can be used in this case such as the trapezoid rule for first-order or the Simpson rule for third order.

It is important to notice that the partial differential equation (2.1) is hyperbolic, which means that discontinuities are propagated with time along the a-axis. This fact must be taken into consideration for choosing an appropriate finite difference operator. As the propagation is directed towards the positive a-axis, the simplest operator is the first-order two-point upwind approximation. Suitable operators of higher order can be derived from the Taylor series. It is recommended to use a combination of upwind and centered approximations. At the boundaries slightly different approximations are used in order to avoid points outside the grid.

The simulation experiments described below were performed with the following parameters:

age = 10 - 60	(age range)
$k = 1$	(birth cohort size)
$P_0(a) \equiv 1$	(initial population distribution)
$w(t, a) \equiv 0$	(prevention)
$i_{\text{loc}} = 0.75$	(influence of local reputation)
$i_u = 0.9$	(user – non-user relation for the reputation)

In particular note that we start our experiments by neglecting any form of prevention, i.e., we are first interested in the uncontrolled dynamics of our model. In the case of the marijuana data the results of the simulation experiments show that the system converges either to a limit cycle (figure 4), if there is a high response to changes of the drug's reputation ($d_3 = 3$), or to a stable equilibrium (figure 5), if the response is low ($d_3 = 0.5$). We did not do any analytical investigations of this phenomenon, but for practical use of the results it makes not much difference if there occurs a real limit cycle, or the cycles fade out in 10000 years.

But the behavior of the solution depends not only on the steepness of the reputation function (see figure 3), but also on the form of the basic initiation rate $\bar{\mu}(a)$. In order to get cycles it is necessary to have age groups with high initiation rates, and others with very low or zero basic initiation. If $\bar{\mu}(a)$ is set constant for the whole age range, then the system always converges to an

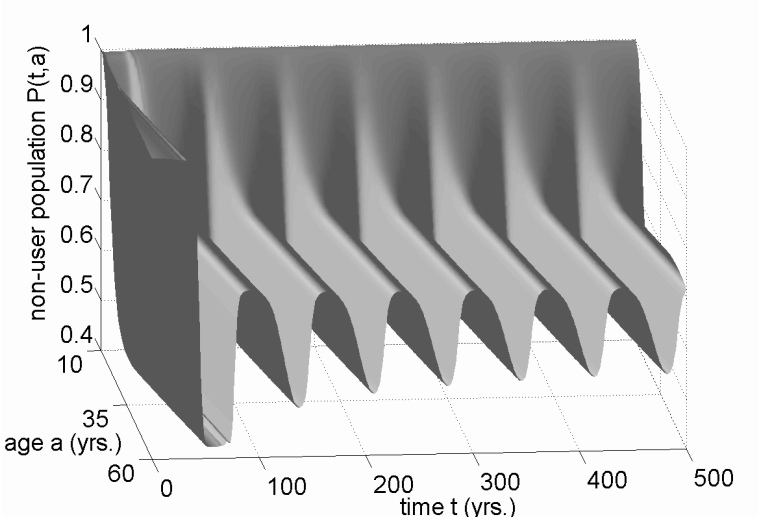

Figure 4: $P(t, a)$ in the case of a limit cycle ($d_3 = 3$) using marijuana data.

