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Optimal Prevention and Treatment in a Dynamic Drug Model with Diverse Feedbacks and Relapse

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Abstract

This thesis aims at modelling and analysing the implications of potential relapse in a dynamic drug model. More precisely, an optimal control model incorporating the two states "drug users" and "teetotallers" is formulated. The main feature of the model is the fact that individuals quitting drug use do not simply leave the system but rather end up in the precarious state of a teetotaller with a certain risk of relapse. Relapsing teetotallers constitute a second inflow of drug users in addition to ordinary initiation. However, teetotallers are also assumed to have a dissuasive effect on initiation. Consequently, the number of teetotallers has both a positive and a negative effect on the overall drug problem. Moreover, the dynamical system is influenced by the controls "prevention" and "treatment". Especially the use of treatment in consideration of high relapse rates is of substantial interest.

After an introductory exploration of the underlying uncontrolled dynamics, Pontryagin's Maximum Principle is applied in order to solve the optimal control model. As a consequence of the model's complexity, the optimal solution and stable paths are calculated numerically using the MATLAB-toolbox OCMAT. Furthermore, a sensitivity analysis, describing the impact of variations of model parameters on the long-run solution, is conducted.

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Chapter 1

Introduction

Even though there are numerous works dealing with the mathematical modelling and the optimization of drug dynamics, there seem to be few models incorporating directly the occurrence of relapse. Nonetheless, relapse is a very prominent feature of addiction: According to the National Institute on Drug Abuse (NIDA), the relapse rate of drug users ranges from 40% to 60% (see [13]). However, for certain drugs like Alcohol, Heroin, or Crack Cocaine, relapse rates can be much higher (see [5]). But what is the reason for that? The main cause is that addiction is not simply a bad habit which can be aborted at will, but rather a disease. The NIDA describes addiction as follows:

"Many people do not understand why or how other people become addicted to drugs. It is often mistakenly assumed that drug abusers lack moral principles or willpower and that they could stop using drugs simply by choosing to change their behavior. In reality, drug addiction is a complex disease, and quitting takes more than good intentions or a strong will. In fact, because drugs change the brain in ways that foster compulsive drug abuse, quitting is difficult, even for those who are ready to do so."[14]

Even when a drug user receives professional treatment, a successful cure is not guaranteed. Nevertheless, according to the NIDA the frequent occurrence of relapse does not suggest uselessness of treatment measures:

"The chronic nature of the disease means that relapsing to drug abuse at some point is not only possible, but likely. Relapse rates (i.e., how often symptoms recur) for people with addiction and other substance use disorders are similar to relapse rates for other well-understood chronic medical illnesses such as diabetes, hypertension, and asthma, which also have both physiological and behavioral components. Treatment of chronic

diseases involves changing deeply imbedded behaviors, and relapse does not mean treatment has failed. For a person recovering from addiction, lapsing back to drug use indicates that treatment needs to be reinstated or adjusted or that another treatment should be tried."[13]

However, in a world with limited resources and especially scarcity of money, the question arises if treatment measures make sense economically in case of high relapse rates. In other words, what is more expensive: more drug users or more treatment expenditures in the light of potential relapse. From a moral point of view, it can be argued that money should not play a role and addicts should be treated in the best possible way in order to give them the highest chance of recovery. As mentioned above, addiction can be seen as a disease, hence society typically pays the corresponding costs (at least in Central Europe). On the other hand, it can also be deemed immoral to impose costs on society for the treatment of addicts when chances of cure are small and the addiction is considered to be more or less the fault of the addicts themselves. After all, one does not get addicted to substances without consuming them, and one may assume that this consumption is based on a free will.

Fortunately, this thesis is not concerned with moral considerations, which cannot be answered satisfactorily anyway. Instead, it focuses solely on the financial aspects of drug use and the measures against it. The central question that will be discussed is the following: What level of treatment is reasonable when relapse is frequent, given the costs of therapy and the costs arising from drug use? In order to answer that question, an optimal control problem, based on a proposal by Fouad El Ouardighi, will be formulated and examined.

The thesis is organised as follows: In Chapter 2 the model formulation will be introduced and motivated. Moreover, a baseline parametrisation will be chosen. In Chapter 3 the state equations without governmental intervention will be analysed. This serves the purpose of getting a general understanding of the underlying dynamics. A special case with simplifying parameters will be considered where stability properties can be derived analytically. For the general case, phase portraits give a good idea of the analytic properties. Moreover, the obtained results also give an understanding of the dynamics with fixed controls rather than only explaining a zero-control policy. In Chapter 4 the optimal solution of the control problem will be calculated and illustrated. In Chapter 5

an extensive sensitivity analysis will be conducted, illustrating the impacts of the model parameters on the long-run solution. Finally, a conclusion will be given in Chapter 6. The source codes used for the numerical calculations can be found in Appendix A.

Chapter 2

Setting up the Model

An optimal control problem primarily consists of two components. First, an objective function that should be minimised or maximised and second, system dynamics which influence the objective function and in return are influenced by the controls as described in [6]. I start building the model with the latter.

2.1. The Dynamical System

The course of the drug epidemic is described by two differential equations, which model the behaviour of the two states: the number of drug users $A(t)$ and the number of teetotallers $T(t)$ at time t. The evolution of the states is governed primarily by three terms:

- initiation,
- desistance, and
- relapse.

In terms of initiation, it is assumed that the number of current drug users is crucial for further initiation, since most initiates are tempted into using drugs by other users, as stated in [8]. Consequently, more users promoting drug use imply heavier initiation at the beginning of an epidemic. However, according to [12] this changes when drug use becomes rampant and its negative effects come forward. In that situation initiation shall become a decreasing function of the number of users. Apparently a logistic function with a carrying capacity a serves this purpose well. For the sake of more modelling flexibility I insert the exponent ω. Additionally, former drug users (similar to heavy users in [1])

constitute a dissuasive force as they clearly reveal the hardships of overcoming an addiction. Instead of simply dividing by the number of teetotallers, I choose a more general approach by multiplying with $T(t)^\gamma$ with a negative exponent γ. Thus, the role of the dissuasive influence of teetotallers can be examined more deeply later on.

Desistance is described by the outflow of a constant fraction δ of drug users.

Relapse, finally, depends positively on both consumers and teetotallers. Drug consumers, just like in initiation, have a persuasive power over teetotallers. However, this persuasion is not affected by an epidemic outbreak of addiction ravaging society since teetotallers have entirely different reasons for taking drugs again than completely new consumers. Putting all parts together, the following equation is obtained for the development of the number of drug users:

$$\dot{A}(t) = \alpha A(t)^\omega T(t)^\gamma \left(a - A(t)\right) - \delta A(t) + \beta A(t)^{b_1} T(t)^{b_2},$$

where α and β are positive proportionality constants and b_1 and b_2 are the positive exponents in the *Cobb-Douglas*-type function constituting the core of the relapse term.

Drug users who quit consumption become teetotallers. Therefore, the outflow of A is the inflow of T. The number of relapsing teetotallers has already been discussed above. Last but not least, it is reasonable to assume that teetotallers do not stay at risk of relapsing for the rest of their lives. So there is an additional outflow at a rate ρ:

$$\dot{T}(t) = \delta A(t) - \beta A(t)^{b_1} T(t)^{b_2} - \rho T(t).$$

That would be the dynamics of A and T in a world without governmental intervention. The two controls considered in this thesis are prevention $p(t)$ and treatment $w(t)$. More precisely, $p(t)$ is the amount of money spent for prevention measures, whereas $w(t)$ describes the effectiveness of treatment. Following [1], prevention reduces initiation by a certain percentage which is dependent on the money $p(t)$ put into prevention methods. This process is described by the function

$$\Psi(p(t)) = h + (1 - h)e^{-mp(t)}.$$

For example, $\Psi(x) = 0.90$ implies that, at an effort of x, initiation will be reduced by

10%. In other words, initiation is brought down to a level of 90% of its uncontrolled value. Moreover, the effectiveness of prevention spending is assumed to be bounded by h:

$$\lim_{p\to\infty} \Psi(p) = h.$$

This means that even with infinite resources, initiation can only be lowered by $(1-h)\times 100\%$.

For treatment it is assumed that $w(t) = y$ implies an additional outflow of consumers at the rate y. However, it is assumed that the total outflow rate of drug users $w(t) + \delta$ is bounded from above by 1: This assumption stems from the fact that the government cannot reach every user directly after the initiation of drug consumption. Most drugs do not show their destructive properties instantaneously, so a new user will not seek help at once. Given an outflow rate of y, the average time spent in the corresponding state is $1/y$. Consequently, allowing too high outflow rates would lead to unrealistically short resting times. The resulting constraint for treatment is therefore $w(t) \leq 1 - \delta$. Hence, the state dynamics including the two controls are given by

$$\begin{aligned}
\dot{A}(t) &= \alpha A(t)^{\omega} T(t)^{\gamma} \left(a - A(t)\right) \Psi(p(t)) - (\delta + w(t))A(t) + \beta A(t)^{b_1} T(t)^{b_2}, \\
\dot{T}(t) &= (\delta + w(t))A(t) - \beta A(t)^{b_1} T(t)^{b_2} - \rho T(t).
\end{aligned}$$

Obviously the numbers of drug users and teetotallers should not be negative. However, a non-negativity condition is not needed as the dynamics of A and T imply that, once positive, A and T cannot become negative. If the carrying capacity a is exceeded ($A(t) > a$), initiation becomes negative and turns from being an inflow to being an outflow. In that case, spending money on prevention would, contrary to common sense, result in a lower outflow of consumption. So the optimal control has to be no prevention at all if $A(t) > a$. In reality, this feature of prevention lowering the decrease of A does not make a lot of sense on the one hand. On the other hand, why should a policy maker invest scarce money into prevention, when there is no positive initiation at all? This is similar to the results in [1] where prevention is zero when there are many heavy users and therefore have a strong dissuasive influence on initiation. Therefore, one could say that this behaviour (not investing money when there is no positive initiation at all) is a reasonable property of the model.

2.2. The Objective Function

The decision maker's objective is the minimisation of social costs caused by the use of drugs. Firstly, costs arise from the drug consumption itself which is assumed to be proportional to the number of drug users. Secondly, the decision maker has to set the controls to intervene and steer the course of the drug epidemic, which also causes costs. The parameter c denotes the average annual costs per user. The costs of prevention measures are simply the control itself, $p(t)$. For the second control, treatment efficiency $w(t)$, a convex cost function is assumed:

$$C(w) = f_0 w + \frac{f_1 w^2}{2}.$$

The objective is to minimise the stream of the discounted overall costs:

$$\min_{p(\cdot),w(\cdot)} \int_0^\infty e^{-rt} \left(cA(t) + p(t) + f_0 w(t) + \frac{f_1 w(t)^2}{2} \right) dt$$

An infinite time horizon and a positive discount rate r are chosen. The discounting formalises the human nature of not caring so much about future costs.

2.3. The Optimal Control Problem

Summing up, the following model was derived:

$$\min_{p(\cdot),w(\cdot)} \int_0^\infty e^{-rt} \left(cA(t) + p(t) + f_0 w(t) + \frac{f_1 w(t)^2}{2} \right) dt$$

subject to

$$
\begin{aligned}
&\dot{A}(t) = \alpha A(t)^{\omega} T(t)^{\gamma} \left(a - A(t)\right) \Psi(p(t)) - \left(\delta + w(t)\right) A(t) + \beta A(t)^{b_1} T(t)^{b_2} \\
&\dot{T}(t) = \left(\delta + w(t)\right) A(t) - \beta A(t)^{b_1} T(t)^{b_2} - \rho T(t) \\
&A(0) = A_0 \geq 0 \\
&T(0) = T_0 \geq 0 \\
&p(t) \geq 0 \\
&1 - \delta \geq w(t) \geq 0 \\
&a, r > 0 \\
&c, f_0, f_1, \alpha, \omega, b_1, b_2, \delta, \beta, \rho, m, h \geq 0 \\
&\gamma \leq 0
\end{aligned}
$$

with $\Psi(p(t)) = h + (1 - h)e^{-mp(t)}$.

2.4. Parametrisation

The baseline parameter values used in this thesis are listed in Table 2.1. r, a, α are taken from [4]. ω and γ are chosen so that initiation is similar to [4] so that the choice of α makes sense. Parameters m and h are taken from [1]. In [3] it is assumed that heavy and light users quit at a rate of 0.062 and 0.163, respectively. I assume the approximate average of 0.11 for the quitting-parameter δ. Regarding relapse, according to [11] $40\% - 60\%$ of teetotallers that received treatment relapse, so I assume $\beta = 0.5$. Furthermore, $b_2 = 1$, $b_1 = 0.05$, and $\rho = 0.10$ are assumed.

[10] calculated the economic cost of heroin addiction which is, subtracting treatment costs, approximately US$ 21 010 millions in 1996 with 600 000 heroin addicts. This means that the cost of one addict is approximately US$ 35 000. However, consumption of heroin is known to be particularly devastating to the body compared to other drugs, so I choose a value of $c = 10000$. This value is more in line with for example [2].

Lastly, the cost function of treatment has to be estimated. For example in [15] and [1] the outflow of treatment is modelled as $-c\left(\frac{u}{A+\varepsilon}\right)^z$, where u is the amount of money invested in treatment measures and A is the number of drug users. Using this thesis'

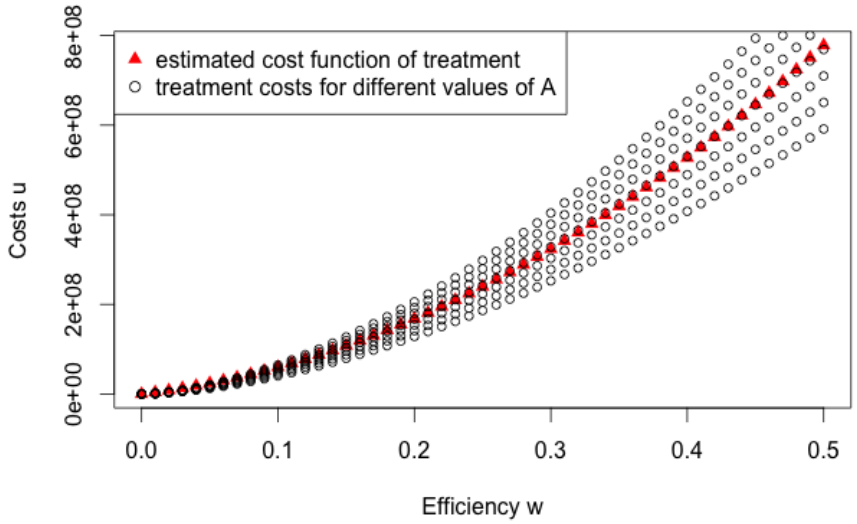

Figure 2.1.: Regression of cost function

notation of w as the additional outflow because of treatment, this means

$$w = c\left(\frac{u}{A+\varepsilon}\right)^z$$
$$\Leftrightarrow u = (A+\varepsilon)\left(\frac{w}{c}\right)^{\frac{1}{z}}.$$

However, we want $u = f_0 w + \frac{f_1 w^2}{2}$. Using a least squares regression for different values of A, as illustrated in Figure 2.1, and taking the mean of the respective parameters, estimates of f_0 and f_1 are obtained, where the corresponding values of c, ε, z are taken from [15].

Table 2.1.: Baseline parameter values

Parameter	Value	Description
a	16 250 000	carrying capacity
r	0.04	annual discount rate (time preference rate)
c	10 000	social cost of drug consumption
f_0	3.618569×10^8	coefficient in treatment cost function
f_1	4.778180×10^9	coefficient in treatment cost function
α	1.581272×10^{-8}	initiation rate proportionality constant
ω	1	constant measuring the persuasive influence of drug users on initiation
γ	-0.05	constant measuring the dissuasive influence of teetotallers on initiation
b_1	0.05	constant measuring the persuasive influence of drug users on relapse
b_2	1	constant measuring the persuasive influence of teetotallers on relapse
δ	0.11	rate at which users quit
β	0.5	proportionality constant of relapse
ρ	0.10	rate at which teetotallers quit their precarious status
m	2.37×10^{-9}	constant measuring efficiency of prevention spending
h	0.84	minimum percentage of baseline to which initiation can be cut by prevention

Chapter 3

The Uncontrolled System

I start the analysis of the dynamical system of drug consumers and teetotallers by assuming that there is no intervention of any social planner. This means $p(t) = w(t) = 0, \forall t$. Setting p equal to zero results in $\Psi(p(t)) = 1$. Hence, the system rewrites:

$$\begin{aligned}
\dot{A}(t) &= \alpha A(t)^{\omega} T(t)^{\gamma} (a - A(t)) - \delta A(t) + \beta A(t)^{b_1} T(t)^{b_2} \\
\dot{T}(t) &= \delta A(t) - \beta A(t)^{b_1} T(t)^{b_2} - \rho T(t) \\
A(0) &= A_0 \geq 0 \\
T(0) &= T_0 \geq 0
\end{aligned}$$

The calculation of steady states as well as further analyses of the system are non-trivial due to the rather complex functional forms. First I will explain a simplified version of the model with convenient parameters. Afterwards I will discuss the general case.

3.1. The Simple Case $b_1 = 0, b_2 = 1, \gamma = 0, \omega = 1$

By setting $b_1 = 0, b_2 = 1, \gamma = 0, \omega = 1$ the following system is obtained:

$$\dot{A}(t) = \alpha A(t) (a - A(t)) - \delta A(t) + \beta T(t) \tag{3.1}$$

$$\dot{T}(t) = \delta A(t) - \beta T(t) - \rho T(t) \tag{3.2}$$

$$A(0) = A_0 \geq 0 \tag{3.3}$$

$$T(0) = T_0 \geq 0 \tag{3.4}$$

$\gamma = 0$ implies that teetotallers have no negative influence on initiation. Similarly, $b_1 = 0$ means that drug users have no influence on the relapse of teetotallers. This could be

interpreted as a retreat from social interaction by teetotallers. In order not to relapse, the teetotallers avoid allurement by current consumers. Therefore, they are to some extent isolated and consequently have no effect on initiation as mentioned before. Finally, $b_2 = 1$ means linear dependence of relapse on the number of teetotallers, while $\omega = 1$ implies that initiation is described by a logistic growth function.

3.1.1. Steady States

Setting $\dot{A}(t) = \dot{T}(t) = 0$ leads to the following system, which has to be solved in order to get the steady states of the dynamical system:

$$\alpha A\,(a - A) - \delta A + \beta T = 0 \tag{3.5}$$

$$\delta A - \beta T - \rho T = 0 \tag{3.6}$$

Obviously, $(A_1^*, T_1^*) = (0, 0)$ is one steady state. The second steady state can also be retrieved. From (3.6) we get

$$T = \frac{\delta}{\rho + \beta} A. \tag{3.7}$$

By plugging (3.7) into (3.5) and assuming that $A \neq 0$, the steady state value of A can be calculated as follows:

$$\begin{aligned} 0 &= \alpha A(a - A) - \delta A + \frac{\beta\delta}{\rho + \beta} A \\ \Leftrightarrow A &= a - \frac{\delta}{\alpha} + \frac{\beta\delta}{\alpha(\rho + \beta)} \\ \Leftrightarrow A &= a - \frac{\delta\rho}{\alpha(\rho + \beta)} \end{aligned}$$

Therefore, the second steady state of the system (3.1) - (3.4) is given by

$$(A_2^*, T_2^*) = \left(a - \frac{\delta\rho}{\alpha(\rho + \beta)}, \left(a - \frac{\delta\rho}{\alpha(\rho + \beta)} \right) \frac{\delta}{\rho + \beta} \right).$$

We notice that the steady state value of A is smaller than the carrying capacity a, due to the fact that all parameters are positive. If drug users only very rarely stop taking drugs (small δ), the steady state will be close to a. On the other hand, if relapse or

initiation are not very likely (small β or α, respectively), the steady state will be farther away from a. The influence of ρ can be seen by calculating

$$\frac{\partial}{\partial \rho}\left(\frac{\delta\rho}{\alpha(\rho+\beta)}\right) = \frac{\delta\beta}{\alpha(\rho+\beta)^2} > 0.$$

This shows that a larger ρ implies a smaller steady state, which also makes sense intuitively: The more people leave there status as a teetotaller without relapsing, the less people start taking drugs again.

