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DIPLOMARBEIT

Supply and Demand in Three-State Dynamic Models
of Australian IDU and U.S. Cocaine Use

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unter der Anleitung von
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durch

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Abstract

This thesis deals with a three-state dynamic model of illicit drug consumption. In addition to susceptible non-users and current users of a drug, for the first time also the supply capacity is considered in a separate state variable. The model is analyzed with parameters derived from data for the U.S. cocaine epidemic and for injection drug use (IDU) in Australia.
In the first part the uncontrolled base model is described and analyzed. Initiation into drug use is assumed to depend primarily on the interaction between susceptibles and users. For the U.S. cocaine epidemic the positive feedback effect from users acting on susceptibles is best described by a convex function, which allows for two stable equilibria separated by a "tipping curve". However, for Australian IDU the best fit is obtained for a concave initiation function, for which only one (stable) steady state occurs. That is one reason why overall the U.S. parameterization yields somewhat more interesting and insightful results.
The second part of this thesis presents some "strategic examinations", which are assumed to provide decision support for public policy and can be seen as a preliminary step towards an optimal control model formulation. In this regard the relative efficiency and cost-effectiveness of treatment and supply shocks at different stages of the epidemic are determined. One preliminary conclusion is that treatment or a supply shock early in the epidemic is more valuable than later in the epidemic.

Contents

Chapter 1

Introduction

The abuse of illicit drugs poses a great challenge for societies and decision makers all over the world. Consequently, over the past years several models have been accumulated in the field of optimal dynamic control of drug use. One of the most recent models is the so-called SA model in which the group of susceptible non-users and the group of users are the states (see, e.g., [Caulkins et al., 2009a], [Caulkins et al., 2009b], [Wallner, 2008]). This thesis deals with an extension of this model based on [Caulkins, 2008]. One motivation is that the SA model is an appealing vehicle for extensions because it is a form of the well-accepted SIR models used in mathematical epidemiology and there are parameterizations for U.S. cocaine and Australian injection drug use (IDU). Our analyses provide a basis for (1) looking at the question of optimal timing of enforcement versus treatment with a richer model of drug use and (2) examining how the optimal response to supply shocks may vary over the course of an epidemic.

The extension involves adding a third state variable, C, which can be understood as the "capital stock" of the drug smuggling industry. However, this capital stock represents social/relational capital and tacit knowledge in comparison with capital stocks in a manufacturing context. Thus, the model is a dynamic three-state model of drug use in which drug supply, price and demand are connected. We will refer to this model as the SAC model.

The first part of this thesis concentrates on an extensive analysis of the

uncontrolled model whereas the second part consists of some strategic examinations which can be seen as a preliminary stage to an optimal control model approach.

Chapter 2 contains the mathematical formulation of our base model as well as the parameter values for the U.S. cocaine and Australian IDU epidemics.

In Chapter 3 this base model is analyzed. First of all, the steady states and their stability behavior are determined. Both numerical values and graphical illustrations are presented. Furthermore, some time paths are displayed.

The results of the sensitivity and bifurcation analyses are represented in Chapter 4. At the beginning, only small modifications of the parameter values are investigated. Later on, we consider wide ranges of parameter values and find the blue sky bifurcation points.

Chapter 5 deals with a comparison between the SAC model and the original SA model. In particular, the two system dynamics are compared to each other. The purpose of this exercise is to find out the value of adding one more state and under which circumstances one may consider the reduced SA model without losing too much information.

Chapter 6 is then dedicated to some strategic analyses with respect to the control intervention treatment. Here, the goal is to be able to say something like, "Treatment early in the epidemic is more valuable than later in the epidemic". Furthermore, the effects of a supply shock are examined.

Finally, Chapter 7 summarizes the most important results and provides a proposal for further studies in this direction.

Please note that all numerical calculations were done using Wolfram's Mathematica 6.0 (see [11]).

Chapter 2

The Model

2.1 General Formulation

The SAC model considered in this thesis has three states with t denoting the time argument. $S(t)$ tracks the number of people who are not consuming illicit drugs, but who are susceptible to start using. $A(t)$ tracks the number of drug users over time and $C(t)$ represents the current "throughput capacity" of the supply network. Within the use state, frequency of consumption or degree of addiction are not taken into account explicitly.

People enter the S-pool via a constant inflow rate k (which can be understood as reaching an age when susceptibility to drug use starts) and "mature out" of the pool with a constant outflow rate δ. The constant per capita rate μ can be interpreted as the exit from active use. Reasons for such an exit may be the successful participation in a treatment program, death or other reasons. This model approach makes initiation price dependent (cf. [Tragler et al., 2001]), where price itself depends on A and C and $a < 0$ is the elasticity of initiation with respect to the price.

The instantaneous growth rate of the supply network $g(p)$ as well as one specific functional form for the initiation function $f(A)$ will be discussed in detail below.

To simplify matters, the time argument t is mostly omitted. Hence, the

dynamic system we consider is

$$\begin{aligned} \dot{S} &= k - \delta\ S - f(A)\ S\ p(A,C)^a \\ \dot{A} &= f(A)\ S\ p(A,C)^a - \mu\ A \\ \dot{C} &= g(p)\ C. \end{aligned} \tag{2.1}$$

2.1.1 Price Function

In contrast to the SA dynamics our base model is now dependent on the price p which is derived by equating the short-run demand and supply curves. Thus, we obtain

$$Q_D(p) = d\ A\ p^{\eta_D} = s\ C\ p^{\eta_S} = Q_S(p)$$

where $Q_D(p)$ and $Q_S(p)$ are the quantities demanded and supplied. Furthermore, $\eta_D < 0$ is the short-run price elasticity of demand and $\eta_S > 0$ is that of supply. Solving for p implies

$$p(A,C) = \tilde{d}\ \left(\frac{A}{C}\right)^{\frac{1}{\eta_S - \eta_D}}$$

with $\tilde{d} = 1$ so that prices are normalized to 1 when supply matches demand.

2.1.2 Growth Rate

For the growth rate of the supply network we choose the following assumptions. If C is smaller than A, then $p > 1$ and $g(p) > 0$ so $\dot{C} > 0$. Otherwise, if C is greater than A, then $p < 1$ and $g(p) < 0$ so $\dot{C} < 0$. Moreover, $g(p) = 0$ should result from $p = 1$. Consequently, one specific functional form of $g(p)$ is $g(p) = c\ \ln(p)$. Given this, the growth rate reduces down to $g(p) = c'\ (\ln(A) - \ln(C))$ with $c' = \dfrac{c}{\eta_S - \eta_D}$.

2.1.3 Initiation Function

Finally, we pay our attention to the initiation function $f(A)$. The interaction between the pool of susceptibles and active users leads to new "infections"

over the course of a drug epidemic. This flow from the S-state to the A-state is reflected in the so-called initiation function. As in other models (cf. [Tragler, 1998]), we use the approach

$$f(A) = \alpha \; A^{\beta} \tag{2.2}$$

with $\alpha > 0$ and $\beta > 0$. Please note that in contrast to others (e.g., [Behrens et al., 1999], [Behrens et al., 2000]) only "imitators" are taken into account here.

2.2 Parameter Values

Table 2.1 summarizes the base parameter values as described in [Caulkins et al., 2009a] for the U.S. cocaine and Australian IDU epidemics. One crucial parameter is the exponent β in the initiation function. For the U.S. cocaine epidemic, $\beta > 1$, and so initiation is a convex function of A, which allows for multiple stable equilibria separated by a tipping point. However, for Australian IDU, $\beta < 1$, and on this account there we will find only one steady state with a positive amount of drug use which is stable (cf. [Caulkins et al., 2009a], [Caulkins et al., 2009b]).

Description	Symbol	U.S. Cocaine	Australian IDU
inflow into S-state	k	1.3417	0.0526
exit rate from S-state	δ	0.0605	0.0952
coefficient in initiation function	α	0.0090	0.5112
exponent in initiation function	β	1.5604	0.8622
price elasticity of supply	η_S	0.5	0.5
price elasticity of demand	η_D	-0.5	-0.5
elasticity of initiation	a	-0.25	-0.25
exit rate from active use	μ	0.1661	0.1136
coefficient in growth rate	c	0.15	0.15
annual discount rate	r	0.04	0.04

Table 2.1: Base parameter values.

Chapter 3

Analysis of the Uncontrolled Model

First, we look at the generalized system, so before the respective parameter values are used for the U.S. or Australia. In order to determine the steady state values and their stability behavior we consider (2.1) with the specification (2.2). Thus, the formulation we get is

$$\begin{aligned} \dot{S} &= k - \delta\ S - \alpha\ A^{\beta}\ S\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S - \eta_D}} \\ \dot{A} &= \alpha\ A^{\beta}\ S\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S - \eta_D}} - \mu\ A \\ \dot{C} &= c'\ (\ln(A) - \ln(C))\ C. \end{aligned} \tag{3.1}$$

The steady states $(\hat{S}, \hat{A}, \hat{C})$ of (3.1) are given by the solutions to ($\dot{S} = 0$, $\dot{A} = 0$, $\dot{C} = 0$). Note that we need to assume that $C > 0$, because the ln(.) is not defined for $C = 0$. Hence, in this formulation $\dot{C} = 0 \Leftrightarrow \hat{C} = \hat{A}$, implying that $p = 1$, so the price effect drops out of the model. Consequently, adding the first two state equations leads us to

$$\dot{S} + \dot{A} = k - \delta\ S - \mu\ A.$$

This implies that the steady state values satisfy the linear relation

$$\hat{S} = \frac{k - \mu\ \hat{A}}{\delta}.$$

Inserting this expression into the equation for S and setting it to zero results in

$$\hat{A}\ (-\alpha\ \mu\ \hat{A}^{\beta} + \alpha\ k\ \hat{A}^{\beta-1} - \delta\ \mu) = 0.$$

Consequently, there are two possibilities. However, the first one, $\hat{A} = 0$, is not feasible because of the ln(.) function within the equation for C. On this account we have to solve

$$\alpha_1\ \hat{A}^{\beta} + \alpha_2\ \hat{A}^{\beta-1} + \alpha_3 = 0 \tag{3.2}$$

with $\alpha_1 = -\alpha\ \mu$, $\alpha_2 = \alpha\ k$ and $\alpha_3 = -\delta\ \mu$.