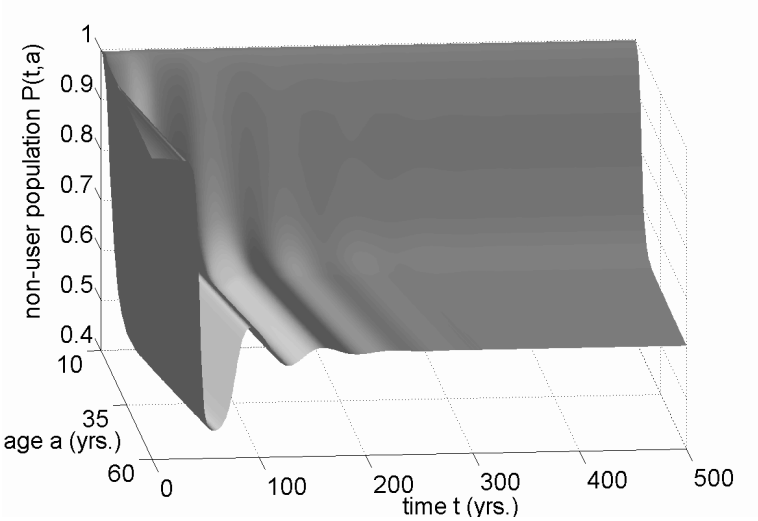

Figure 5: $P(t, a)$ in the case of an equilibrium ($d_3 = 0.5$) using marijuana data.

equilibrium. But if a simple step function is used (see figure 1), it is again possible to have cycles, where the exact behavior depends as before on the drug's reputation (cf. table 1).

The explanation for this behavior lies in the form of the influence function $m(a, a')$, because the negative influence of old users on young non-users can

block the high initiation rate of young people, if the group of older users is large enough (see figure 2). So the number of young users stays low, but if the initiation rate for older non-users is still high, the number of users will increase immediately when they grow too old to be influenced from others. Hence, cycles can only arise if the initiation rate for older individuals is low, so that this low number of users can travel through all age groups. This low number of old users cannot decrease the initiation rate of younger non-users, so a new wave of many users is created.

If the age range, in which the initiation is high, is decreasing the amplitude of the resulting waves gets very large. The application of different prevention programs can reduce this amplitude only marginally, but the number of users as a whole is reduced. The total social costs in the last column of table 1 suggest, that for a given total expenditure the effectiveness of different types of prevention depends on the allocation of resources over different age classes.

Therefore it is necessary to investigate the influence of the prevention factor and how to optimize it, which is the contents of the following section.

Test parameters			**Results**				
$\bar{\mu}(a)$[1]	d_3	$w(t,a)$[2]	LC[3]	U60[4]	MI[5]	aMI[6]	TSC[7]
const.	0.5	0	0	0.37	0.0077	43	0.58
const.	3	0	0	0.34	0.0095	47	0.55
step	0.5	0	0	0.49	0.033	10	1.22
step	3	0	62.5	0.36-0.51	0.043	21	1.26
mari.	0.5	0	0	0.39	0.078	16	0.96
mari.	3	0	71.4	0.28-0.46	0.096	16	1.00
mari.	0.5	step	0	0.38	0.075	16	0.94
mari.	3	step	71.4	0.26-0.44	0.093	16	0.98
mari.	0.5	init	0	0.37	0.071	16	0.92
mari.	3	init	71.4	0.26-0.42	0.081	16	0.96

[1] Three different forms for the basic initiation rates $\bar{\mu}(a)$ are used (const. – constant; step – constant between 0 and 25, otherwise 0; mari. – marijuana initiation; cf. figure 1).

[2] For the prevention $w(t,a)$ we tested three different cases, which are all independent of t (0 – no prevention; step – constant between 10 to 25, otherwise 0; init – prevention expenditure proportional to basic initiation rate between 10 and 25). The *step* and *init* forms are chosen to have the same total prevention expenditure.

[3] LC — limit cycle for the last age class (60 yrs.). The value in this column indicates the duration of the cycles (in years). If the system converges to an equilibrium, the entry is 0.

[4] U60 — the relative number of drug users at age 60.

[5] MI — the maximal incidence rate.

[6] aMI — age of maximal incidence rate.

[7] TSC — total social costs according to equation (4.1) relative to the 6th test(marijuana data, $d_3 = 3$, no prevention). The effectiveness of prevention programs w.r.t. the total social costs depends on the amount of social costs assumed to be generated by one drug user per year.

Table 1: Results of different simulation experiments.