Naturally, we are only interested in positive values of A and T. To ensure a positive value of A_2^* and thereof a positive value of T_2^* the following assumption is sufficient:

$$a > \frac{\delta\rho}{\alpha(\rho+\beta)} \tag{3.8}$$

Given the parametrisation of Table 2.1 this condition is easily fulfilled.

3.1.2. Stability

I use the principle of linearised stability to identify conditions under which the steady state (A_2^*, T_2^*) is asymptotically stable. The Jacobian matrix of the right-hand side of the system (3.1) - (3.2) is given by

$$J(A,T) = \begin{pmatrix} -2\alpha A + \alpha a - \delta & \beta \\ \delta & -\beta - \rho \end{pmatrix}.$$

Inserting $(A,T) = (A_2^*, T_2^*)$ we obtain

$$J(A_2^*, T_2^*) = \begin{pmatrix} \frac{2\delta\rho}{(\rho+\beta)} - \alpha a - \delta & \beta \\ \delta & -\beta - \rho \end{pmatrix}.$$

The principle of linearised stability implies that if the real parts of all eigenvalues of $J(A_2^*, T_2^*)$ are negative, (A_2^*, T_2^*) is an asymptotically stable steady state. The eigenvalues

are determined by the roots of the characteristic polynomial of $J(A_2^*, T_2^*)$:

$$\begin{aligned}
&\begin{vmatrix} \frac{2\delta\rho}{(\rho+\beta)} - \alpha a - \delta - X & \beta \\ \delta & -\beta - \rho - X \end{vmatrix} = \\
&= \left(\frac{2\delta\rho}{(\rho+\beta)} - \alpha a - \delta - X \right) (-\beta - \rho - X) - \beta\delta = \\
&= X^2 + X \underbrace{\left[\rho + \beta + \delta + \alpha a - \frac{2\delta\rho}{(\rho+\beta)} \right]}_{=:p} + \underbrace{\left[(\rho+\beta) \left(\alpha a - \frac{2\delta\rho}{(\rho+\beta)} \right) + \delta\rho \right]}_{=:q} \stackrel{!}{=} 0
\end{aligned}$$

The eigenvalues are the solutions of the last equation. The solution is given by

$$X_{1,2} = -\frac{p}{2} \pm \sqrt{\left(\frac{p}{2}\right)^2 - q}$$

If $-p/2 \geq 0$, the real part of $-\frac{p}{2} + \sqrt{\left(\frac{p}{2}\right)^2 - q}$ is definitely non-negative since the square root is non-negative. If $q \leq 0$, the square root is real and larger than or equal to $|-p/2|$, which again implies a non-negative solution. Consequently, $\mathrm{Re}(X_1)$ and $\mathrm{Re}(X_2)$ are both smaller than zero if and only if $-p/2 < 0$ and $q > 0$. The constraint $-p/2 < 0$ yields the following:

$$\begin{aligned}
-\frac{p}{2} &= \frac{\delta\rho}{(\rho+\beta)} - \frac{\rho+\beta+\delta+\alpha a}{2} < 0 \\
&\Leftrightarrow \ldots \\
&\Leftrightarrow \delta(\rho-\beta) < (\rho+\beta)(\alpha a + \rho + \beta) \\
&\Leftrightarrow \delta \begin{cases} < (\alpha a + \rho + \beta)\frac{(\rho+\beta)}{(\rho-\beta)} & \text{if} \quad \rho - \beta > 0 \\ > (\alpha a + \rho + \beta)\frac{(\rho+\beta)}{(\rho-\beta)} & \text{if} \quad \rho - \beta < 0 \end{cases}
\end{aligned}$$

In the second case, the right-hand side is negative because of $\rho - \beta < 0$, whereas δ is positive. Therefore, the inequality is fulfilled without any assumptions. In the first case, it has to be assumed that

$$\delta < (\alpha a + \rho + \beta)\frac{(\rho+\beta)}{(\rho-\beta)} \tag{3.9}$$

in order to satisfy $-p/2 < 0$.

Looking at the condition $q > 0$:

$$\begin{aligned} q &= (\rho+\beta)\left(\alpha a - \frac{2\delta\rho}{(\rho+\beta)}\right) + \delta\rho > 0 \\ &\Leftrightarrow \rho\alpha a + \beta\alpha a - \delta\rho > 0 \\ &\Leftrightarrow \delta < \alpha a \frac{\rho+\beta}{\rho} \end{aligned} \tag{3.10}$$

For $\rho - \beta > 0$ it holds that

$$\alpha a \frac{\rho+\beta}{\rho} < \alpha a \frac{\rho+\beta}{\rho-\beta} < (\alpha a + \rho + \beta)\frac{(\rho+\beta)}{(\rho-\beta)},$$

which means that (3.10) renders (3.9) redundant. Therefore, the single assumption (3.10) is sufficient to make (A_2^*, T_2^*) an asymptotically stable equilibrium. Furthermore, (3.10) is equivalent to the condition (3.8) for a positive steady state. Consequently, all assumptions of this section boil down to

$$a > \frac{\delta\rho}{\alpha(\rho+\beta)}, \tag{3.11}$$

which is, as already mentioned, not very restrictive but is yet enough to make (A_2^*, T_2^*) a positive and therefore feasible asymptotically stable equilibrium.

For the second steady state at $(0, 0)$ the eigenvalues of the corresponding Jacobian are calculated as follows:

$$\begin{aligned} &\begin{vmatrix} \alpha a - \delta - X & \beta \\ \delta & -\beta - \rho - X \end{vmatrix} = \\ &= (\alpha a - \delta - X)(-\beta - \rho - X) - \beta\delta = \\ &= X^2 + X\underbrace{[\rho + \beta + \delta - \alpha a]}_{=:p} + \underbrace{[\delta\rho - \alpha a(\beta+\rho)]}_{=:q} = 0 \end{aligned}$$

Like before, the properties of q are reviewed:

$$q = \delta\rho - \alpha a(\beta+\rho) \overset{(3.11)}{<} \delta\rho - \alpha\frac{\delta\rho}{\alpha(\beta+\rho)}(\beta+\rho) = 0$$

Given condition (3.11), we may hence conclude that $q < 0$, the square root $\sqrt{p^2/4 - q}$

is real and positive, and we have that $\sqrt{p^2/4 - q} > |-p/2|$. Therefore, at least one eigenvalue of $J(0,0)$ has a positive real part and according to the principle of linearised stability, this implies that the steady state $(0,0)$ is unstable.

3.1.3. Phase Portrait

To deepen the understanding of the system (3.1) - (3.4), taking a look at the phase portrait is always recommendable. In the simple case considered here, the isoclines can be calculated explicitly:

$$\dot{A} = 0 \Leftrightarrow T = \frac{1}{\beta}\left(\alpha A^2 + (\delta - \alpha a)A\right) \tag{3.12}$$

$$\dot{T} = 0 \Leftrightarrow T = \frac{\delta}{\beta + \rho}A \tag{3.13}$$

(3.13) is a linear function in A. (3.12) is a U-shaped quadratic function. Both functions share a root at $A = 0$. The second root of (3.12) is $a - \frac{\delta}{\alpha}$, which is positive if

$$a > \frac{\delta}{\alpha} > \frac{\delta\rho}{\alpha(\rho + \beta)},$$

which also implies (3.11).

Analogously we obtain

$$\dot{A} > 0 \Leftrightarrow T > \frac{1}{\beta}\left(\alpha A^2 + (\delta - \alpha a)A\right)$$

$$\dot{T} > 0 \Leftrightarrow T < \frac{\delta}{\beta + \rho}A.$$

Figure 3.1 illustrates the case $a > \delta/\alpha$. There are two steady states where the isoclines intersect. Both are feasible under assumption (3.11), and A_2^* is smaller than a. The arrows indicate the behaviour of the dynamical system (3.1)-(3.4) in the corresponding areas. For example, in the top left part above the two isoclines where there are many teetotallers but rather few users, the number of users increases whereas the number of teetotallers decreases. The other arrows may be interpreted analogously. Obviously, everything points in the direction of the positive steady state indicating its asymptotic stability. Figure 3.1 also shows that the number of drug users can temporarily grow so much that it exceeds the carrying capacity a. This happens when there is a sufficiently

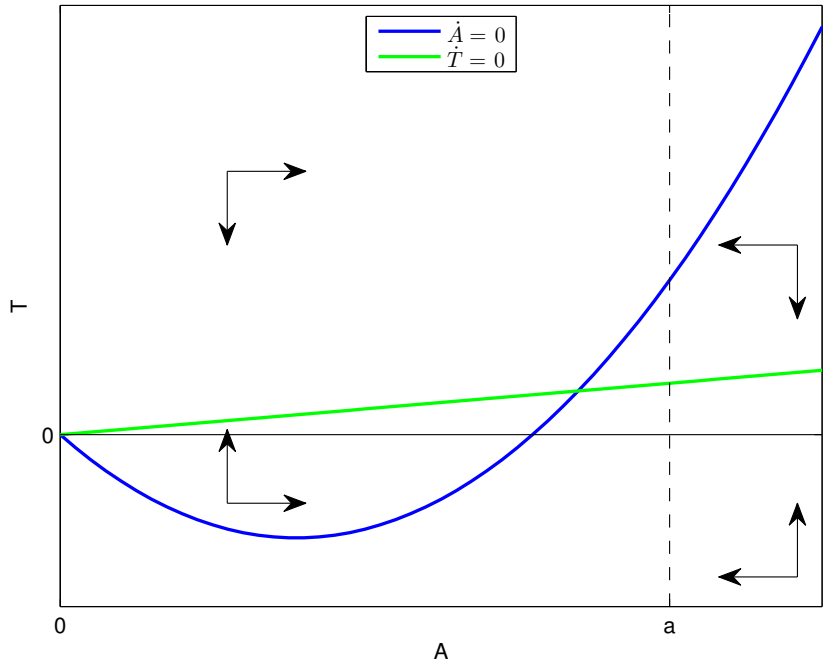

Figure 3.1.: The uncontrolled system and its two isoclines. The arrows show the directions of the vector field in the corresponding areas. The carrying capacity a is indicated by the dashed line.

high amount of drug users and teetotallers alike. Because of a high value of A, initiation is already low due to the factor $(a - A)$ in the initiation term. Nevertheless, there is a high inflow of new consumers because of a high amount of relapsing teetotallers. But as the number of teetotallers decreases, the number of consumers starts to decrease again as soon as the relapsing teetotallers are outnumbered by the people leaving addiction behind as a result of the breach of the carrying capacity.

Figures 3.2 - 3.5 show detailed phase portraits as well as some generic trajectories calculated with MATLAB. The circles mark initial values, and the boxes mark the end values after several time steps.

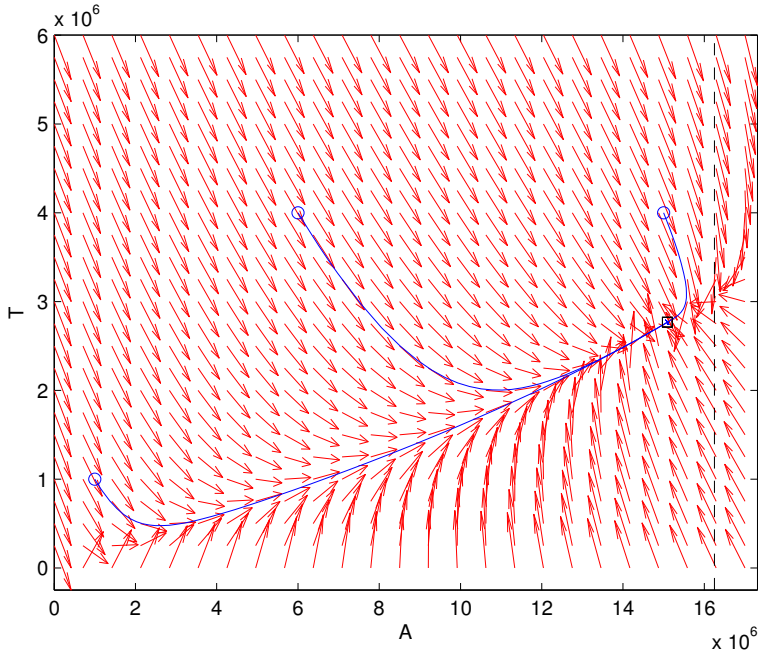

Figure 3.2.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 1.58 \times 10^{-8}, \delta = 0.11, \beta = 0.5, \rho = 0.1$ and some trajectories.

The asymptotic stability is apparent. No matter what feasible initial value is chosen, the trajectories clearly converge to the steady state (A_2^*, T_2^*). For different sets of parameters, the trajectories look quite similar. The overall look of the phase portrait is primarily determined by the slope of the $\dot{T} = 0$ - isocline. The main differences can be seen in the location of the steady state and how far the carrying capacity a can be overshot. The relapse parameter β plays a vital role in this overshooting. A higher level of natural desistance (δ) significantly increases T in its steady state whereas A is not influenced as heavily. A reduction in initiation (α) reduces A comparably more than T.

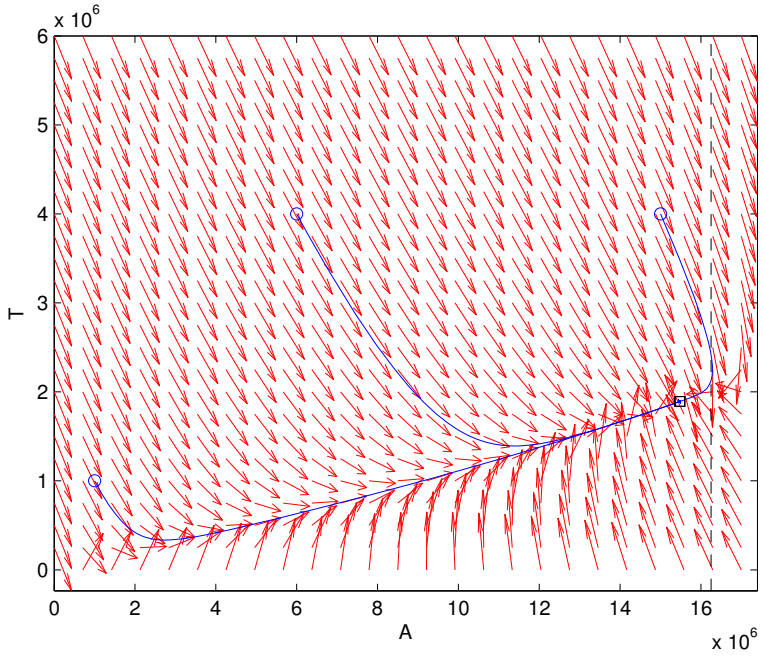

Figure 3.3.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 1.58 \times 10^{-8}, \delta = 0.11, \beta = 0.8, \rho = 0.1$ and some trajectories.

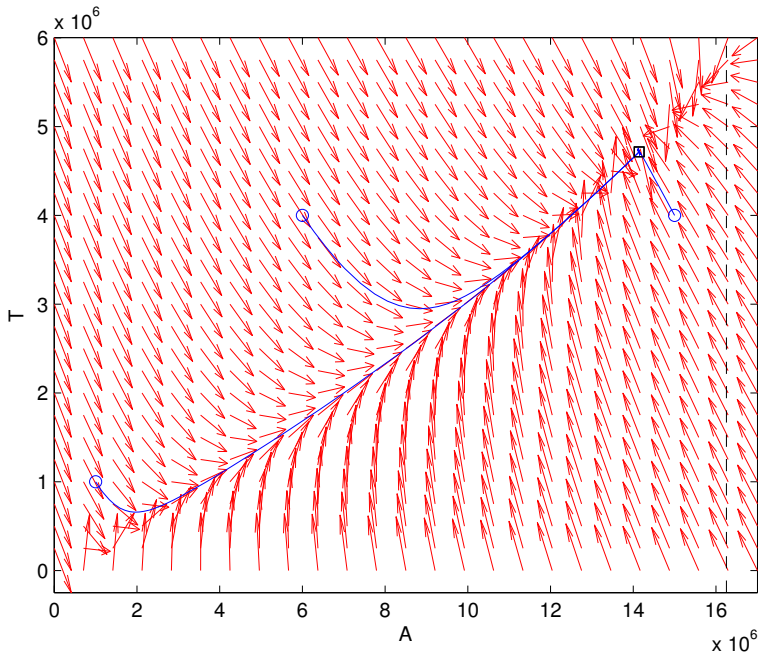

Figure 3.4.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 1.58 \times 10^{-8}, \delta = 0.20, \beta = 0.5, \rho = 0.1$ and some trajectories.

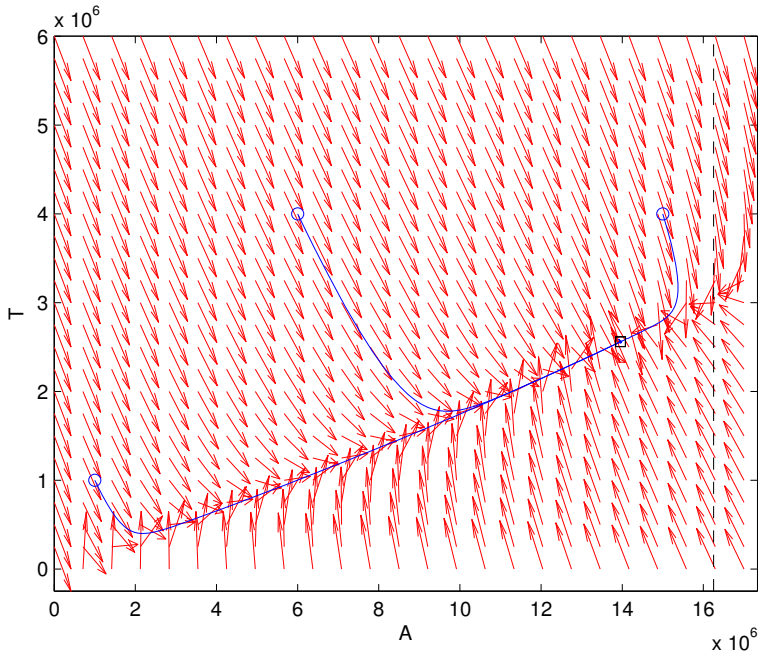

Figure 3.5.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 0.8 \times 10^{-8}, \delta = 0.11, \beta = 0.5, \rho = 0.1$ and some trajectories.

3.2. The General Case

In the general case the parameters $b_1, b_2, \omega \geq 0$ and $\gamma \leq 0$ are considered without specifications as well, so we consider the dynamics in the most general case:

$$\begin{aligned}
\dot{A}(t) &= \alpha A(t)^{\omega} T(t)^{\gamma} (a - A(t)) - \delta A(t) + \beta A(t)^{b_1} T(t)^{b_2} \\
\dot{T}(t) &= \delta A(t) - \beta A(t)^{b_1} T(t)^{b_2} - \rho T(t) \\
A(0) &= A_0 \geq 0 \\
T(0) &= T_0 \geq 0
\end{aligned}$$

In contrast to the simple case considered in section 3.1, the teetotallers' negative influence on initiation as well as their positive influence on relapse is featured in the model. The positive influence of drug users on initiation and relapse can be tuned by the corresponding exponents as well.