3.1 U.S. Cocaine Use

3.1.1 Steady States, Stability Behavior, and Phase Portraits

By solving the equation (3.2) for the U.S. base case parameter set one obtains two different solutions, $\hat{A}_1 = 0.8867$ and $\hat{A}_2 = 5.4888$. Figure 3.1 shows these roots, where the values of the steady state solutions are denoted in millions.

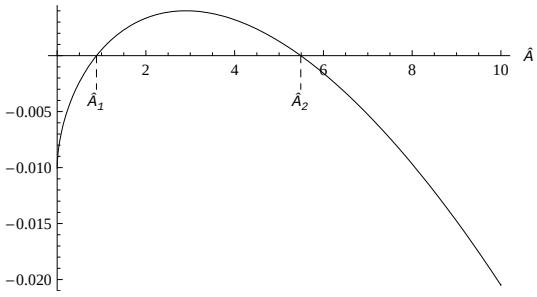

Figure 3.1: Solutions for equation (3.2) when using the U.S. parameterization.

Hence, we get two steady states located at

$$\begin{aligned} \hat{E}_1 &= (\hat{S}_1, \hat{A}_1, \hat{C}_1) &&= (19.7426, 0.8867, 0.8867) \\ \hat{E}_2 &= (\hat{S}_2, \hat{A}_2, \hat{C}_2) &&= (7.1076, 5.4888, 5.4888). \end{aligned}$$

The analysis of the stability behavior uses the Jacobian matrix

$$\mathbf{J} = \begin{pmatrix} \dot{S}_S & \dot{S}_A & \dot{S}_C \\ \dot{A}_S & \dot{A}_A & \dot{A}_C \\ \dot{C}_S & \dot{C}_A & \dot{C}_C \end{pmatrix}$$

with the generalized entries

$$\begin{aligned}
\dot{S}_S &= -\delta - \alpha\ A^{\beta}\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}} \\
\dot{S}_A &= -\alpha\ S\ \beta\ A^{\beta-1}\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}} - \frac{\alpha\ S\ A^{\beta}\ a\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}-1}}{C\ (\eta_S-\eta_D)} \\
\dot{S}_C &= \frac{\alpha\ S\ A^{\beta+1}\ a\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}-1}}{C^2\ (\eta_S-\eta_D)} \\
\dot{A}_S &= \alpha\ A^{\beta}\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}} \\
\dot{A}_A &= -\mu + \alpha\ S\ \beta\ A^{\beta-1}\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}} + \frac{\alpha\ S\ A^{\beta}\ a\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}-1}}{C\ (\eta_S-\eta_D)} \\
\dot{A}_C &= -\frac{\alpha\ S\ A^{\beta+1}\ a\ \left(\frac{A}{C}\right)^{\frac{a}{\eta_S-\eta_D}-1}}{C^2\ (\eta_S-\eta_D)} \\
\dot{C}_S &= 0 \\
\dot{C}_A &= \frac{c'\ C}{A} \\
\dot{C}_C &= c'\ (\ln(A) - \ln(C)) - c'.
\end{aligned}$$

Using the parameter values and evaluating at the first fixed point $\hat{E}_1$ leads to

$$\mathbf{J} = \begin{pmatrix} -0.0679597 & -0.217657 & -0.041525 \\ 0.00745974 & 0.0515574 & 0.041525 \\ 0 & 0.15 & -0.15 \end{pmatrix}$$

with the Eigenvalues

$$\begin{aligned}
\lambda_1 &= -0.177302 \\
\lambda_2 &= 0.0666524 \\
\lambda_3 &= -0.0557528.
\end{aligned}$$

All Eigenvalues are real and we find opposite signs. Therefore, this equilibrium is a saddle point.

For the second steady state $\hat{E}_2$ we get

$$\mathbf{J} = \begin{pmatrix} -0.188769 & -0.217657 & -0.041525 \\ 0.128269 & 0.0515574 & 0.041525 \\ 0 & 0.15 & -0.15 \end{pmatrix}.$$

The Eigenvalues are now given by

$$\begin{aligned} \lambda_1 &= -0.178759 \\ \lambda_{2,3} &= -0.0542265 \pm 0.101054\ i \end{aligned}$$

implying that the second equilibrium is a stable focus.

Henceforward, we concentrate on analyzing the qualitative stability behavior of the steady states graphically. For this reason, the phase portraits of the system were created in a neighborhood of the equilibria. Figure 3.2 shows some trajectories around $\hat{E}_1$ within the (A, S, C)-plane, while the phase portrait around the stable focus can be found in Figure 3.3.

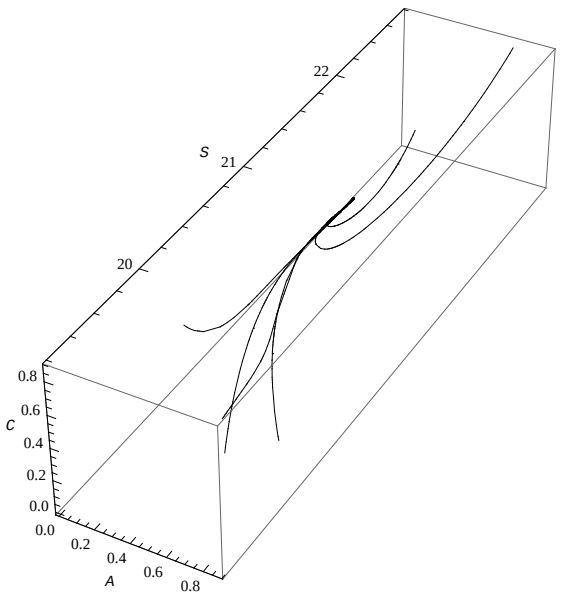

Figure 3.2: Phase portrait around $\hat{E}_1$ for the U.S. parameter values.

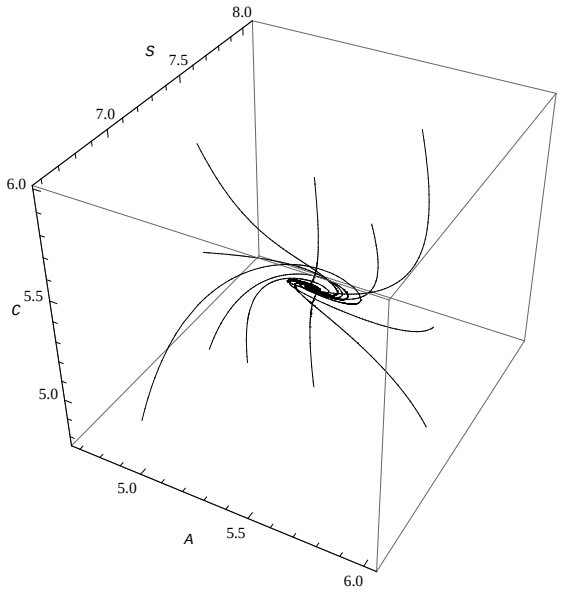

Figure 3.3: Phase portrait around $\hat{E}_2$ for the U.S. parameter values.

For a better representation of the stability behavior we next restrict our examinations to the two-dimensional (A, S)-plane. Since $\hat{C} = \hat{A}$ is valid in the stationary points, we reduce the system (3.1) to

$$\begin{aligned} \dot{S} &= k - \delta\ S - \alpha\ A^{\beta}\ S \\ \dot{A} &= \alpha\ A^{\beta}\ S - \mu\ A. \end{aligned}$$

Note that this corresponds exactly to the SA dynamics as described in [Wallner, 2008]. Adding $\dot{S} = 0$ and $\dot{A} = 0$ up, we obtain $k - \delta\ \hat{S} - \mu\ \hat{A} = 0$ and solving for $\hat{S}$ yields

$$\hat{S} = \frac{k - \mu\ \hat{A}}{\delta}. \tag{3.3}$$

This is a downward sloping line between $(A, S) = (0, \frac{k}{\delta}) = (0, 22.1769)$ and $(A, S) = (\frac{k}{\mu}, 0) = (8.0777, 0)$.

The isoclines $\dot{S} = 0$ and $\dot{A} = 0$, given by

$$S = \frac{k}{\delta + \alpha\ A^{\beta}} \qquad \text{and}$$

$$S = \frac{\mu\ A}{\alpha\ A^{\beta}},$$

are shown in Figure 3.4. The black straight line represents the linear relation (3.3) between $\hat{S}$ and $\hat{A}$.