4 Optimal prevention expenditures — applying Pontryagin's maximum principle on two model variants

Before starting investigations on optimal prevention programs it is necessary to define an objective which should be minimized (or maximized). In our case we have chosen the discounted total social costs as the sum of costs induced by the drug consumption and the prevention expenditures,

$$J = \int_0^T e^{-rt} \int_0^\omega \Big(\rho\big(k - P(t,a)\big) + w(t,a)\Big) da dt \longrightarrow \min_{w(t,a)}, \tag{4.1}$$

where ρ subsumes the social costs of one drug user. Of course, also other forms of the objective are possible, which, e.g., incorporate the incidence or the number of drug users.

In this section the maximum principle is applied to two simplified versions of the model with modified reputation terms, because existing methods described in literature are not general enough to cover the full model.

4.1 Interaction only with individuals of the same age

In this first case we take the control model (2.1)-(2.9), (4.1), but replace the interaction between users and non-users of different ages with the interaction between individuals only of the same age. So the integral in (2.6) vanishes and we have a new initiation rate of the form

$$\mu(t,a) = \bar{\mu}(a)\Theta(P(t,a))\Psi(w(t,a)) \tag{4.2}$$

where $\Theta(P(t,a))$ represents the reputation of the drug as it is transmitted by a-years old users and non-users to non-users of the same age.

The following theorem describes the necessary conditions of this simplified problem. (From now on we write the function arguments only if their omitting would cause misinterpretations.)

Theorem 1. *If $w^*(t,a)$ is the optimal control for the dynamic system described through (2.1), (2.2), (2.9), (4.1), and (4.2), then there exists functions $P^*(t,a)$ defined through (2.1), (2.2), (2.9), (4.2) and $q^*(t,a)$ defined through*

$$q_t^* + q_a^* = -1 + rq^* + \bar{\mu}\Psi(w^*)\big(P^*\Theta'(P^*) + \Theta(P^*)\big)q^* \tag{4.3}$$

together with

$$\begin{aligned} q^*(t,\omega_a) &= 0 \quad \textit{for } 0 \le t \le T \\ q^*(T,a) &= 0 \quad \textit{for } 0 \le a \le \omega_a \end{aligned} \tag{4.4}$$

and

$$w^* = \begin{cases} 0 & \textit{for } (1-c)\varepsilon\rho\bar{\mu}q^*P^*\Theta(P^*) \le 1 \\ \frac{1}{\varepsilon}\ln\big((1-c)\varepsilon\rho\bar{\mu}q^*P^*\Theta(P^*)\big) & \textit{otherwise} \end{cases} \tag{4.5}$$

which hold for $(t,a) \in [0,T] \times [0,\omega]$.

Proof. The above maximum principle can be proofed using the methods described in [11] and [12], where the optimal control problem is solved in a more general form.

The present value Hamiltonian for this problem is

$$H = -e^{-rt}\rho\,(k-P) - e^{-rt}w + \lambda\big(-P_a - \bar{\mu}\Theta(P)\Psi(w)P\big), \tag{4.6}$$

where λ denotes the costate variable. The adjoint equation is given by

$$\frac{\partial\lambda}{\partial t} = -e^{-rt}\rho + \lambda\bar{\mu}\Psi(w)\big(P\Theta'(P) + \Theta(P))\big) - \frac{\partial\lambda}{\partial a}. \tag{4.7}$$

Moving the last term $\partial\lambda/\partial a$ to the left hand side and substituting $\lambda = e^{-rt}\rho q^*$ yields equation (4.3). For calculating the maximum of the Hamiltonian $\max_w H$ all terms independent of $w(t,a)$ can be removed. So the remaining part is

$$F(w) := -w - \bar{\mu}\rho q^*P^*\Theta(P^*)\Psi(w). \tag{4.8}$$

The first and second order derivatives are (consider equation (2.9))

$$F'(w) = -1 + (1-c)\varepsilon\bar{\mu}\rho q^*P^*\Theta(P^*)e^{-\varepsilon w}, \tag{4.9}$$

$$F''(w) = -(1-c)\varepsilon^2\bar{\mu}\rho q^*P^*\Theta(P^*)e^{-\varepsilon w}. \tag{4.10}$$

Solving $F'(w) = 0$ gives the optimal control of (4.5), and verifying that it is a maximum, we have to distinguish between two different cases:

1. $q^* \le 0 \Rightarrow F'(w) < 0$, implying that $w^* = 0$ is a maximum on the boundary, or
2. $q^* > 0 \Rightarrow F''(w^*) < 0$, implying that w^* is a maximum in the interior.