3.2.1. Steady States

The steady states are given by the solutions of

$$\alpha A^{\omega} T^{\gamma} (a - A) - \delta A + \beta A^{b_1} T^{b_2} = 0 \tag{3.14}$$

$$\delta A - \beta A^{b_1} T^{b_2} - \rho T = 0. \tag{3.15}$$

$T = 0$ cannot be part of a solution because in that case T^{γ} is not defined due to the assumption that $\gamma \leq 0$. Therefore, $(0, 0)$ is no solution any more. Inserting (3.15) into (3.14) yields $T = \left[\frac{\alpha}{\rho} A^{\omega}(a - A)\right]^{\frac{1}{1-\gamma}}$, and putting that expression back in (3.15) yields

$$\underbrace{\delta A - \beta A^{b_1} \left[\frac{\alpha}{\rho} A^{\omega}(a - A)\right]^{\frac{b_2}{1-\gamma}} - \rho \left[\frac{\alpha}{\rho} A^{\omega}(a - A)\right]^{\frac{1}{1-\gamma}}}_{=:g(A)} = 0.$$

It holds that $g(0) = 0$ and $g(a) = \delta a > 0$. Furthermore, g is continuous and real in $(0, a)$. Consequently, $g'(0) < 0$ would be a sufficient condition for the existence of a solution $A^* > 0$ with $A^* < a$ and $g(A^*) = 0$.

$$\begin{aligned} g'(0) &= \lim_{h \to 0} \frac{g(0 + h) - g(0)}{h} = \lim_{h \to 0} \frac{g(h)}{h} = \\ &= \lim_{h \to 0} \delta - \beta h^{\frac{(b_1 - 1)(1-\gamma) + \omega b_2}{1-\gamma}} \left[\frac{\alpha}{\rho}(a - h)\right]^{\frac{b_2}{1-\gamma}} - \rho h^{\frac{\omega + \gamma - 1}{1-\gamma}} \left[\frac{\alpha}{\rho}(a - h)\right]^{\frac{1}{1-\gamma}} \end{aligned}$$

Depending on the signs of the exponents of h, $g'(0)$ is either δ or $-\infty$. For $-\infty$, at least one of the exponents needs to be negative.

$$g'(0) < 0 \Leftrightarrow ((b_1 - 1)(1 - \gamma) + \omega b_2 < 0 \vee \omega + \gamma - 1 < 0) \tag{3.16}$$

Under this condition the existence of a positive steady state A^* is sure. The baseline parameters of Table 2.1 fulfil (3.16). However, if the condition does not hold, this does not mean that there is no positive solution. Indeed, Figures 3.6 - 3.8 show that further scenarios are possible.[1]

[1] For Figures 3.6 - 3.8, the parameters were chosen in order to demonstrate the different cases. They are very different from the baseline parametrisation. However, for values similar to the baseline parametrisation, the graph of g looks similar to Figure 3.6.

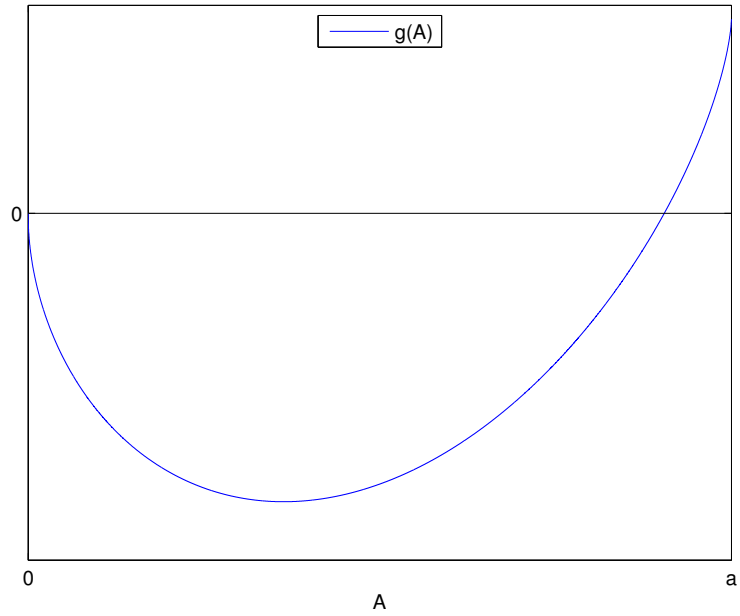

Figure 3.6.: Plot of $g(A)$ with parameters $a = 7, \alpha = 0.1, \delta = 0.2, \beta = 0.7, \rho = 0.2, \omega = 0.5, \gamma = -0.4, b_1 = 0.3, b_2 = 0.9$. (3.16) is satisfied. One root at $A \approx 6.33$.

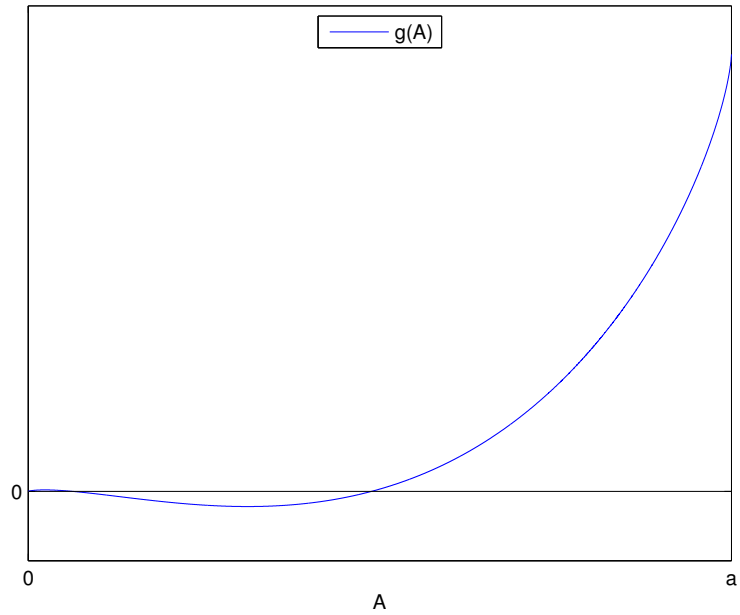

Figure 3.7.: Plot of $g(A)$ with parameters $a = 7, \alpha = 0.1, \delta = 0.9, \beta = 0.2, \rho = 0.1, \omega = 1.5, \gamma = -0.4, b_1 = 0.3, b_2 = 0.9$. (3.16) is not satisfied. Two roots at $A_1 \approx 0.44$ and $A_2 \approx 3.42$.

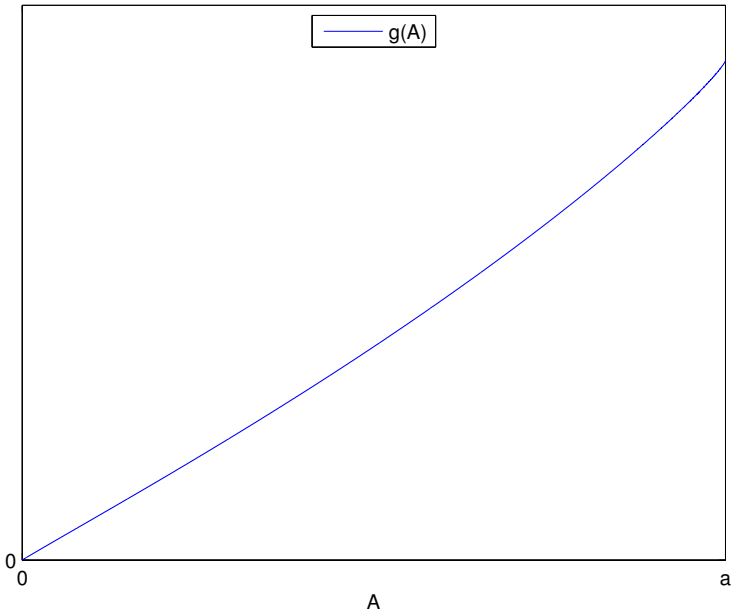

Figure 3.8.: Plot of $g(A)$ with parameters $a = 7, \alpha = 0.01, \delta = 0.9, \beta = 0.2, \rho = 0.8, \omega = 1.5, \gamma = -0.4, b_1 = 0.3, b_2 = 0.9$. (3.16) is not satisfied. No roots.

Furthermore, a steady state value of A cannot exceed a: by putting (3.15) into (3.14) it is obtained that

$$\alpha A^{\omega} T^{\gamma}(a - A) - \rho T = 0.$$

This however, cannot be true if $A > a$, because in that case the left side is negative and not equal to zero.

3.2.2. Phase Portraits

As the steady states in general cannot be written in an explicit form, the principle of linearised stability cannot be applied like in the simple case before. In order to get an understanding of the system's behaviour nonetheless, we investigate a few phase portraits. Figures 3.9 - 3.12 show phase portraits with different sets of parameters and several trajectories.

The most striking feature is that all trajectories clearly converge to an asymptotically stable steady state. Moreover, the general structure of the phase portrait is practically the same as before in the simple case. Due to the negative feedback imposed by tee-totallers on initiation, steady state values of A are comparably lower throughout all

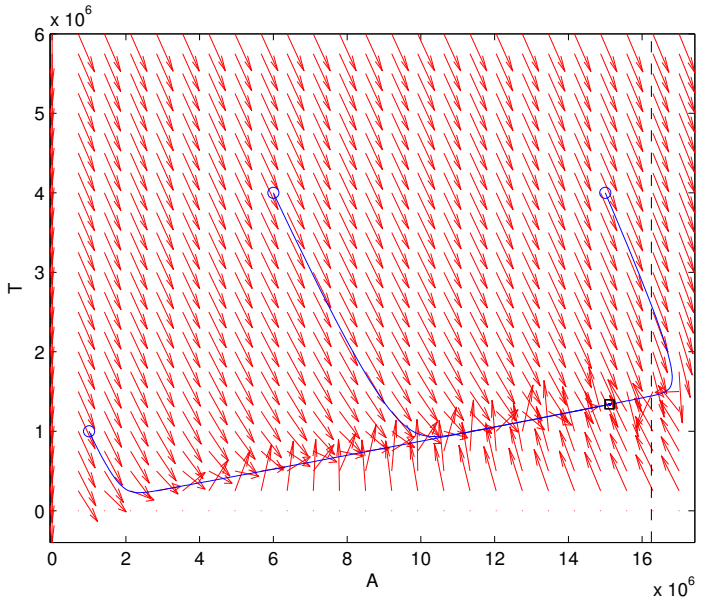

Figure 3.9.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 1.58 \times 10^{-8}, \delta = 0.11, \beta = 0.5, \rho = 0.1, \omega = 1, \gamma = -0.05, b_1 = 0.05, b_2 = 1$ and some trajectories (baseline parameters).

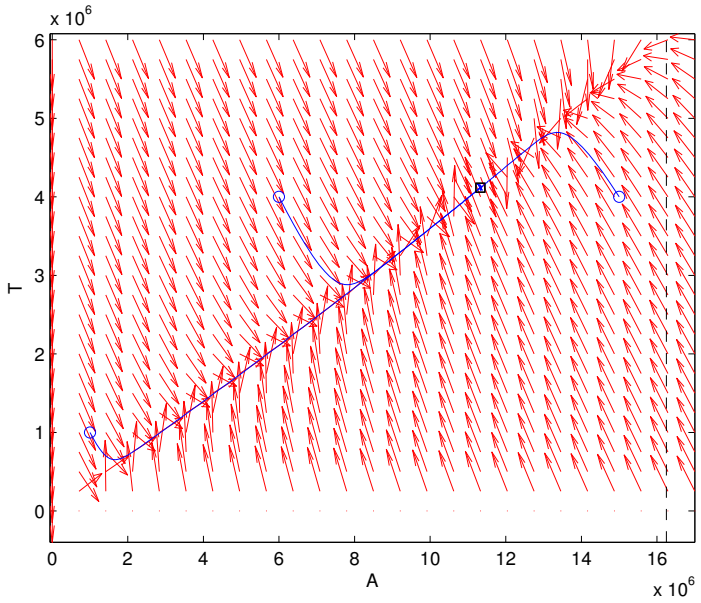

Figure 3.10.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 1.58 \times 10^{-8}, \delta = 0.2, \beta = 0.2, \rho = 0.1, \omega = 1, \gamma = -0.05, b_1 = 0.05, b_2 = 1$ and some trajectories.

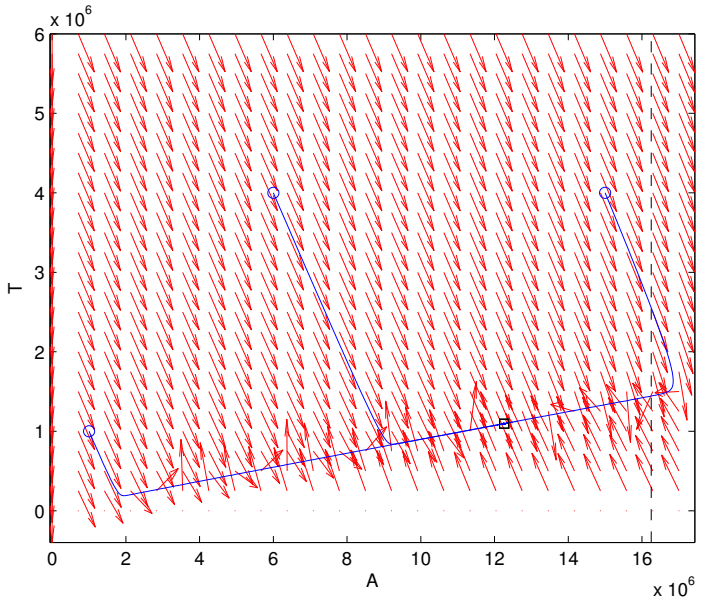

Figure 3.11.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 0.9 \times 10^{-8}, \delta = 0.11, \beta = 0.5, \rho = 0.1, \omega = 1, \gamma = -0.1, b_1 = 0.05, b_2 = 1$ and some trajectories.

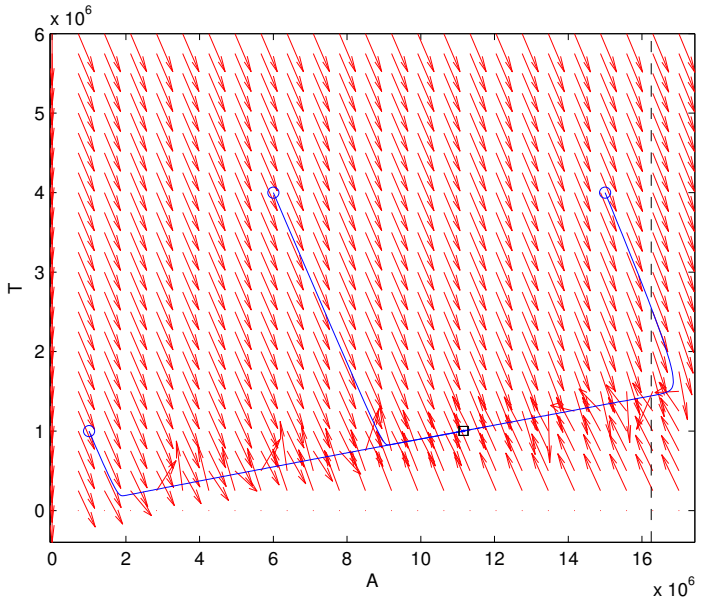

Figure 3.12.: Plot of the phase portrait with parameters $a = 16250000, \alpha = 1.58 \times 10^{-8}, \delta = 0.11, \beta = 0.5, \rho = 0.1, \omega = 0.95, \gamma = -0.1, b_1 = 0.05, b_2 = 1$ and some trajectories.

figures. The exponents in initiation are vital to the model: Comparing Figures 3.9 and 3.12, where ω and γ were both decreased by 0.05, the positions of the steady states differ significantly. For sufficiently high starting values of A and T, overshooting of the carrying capacity a is possible.

Last but not least it is remarkable that the trajectories in all the figures do not take a monotonous way towards the steady state. Instead, at first, the number of teetotallers changes rapidly whereas the number of drug users only changes a bit. Then the trajectories bend sharply and the adjustment of A takes over whereas T is not changing a lot any more. Furthermore, looking at the two starting points on the left-hand side of the figures, one can see that their trajectories follow the same track near the steady state.[2] As long as the initial values are sufficiently similar, the two trajectories only differ remarkably at the beginning. Once their rapid "teetotaller-adjustment" is done, they move on more or less the same path towards the steady state.

It is worth highlighting that the results of this chapter can also be applied to the system with constant controls. A constant level of prevention can be simulated by lowering α, while the effects of a constant level of treatment efficiency can be produced by an increase of δ. Therefore, the effects of constant controls can also be seen partly in Figures 3.9 - 3.12.

[2]Due to the uniqueness of solutions of initial value problems, trajectories cannot intersect, so the trajectories do not really follow the same track. Yet they are so close to each other that they cannot be distinguished visually.

Chapter 4

The Optimal Control Model

In this chapter the optimal solution of the control problem will be calculated. This calculation will be conducted partly analytically and partly numerically using the MATLAB-toolbox OCMAT.

4.1. The Necessary Optimality Conditions

In order to solve and analyse the optimal control problem, Pontryagin's Maximum Principle is applied (see for example [6]). First of all, I restate the system dynamics as they will be needed in the following pages, where the time argument t will be partly omitted for the sake of better understanding:

$$\dot{A} = \alpha A^{\omega} T^{\gamma} (a - A) \Psi(p) - (\delta + w)A + \beta A^{b_1} T^{b_2} \tag{4.1}$$

$$\dot{T} = (\delta + w)A - \beta A^{b_1} T^{b_2} - \rho T \tag{4.2}$$

The current value Hamiltonian $\mathcal{H}$ is then given by [1]

$$\begin{aligned} \mathcal{H}(A, T, p, w, \lambda_1, \lambda_2) = &- cA - p - f_0 w - \frac{f_1 w^2}{2} \\ &+ \lambda_1 \left[\alpha A^{\omega} T^{\gamma} (a - A) \Psi(p) - (\delta + w)A + \beta A^{b_1} T^{b_2}\right] \\ &+ \lambda_2 \left[(\delta + w)A - \beta A^{b_1} T^{b_2} - \rho T\right]. \end{aligned}$$

[1] Note that the optimal control problem has been reformulated as a maximisation problem by multiplying the objective function by -1. Therefore, the costate variables λ_1, λ_2 are going to be negative in economically meaningful cases. However, when I write about the shadow price interpretation of these variables, I will treat them as positive because it would be odd to talk about negative prices. A high shadow price means a high absolute value of λ_i, and $\lambda_1 < \lambda_2$ means that state 1 has a higher shadow price than state 2.

According to the maximum principle, the optimal control maximises the Hamiltonian. As there are three control constraints, the Lagrangian $\mathcal{L}$ has to be considered in order to solve this static maximisation problem. Note that the inequality constraints have to be transformed to the form $g(u) \geq 0$.

$$\mathcal{L}\left(A, T, p, w, \lambda_1, \lambda_2, \mu_1, \mu_2, \mu_3\right) = \mathcal{H}\left(A, T, p, w, \lambda_1, \lambda_2\right) + \mu_1 p + \mu_2 w + \mu_3\left(1 - \delta - w\right)$$

The necessary optimality conditions for a maximum of $\mathcal{H}$ are:

$$\mathcal{L}_p = 0 \tag{4.3}$$
$$\mathcal{L}_w = 0 \tag{4.4}$$
$$\mu_i \geq 0, \quad i = 1, 2, 3 \tag{4.5}$$
$$\mu_1 p = 0 \tag{4.6}$$
$$\mu_2 w = 0 \tag{4.7}$$
$$\mu_3\left(1 - \delta - w\right) = 0 \tag{4.8}$$

Note also that $\mathcal{H}$ is concave with respect to both controls p and w. The concavity with respect to w is obvious because of the negative quadratic term in the objective function. Moreover, $\Psi(p)$ is convex, but the whole term is concave since λ_1 is negative.