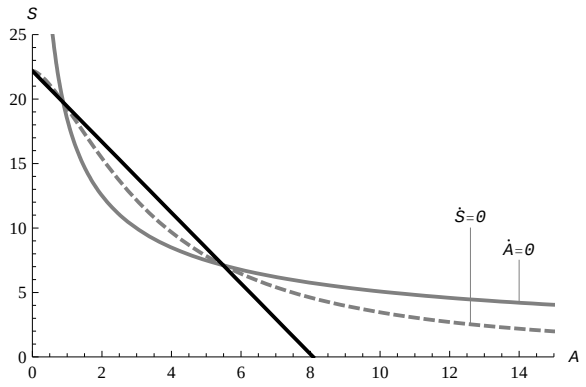

Figure 3.4: Isoclines and the linear relation between the steady state values for the U.S. parameter set.

In order to illustrate the stability behavior within the (A, S)-plane, again, some trajectories were determined. In Figure 3.5, the equilibria $\hat{E}_1$ and $\hat{E}_2$, which are located at the intersection of the isoclines, are depicted as black dots. Below the isocline $\dot{S} = 0$, the number of susceptibles increases. Above this curve, S decreases. For $\dot{A} = 0$ it is the other way around. Below, the number of users drops down while it is rising above. This is indicated by the red arrows.

As stated at the beginning of this chapter, the no-use state, $A = 0$, cannot be computed as steady state value because of the ln(.) function within the state equation for C. However, Figure 3.5 shows that some trajectories converge towards the point $(A, S) = (0, \frac{k}{\delta}) = (0, 22.1769)$. Indeed, this is not a surprise, since we actually deal with the SA system, in which $\hat{A} = 0$ is an equilibrium (see [Wallner, 2008], [Caulkins et al., 2009a], [Caulkins et al., 2009b]). In our extended model, A and C cannot reach the value 0, but if we choose the two close to zero and assume that $\hat{C} = \hat{A}$, we find that the system (3.1) converges towards zero for $(S, A, C) = (\frac{k}{\delta}, A \to 0, C = A \to 0)$ (cf. the analysis carried out in [Feichtinger et al., 2002]). Next, we will analyze the stability of this "steady state". For that purpose, we look at the

two-dimensional system with its partial derivatives

$$\begin{aligned}
\dot{S}_S &= -\delta - \alpha\ A^{\beta} \\
\dot{S}_A &= -\alpha\ \beta\ A^{\beta-1}\ S \\
\dot{A}_S &= \alpha\ A^{\beta} \\
\dot{A}_A &= \alpha\ \beta\ A^{\beta-1}\ S - \mu.
\end{aligned}$$

If $A \to 0$, then the Jacobian matrix becomes

$$\mathbf{J} = \begin{pmatrix} -\delta & 0 \\ 0 & -\mu \end{pmatrix}.$$

Hence, one concludes directly that it has the stability properties of a stable node. Overall, this means that for the U.S. cocaine epidemic we have two stable equilibria which are separated by a saddle point.

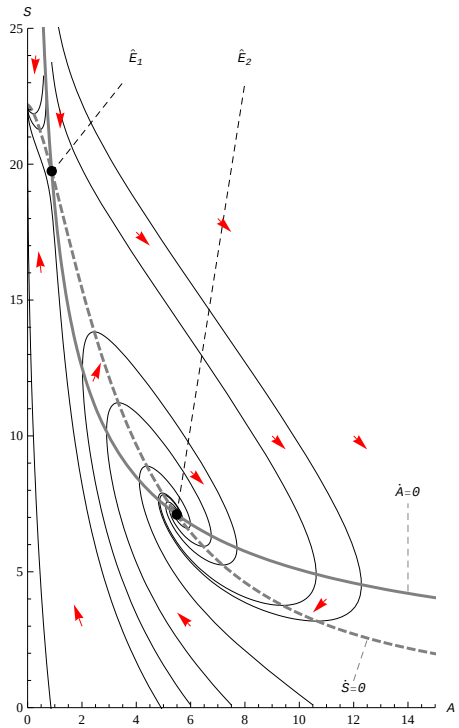

Figure 3.5: Phase portrait for the U.S. base case parameter set within the (A, S)-plane.

3.1.2 Time Paths

Finally, we extend our picture by looking at a few time paths. For this purpose, three trajectories with different initial values were designed. Trajectory T_1 arises from $(S(0), A(0), C(0)) = (1, 2, 2)$. The second trajectory T_2 satisfies the conditions $(S(0), A(0), C(0)) = (5, 3, 3)$ and the third trajectory T_3 is based on high initial values, namely $(S(0), A(0), C(0)) = (22, 8, 8)$. Here, $A(0) = C(0)$ was chosen intentionally, so that one can see that the two states develop differently over a short period before they then converge again when approaching the corresponding equilibrium.

Figure 3.6 shows the evolution of $S(t)$, $A(t)$, $C(t)$ and of the price within the first 150 years along T_1, where the number of users and susceptibles is still very low at the beginning of the epidemic. The pool of susceptibles increases while the number of users is dropping. The number of drug users monotonously converges to zero, confirming again that $\hat{A} = 0$ is a steady state.

Figure 3.7 illustrates the time paths along T_2. Please note that here the time ranges from $t = 0$ to $t = 200$. The development of $S(t)$ is oscillating as follows: Strongly increasing at the beginning, then strongly falling, increasing again and flattening out. The progression of A and C is in the opposite direction. In this case, the values converge towards the high-use equilibrium.

In Figure 3.8 the time span goes up to $t = 130$. $S(t)$ declines at the early stages, then slightly grows and then smoothly converges to its steady state value. A and C, however, increase first, then drop quickly close to their steady state values.

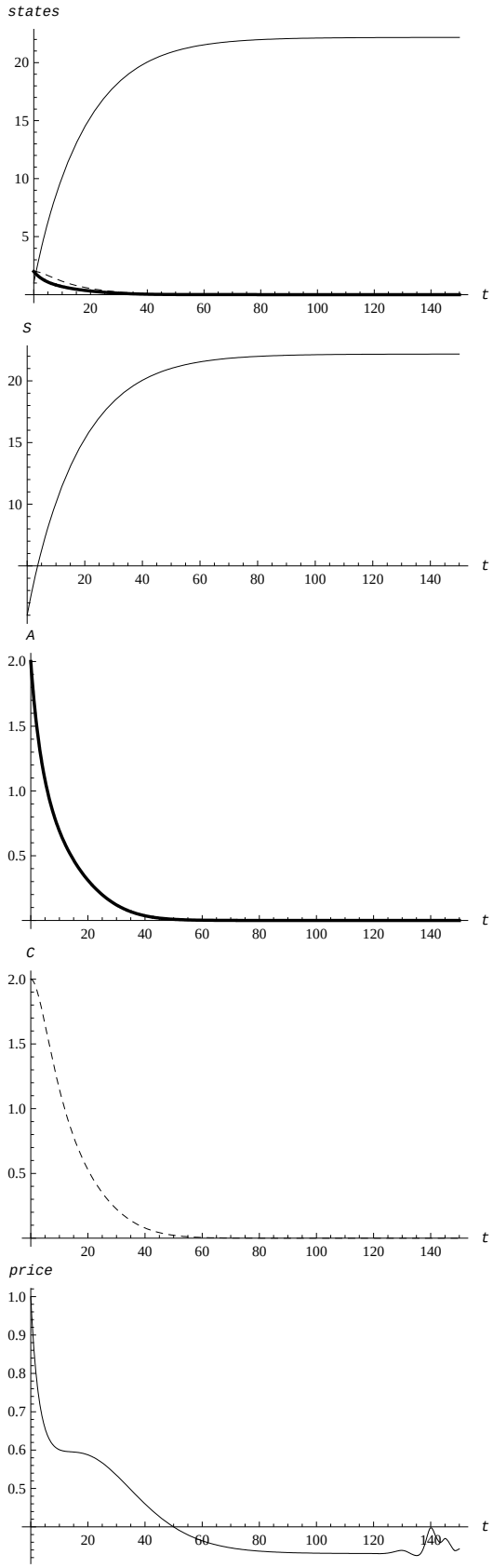

Figure 3.6: Time paths relating to T_1 for the U.S. parameterization.

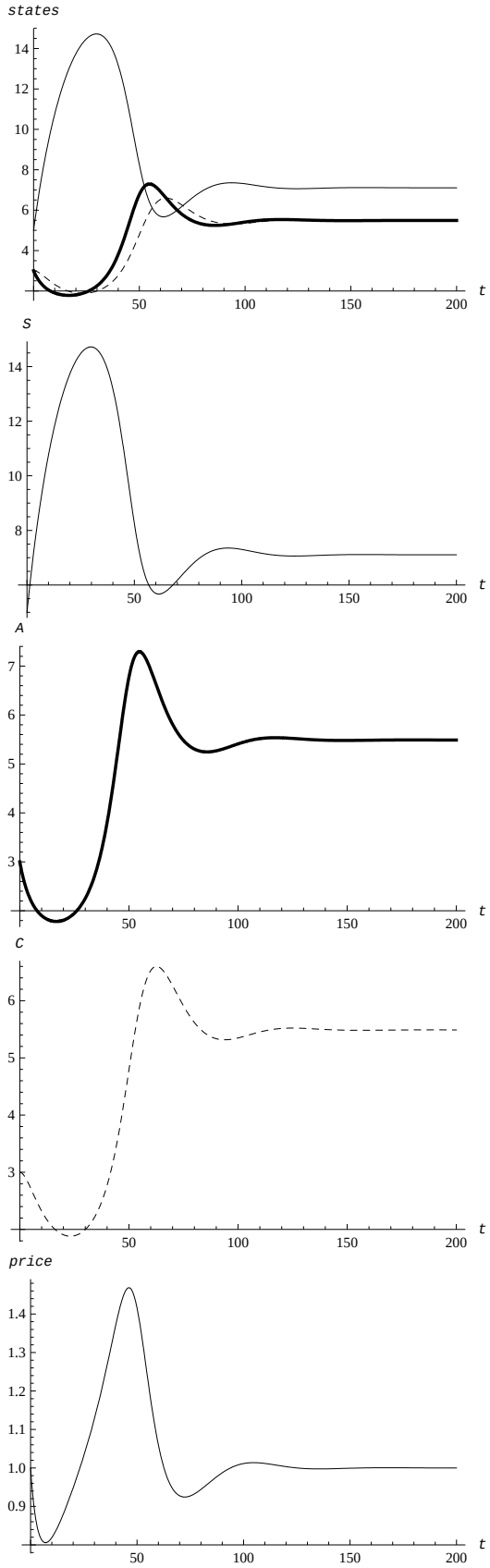

Figure 3.7: Time paths relating to T_2 for the U.S. parameterization.