□

The terms of the adjoint equation (4.3) can be interpreted in our drug initiation context. The adjoint variable $q^*(t,a)$ can be regarded as the marginal value at time t of the non-user population $P^*(t,a)$ of age a relative to the social costs ρ of a drug user. So the effect of an additional non-user in the next time step can be split up term for term:

1. $q_t^* + q_a^*$ is the change of valuation of a non-user. It is equal to the sum of the following terms.
2. $-1 + rq^*$ are the effects due to the social costs (-1) and the discount rate (rq^*).
3. $\bar{\mu}\Psi(w^*)\Theta(P^*)q^*$ is the effect of the additional initiation $\bar{\mu}\Psi(w^*)\Theta(P^*)$ valued with q^*, because an additional non-user is also a potential new user.
4. $\bar{\mu}\Psi(w^*)\Theta'(P^*)P^*q^*$ is the effect of the change of the initiation rate due to the additional non-user weighted with q^*.

4.2 Global reputation only ($i_{\mathbf{loc}} = 0$)

Going back to the original problem of (2.1)-(2.9), (4.1), but with $i_{\text{loc}} = 0$, means that we consider only the global reputation R_{glob}. So we have a simplified reputation function:

$$R_{\text{glob}}(t,a) = m_1(a) \int_0^\omega m_2(a') \left(i_u - \frac{P(t,a')}{k} \right) da', \tag{4.11}$$

and as initiation rate

$$\mu(t,a) = \bar{\mu}(a)\Phi(R_{\text{glob}}(t,a))\Psi(w(t,a)). \tag{4.12}$$

Now we need a new method in order to apply the maximum principle, which is described in the next theorem.

Theorem 2. *If $w^*(t,a)$ is the optimal control for the dynamic system described through (2.1), (2.2), (2.9), (4.1), (4.11), and (4.12), then there exists functions $P^*(t,a)$ and $R^*_{glob}(t,a)$ defined through (2.2), (2.9), (4.11), and (4.12) and $q^*(t,a)$ and $\theta^*(t)$ defined through*

$$q_t^* + q_a^* = -1 + rq^* + \bar{\mu}\Phi(R^*_{glob})\Psi(w^*)q^* + \frac{m_2}{k}\theta \tag{4.13}$$

$$\theta(t) = -\int_0^\omega \bar{\mu}(a)\Phi'(R^*_{glob}(t,a))m_1(a)\Psi(w^*(t,a))q^*(t,a)P^*(t,a)da \tag{4.14}$$

together with

$$\begin{aligned} q^*(t,\omega_a) &= 0 \quad \textit{for } 0 \le t \le T \\ q^*(T,a) &= 0 \quad \textit{for } 0 \le a \le \omega_a \end{aligned} \tag{4.15}$$

and

$$w^* = \begin{cases} 0 & \textit{for } (1-c)\varepsilon\rho\bar{\mu}q^*P^*\Phi(R^*_{glob}) \le 1 \\ \frac{1}{\varepsilon}\ln\left((1-c)\varepsilon\rho\bar{\mu}q^*P^*\Phi(R^*_{glob})\right) & \textit{otherwise} \end{cases} \tag{4.16}$$

which hold for $(t,a) \in [0,T]\times[0,\omega]$.

Proof. In [13] a more general control problem is solved. So we can use those results to proof the theorem, but we have to do some transformations, because in [13] a minimizing instead of a maximizing problem was the starting basis.