Differentiating the Lagrangian, the conditions (4.3) - (4.4) yield

$$\mathcal{L}_p = -1 + \lambda_1 \alpha A^{\omega} T^{\gamma}(a - A)\Psi'(p) + \mu_1 = 0 \tag{4.9}$$
$$\mathcal{L}_w = -f_0 - f_1 w - \lambda_1 A + \lambda_2 A + \mu_2 - \mu_3 = 0. \tag{4.10}$$

The adjoint equations defined by the maximum principle as $\dot{\lambda} = r\lambda - \mathcal{L}_x$, with $x = (A, T)$ read as:

$$\begin{aligned}\dot{\lambda_1} &= r\lambda_1 + c - \lambda_1\left[\alpha T^{\gamma}\Psi(p)A^{\omega-1}(\omega(a - A) - A) - (\delta + w) + \beta b_1 A^{b_1 - 1}T^{b_2}\right] \\ &\quad - \lambda_2\left[\delta + w - \beta b_1 A^{b_1 - 1}T^{b_2}\right]\end{aligned} \tag{4.11}$$

$$\begin{aligned}\dot{\lambda_2} &= r\lambda_2 - \lambda_1\left[\alpha A^{\omega}\gamma T^{\gamma - 1}(a - A)\Psi(p) + \beta b_2 A^{b_1}T^{b_2 - 1}\right] \\ &\quad - \lambda_2\left[-\beta b_2 A^{b_1}T^{b_2 - 1} - \rho\right]\end{aligned} \tag{4.12}$$

The adjoint equations (4.11) - (4.12) and the conditions (4.3) - (4.8) constitute the necessary conditions for the solution of the optimal control problem. Given the three

control constraints and the fact that w cannot be equal to 0 and $1-\delta$ at the same time, there are six different arcs which have to be distinguished: one interior and five boundary arcs.

Interior Arc

In the interior of the admissible region no constraint is active. Therefore, the complementary slackness conditions yield $\mu_1 = \mu_2 = \mu_3 = 0$. Some simple transformations from (4.9) and (4.10) then reveal the following optimal controls:

$$p = \frac{1}{m} \ln\left(-m(1-h)\lambda_1 \alpha A^{\omega} T^{\gamma}(a-A)\right)$$
$$w = \frac{-f_0 + A\left(\lambda_2 - \lambda_1\right)}{f_1}$$

Obviously, the optimal choice of p primarily depends on initiation. When there are lots of teetotallers, p will be low because γ is negative. When the maximum effectiveness of prevention, given by $(1-h)$ rises, p rises as well. The reactions of p to changes in m or A depend on the combination of the other values and can be positive or negative. The only factor in the formula for p that does not come directly from initiation is λ_1. The formula shows that λ_1 indeed has to be negative. Otherwise the argument of the logarithm would be negative and hence the logarithm would not exist. For a smaller value of λ_1 (thus for a larger absolute value and thereof shadow price) p grows. The interpretation is straightforward: When an additional drug user costs a lot of money, the social planner is willing to invest more into prevention measures. Contrary, when an additional drug user costs less money, the decision maker will not put as much effort into prevention as it is cheaper to accept more drug users in the system.

The formula for the treatment control w is simpler. Both cost parameters f_0 and f_1 naturally have an adverse effect on w. The effect of A is clearly positive now. If λ_2 is not sufficiently larger than λ_1, w becomes negative and therefore infeasible. For example, if $\lambda_1 = \lambda_2$, w would be negative and had to be set to 0. This absolutely makes sense: When the shadow prices are the same, we do not care whether we get an additional drug user or an additional teetotaller as they cost the same. In such a situation there is no incentive to start treatment in order to transform drug users into teetotallers. However, the formula shows that, depending on the values of A and f_0, the difference in the shadow prices has to be high enough for treatment to be used at all.

Putting these formulas for p and w into the adjoint equations (4.11) - (4.12) and into the system dynamics (4.1) - (4.2) yields the canonical system which I will not describe

here in detail due to its very lengthy form.

Boundary Arc 1: no prevention / $p = 0$

When p equals zero, μ_1 becomes free while the other Lagrange multipliers remain zero. Some transformations yield

$$\mu_1 = 1 + \lambda_1 \alpha A^{\omega} T^{\gamma} (a - A) m (1 - h).$$

Due to the nonnegativity condition for μ_1, the following inequality has to be satisfied (assuming $A < a$):

$$\lambda_1 \geq -\frac{1}{\alpha A^{\omega} T^{\gamma} (a - A) m (1 - h)}$$

This means that when the shadow price is too high (when λ_1 is too small), the choice of $p = 0$ cannot be optimal. The optimal choice for w is the same as in the case of the *Interior Arc*.

Boundary Arc 2: no prevention, no treatment / $p = w = 0$

When both controls are zero, $\mu_3 = 0$ has to be fulfilled. For the other Lagrange multipliers we get:

$$\begin{aligned} \mu_1 &= 1 + \lambda_1 \alpha A^{\omega} T^{\gamma} (a - A) m (1 - h) \\ \mu_2 &= f_0 + A (\lambda_1 - \lambda_2) \end{aligned}$$

μ_1 is the same as in *Boundary Arc 1* and so is the condition for λ_1. The nonnegativity condition for μ_2 yields

$$\lambda_1 - \lambda_2 \geq -\frac{f_0}{A}$$

This condition means that λ_1 should not be too small compared to λ_2: If the shadow price of drug users is too high compared to the shadow price of teetotallers, it is not optimal to choose $w = 0$. Setting $w > 0$ will turn more drug users into teetotallers, which is only beneficial when a drug user costs sufficiently more than a teetotaller. This sufficient difference is given by f_0/A.

Boundary Arc 3: no prevention, maximum treatment / $p = 0$, $w = 1 - \delta$

When treatment is used to the maximum extent ($w = 1 - \delta$) and p equals zero, the complementary slackness condition yields $\mu_2 = 0$. From conditions (4.9) - (4.10) the

following equations for μ_1 and μ_3 can be derived:

$$\mu_1 = 1 + \lambda_1 \alpha A^{\omega} T^{\gamma} (a - A) m (1 - h)$$
$$\mu_3 = -f_0 - f_1(1 - \delta) + A\,(\lambda_2 - \lambda_1)$$

μ_1 is the same as before and so is the condition for λ_1. The nonnegativity condition for μ_3 yields

$$\lambda_1 - \lambda_2 \leq -\frac{f_0 + f_1(1 - \delta)}{A}.$$

The interpretation is almost the exact opposite from the interpretation in *Boundary Arc 2*: In order to choose maximum treatment efficiency, the shadow price of drug users has to be sufficiently higher than the shadow price of teetotallers. Otherwise, in terms of costs, it would not be beneficial to transform so many drug users into teetotallers by means of treatment.

Boundary Arc 4: no treatment / $w = 0$

p can be calculated as in the case of the *Interior Arc* and μ_2 is the same as in *Boundary Arc 2*.

Boundary Arc 5: maximum treatment / $w = 1 - \delta$

p can be calculated as in the case of the *Interior Arc* and μ_3 is the same as in *Boundary Arc 3*.

To sum up, which arc is chosen in order to maximise the Hamiltonian $\mathcal{H}$ depends to a great extent on the costate variables λ_1 and λ_2. Considering prevention, only λ_1 plays a role, whereas λ_2 does not matter. As long as the shadow price is low enough (in other words as long as λ_1 is large enough), there will be no investment into prevention as it costs more than it helps. In terms of treatment, the difference between the shadow prices has to be considered. As long as they are close together (closer than f_0/A), no treatment is deployed. When they are further away from each other than $(f_0 + f_1(1-\delta))/A$, treatment measures are at full capacity. It is of course not surprising that these threshold values for the usage or non-usage of treatment depend on the treatment cost parameters f_0 and f_1.

It is also worth highlighting that in all cases in which $w > 0$ the condition $\lambda_2 > \lambda_1$ must hold. Otherwise, in the interior arc w would be negative and therefore infeasible, and in the case $w = 1 - \delta$ the nonnegativity condition of μ_3 would be violated. In other words, the shadow price of drug users is usually higher than the shadow price

of teetotallers, which was to be expected since drug users cause direct costs whereas teetotallers only cause costs in case of a relapse.

4.2. The Optimal Paths

The canonical system given by the adjoint equations (4.11) - (4.12) and the state equations (4.1) - (4.2) is the next point of focus:

$$
\begin{aligned}
\dot{A} &= \alpha A^{\omega} T^{\gamma} (a - A) \Psi(p) - (\delta + w)A + \beta A^{b_1} T^{b_2} \\
\dot{T} &= (\delta + w)A - \beta A^{b_1} T^{b_2} - \rho T \\
\dot{\lambda_1} &= r\lambda_1 + c - \lambda_1 \left[\alpha T^{\gamma} \Psi(p) A^{\omega-1} (\omega(a - A) - A) - (\delta + w) + \beta b_1 A^{b_1 - 1} T^{b_2}\right] \\
&\quad - \lambda_2 \left[\delta + w - \beta b_1 A^{b_1 - 1} T^{b_2}\right] \\
\dot{\lambda_2} &= r\lambda_2 - \lambda_1 \left[\alpha A^{\omega} \gamma T^{\gamma - 1} (a - A) \Psi(p) + \beta b_2 A^{b_1} T^{b_2 - 1}\right] \\
&\quad - \lambda_2 \left[-\beta b_1 A^{b_1} T^{b_2 - 1} - \rho\right]
\end{aligned}
$$

Theoretically the task is now to insert the Hamiltonian-maximising values of p and w and to solve the differential equation system with the initial value $(A(0), T(0)) = (A_0, T_0)$ while considering all different arcs. However, given the complexity of this system (which is even additionally enhanced by the terms for p and w), a further purely analytical examination does not seem very promising. Hence, to carry out the following calculations I use the already mentioned MATLAB-toolbox OCMAT, which was created for that very purpose. Details concerning the use and application of this toolbox in order to solve optimal control problems are given in the Appendix.

4.2.1. The Optimal Path in the Base Case

The first step is the calculation of equilibria of the canonical system. This means that the system of equations $\dot{A} = \dot{T} = \dot{\lambda_1} = \dot{\lambda_2} = 0$ has to be solved, which can only be done numerically in this case. Using the baseline parameters given in Table 2.1 the following equilibrium is obtained:

$$
\begin{pmatrix} A^* \\ T^* \\ \lambda_1^* \\ \lambda_2^* \end{pmatrix} = \begin{pmatrix} 2.8043 \\ 2.4376 \\ -0.1144 \\ -0.1004 \end{pmatrix} \times 10^6
$$

In fact, there were several equilibra found, but after checking all of them for admissibility, only the one stated above remained. The criteria for admissibility are the following: The canonical system at the equilibrium has to be zero or at least very close to zero. This might seem redundant as the utilized MATLAB-function ought to look for exactly such points, but due to numerical errors it is possible that points which are not steady states at all are returned. Moreover, the states have to be positive and all variables have to be real. Lastly, the controls and the corresponding Lagrange multipliers have to lie in the feasible region. For the vector stated above, all these criteria were met:

$$\begin{pmatrix} p^* \\ w^* \end{pmatrix} = \begin{pmatrix} 1.0621 \times 10^9 \\ 0.89 \end{pmatrix}, \quad \begin{pmatrix} \mu_1^* \\ \mu_2^* \\ \mu_3^* \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 3.4446 \times 10^{10} \end{pmatrix}$$

Consequently, the steady state is indeed admissible. The values of A and T are quite low compared to the steady state of the uncontrolled system with $p = w = 0$ under baseline parametrisation, which can be seen in Figure 3.9 in Chapter 3. In Figure 3.9, the number of drug users was close to the carrying capacity of $a = 16250000$. Now, the number of drug users is significantly lower at around three millions. In return, the number of teetotallers is about twice as much compared to the uncontrolled case. The reason for this hefty reduction in drug consumption is of course to be seen in the applied controls: Treatment is used to the maximum extent which means an expenditure of around 2.2 billion dollars. Prevention expenditure is around 1.1 billion dollars, which means $\Psi(p^*) = 0.85$ is very close to the efficiency limit of $h = 0.84$. So it is safe to say that both available controls are deployed pretty massively. Apparently, the social cost of uncontrolled drug use is so high that huge control costs are preferred. Or, to see it from a different angle, the controls are so cheap that it is better to use them as much as possible rather than accept wide spread drug use. However, it also has to be highlighted that the controls, especially treatment, are simply very powerful by design. Giving the social planner the possibility to treat every single drug user no matter how many there are, was a critical point in the model formulation.

The costate variables λ_1, λ_2, and the Lagrange multiplier μ_3 fulfil the conditions stated in the discussion of *Boundary Arc 3*. λ_1 is sufficiently smaller than λ_2, which means that drug users have a higher shadow price than teetotallers in the steady state. In fact, the difference is so high that it makes sense to apply treatment as much as possible.

Next, the stability of the steady state is to be considered. The eigenvalues of the

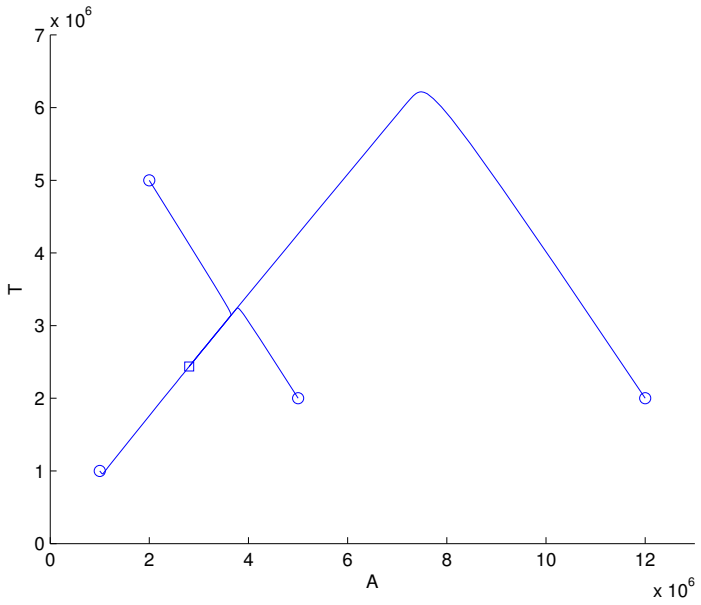

Figure 4.1.: Plot of some optimal trajectories. The steady state is indicated by the square. The initial values are indicated by the circles.

corresponding Jacobian are

$$\begin{pmatrix} -2.0260 \\ 2.0660 \\ -0.0107 \\ 0.0507 \end{pmatrix}.$$

Therefore, the equilibrium is a saddle point. The calculation of the stable paths leading into the equilibrium is carried out by OCMat by continuing the solutions of boundary value problems. The initial solution for the continuation process is trivially given by the steady state itself.

The results of several continuation processes are presented in Figure 4.1. The exemplary initial values (A_0, T_0) are $(1, 1) \times 10^6$, $(12, 2) \times 10^6$, $(5, 2) \times 10^6$ and $(2, 5) \times 10^6$. The trajectories qualitatively look very similar to those in the uncontrolled case presented in Chapter 3. There is one diagonal to which the trajectories move more or less directly. Once on this diagonal, the trajectories take a sharp bend and move directly towards the steady state. One can only reach the equilibrium from the top right and the bottom left. This means that A and T have to rise or fall together in order to reach the steady state. If one state is increasing and the other is decreasing one can be sure that the trajectory will experience a sharp bend at some point in the future.

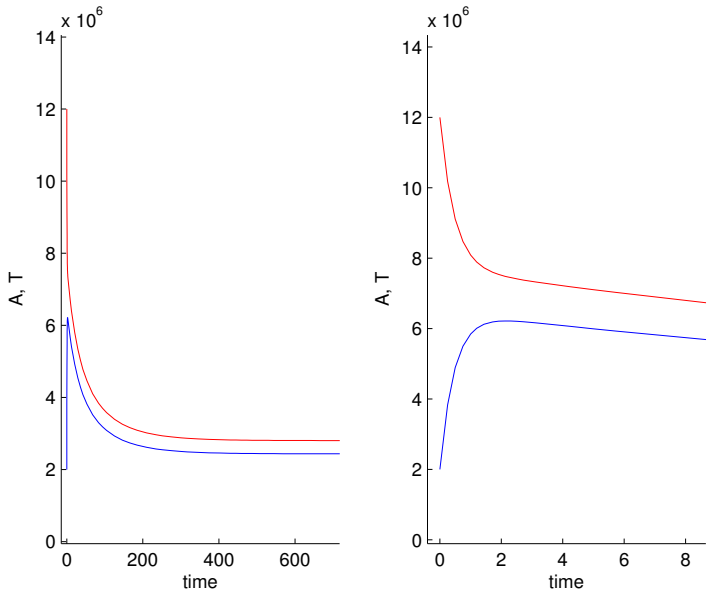

Figure 4.2.: Plot of the optimal state path for the initial value $(A_0, T_0) = (12, 2) \times 10^6$. A is red. T is blue.

Figure 4.2 depicts the optimal path of the two states with initial value $(12, 2) \times 10^6$. The left panel shows a longer time period, whereas the right panel focuses on the first few time steps. Obviously, both states experience a rapid change at first: The number of users falls from 12 to under 8 millions, and the number of teetotallers more than triples from 2 to over 6 millions. Once they are closer to each other, the difference between the two states remains about constant. Comparing the graphs with Figure 4.1 reveals that the first phase of extremely fast change stops when the trajectory reaches the diagonal at approximately $(8, 6) \times 10^6$ and turns in the direction of the steady state. For the other initial values, the situation revealed to be the same: At first there is a very fast movement towards the diagonal. Once the diagonal is reached, the system converges to the equilibrium comparatively slowly.

The optimal controls along the trajectories with initial values $(1, 1) \times 10^6$ and $(12, 2) \times 10^6$ are shown in Figures 4.3 and 4.4, respectively. We see that in both cases $w = 0.89$ for the whole time. So even when the system starts with very low numbers of A and T and treatment is used to a maximum extent, initiation is so strong that both values rise. In the case of a high initial value it is more understandable that w is maximal, as A has to decrease a lot in order to reach the equilibrium. As A decreases, drug users are transformed into teetotallers, whose numbers consequently grow. Because of the low value of T in that high initial point, prevention expenditures are high at first and then

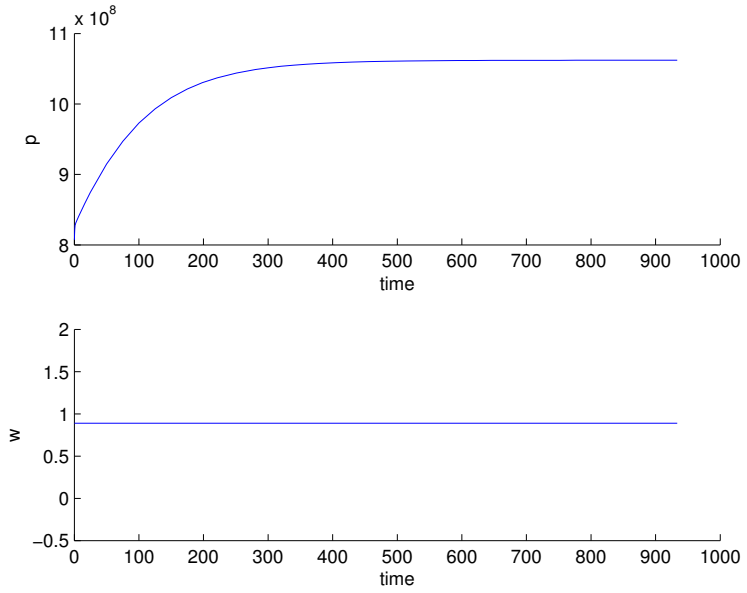

Figure 4.3.: Plot of the optimal control path for the initial value $(A_0, T_0) = (1, 1) \times 10^6$.

decrease as the growing number of teetotallers have a stronger dissuasive influence on initiation. For the low starting point, p behaves the opposite way and grows towards its steady state value. A has to grow anyway in that case, so it is not ideal to hinder that growth by spending too much on prevention at first.