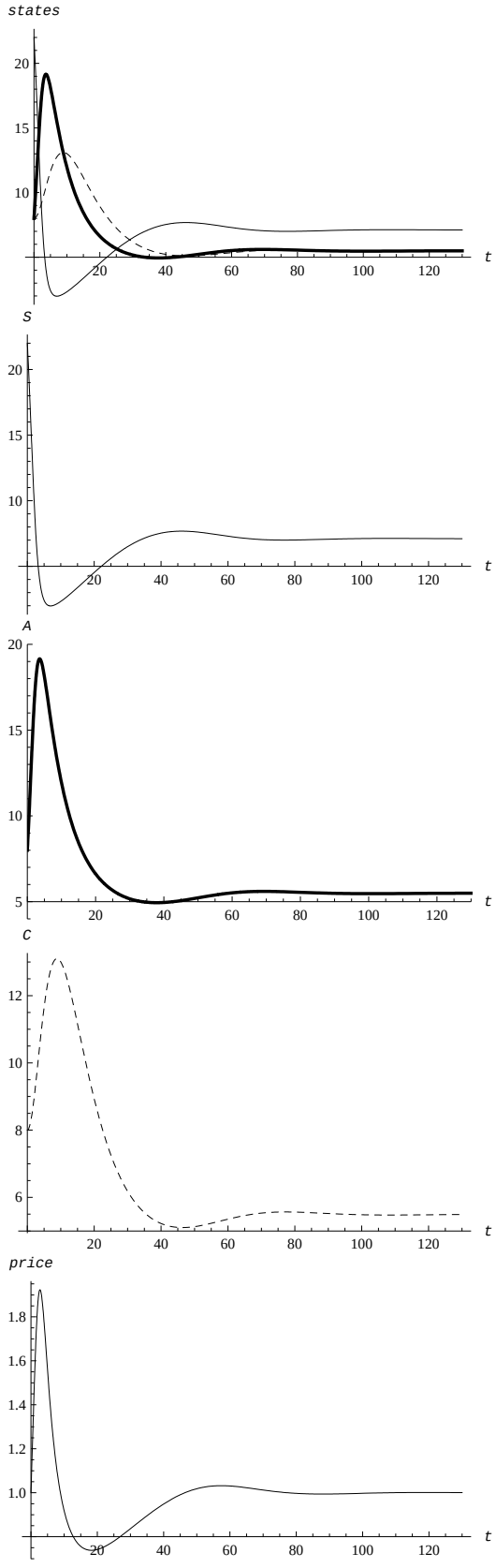

Figure 3.8: Time paths relating to T_3 for the U.S. parameterization.

3.2 Australian Injection Drug Use

3.2.1 Steady States, Stability Behavior, and Phase Portraits

Again, we have to solve equation (3.2). In the context of the base case parameter values for the Australian IDU epidemic the solution is unique, to be more precise $\hat{A} = 0.304916$ (see Figure 3.9).

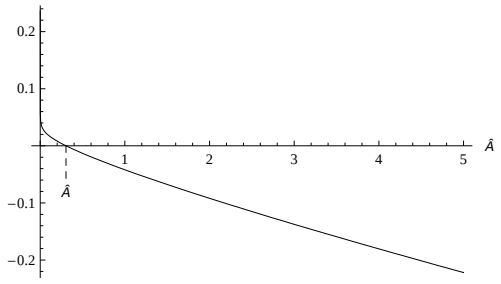

Figure 3.9: Solution for equation (3.2) when using the Australian parameterization.

Thus, we obtain the steady state

$$\hat{E} \;=\; (\hat{S}, \hat{A}, \hat{C}) \;=\; (0.188672, 0.304916, 0.304916).$$

Please note that $\hat{S} < \hat{A}$ here. Usually, we should expect to find more susceptibles than active users in the steady state, particularly since for the Australian parameterization the A-state refers to injection drug use and not just any use. This suggests that maybe one should reconsider the Australian parameter values.

The Eigenvalues of the Jacobian matrix

$$\mathbf{J} = \begin{pmatrix} -0.278791 & -0.0695459 & -0.0284 \\ 0.183591 & -0.0440541 & 0.0284 \\ 0 & 0.15 & -0.15 \end{pmatrix}$$

are given by

$$\begin{aligned} \lambda_1 &= -0.248776 \\ \lambda_{2,3} &= -0.112034 \pm 0.0303643\ i. \end{aligned}$$

Therefore, the equilibrium is a stable focus. Figure 3.10 depicts some trajectories within the (A, S, C)-plane.

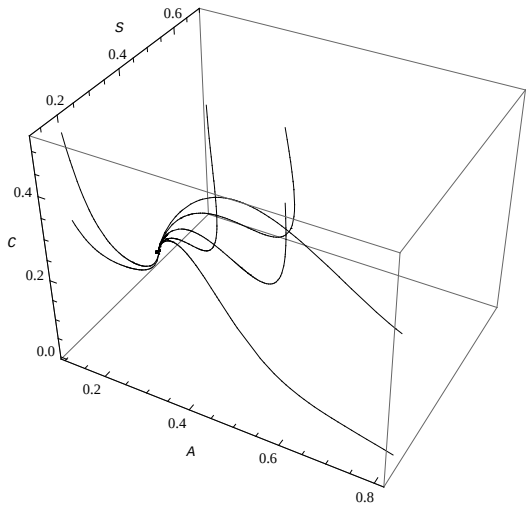

Figure 3.10: Phase portrait around $\hat{E}$ for the Australian parameter set.

Further analyses are carried out again only for the two-dimensional system. Figure 3.11 represents the isoclines $\dot{S} = 0$ and $\dot{A} = 0$ as well as the downward sloping line between $(A, S) = (0, \frac{k}{\delta}) = (0, 0.552521)$ and $(A, S) = (\frac{k}{\mu}, 0) = (0.463028, 0)$. One sees that there is only one intersection and so the steady state is unique.

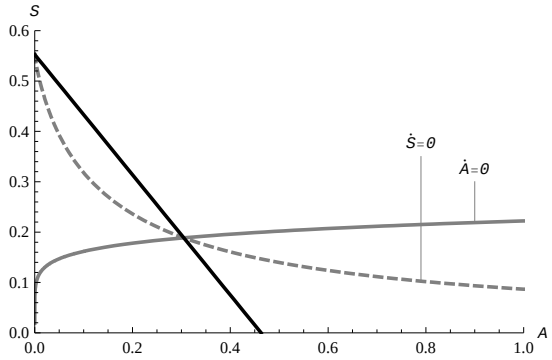

Figure 3.11: Isoclines and the linear relation (3.3) between $\hat{S}$ and $\hat{A}$ for the Australian base case parameter set.

Finally, Figure 3.12 shows the phase portrait within the (A, S)-plane. The stable focus is depicted as a black dot. The little red arrows trace the vector field and in order to get a better visualization of the system dynamics some trajectories are depicted as black curves. Here, all trajectories converge to $\hat{E}$. The no-use state is unstable and cannot be reached for initial values with $A(0) > 0$. Again, the system is not defined at $A = 0$ because of the ln(.) function within the state equation for C.

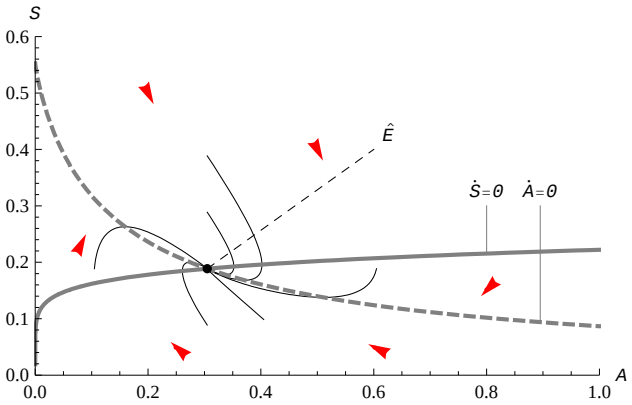

Figure 3.12: Phase portrait for the Australian parameterization within the (A, S)-plane.

3.2.2 Time Paths

The concluding section of this chapter presents some time paths for the Australian base case parameter set. The chronological sequence of $S(t)$, $A(t)$, $C(t)$ and of the price is represented with the help of two trajectories. Trajectory T_1 originates from $(S(0), A(0), C(0)) = (0.01, 0.01, 0.01)$. The second trajectory T_2 has high initial values which are given by $(S(0), A(0), C(0)) = (0.5, 0.7, 0.7)$. Also here, $A(0) = C(0)$ was chosen.