Using our notation, the adjoint equations for the minimizing problem can be written as

$$\begin{aligned} \tilde{q}(t,a) &= \int_t^T -e^{-r\tau}\rho - \bar{\mu}\Phi\left(R^*_{\text{glob}}(\tau,a)\right)\Psi\big(w^*(\tau,a)\big)\tilde{q}(\tau,a) - \frac{m_2}{k}\tilde{\theta}(\tau)d\tau, \\ \tilde{q}(T,a) &= 0, \\ \tilde{\theta}(t) &= -\int_0^\omega \bar{\mu}(a)\Phi'\left(R^*_{\text{glob}}(t,a)\right)m_1(a)\Psi\big(w^*(t,a)\big)P^*(t,a)\tilde{q}(t,a)da. \end{aligned} \tag{4.17}$$

Transforming the integral equation for $\tilde{q}$ into a differential equation and substituting $\tilde{q} = -e^{-rt}\rho q$, we get equations (4.13) and (4.14).

For calculating the optimal control $w^*(t,a)$ it is necessary to maximize

$$F(w) := -w - \bar{\mu}\rho q^*P^*\Phi(m_1\bar{R}^*)\Psi(w). \tag{4.18}$$

The same arguments as in the previous model hold here, too. So the optimal control of (4.16) maximizes the above function. □

Remark. *Applying this method of Brokate [13] to the simplified model, where interaction takes place only between individuals of the same age, only slight changes of the proof are necessary to get the same results.*

The interpretation of the necessary conditions is pretty much the same as in the previous case. The integral term here represents the effects of the change of the initiation rate, due to the change of the reputation.

Numerical Results

Exemplarily we discuss some numerical results for theorem 2, but the results for the simpler model of section 4.1 do not differ very significantly from those presented here, because due to out parameter choice for these experiments the influence of the reputation term on the model dynamics is low.

To solve the initial-boundary value problem described through the model equations and the necessary conditions of theorem 2 again the method of lines is used to transform it into an ordinary boundary value problem by discretizing the age. Furthermore, a finite difference method of the *NAG Fortran Library* was used to solve those systems of ODEs.

With some exceptions the same parameters as for the simulation experiments described in section 3 are used. Those exceptions are:

$P_0(a) = \text{equil.}$	(initial values are set equal to the equilibrium)
$i_{\text{loc}} = 0$	(due to the requirements of theorem 2)
$d_3 = 2$	(high response to changes of the drug's reputation)
$\bar{\mu}(a) = \text{marij./step}$	(two variants of the basic initiation rate, cf. table 1)

The results of the numerical experiments are unspectacular, because the effectiveness of prevention was assumed to be very low, i.e. the parameter ε in equation (2.9) is very low and $c = 0.84$, which provokes a maximal possible reduction of the initiation rate of 16%. Nevertheless the application of prevention reduces not only the number of users, but also the total social costs. In the case of the marijuana data, the optimal prevention program reduces the total social cost by 2% while the number of users can be decreased by about 5% (cf. table 2).

$w(t,a)$	U60	MI	aMI	TSC
0	0.29	0.057	16	1.000
step	0.28	0.055	16	0.993
init	0.27	0.052	16	0.983
optimal	0.27	0.051	16	0.981

Table 2: Comparison of results using optimal prevention and heuristic or no prevention. (For a description of the table entries see the footnotes of table 1)

Due to the instant effect of prevention programs on the initiation rate without any time delay, the optimal prevention expenditure follows more or less the basic initiation rate. Because of the discount rate and the finite time horizon the total sum of the prevention expenditures over all ages decreases

with time. Close to the terminal time it is not useful to do any prevention at all, because there is no salvage value assumed and therefore it is too costly in comparison with the savings due to a lower number of drug users (see figures 6 - 8 for further details).