The most striking feature in my opinion is that treatment is used as much as possible not only in the steady state but also in every trajectory at all times. Even when the drug problem starts at smaller values, this does not change.

4.2.2. The Optimal Path with Higher Treatment Costs

It seems to be interesting to see, how the optimal control paths change when it is not optimal to use treatment so much. This can be achieved, for example, by increasing the costs of treatment. More precisely, I set $f_1 = 6 \times 10^{10}$ while leaving the other parameters the same. An increase in f_1 implies a decrease in w in the case of an interior solution. In the case of $w = 1 - \delta$, like in *Boundary Arc 3*, an increase in f_1 means that the difference in shadow prices must be even larger to make this arc optimal. Like before, the first thing to do is to calculate the steady states of the canonical system. It turns out that

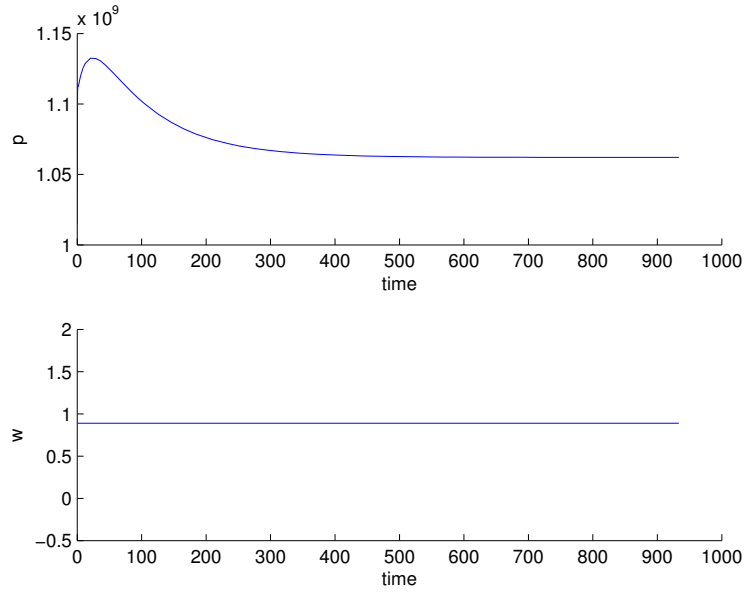

Figure 4.4.: Plot of the optimal control path for the initial value $(A_0, T_0) = (12, 2) \times 10^6$.

there is again only one admissible steady state which is given by

$$\begin{pmatrix} A^* \\ T^* \\ \lambda_1^* \\ \lambda_2^* \end{pmatrix} = \begin{pmatrix} 3.7479 \\ 2.9913 \\ -0.1100 \\ -0.0968 \end{pmatrix} \times 10^6.$$

The values of A and T are naturally higher than before, but considering the fact that f_1 was multiplied more than tenfold, the increase is not that severe. The number of drug users grew by approximately one million. The number of teetotallers grew by about half a million. The costate variable stayed roughly the same. The corresponding eigenvalues are

$$\begin{pmatrix} 2.6309 \\ -2.5909 \\ 0.0644 \\ -0.0244 \end{pmatrix}.$$

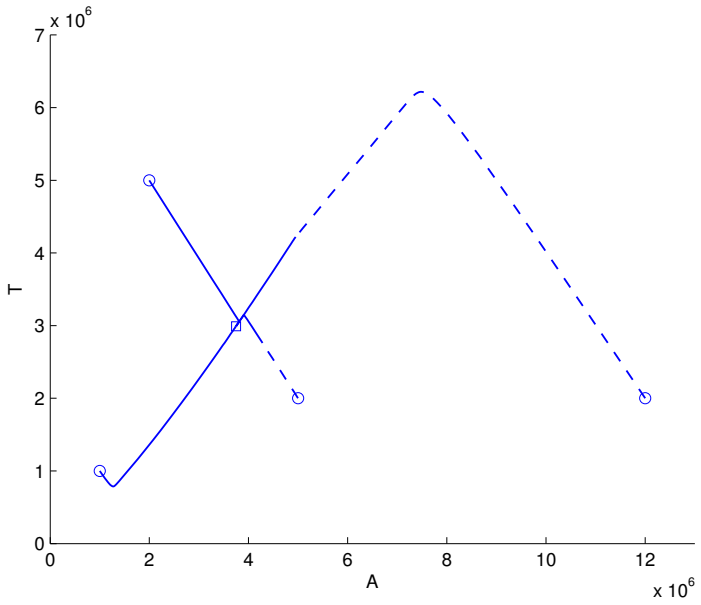

Figure 4.5.: Plot of some optimal trajectories with $f_1 = 6 \times 10^{10}$. The steady state is indicated by the square. The initial values are indicated by the circles.

Hence, the steady state is again a saddle point. The control values and Lagrange multipliers in the equilibrium are given by

$$\begin{pmatrix} p^* \\ w^* \end{pmatrix} = \begin{pmatrix} 1.1332 \times 10^9 \\ 0.8204 \end{pmatrix}, \quad \begin{pmatrix} \mu_1^* \\ \mu_2^* \\ \mu_3^* \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}.$$

The long-run solution is to be found in the interior of the admissible region. While w^* is lower than before, p^* is higher. What does that mean for the optimal stable paths? Figure 4.5 depicts the trajectories for the same initial values as in the baseline case.

The phase portrait looks qualitatively more or less the same as before in the base case. The only big difference can be seen in the location of the equilibrium. However, there is a major difference when it comes to the applied controls, as the two paths coming from the right side switch arcs at some point. The dashed sections of the lines indicate *Boundary Arc 5* ($p > 0$, $w = 1 - \delta$) whereas the continuous parts indicate the *Interior Arc*. The interpretation is straightforward: As long as there are many drug users, a maximum amount of treatment is delivered to decrease A as fast as possible. Once near the equilibrium, less treatment is more cost efficient, as the high treatment effort costs more than a few drug users. The optimal control paths can be seen in more detail in

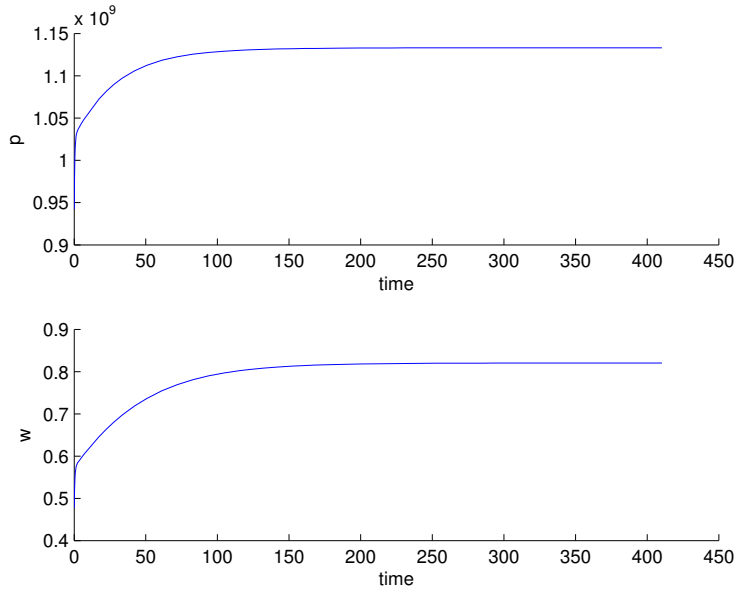

Figure 4.6.: Plot of the optimal control path for the initial value $(A_0, T_0) = (1, 1) \times 10^6$.

Figures 4.6 and 4.7, which show the optimal controls for the lowest and highest initial values, respectively.

In the case of the low starting value, both p and w rise in a concave fashion towards their long-run value. So, when at first there are few users, there is not so much treatment in order to let the numbers grow to their steady state value. In the case of the high initial value, it is the other way round. w starts off at the maximum of 0.89 and stays there for a while. After that, w falls, forming a convex graph. Interestingly, prevention increases as time goes by. The only difference to the path of the low initial value is the fact that p already starts at a higher level.

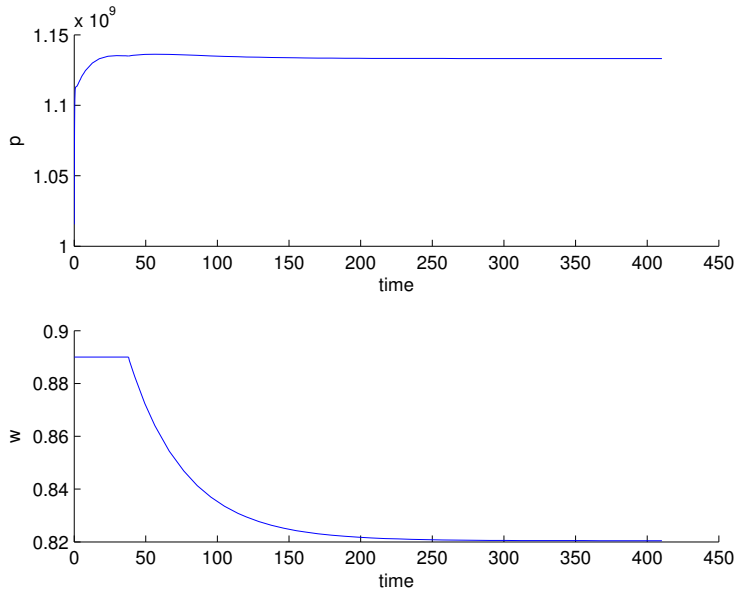

Figure 4.7.: Plot of the optimal control path for the initial value $(A_0, T_0) = (12, 2) \times 10^6$.

Chapter 5

Sensitivity Analysis

In this chapter I will show how the problem's long-term solution depends on the system's parameters. For this purpose, one single parameter will be varied while the others remain constant at their baseline values (see Table 2.1). For each set of parameters, the optimal steady state and the corresponding controls are then calculated. One can conduct this analysis in MATLAB either by using a loop or by deploying the MATCONT-toolbox (see [7]), which is a toolbox generally used for differential equations but not for optimal control problems in the first place. The corresponding commands are listed in the Appendix.

5.1. Social Cost c

The social cost of drug consumption per user, represented by the parameter c, is central to the model and ultimately the model's raison d'être. Without costs arising from drug consumption there is actually nothing to think about, as the solution is then simply $w \equiv p \equiv 0$ (at least as long as the policy maker is interested exclusively in cost reduction rather than harm reduction). A phase portrait of this situation was illustrated in Figure 3.9. With different costs stemming from consumption, the bigger picture changes substantially. Figure 5.1 illustrates the role that social cost c is playing. The figures are split in two, so that one can better see the behaviour for small values of c. The dashed vertical lines mark those values, where a control constraint changes from active to inactive or vice versa.

As already mentioned, for $c = 0$ both controls are equal to 0, and hence there are tremendous amounts of drug users and also quite a few teetotallers. This remains unchanged until $c \approx 34$, which is the location of the first dashed line. Here the policy maker starts deploying treatment. The higher c becomes, the more treatment is used.

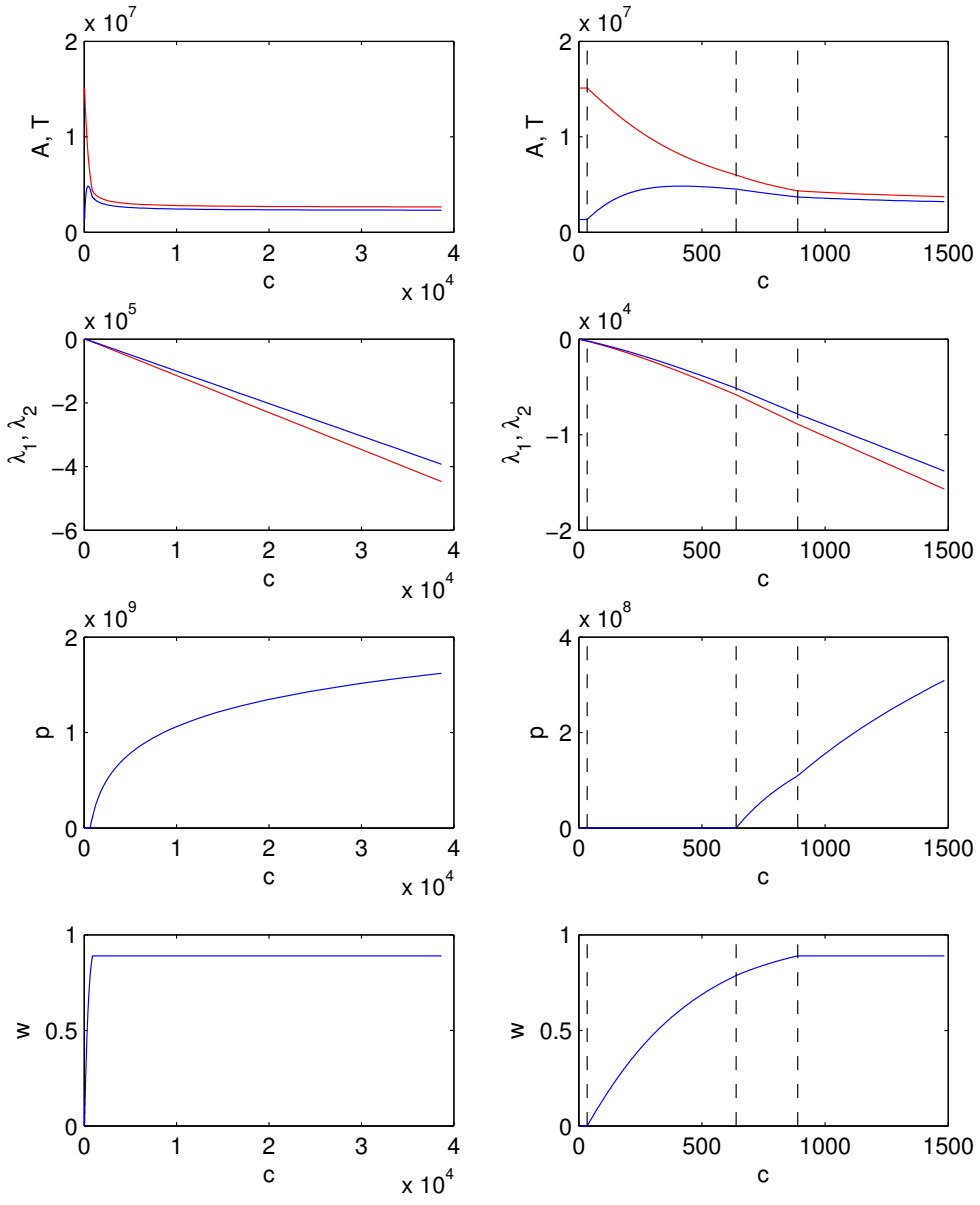

Figure 5.1.: The left panel shows c on the entire considered interval, while the right panel shows only comparably small values of c. The dashed lines at 34, 639, and 889 mark values where a control constraint becomes active or inactive. A and λ_1 are red. T and λ_2 are blue.

Prevention is still not considered. The number of drug users decreases significantly with growing costs due to the use of treatment measures. The number of teetotallers on the other hand increases simply because there are more people quitting drug consumption. However, at some point their quantity starts to decrease again when the inflow of quitting consumers becomes weaker. At $c \approx 639$ the next dashed line symbolizes the start of prevention measures. Treatment is still increasing but not maximal yet. Finally, at $c \approx 889$ treatment is used to the maximum extent and cannot be increased any further. From that point on, the number of drug users is not decreasing as rapidly as before any more, since the only possibility the policy maker has is to further increase spendings on prevention. However, this increase in prevention spendings is slowing down, which can be seen in the concave form of the curve. This is also not surprising, given the modelling of prevention with a maximum efficiency given by $1-h$. Spending more and more money on prevention is becoming less and less effective.

Looking at the costates λ_1, λ_2, one can see that they are decreasing all the time with no visible qualitative change at the dashed lines. The figure illustrates the obvious: the higher the social cost, the higher the impact of one additional consumer or teetotaller. Additionally, it can be seen that $\lambda_1 < \lambda_2, \forall c$, which means that one additional drug user costs more than one additional teetotaller.

To put it in a nutshell, variations of c are only relevant for a rather small value of c. Considering the base value $c = 10000$, variations of several thousand dollars upwards or downwards are pretty much irrelevant in terms of the long-term optimal policy and location of the equilibrium. Treatment will be given to anyone, as it is cheaper to treat all users than to accept a higher equilibrium than necessary.

5.2. Cost of Treatment Coefficient f_1

The analysis of c revealed once more that treatment is assumed to be too cheap not to be used to the full extent. But what happens, if treatment measures become more expensive? This question was already partly answered in Section 4.2.2, where I set $f_1 = 6 \times 10^{10}$. Here, the analysis is extended to a much larger variety of possible values for f_1.

Figure 5.2 reveals that at $f_1 \approx 4.3 \times 10^{10}$ the treatment control constraint becomes inactive. Beneath that point, no matter what the cost, both controls remain constant with treatment at its maximum level. Since both controls remain unchanged, the same holds true for the states and the costates. Once the threshold illustrated by the dashed

line is surpassed, treatment is so expensive that it is not optimal to use it to the full extent any more. Simultaneously, more money is put into prevention leading again to a concave graph. The number of drug users and teetotallers both rise with A showing the steeper incline. Obviously the higher prevention efforts cannot compensate for the reduction in treatment. The costates λ_1, λ_2 rise. λ_1 is always lower than λ_2 and their difference remains fairly equal.

5.3. Discount Rate r

The discount rate r determines how much the policy maker values future costs and future cost reductions. A large discount rate means that the future is not valued very highly, whereas a small r reflects a policy maker's far-sightedness. Given the importance of the discount rate in any infinite horizon optimal control problem, the results presented in Figure 5.3 are unspectacular. It can be seen that the discount rate r has not too much impact on the long-run solution. Treatment is always used to the full extent and only prevention payments are decreased with growing r. Consequently, the numbers of drug users and teetotallers rise as there are simply more people in the drug system since initiation is higher due to less prevention. However, the number of drug users never exceeds 5 millions, which is still far below the carrying capacity of 16.25 millions. The only dramatic changes are to be seen in the panel illustrating the costate variables. Initially they rise very sharply to remain almost constant starting at $r \approx 0.2$. The interpretation is simple and intuitive: the less I care about the future, the less I care about additional drug users or teetotallers in the long-term equilibrium. However, it is still interesting that the increase in shadow prices slows down significantly. At $r \approx 1.09$ prevention payments stop. Both controls remain unchanged from that point on, which means that the state variables stay constant as well while the costate variables continue to grow very slowly. Nevertheless, treatment plays a significant role. No matter how little we care about the future, treatment is still worth the extra spending.