Figure 3.13 shows the paths for trajectory T_1 over the first 100 years. The number of susceptibles is strongly increasing first but then, after reaching a peak, it is dropping. The increase in use is moderate within the first decade, but then use begins to grow more strongly until the steady state value is reached.

Figure 3.14 demonstrates the evolution along T_2 in which only the first 80 years are looked at. The pool of susceptibles is declining first and then slightly growing towards its steady state value. The number of users briefly goes up and then falls down rather rapidly.

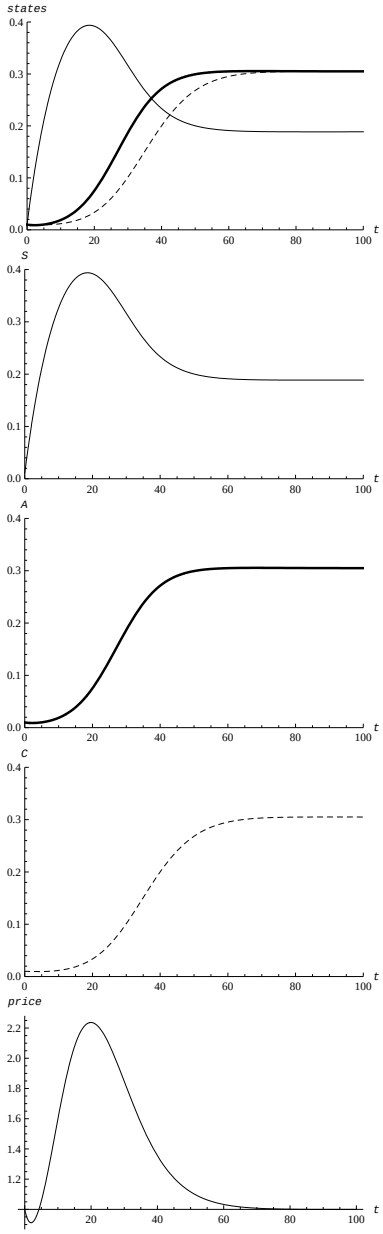

Figure 3.13: Time paths relating to T_1 for the Australian parameterization.

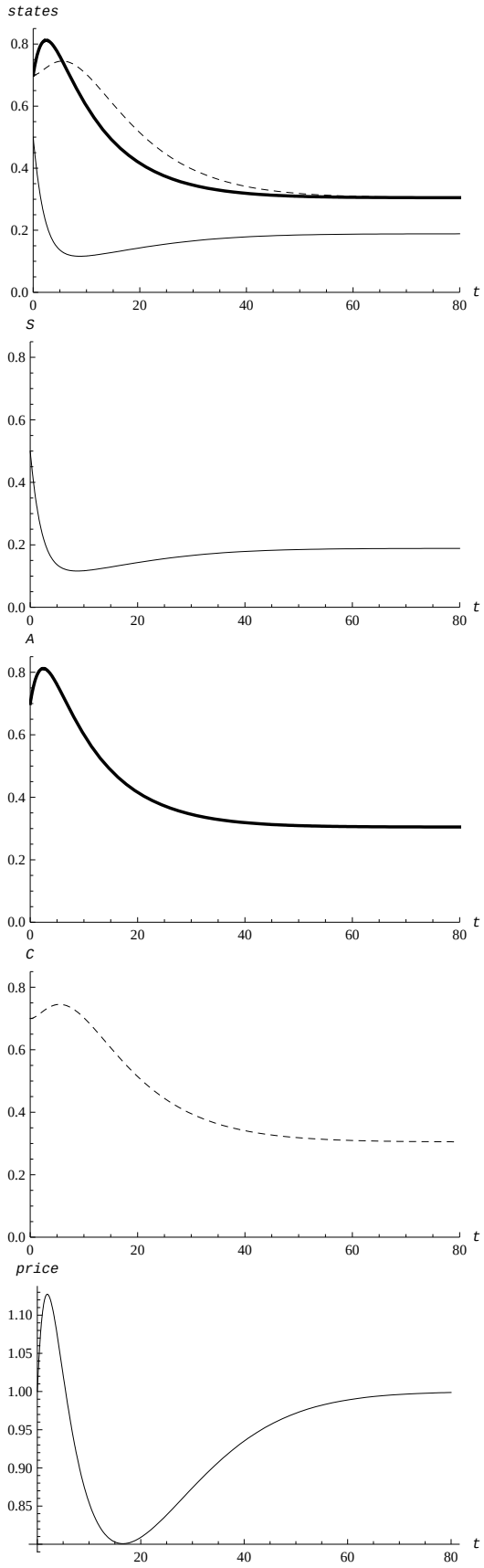

Figure 3.14: Time paths relating to T_2 for the Australian parameterization.

Chapter 4

Sensitivity and Bifurcation Analysis

4.1 Sensitivity Analysis

Our base model contains a lot of parameters. Unfortunately, such values are not exact in most cases. Sensitivity analysis deals with the question how results vary when parameters are changed. First of all, we consider a 1% variation of the parameter values. This means that one of the base case parameters is increased by 1% while the other values are kept the same. The steady state values are then recalculated and the respective effect is expressed as percentage.

4.1.1 United States

Table 4.1 summarizes the effects on the first steady state $\hat{E}_1 = (\hat{S}_1, \hat{A}_1, \hat{C}_1) = (19.7426, 0.8867, 0.8867)$ and Table 4.2 gives the results of the sensitivity analysis for the second steady state $\hat{E}_2 = (\hat{S}_2, \hat{A}_2, \hat{C}_2) = (7.1076, 5.4888, 5.4888)$.

Some parameters (more precisely η_S, η_D, a, and c) do not have influence on the values since in every stationary point $\hat{C} = \hat{A}$ must be valid and therefore the price effect drops out of the model. These parameters were left out in the tables. Please note that a positive value means an increase relative to the base case. Contrariwise, a negative value stands for a decrease.

Parameter	$\hat{S}_1$	$\hat{A}_1$	$\hat{C}_1$
k	1.43	-2.51	-2.51
δ	-1.27	2.31	2.31
α	0.277	-2.24	-2.24
β	-0.0513	0.416	0.416
μ	-0.448	2.61	2.61

Table 4.1: Effects of a 1% increase of the parameter values on the steady state $\hat{E}_1$ for the U.S. parameterization.

This table shows that a 1% increase in the parameter values brings about relatively great consequences for all parameters apart from β. For β only the modification is less than 1% and that for all values. The effects on the steady state values are mostly as one would expect in respect of the dynamics $\dot{S}$ and $\dot{A}$.

Parameter	$\hat{S}_2$	$\hat{A}_2$	$\hat{C}_2$
k	-1.1	1.99	1.99
δ	0.363	-0.645	-0.645
α	-1.34	0.631	0.631
β	-3.55	1.67	1.67
μ	2.15	-1.99	-1.99

Table 4.2: Effects of a 1% increase of the parameter values on the steady state $\hat{E}_2$ for the U.S. parameterization.

Here, the strongest impacts on the steady state values are encountered when the parameters k, β, or μ are modified.

We have already mentioned many a time that $\hat{A} = 0$ is a steady state that does not exist mathematically because $\ln(0)$ is undefined. Nevertheless, some trajectories converge towards this state and therefore also here a sensitivity analysis was made which is shown in Table 4.3. Please note that only $\hat{S} = \frac{k}{\delta}$ is influenced in this case.

Parameter	$\hat{S}$
k	1.00
δ	-0.9901

Table 4.3: Effects of a 1% increase of the parameter values on the no-use state for the U.S. parameterization.

In conclusion, we still want to give a sensitivity analysis concerning the drug initiation. For it, we first deal with the base parameter values and calculate the initiation force at the respective steady states. Afterwards, α and β are adjusted simultaneously to keep the force of initiation the same at the equilibria. This means that α is increased by 1% and then the appropriate β is determined. Now, the results. For $\hat{A}_1 = 0.8867$ this examination shows that β must be increased by 5.301%. A reduction of β by 0.375% is necessary at the second steady state value $\hat{A}_2 = 5.4888$ and, of course, this analysis is not relevant for $\hat{A} = 0$.

4.1.2 Australia

In this case, the steady state is unique and given by $\hat{E} = (\hat{S}, \hat{A}, \hat{C}) = (0.188672, 0.304916, 0.304916)$. The results of the sensitivity analysis can be found in the following table.

Parameter	$\hat{S}$	$\hat{A}$	$\hat{C}$
k	0.194	1.42	1.42
δ	-0.0668	-0.484	-0.484
α	-0.925	0.48	0.48
β	0.964	-0.5	-0.5
μ	0.804	-1.4	-1.4

Table 4.4: Effects of a 1% increase of the parameter values on the steady state $\hat{E}$ for the Australian parameterization.

The strongest effects on the values arise by the increase of k and μ. When k increases by one percent, $\hat{A}$ and $\hat{C}$ go up by more than 1.4%. A 1% increase of μ reduces the values by 1.4%.

At this point, it still is mentioned that β must be increased by 0.972% after a 1% increase of α, so that the initiation force at $\hat{E}$ is the same as with the Australian base parameterization.

4.2 Bifurcation Analysis

A bifurcation of a dynamical system is a qualitative change in its dynamics produced by varying parameters. Examples are the creation or destruction of steady states or the exchange of the stability behavior of equilibria. Bifurcation theory provides a procedure for investigating such disruptions. A parameter value where a bifurcation occurs is called a critical value of the system and we will denote it by $parameter_c$.