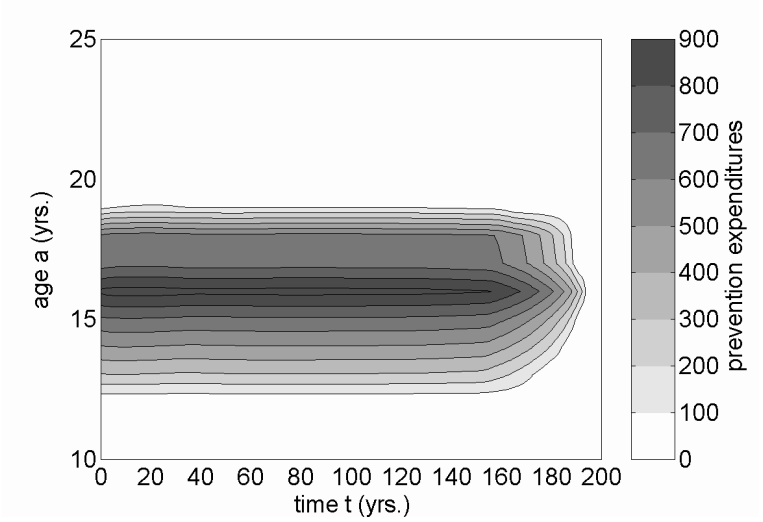

Figure 6: Optimal allocation of resources to prevention programs over different age classes over a time horizon of 200 years (using the marijuana data for the basic initiation rate).

The application of a prevention program leads to a significant reduction of the number of young drug users of about 7-8% within a few years. The older age groups change in the same way, but according to the age difference many years later. At the end of the planning horizon, the number of young users increases again and ends up at an even higher level than at the beginning, while the older groups keep staying at lower levels (see figure 10). The reason for this strong rise of younger drug users is not only the fact that prevention does not pay off at the end, but also the very low number of drug users in older age groups, which causes a higher reputation of the drug than at the beginning.

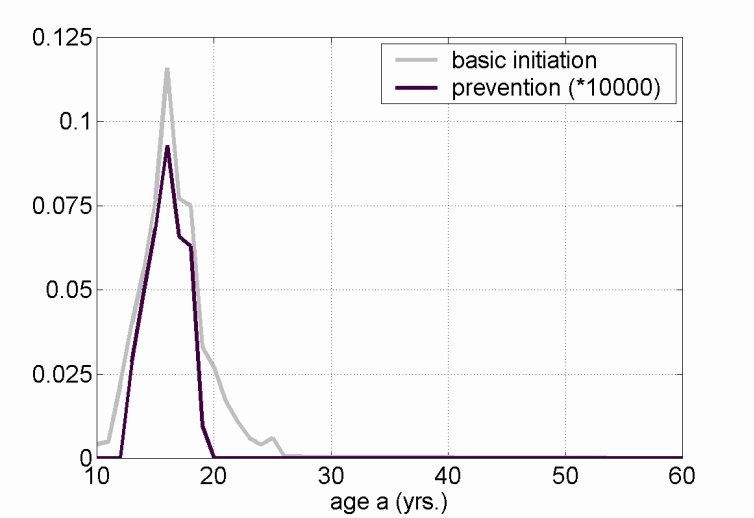

Figure 7: Optimal allocation of resources to prevention programs over different age classes at $t = 100$ years (using the marijuana data for the basic initiation rate, cf. figure 6).