5.4. Relapse Coefficient β

The occurrence of relapse is one of the central aspects of the model presented in this thesis. Important questions are: How do relapse and treatment coincide? Does treatment make sense when most teetotallers lapse back into drug use anyway? The answer given in Figure 5.4 is surprising: The higher the relapse rate, the higher the treatment

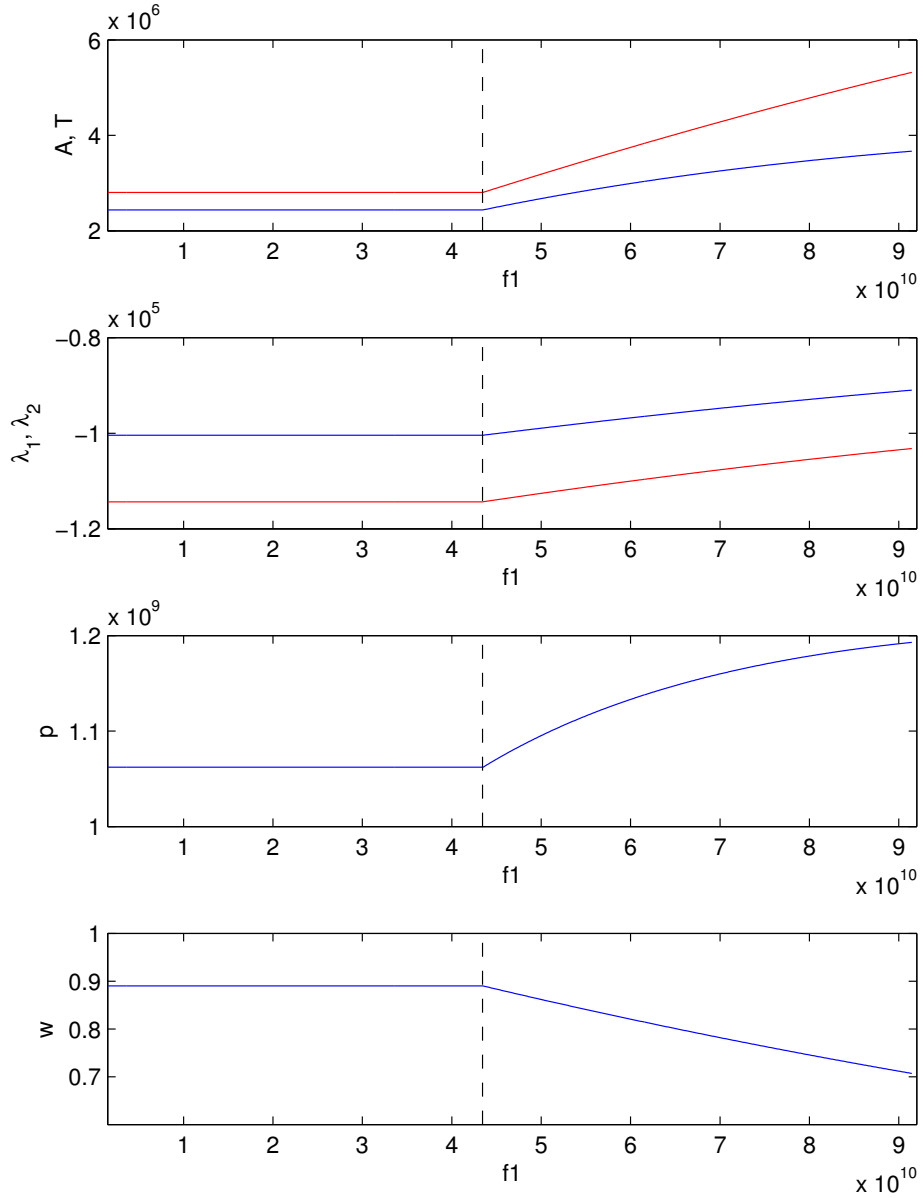

Figure 5.2.: The dashed line at 4.3×10^{10} marks the point where w starts to decrease. A and λ_1 are red. T and λ_2 are blue.

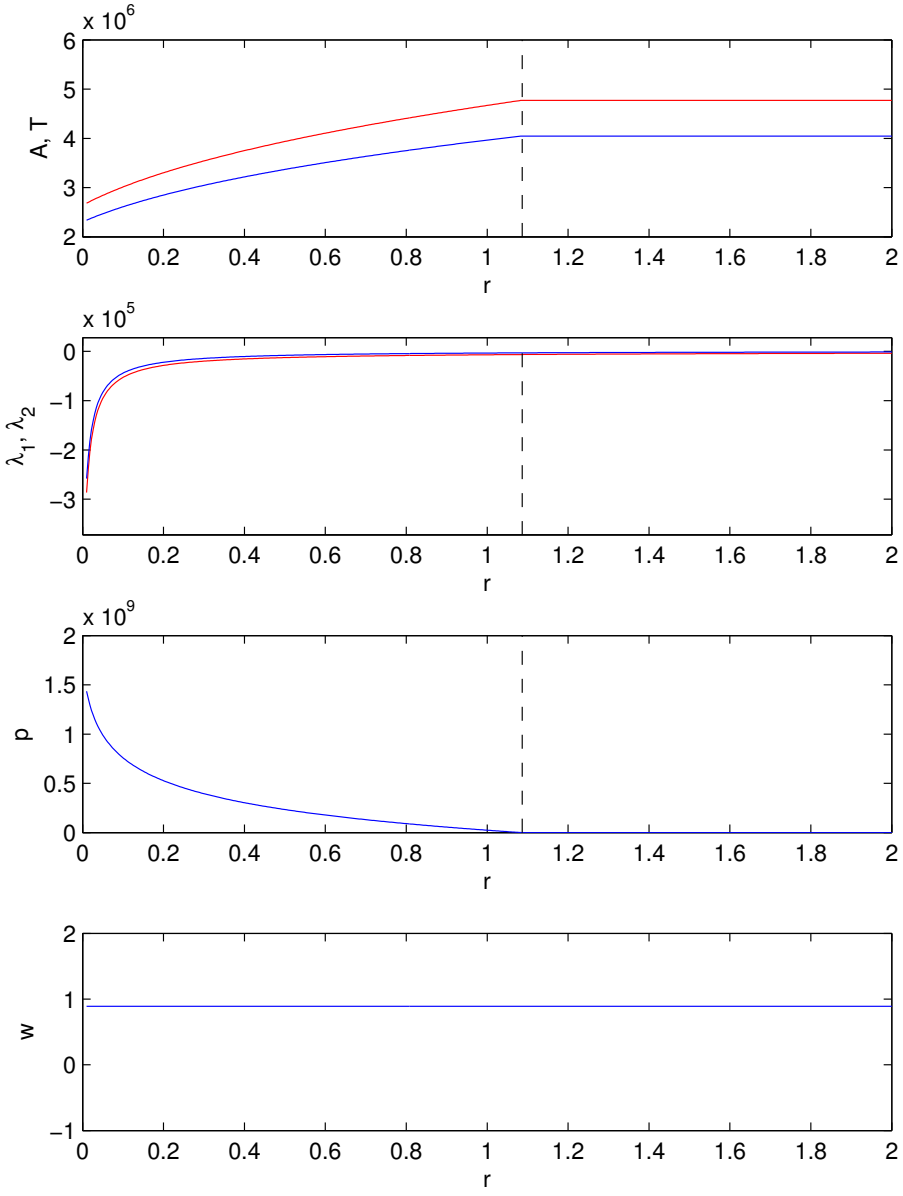

Figure 5.3.: The dashed line at $r \approx 1.09$ marks the point where prevention payments stop. A and λ_1 are red. T and λ_2 are blue.

efforts. The policy maker is not willing to accept high numbers of users, and even with high rates of relapse this can be achieved with treatment. Even in the case of $\beta = 1$ we have maximal use of treatment. The number of drug users rises of course with growing β, but A is always much lower than in the uncontrolled case. If $\beta = 0$, we have $\lambda_2 > 0$, which means the shadow price of teetotallers is positive and additional teetotallers are desirable. This is because they have an adverse affect on initiation, but without relapse they cannot lead to more costs. The states A and T are extremely low until the point where w cannot grow any more. From that point on, even though prevention is increased massively, A and T rise significantly with growing β. Prevention, however, does not rise forever and stagnates once β is above 0.5.

Since relapse is vital to the model, I want to take a look at the equilibrium when treatment costs are higher. Figure 5.5 illustrates the behaviour of the equilibrium with $f_1 = 12 \times 10^{10}$. Now the situation looks different. Treatment w never reaches its maximum use of 0.89. At first, w increases with β, but at $\beta \approx 0.53$ it starts to decrease. So, if treatment is expensive and relapse is probable, it is not optimal to treat everyone. Moreover, in such a case with high treatment costs, prevention is already used at a much lower level of β. Nonetheless, at higher levels of β, p is comparable to before. This once more shows that prevention cannot be used as a substitute for treatment, as it is by far less effective. The values of A and T are comparably higher of course.

Figures 5.6 - 5.8 describe the phase portraits for $f_1 = 12 \times 10^{10}$ and three different values of β, namely $0.5, 0.7$, and 0.9. As already mentioned, for growing β, the number of drug users in the steady state rises. However, the diagonal followed by the different paths on their way there becomes less steep. This means that the ratio of teetotallers to drug users in the steady state becomes smaller, which fully makes sense.

5.5. Teetotaller Oblivion Rate ρ

The parameter ρ determines how fast teetotallers leave their precarious state and go back to a normal life without the risk of relapsing. A smaller ρ implies a longer period in which former drug users stay at risk of relapsing. The longer a former drug user is at risk, the higher the probability of relapse. Figure 5.9 reveals the strong influence ρ has on the long-run solution. For small values of ρ, the number of drug users and teetotallers is extremely high and near the system's carrying capacity. However, these numbers plunge dramatically when ρ is increased and for $\rho \approx 0.12$, A is already below one million. Treatment efforts, measured by w, are at their maximum level until $\rho \approx 0.14$.

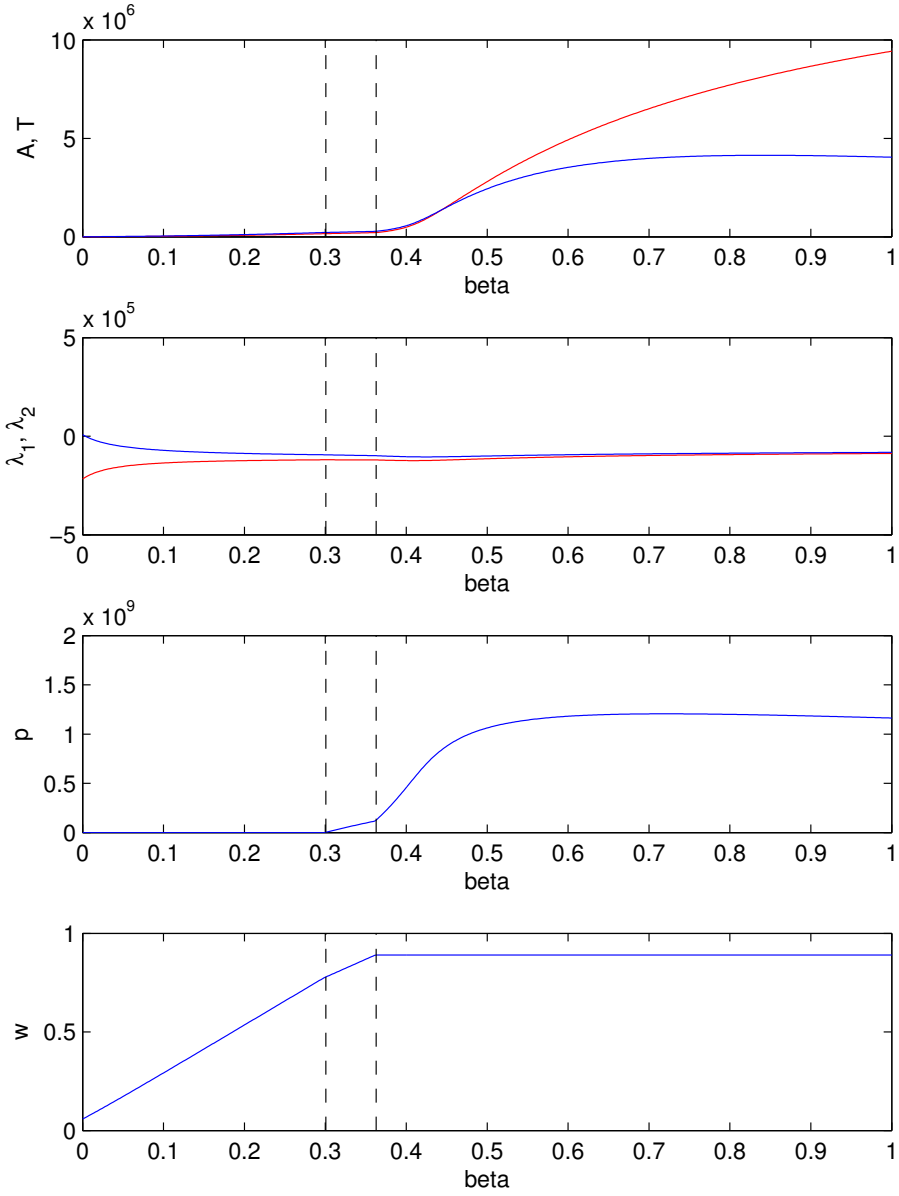

Figure 5.4.: At the first dashed line at $\beta \approx 0.30$, prevention starts. At the second line at $\beta \approx 0.36$, treatment gets maximal. A and λ_1 are red. T and λ_2 are blue.

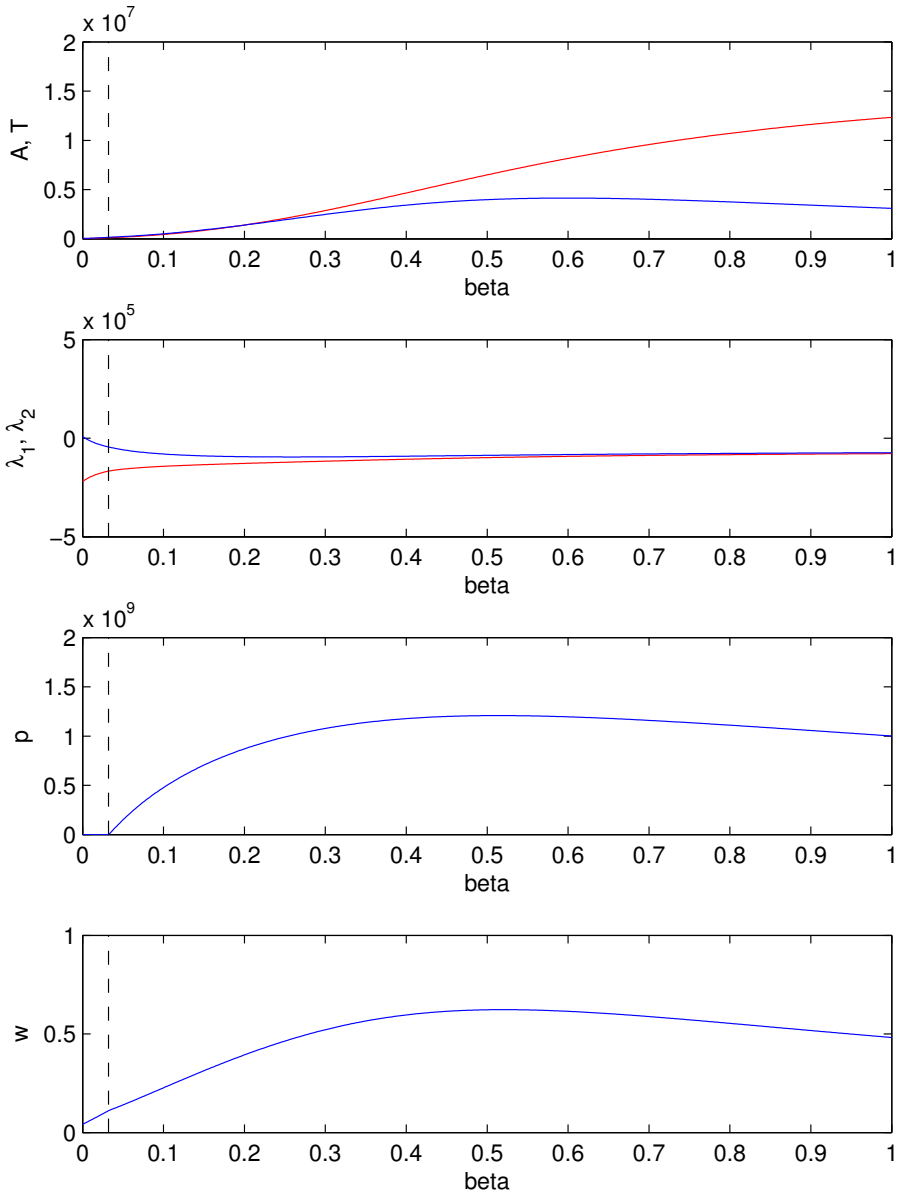

Figure 5.5.: Steady state values for $f_1 = 12 \times 10^{10}$. The dashed line at $\beta \approx 0.03$ marks the point where prevention starts. A and λ_1 are red. T and λ_2 are blue.

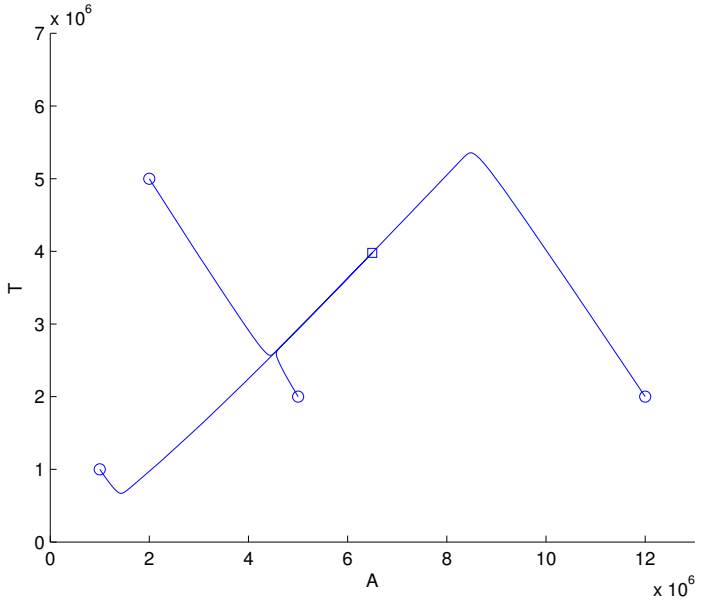

Figure 5.6.: Some optimal trajectories with $f_1 = 12 \times 10^{10}$ and $\beta = 0.5$.

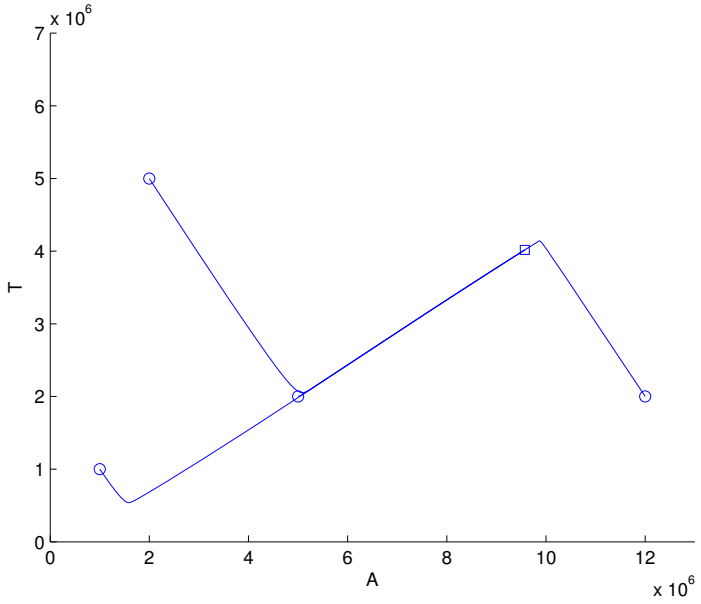

Figure 5.7.: Some optimal trajectories with $f_1 = 12 \times 10^{10}$ and $\beta = 0.7$.