In this area, a saddle-node bifurcation or tangent bifurcation is a local bifurcation in which fixed points of a dynamical system are created or destroyed. Another denomination is blue sky bifurcation in reference to the sudden creation of two fixed points. We will find this type of bifurcation in our current model. More details on this topic can be found in [Grass et al., 2008].

4.2.1 United States

4.2.1.1 Flow into Pool of Susceptibles (k)

If the inflow rate into the S-state, k, is changed to a greater extent, then a blue sky bifurcation occurs. Figure 4.1 shows the bifurcation plots for $\hat{S}$ and $\hat{A}$. Please note that the depiction for $\hat{C}$ is the same as that for $\hat{A}$ and can hence be omitted here.

The saddle-node bifurcation turns up at the point $BS = (\hat{S}_c, \hat{A}_c, \hat{C}_c) = (11.462, 2.33961, 2.33961)$ and the respective critical parameter value is given by $k_c = 1.08206$. At the base case parameter value $k_{bc} = 1.3417$ there is a

vertical dashed line and the two black dots indicate our previous steady state values.

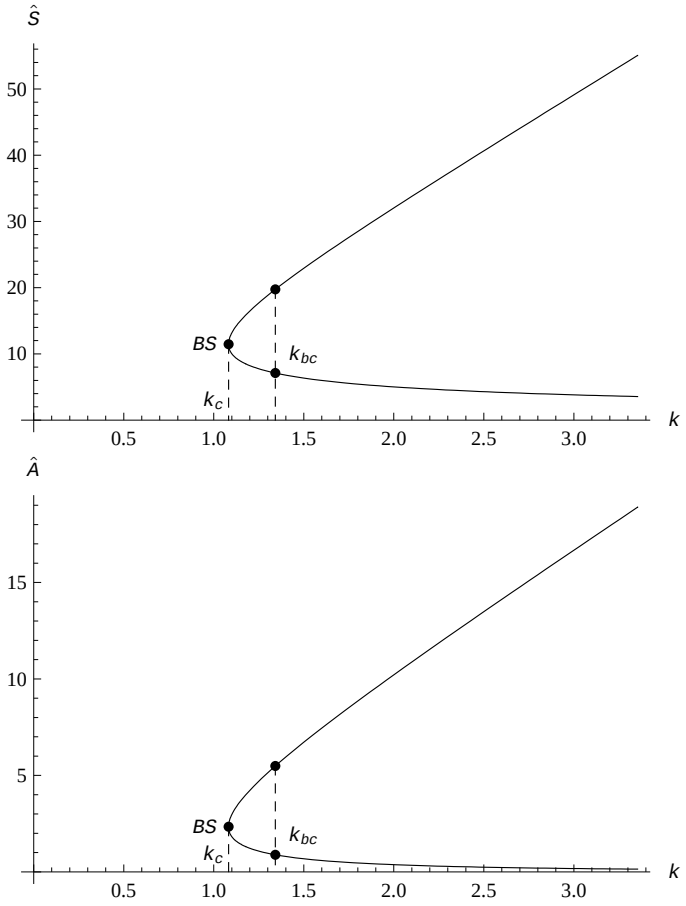

Figure 4.1: Bifurcation diagrams with respect to the parameter k for the U.S. cocaine epidemic.

4.2.1.2 Exit Rate from Pool of Susceptibles (δ)

The results from the bifurcation analysis for the cocaine epidemic in the United States with respect to the parameter δ are shown in Figure 4.2.

Again, a saddle-node bifurcation occurs. The critical parameter value is located at $\delta_c = 0.0846261$ with the corresponding steady state $BS = (\hat{S}_c, \hat{A}_c, \hat{C}_c) = (10.1605, 2.901, 2.901)$.

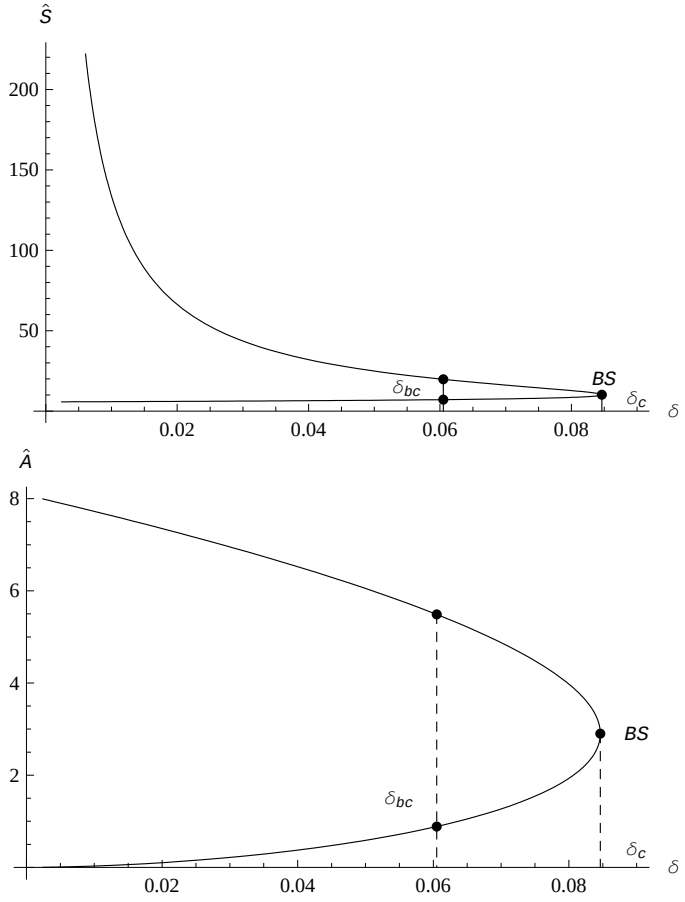

Figure 4.2: Bifurcation diagrams with respect to the parameter δ for the U.S. cocaine epidemic.

4.2.1.3 Initiation Function Coefficient (α)

Figure 4.3 shows the bifurcation diagrams when the parameter α of the U.S. parameter set varies. The dashed line at $\alpha_{bc} = 0.009$ represents the base case coefficient in the initiation function and the black dots denote the base case steady state values. At the point $BS = (\hat{S}_c, \hat{A}_c, \hat{C}_c) = (14.2123, 2.901, 2.901)$ the saddle point $\hat{E}_1$ and the stable focus $\hat{E}_2$ collide. The critical parameter value is $\alpha_c = 0.00643419$.

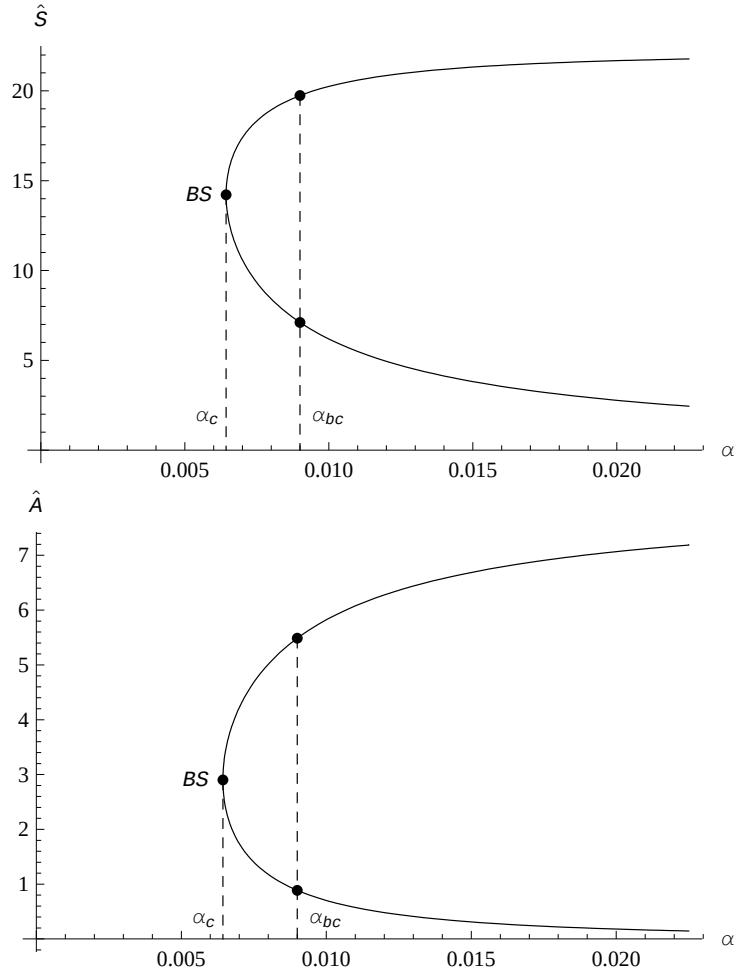

Figure 4.3: Bifurcation diagrams with respect to the parameter α for the U.S. cocaine epidemic.

4.2.1.4 Initiation Function Exponent (β)

We can find the bifurcation plots with regard to the important parameter β in Figure 4.4. If the critical parameter value $\beta_c = 1$ is reached, then there is only one fixed point instead of two (see also Section 2.2). As opposed to the other parameters, this equilibrium also exists if one reduces the exponent in the initiation function further. The bifurcation point was detected at $(\hat{S}_c, \hat{A}_c, \hat{C}_c) = (18.4556, 1.35544, 1.35544)$.