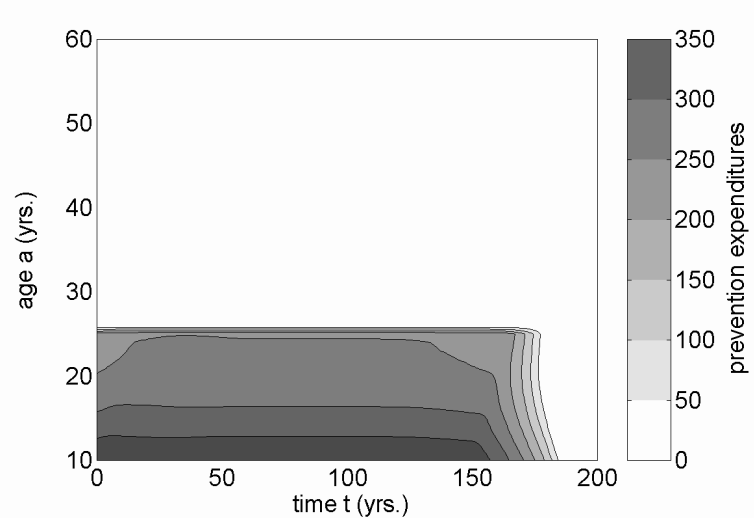

Figure 8: Optimal allocation of resources to prevention programs over different age classes over a time horizon of 200 years (using a constant basic initiation rate for the ages 10 to 25 and 0 otherwise).

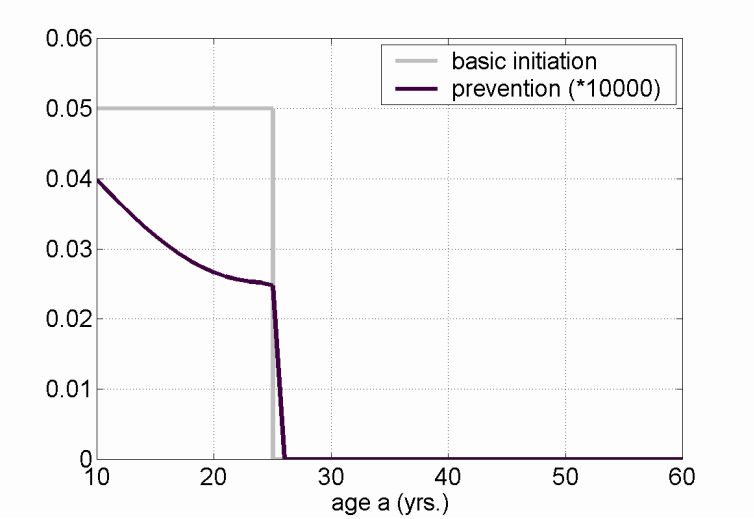

Figure 9: Optimal allocation of resources to prevention programs over different age classes at $t = 100$ years (using a constant basic initiation rate for the ages 10 to 25 and 0 otherwise, cf. figure 8).

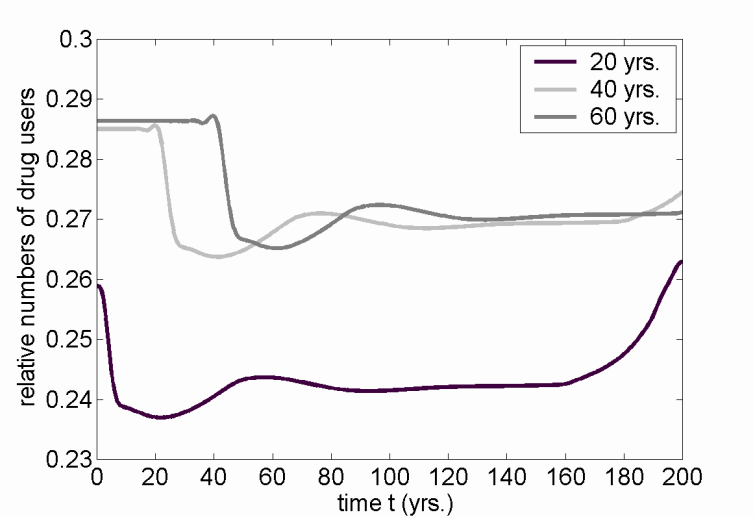

Figure 10: Trends of relative numbers of drug users in 3 different age classes over the whole planning horizon.

5 Conclusions

Introducing age-specific aspects to drug initiation models allows to develop more complex models that give detailed insights into the principles of drug epidemics and their control. The simple single-state model presented in this paper already shows a big advantage of age-structured models: the complex dynamics of a drug epidemic can be simulated using a model based on simple, manageable assumptions, such as distinguishing between just two groups of people. The behavior of the solution (cycles or equilibrium) depends on the type of age-specific feedback. To gain similar results with traditional multi-state models that do not differentiate by age a larger number of groups would be necessary.