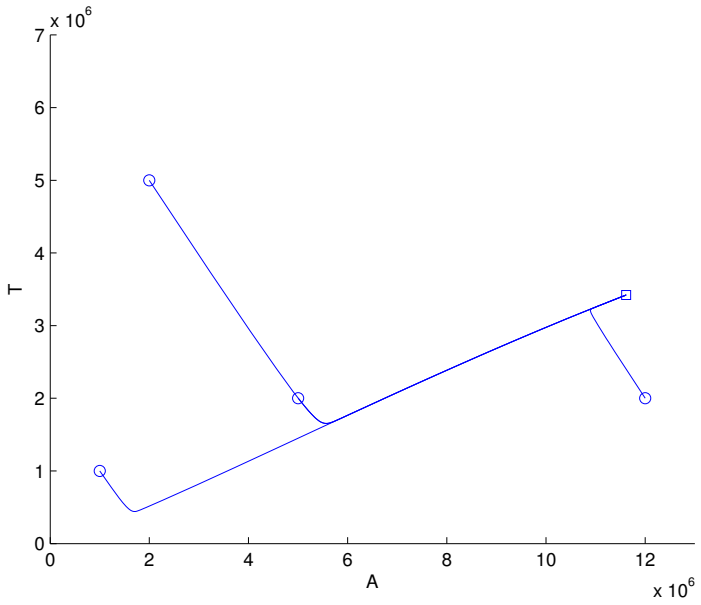

Figure 5.8.: Some optimal trajectories with $f_1 = 12 \times 10^{10}$ and $\beta = 0.9$.

Afterwards w decreases monotonously. Prevention p shows an inverted U-shape and is not used at all for $\rho > 0.19$.

To sum up, the parameter ρ has a huge impact especially on the values of A and T. Of course ρ may vary a lot among different types of drugs and the results here clearly show how important it is to carefully consider these different idiosyncrasies in relapse.

5.6. Relapse Parameter b_1

b_1 measures the persuasive effect of drug users on relapse. More drug users mean a higher probability of relapse. b_1 measures how much higher that is. Figure 5.10 shows that for small b_1, the number of drug users and teetotallers are negligible. However, for a larger b_1, A quickly comes close to the carrying capacity $a = 16,250,000$. T initially rises but becomes very small again for big values of b_1 because most teetotallers relapse since the persuasive effect of the large number of users is so strong. Treatment is used to the maximum extent for the most part. It only declines again at $b_1 \approx 0.247$, where most teetotallers relapse and make treatment nearly pointless. Prevention efforts p behave very similarly. What is interesting to observe in Figure 5.10 is the fact that both costates first fall and then rise again. In short, when looking for a reasonable choice for b_1, the options are very limited and one has to be very thoughtful, because the system is quite

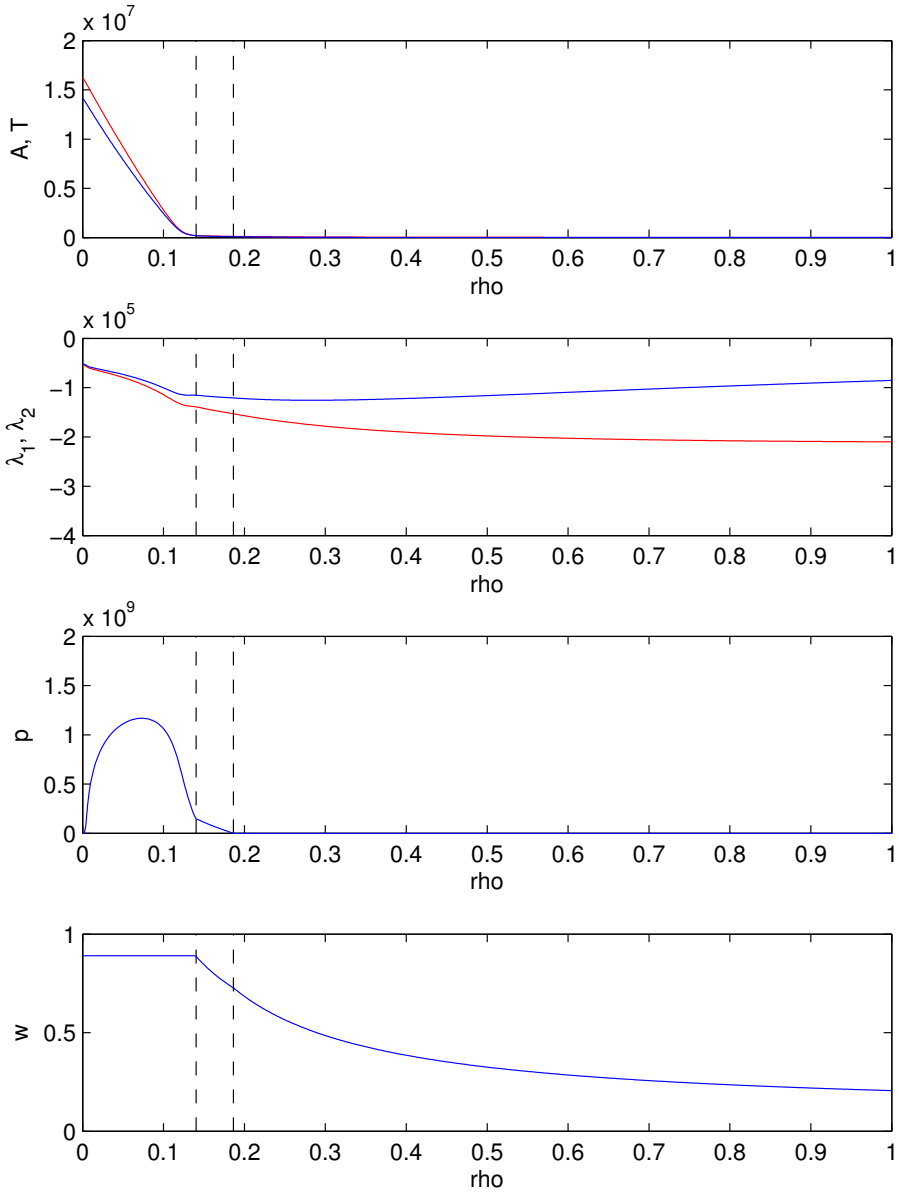

Figure 5.9.: The first dashed line at $\rho \approx 0.14$ marks the point until where treatment is maximal. The second line at $\rho \approx 0.19$ marks the point where prevention stops. A and λ_1 are red. T and λ_2 are blue.

sensitive with respect to changes in b_1.

5.7. Relapse Parameter b_2

Similarly to b_1, b_2 also measures persuasive influence, but in this case influence of teetotallers on themselves. The more teetotallers there are, the more will relapse, and the higher b_2, the larger this effect. Figure 5.11 shows that for small values of b_2, relapse is so low that the number of drug users and teetotallers are negligible. For $b_2 \approx 0$, the shadow price of teetotallers is even positive which means that an additional teetotaller has a positive effect on overall cost because her/his negative effect on initiation is bigger than her/his positive effect on relapse. Prevention is not used and treatment stays at a very low level until b_2 comes close to 1. At $b_2 \approx 0.977$, w becomes maximal, cannot be raised any more, and therefore A and T start skyrocketing. Prevention payments start at $b_2 \approx 0.959$, rise quickly and decrease again. For $b_2 \approx 1.230$, relapse becomes so frequent, that maximal treatment efforts are not optimal any more. Also prevention payments stop at $b_2 \approx 1.248$. A is now close to its carrying capacity, and T is close to 0 as teetotallers more or less instantly relapse. To put it in a nutshell, Figure 5.11 shows that there is only a very small region around the baseline value of $b_2 = 1$ (see Table 2.1) where the choice of b_2 actually seems to make sense. If b_2 is too low, the drug problem does not really exist any more. If it is too high, the drug epidemic is so bad that it does not even make sense to control it.

5.8. Initiation Parameter ω

ω describes the persuasive influence of drug users on initiation and is therefore technically similar to b_2. Figure 5.12 shows that ω influences the system indeed in a very similar way as b_2 does (cf. Figure 5.11). For small values of ω, the drug problem is negligible and there are no controls applied. At $\omega \approx 0.712$, treatment starts and at $\omega \approx 0.961$, also prevention payments start. Shortly after that, w reaches its upper bound and cannot increase any further. At that point A and T significantly climb together. When $\omega > 1$ initiation is heavily influenced by existing users and therefore more and more people start taking drugs. For even higher levels of influence, prevention and treatment are not even used any longer, since initiation is so strong that trying to control the drug epidemic is just a waste of money. Therefore, A comes close to the carrying capacity, whereas T goes back to a very low level. Obviously, for the choice of ω there is only a

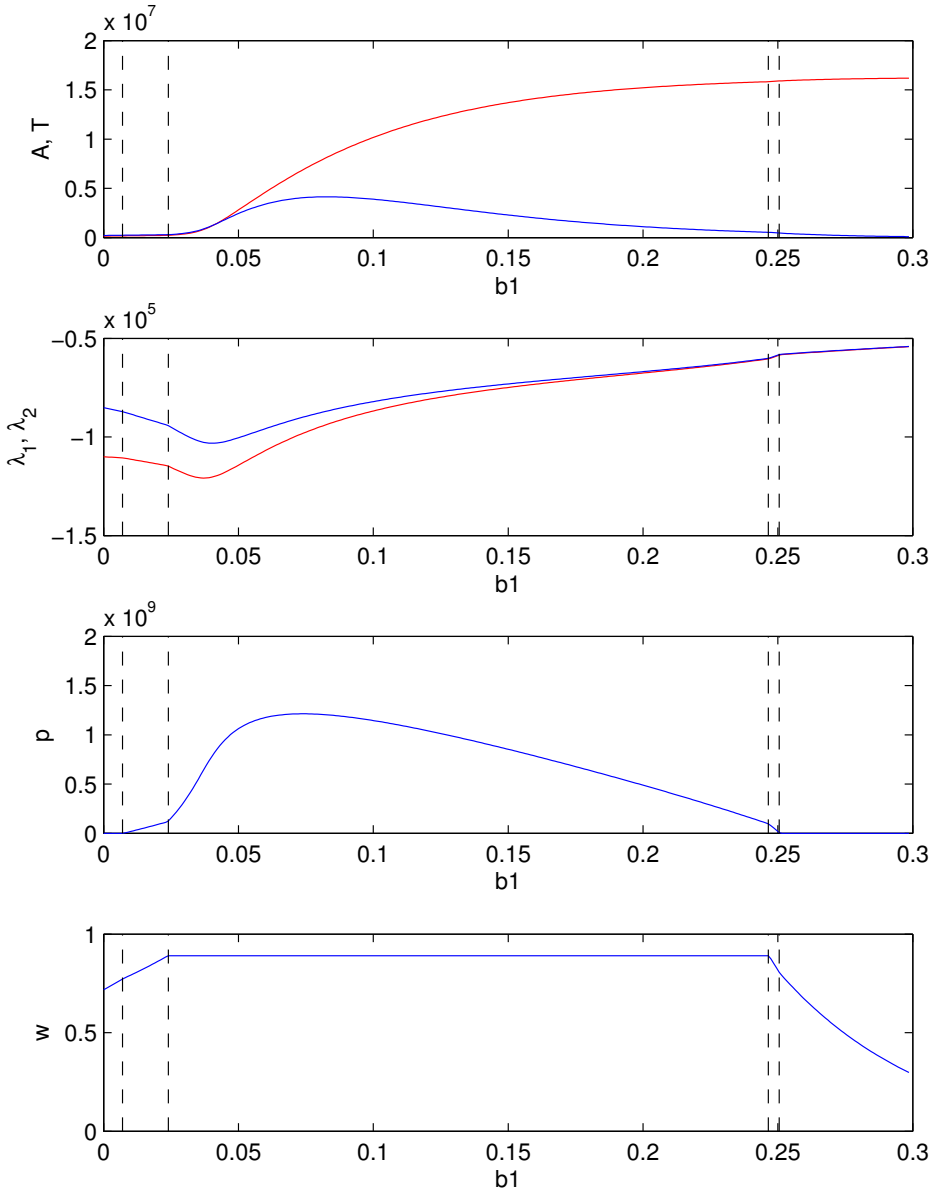

Figure 5.10.: The dashed lines at $0.007, 0.024, 0.247, 0.251$ mark the parameter values where control constraints become active or inactive. A and λ_1 are red. T and λ_2 are blue.

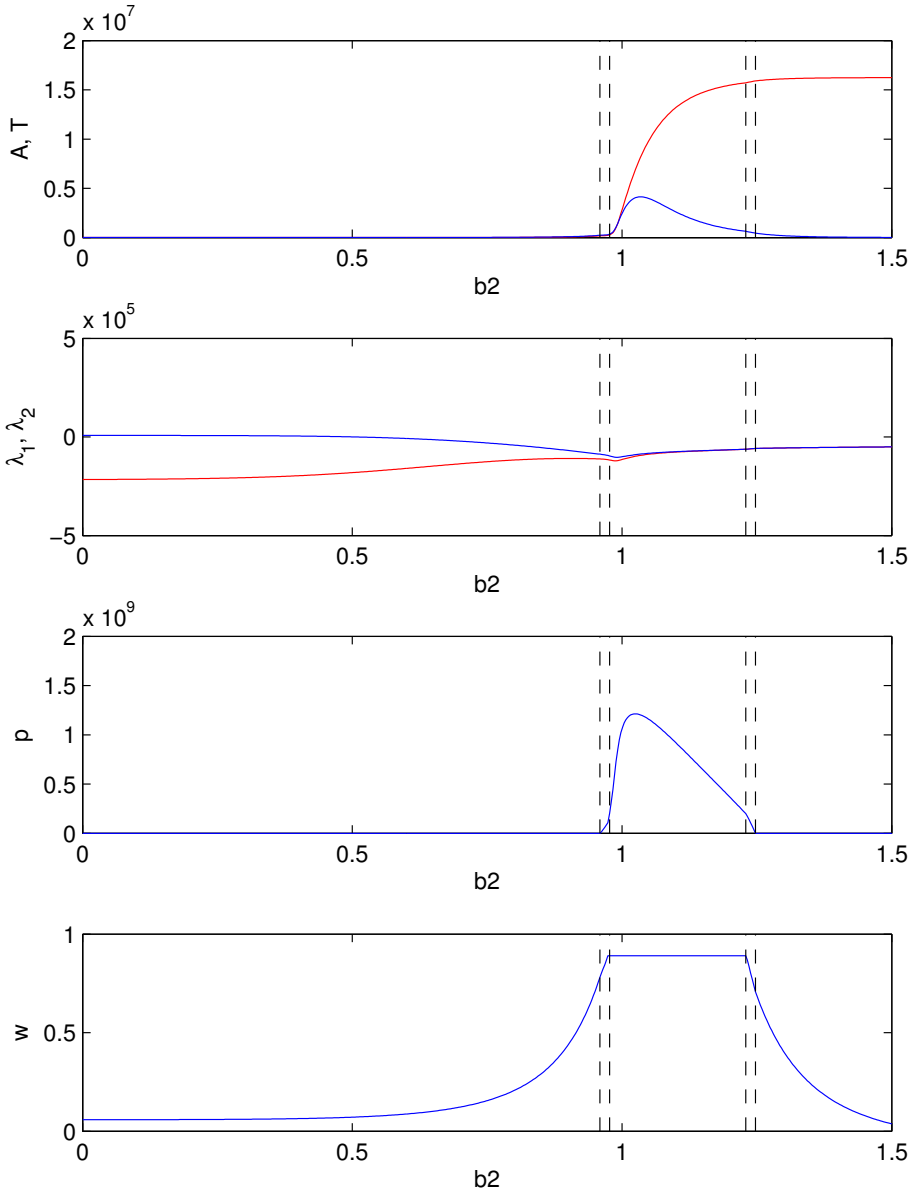

Figure 5.11.: The dashed lines at $0.959, 0.977, 1.230, 1.248$ mark the parameter values where control constraints become active or inactive. A and λ_1 are red. T and λ_2 are blue.

small reasonable region around $\omega = 1$. In all other cases, the drug problem does not exist or is so invincible that it is not controlled at all.

5.9. Initiation Parameter γ

Last but not least, the negative parameter γ measures the dissuasive influence of teetotallers on initiation. The smaller γ is, the more teetotallers hinder initiation. In Figure 5.13 one can see that for $\gamma \leq -0.074$, A and T are close to zero as there is almost no initiation. However, this is only possible due to growing levels of treatment. Once w is maximal, A and T rise quickly. But even with $\gamma = 0$, the drug epidemic is fairly far from its maximal possible spread given by the carrying capacity a.

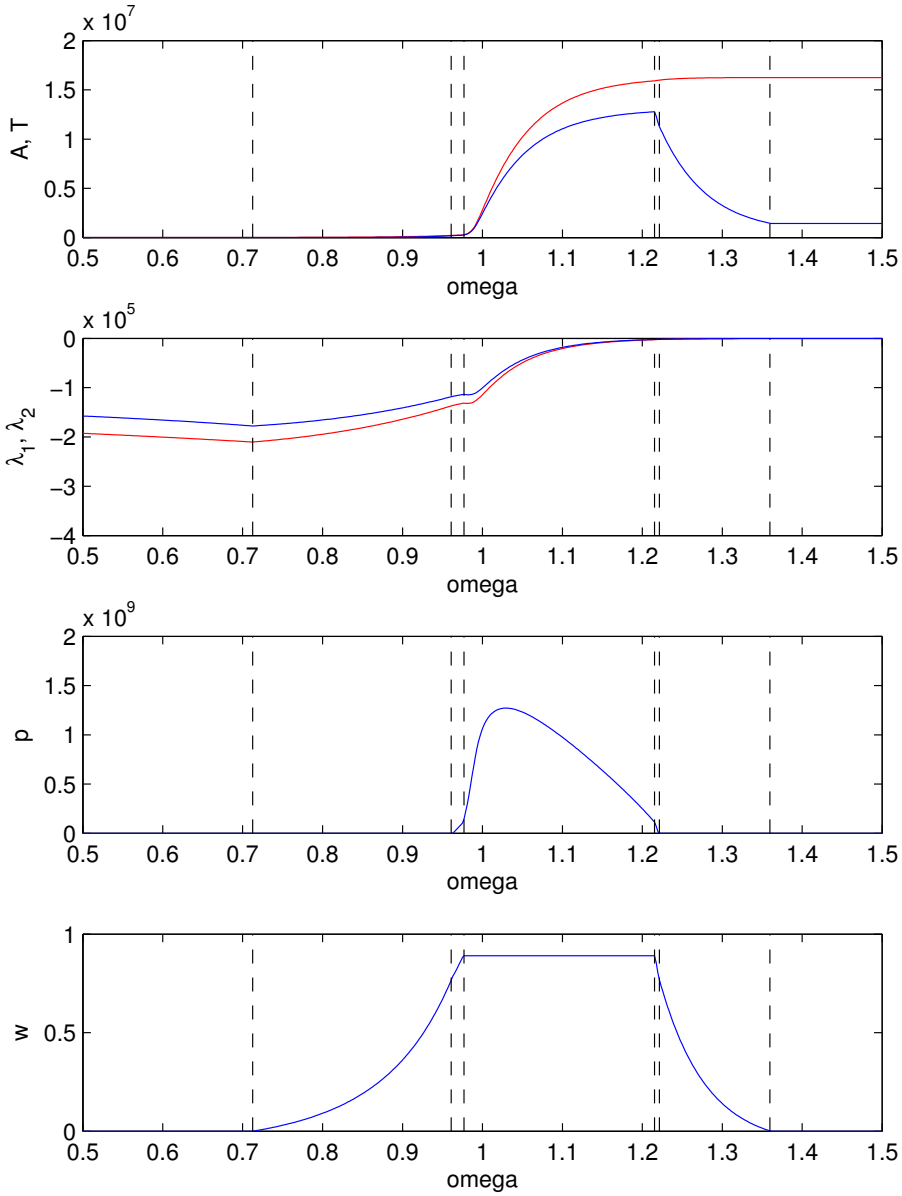

Figure 5.12.: The dashed lines at $0.712, 0.961, 0.977, 1.215, 1.221, 1.360$ mark the parameter values where control constraints become active or inactive. A and λ_1 are red. T and λ_2 are blue.