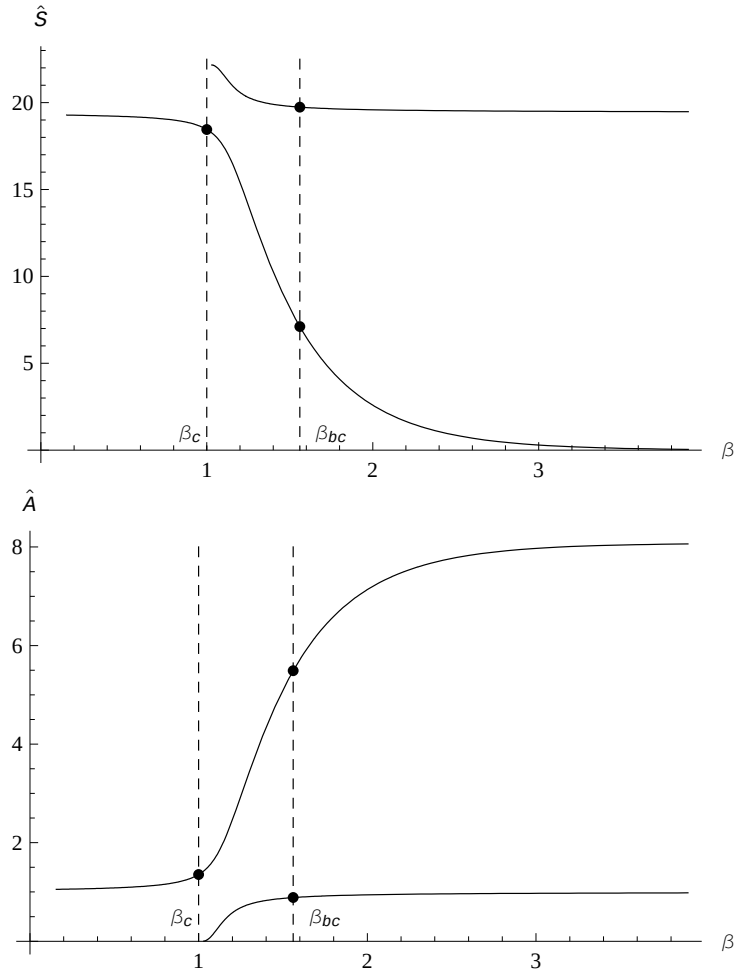

Figure 4.4: Bifurcation diagrams with respect to the parameter β for the U.S. cocaine epidemic.

4.2.1.5 Exit Rate from Active Use (μ)

Finally, we still want to look at the bifurcation plots associated with the exit rate μ. Here, a blue sky bifurcation point appears at $BS = (\hat{S}_c, \hat{A}_c, \hat{C}_c) = (14.2323, 2.33961, 2.33961)$. The corresponding critical parameter value is given by $\mu_c = 0.205956$ and is on the right-hand side of the base case parameter value $\mu_{bc} = 0.1661$.

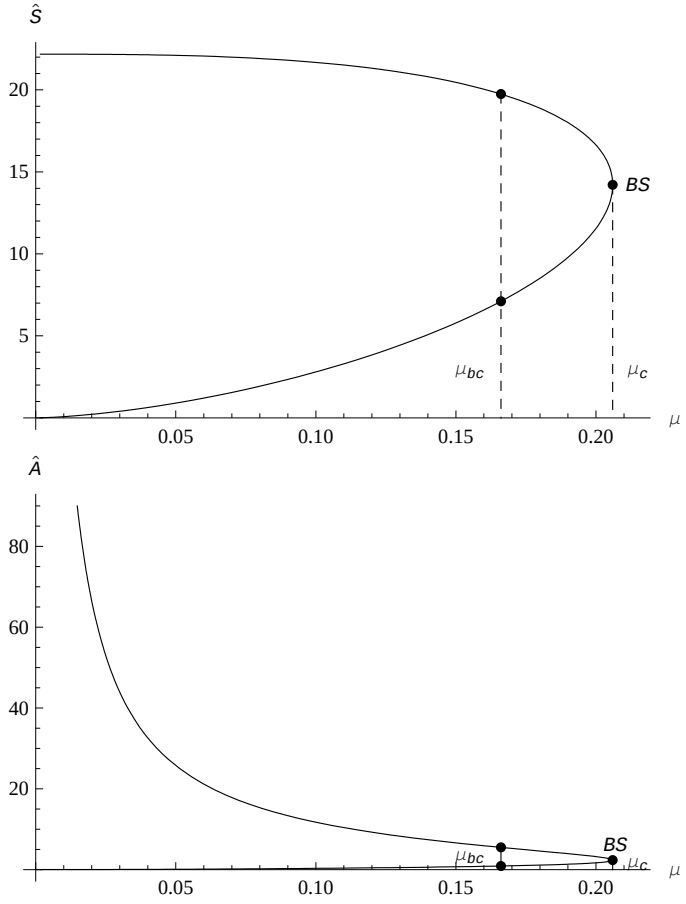

Figure 4.5: Bifurcation diagrams with respect to the parameter μ for the U.S. cocaine epidemic.

4.2.2 Australia

4.2.2.1 Bifurcation Analysis with regard to the Parameters k, δ, α, and μ

For the Australian IDU epidemic only one parameter leads to a disruption in the dynamical behavior under parameter variances. This is the exponent in the initiation function, β. For the other parameters no bifurcations emerge. This section merely shows the growth of the steady state values $\hat{S}$ and $\hat{A}$ if one of the parameters k, δ, α, or μ is changed. The following figures depict the results.

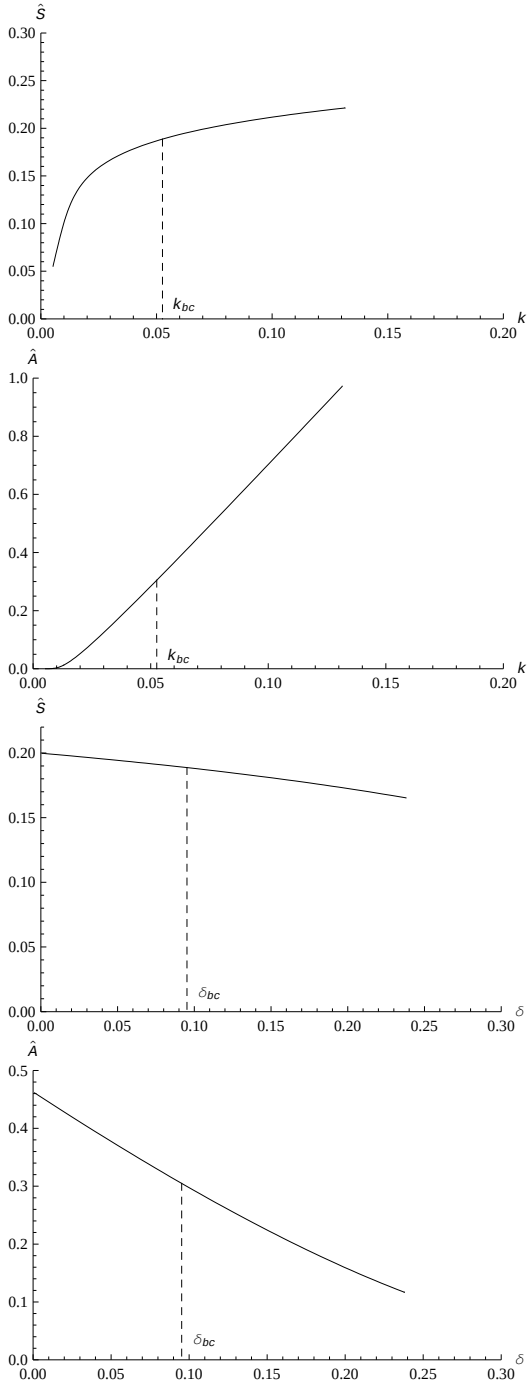

Figure 4.6: Bifurcation plots with respect to the parameters k and δ for the Australian IDU epidemic.

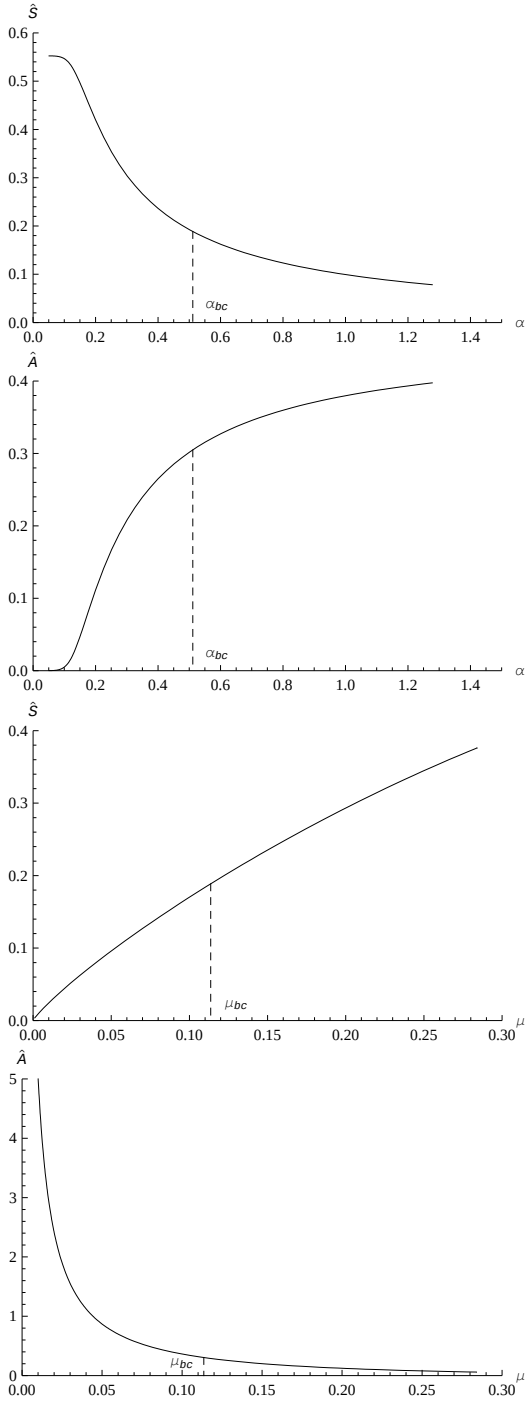

Figure 4.7: Bifurcation plots with respect to the parameters α and μ for the Australian IDU epidemic.