In particular, the age-specific concept allows one to incorporate an age-specific reputation effect (feedback from number of users on the initiation rate), which depends on how much influence a user of age a' has on a non-user of age a, and whether this influence is positive or negative.

Another advantage of age-specificity is the ability to investigate age-structured strategies for prevention programs and to optimize them. The results of the control models are not very spectacular, but they show that it is very important to choose prevention programs according to the problem, because otherwise the produce only higher costs with only a low efficiency. An optimal program causes, besides a lower number of drug consumers, also decreasing social costs. Although the control model introduced in this paper consider only an instantaneous effect of prevention on the initiation rate without a persistent impact, the development of formal models considering other types of prevention is straightforward. But already now the existing maximum principles are not sufficient enough to allow to the application on the model with the full feedback, and increased complexity makes analytical investigations even more complicated.

The next steps for further developments are the formulation of an extended maximum principle for the full feedback and the introduction of more age-specific details. For instance, the efficiency rate and the maximal reduction rate of the initiation due to prevention (ε and c in equation (2.9)) can be regarded as age-specific, so that prevention is more effective in younger age classes. Further extensions would be to use finer group classifications or to introduce the duration of use as a third parameter, because some aspects of drug initiation depend on how long an individual has been in his/her current group. For instance, the probability of an individual to move from light to heavy use may be related to the duration of his/her drug consumption.

In summary, the age-specific model presented in this paper allows more detailed insights into the dynamics of drug epidemics than models neglecting

age-specificity, and by formulating it as an optimal control model it can be used to increase the positive effects of prevention programs by improving their targeting.

References

[1] J. Kaplan. *The Hardest Drug: Heroin and Public Policy*. The University of Chicago Press, 1986.

[2] M.A.R. Kleinman. Enforcement swamping: A positive feedback mechanism in rates of illicit activity. *Math. Comput. Modelling*, 27(2):67–95, 1993.

[3] G.W. Swan. *Applications of Optimal Control Theory in Biomedicine*. Dekker, 1984.

[4] B.L. Keyfitz, N. Keyfitz. The McKendrick partial differential equation and its uses in epidomology and population studies. *Math. Comput. Modelling*, 25(6):1–9, 1997.

[5] J.D. Murray. *Mathematical Biology*. Springer-Verlag, 2nd edition, 1993.

[6] S.N. Busenberg, M. Iannelli, H.R. Thieme. Global behavior of an age-structured epidemic model. *SIAM J. Math. Anal.*, 22(4):1065–1080, 1991.

[7] M. Iannelli, F.A. Milner, A. Pugliese. Analytical and numerical results for the age-structured S-I-S epidemic model with mixed inter-intracohort transmission. *SIAM J. Math. Anal.*, 23(3):662–688, 1992.

[8] J. Müller. Optimal vaccination patterns in age-structured populations. *SIAM J. Appl. Math.*, 59(1):222–241, 1998.

[9] J. Müller. Optimal vaccination patterns in age-structured populations: Endemic case. *Math. Comp.*, 31:149–160, 2000.

[10] W.E. Schiesser. *The Numerical Method of Lines*. Academic Press, 1991.

[11] N. Derzko, S.P. Sethi, G.L. Thompson. Distributed parameter systems approach to the optimal cattle ranching problem. *Optimal Control Appl. Meth.*, 1:3–10, 1980.

[12] J. Muzicant. *Systeme mit verteilten Parametern in der Bioökonomie — Ein Maximumprinzip zur Kontrolle altersstrukturierter Modelle.* PhD thesis, Vienna University of Technology, 1980.

[13] M. Brokate. Pontryagin's principle of control problems in age-dependent population dynamics. *J. Math. Biology*, 23:75–101, 1985.