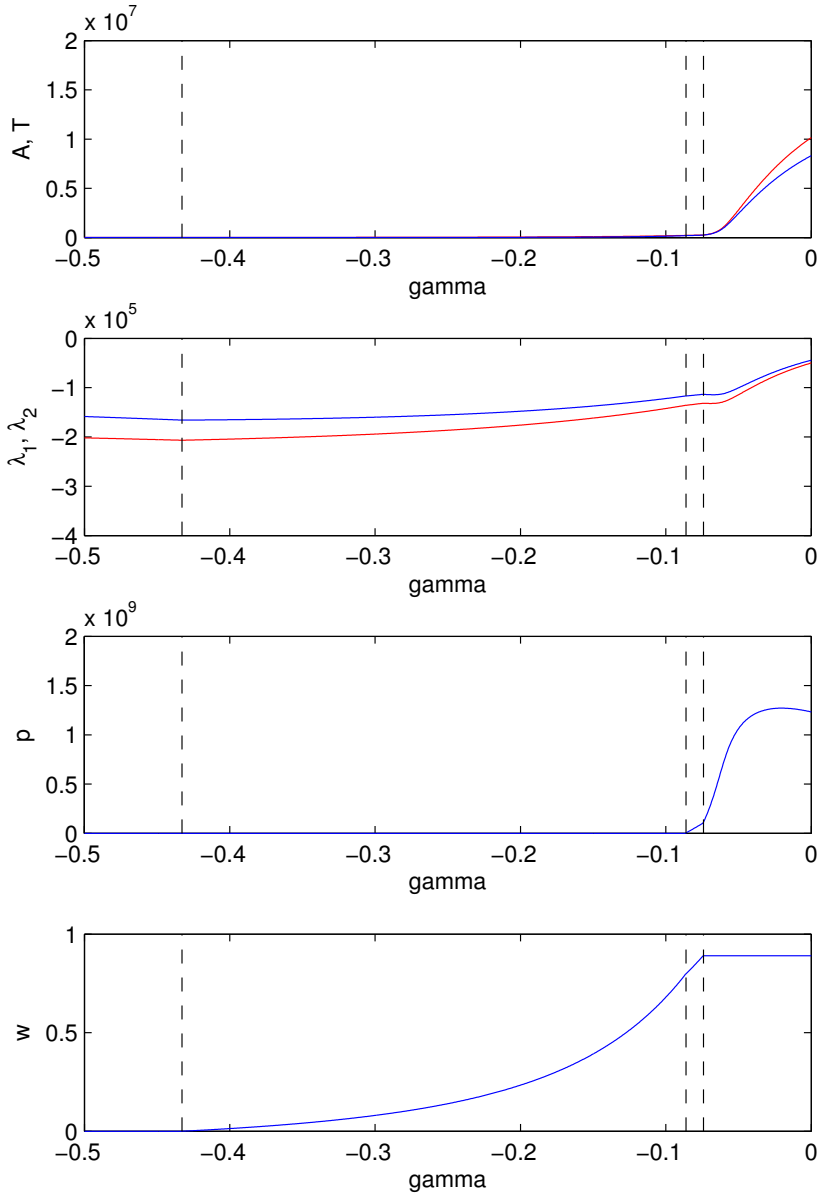

Figure 5.13.: The dashed lines at $-0.433, -0.086, -0.074$ mark the parameter values where control constraints become active or inactive. A and λ_1 are red. T and λ_2 are blue.

Chapter 6

Summary and Conclusions

One important objective of this thesis was to answer the question whether or not treatment of drug users is financially reasonable when relapse is considered. In order to throw some light on this issue, a new dynamic drug model was developed. The model consisted partly of some already well examined dynamics from past works on that field but also introduced some new aspects. The most decisive extension was definitely the incorporation of the state of teetotallers whose relapse induces an additional inflow into drug consumption. The baseline parameters were taken from existing works for the most part.

The first step in the analysis was the exploration of the uncontrolled system dynamics. In a simplified model with convenient parameter choices, steady states could be derived analytically. Moreover, a condition for the asymptotic stability of the feasible positive steady state was deduced. For the general case, a condition for the existence of steady states was obtained. Phase portraits gave good impressions and understanding of the system's behaviour and the stability of its steady state. It is also worth highlighting that the uncontrolled dynamics coincide qualitatively with the dynamics with fixed controls. This means that this section also shows what the implications of a constant policy would be.

The next step was the derivation of the optimal solution. For that purpose, Pontryagin's Maximum Principle was applied. The so-obtained canonical system was analysed using the MATLAB-toolbox OCMAT. One single saddle point equilibrium was found and the stable paths were calculated. The results allow one main conclusion: Given the baseline parametrisation, treatment is deployed as much as possible. Consequently, the equilibrium is rather low compared to the uncontrolled case. This means that even though some treated former drug users lapse back into drug use, the positive effects of treatment still outweigh their costs. Naturally, when the costs of treatment are increased,

the treatment efforts decline but still remain on a relatively high level.

The sensitivity analysis revealed the parameters' impact on the long-run solution. The consequences of varying levels of the relapse parameter β was of primary interest. Surprisingly, it turned out that with growing β the use of treatment increased as well. However, this monotonous relation changed, when the treatment cost was increased. In that case the control-variable w describing treatment exhibited an inverted U-shape for varying β: for a low relapse rate not so much treatment is deployed. When β grows, w grows as well, but when β becomes too large, treatment efforts decline again. Furthermore, it was shown that some parameters like b_1, b_2, and ω only have a small range in which the model yields reasonable results. Therefore, these values should be considered particularly carefully, e.g., when estimating them with an econometric model.

To sum up, the answer to the question whether treatment is reasonable in a scenario with high relapse rates is not a simple yes or no. It depends on the cost structure of treatment and the social costs of drug consumption on the one hand and the relapse rate on the other hand. When costs of treatment are sufficiently high and the relapse rates are high too, treatment efforts decline. However, for reasonable parameter combinations, treatment was never zero. By and large it has to be concluded, that offering treatment to at least some drug users is beneficial in any case.

Appendix A

The Matlab-Toolbox OCMat

The OCMat toolbox provides functions for analysing optimal control problems. It can be accessed and downloaded for free via the Internet.[1] A manual for the toolbox can also be found there (see [9]).

A.1. Initialization

Before one can start the analysis of an optimal control problem with OCMat, the model has to be initialized. For that purpose, an initialization file, in which the optimisation problem is defined, has to be created. The initialization file is a plain ASCII file with the extension `.ocm`. The initialization file looks like that:

```
Type
standardmodel

Description
drugmodel for thesis

Modelname
drugmodel

Variable
state::A,T
control::p,w
```

[1] `http://orcos.tuwien.ac.at/research/ocmat_software/`, last accessed March 11, 2016.

```
Statedynamics
 ode::DA=alpha*A^omega*T^gam*(a-A)*(h+(1-h)*exp(-m*p))-...
         (delta+w)*A+beta*A^b1*T^b2
 ode::DT=(delta+w)*A-beta*A^b1*T^b2-rho*T

Objective
int::-c*A-p-f0*w-f1*w^2/2

Controlconstraint % identifier has to contain an alphabetic character
CC1::ineq::p>=plow
CC2::ineq::w<=1-delta
CC3::ineq::w>=wlow

ArcDefinition
0::[]
1::CC1
2::CC2
3::CC3
4::CC1,CC2
5::CC1,CC3

Parameter
r::0.04
alpha::1.581272e-8
h::0.84
m::2.37e-9
omega::1
gam::-0.05
a::16250000
delta::0.11
beta::0.5
b1::0.05
b2::1
c::10000
f0::3.618569e+8
```

```
f1::4.778180e+9
plow::0
wlow::0
rho::0.1
```

The definition of the variables, state equations, objective function, and parameters is pretty self-explanatory. Every control constraint has to be named individually. These names are then used in the definition of the different arcs. The arcs are numbered, and for each arc, the active constraint has to be defined. Note that `CC2` and `CC3` cannot be active at the same time. The number assigned to each arc is the so-called arc identifier. In the next step, the initialization file is processed and the model files are created and stored in the correct folder.

```
ocStruct=processinitfile('drugmodel');
modelfiles=makefile4ocmat(ocStruct);
moveocmatfiles(ocStruct,modelfiles);
m=stdocmodel('drugmodel');
save(m)
```

In the object `m` all important information is stored. For example, `hamiltonian(m)` returns the Hamiltonian; `control(m)` returns the controls that maximise the Hamiltonian (interior solution).

A.2. Equilibria and Stable Paths

The equilibria of the canonical system are calculated by the function `calcep(m)`. However, in this case the canonical system cannot be solved explicitly. Therefore, this call produces an error. To tell the function that it shall use a numerical search algorithm rather than trying to solve the equation analytically, an initial value has to be provided:

```
ocEP=calcep(m,[5e6; 5e6; -1e3; -1e3]);
b=isadmissible(ocEP,m);
ocEP(~b)=[];
```

In the second and third line, the inadmissible solutions are identified and discarded. It is possible that no admissible solution is found. In that case, the initial values should be altered a bit. Moreover, when an admissible solution is finally found, this does not mean

that no other admissible steady state exists. Therefore, one should systematically vary the initial values in order to scan the entire admissible region. In the case of this thesis' model, there is strong evidence suggesting that there indeed exists only one steady state: After a very thorough search, no second equilibrium could be identified, and even for starting values very far from the steady state the search algorithm converged to that seemingly unique steady state. Basic information about the equilibrium is accessed by typing `ocEP{1}`, which returns

```
ans =

ocmatclass: dynprimitive

modelname: drugmodel

Equilibrium:
  1.0e+06 *

    2.8043
    2.4376
   -0.1144
   -0.1004

Eigenvalues:
   -2.0260
    2.0660
   -0.0107
    0.0507

Arcidentifier:
    2
```

Arcidentifier 2 means that the second control constraint is active with $w = 1 - \delta$. The controls and Lagrange multipliers are accessed via `control(m,ocEP{1})` and `lagrangemultiplier(m,ocEP{1})`, respectively.

To calculate the saddle-path from the initial value $(1, 1) \times 10^6$ to the equilibrium stored in `ocEP{1}`, the following commands are called:

```
opt=setocoptions('OCCONTARG','MaxStepWidth',1e5,'InitStepWidth',1e0,...
'MaxContinuationSteps',inf,'CheckAdmissibility','on','SBVPOC',...
'BCJacobian',0,'MeshAdaptAbsTol',1e-3,'MeshAdaptRelTol',1e-4,...
'NMax',10000,'GENERAL','AdmissibleTolerance',1e-3);
epidx=1;
eigval=real(eig(ocEP{epidx}));
eigval(eigval>-1e-5)=[];
T=10/min(abs(eigval));

sol=initocmat_AE_EP(m,ocEP{epidx},1:2,[1e6;1e6],opt,'TruncationTime',T);
c=bvpcont('extremal2ep',sol,[],opt);
store(m,'extremal2ep');
```

The first block of commands sets out some options conveniently. Afterwards, the continuation process is initialized by `sol`. The third argument tells the function, which parameters are to be continued. In our case these are the first two, which represent A and T. The fourth argument defines the initial value to which the solution shall be continued in the end. The continuation itself is then conducted by `bvpcont` and saved in the object `m`. The result of the continuation can be accessed by calling `m.Result.Continuation{1}`.

This procedure is now repeated for other initial values. It is important to remember to always store the results, as the calculations can take quite some time, so it would be very impractical to do the same calculations all over again. Once all desired calculations are accomplished, they can be illustrated graphically. There are several options how to achieve this. I used the following functions to generate for example Figures 4.1 and 4.3, which show some optimal state and control trajectories.

```
%PHASE PORTRAIT
plotcont(m,'state',1,'state',2,'index',[1 2 3 4],'color','b')
hold on
plot(ocEP{1}.y(1),ocEP{1}.y(2),'Linestyle','none',...
     'Marker','s','Markersize',6)
xlabel('A')
ylabel('T')
xlim([0 13000000])
ylim([0 7000000])
plot(1000000,1000000,'bo')
```

```
plot(12000000,2000000,'bo')
plot(2000000,5000000,'bo')
plot(5000000,2000000,'bo')
hold off

%CONTROLS
subplot(2,1,1)
plot(m.Result.Continuation{1}.ExtremalSolution,1,1,'xdata','time',...
'ydata','control','ocmodel',m,'color','b')
ylabel('p')
xlabel('time')
subplot(2,1,2)
plot(m.Result.Continuation{1}.ExtremalSolution,1,2,'xdata','time',...
'ydata','control','ocmodel',m,'color','b')
ylabel('w')
xlabel('time')
```

The indices provided to `plotcont` in the first line are the indices of the desired solutions stored in `m.Result.Continuation`.

Unfortunately, performing the continuation process does not always go as smooth as above. Problems arise when the arc, the solution is currently on, changes. This change of arcs has to be done manually. That occurred for example in the case with $f_1 = 6 \times 10^{10}$ for the initial value $(12, 2) \times 10^6$: After changing the parameter value of f_1 and calculating the equilibrium, the continuation process is started exactly like before. However, the function `bvpcont` is interrupted when the stepsize is getting too small. When no new admissible solution can be found, the step size is decreased. Once the stepsize is under a certain threshold, it can be assumed that no admissible solution on the same arc exists. This means that the arc has to be changed. Then a new continuation is started that tries to reach the initial value from the point where the last iteration stopped. These two paths are combined to one path:

```
m=stdocmodel('drugmodel');
m=changeparametervalue(m,'f1',6e10);

ocEP=calcep(m,[5e6;5e6;-1e3;-1e3]);b=isadmissible(ocEP,m);
ocEP(~b)=[];
```

```
opt=setocoptions('OCCONTARG','MaxStepWidth',1e5,'InitStepWidth',1e0,...
'MaxContinuationSteps',inf,'CheckAdmissibility','on','SBVPOC',...
'BCJacobian',0,'MeshAdaptAbsTol',1e-3,'MeshAdaptRelTol',1e-4,...
'NMax',10000,'GENERAL','AdmissibleTolerance',1e-3);
epidx=1;
eigval=real(eig(ocEP{epidx}));
eigval(eigval>-1e-5)=[];
T=10/min(abs(eigval));

%calculate path, part 1
sol=initocmat_AE_EP(m,ocEP{epidx},1:2,[12e6;2e6],opt,'TruncationTime',T);
c=bvpcont('extremal2ep',sol,[],opt);
store(m,'extremal2ep');

%part 2
ocEx=extremalsolution(m);n=length(ocEx);

opt0=setocoptions('GENERAL','AdmissibleTolerance',0);

[b infoS newarcpos violarcarg]=testadmissibility(ocEx{n},m,opt0);

ocAsymN=redefinearc(ocEx{n},newarcpos,2);
sol=initocmat_AE_AE(m,ocAsymN,1:2,[12e6;2e6]);

opt=setocoptions('OCCONTARG','MaxStepWidth',1e5,'InitStepWidth',1e2,...
'MaxContinuationSteps',inf,'CheckAdmissibility','off','SBVPOC',...
'BCJacobian',0,'MeshAdaptAbsTol',1e-6,'MeshAdaptRelTol',1e-7,...
'NMax',10000,'GENERAL','AdmissibleTolerance',1e3,'NEWTON',...
'MaxNewtonIters',15,'MaxProbes',10);
c=bvpcont('extremal2ep',sol,[],opt);
store(m,'extremal2ep')
```

Note that in the second part of the path calculation, the tolerances are lower to tackle numeric instability. In `redefinearc(ocEx{n},newarcpos,2)` the last argument provides the new arcidentifier. This has to be chosen manually. By looking at `infoS.constraintvalue`

one can see which Lagrange multiplier became negative and therefore draw conclusions which new arc should be chosen. Alternatively, one can simply do it by trial and error.

For plotting the corresponding phase portrait (Figure 4.5), a slightly different approach than before is chosen in order to illustrate the different arcs by dashed and normal lines:

```
ocEx=extremalsolution(m,[1 3 5 6]);

arcid=[];
for ii=1:length(ocEx)
arcid=[arcid arcargument(ocEx{ii})];
end

h=plotcont(m,'state',1,'state',2,'index',[1 3 5 6]);
for ii=1:length(h)
if arcid(ii)==0
set(h(ii),'Color','b','LineStyle','-','LineWidth',1)
elseif arcid(ii)==2
set(h(ii),'Color','b','LineStyle','--','LineWidth',1)
end
end
hold on
plot(ocEP{1}.y(1),ocEP{1}.y(2),'Linestyle','none',...
     'Marker','s','Markersize',6)
xlabel('A')
ylabel('T')
xlim([0 13000000])
ylim([0 7000000])
plot(1000000,1000000,'bo')
plot(12000000,2000000,'bo')
plot(2000000,5000000,'bo')
plot(5000000,2000000,'bo')
hold off
```

The indices in the first line are chosen that way, because the second and third solutions consist of two different arcs. Consequently, the solutions with indices 2 and 4 consist only of the first half of the respective path. The whole path is stored in 3 and 5, respectively.

Apart from the indices, the plot command is the same as before. The difference is that afterwards the linestyle is defined for the two different arcids.

A.3. Sensitivity

For generating the plots of Chapter 5, one could use the toolbox MatCont. However, this toolbox does not provide any admissibility checks, which makes its usage a bit more complicated. Therefore, I simply used a loop and OCMat to iteratively calculate new admissible equilibria for new parameters. In every step, the important information is stored in a matrix containing the values of the parameter, states, costates, and controls in the equilibrium. The following code shows the process for the parameter β:

```
interval = linspace(0,1,500);
RES_beta=zeros(7,length(interval));
RES_beta(5,:)=interval;

m2=changeparametervalue(m,'beta',interval(1));
ocEP2=calcep(m2,[2e+5; 4e+6; -2e+5; -2e+4]);b=isadmissible(ocEP2,m2);
ocEP2(~b)=[];
RES_beta(1:4,1)=ocEP2{1}.y;
RES_beta(6:7,1)=control(m2,ocEP2{1});

for i=2:length(interval)
m2=changeparametervalue(m,'beta',interval(i));
ocEP2=calcep(m2,RES_beta(1:4,i-1));b=isadmissible(ocEP2,m2);
ocEP2(~b)=[];
RES_beta(1:4,i)=ocEP2{1}.y;
RES_beta(6:7,i)=control(m2,ocEP2{1});
end
```

The last steady state is used as an initial solution for the numeric search for the next steady state. Sometimes `calcep` fails to find an admissible solution. In that case one has to choose a better starting value manually or reduce the step-width in the parameter grid. Once the process is complete, the obtained results can easily be plotted by using `plot`.

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