4.2.2.2 Bifurcation Analysis with regard to the Parameter β

If the parameter β is increased relative to its base case, then two bifurcation points occur. The first one at $\beta_{c_1} = 1.0795$ and the second one at $\beta_{c_2} = 1.29002$. As soon as the first critical value is reached, the number of steady states changes. So far, the equilibrium was unique but now there are two stationary points. These remain upright within a short area until they collide with each other at the second critical parameter value.

If also negative steady state values were permitted, then there would be two equilibria in the complete area until the second critical parameter value is reached.

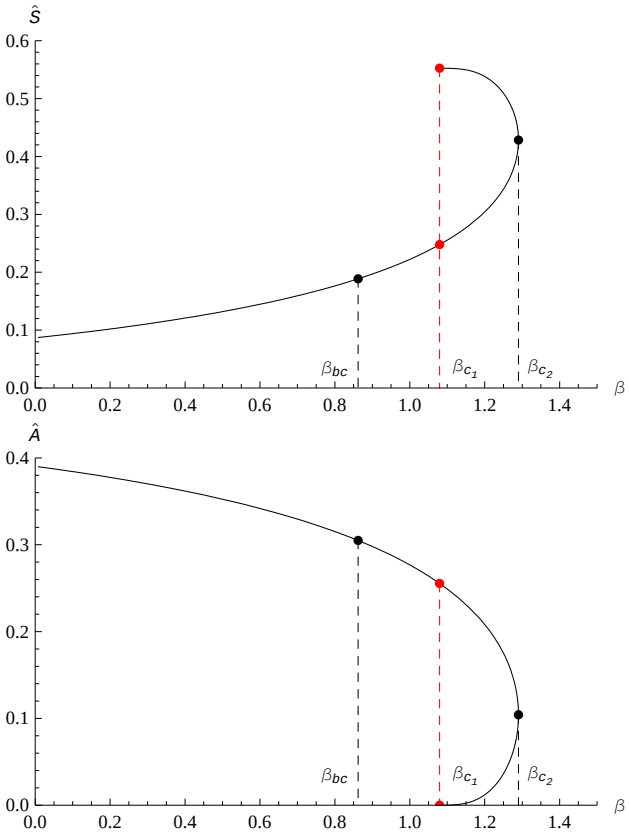

Figure 4.8: Bifurcation plots with respect to the parameter β for the Australian IDU epidemic.

Chapter 5

SAC Model vs. SA Model

Until now, we have already realized a variety of analyses with our SAC model. However, it also makes sense to compare its dynamics with the SA dynamics.

The dynamical system, examined in this thesis is

$$\begin{aligned} \dot{S} &= k - \delta\ S - \alpha\ A^{\beta}\ S\ p^{\frac{a}{\eta_S - \eta_D}} \\ \dot{A} &= \alpha\ A^{\beta}\ S\ p^{\frac{a}{\eta_S - \eta_D}} - \mu\ A \\ \dot{C} &= c'\ (\ln(A) - \ln(C))\ C \end{aligned}$$

and the formulation for the SA model is

$$\begin{aligned} \dot{S} &= k - \delta\ S - \alpha\ A^{\beta}\ S \\ \dot{A} &= \alpha\ A^{\beta}\ S - \mu\ A. \end{aligned}$$

The two systems vary in the additional state variable C and in the price function $p(A, C) = \frac{A}{C}$. C represents the current "throughput capacity" of the supply network and its state equation is dependent on the parameter c' which is given by $c' = \frac{c}{\eta_S - \eta_D}$. With our parameterization the denominator is 1 and so $\dot{C}$ is only depending on the coefficient in the growth rate, c, with the base value 0.15.

In this chapter, we will change this parameter and compare the respective paths and prices with those of the two-state SA model. Please note that the

bigger the parameter value is, the closer is the adaptation to the original SA system. Formulated sloppily this means that if $c = 0.15$ is changed to $c = \infty$, then the third state variable basically disappears.

This analysis is carried out for the U.S. cocaine epidemic with two different initial values, $(S(0), A(0)) = (10, 1.8)$ and $(S(0), A(0)) = (10, 1.6)$. The initial conditions were chosen such that once the high-use-equilibrium is approached and once $\hat{A} = 0$. For $C(0)$ we use $C(0) = A(0)$, $C(0) = \frac{A(0)}{2}$, and $C(0) = 2A(0)$.

The results can be found in Figures 5.1 - 5.6. In some cases, the SAC model does not yield any significant difference in comparison with the SA dynamics. But there are also situations in which the SA model and the SAC model behave very differently. In particular, SA can go to extinction but not SAC and vice versa, if the parameter c is small enough (see Figure 5.2 and Figure 5.4). In Figure 5.6 the two systems approach two different steady states already for the base value $c = 0.15$. Even if the long-run outcome is the same, there may be very different trajectories for getting there. We conclude that it can play a large role depending on which of the two models is used.

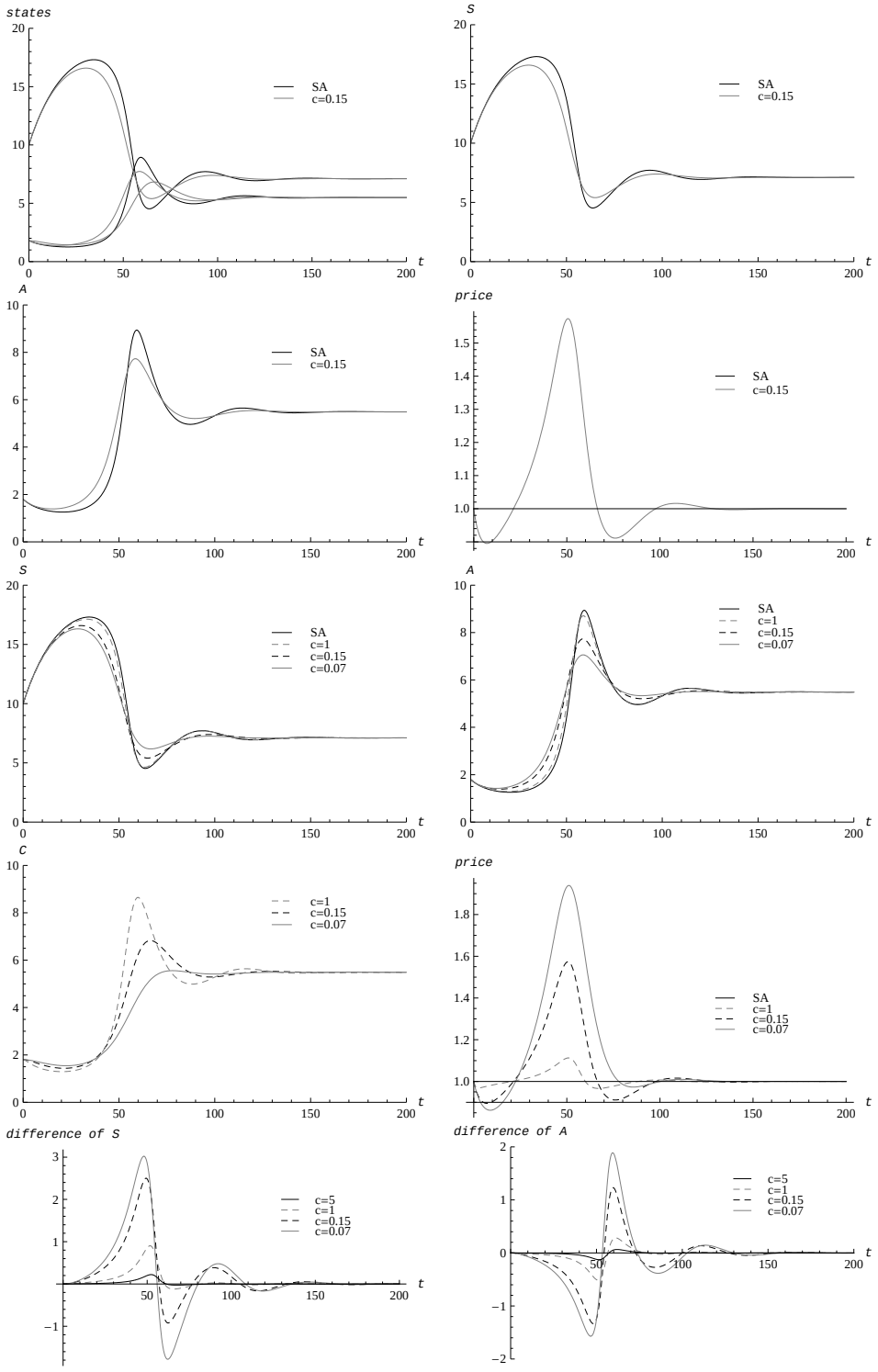

Figure 5.1: Comparison between the SAC and the SA model for the U.S. parameter set with $(S(0), A(0), C(0) = A(0)) = (10, 1.8, 1.8)$.

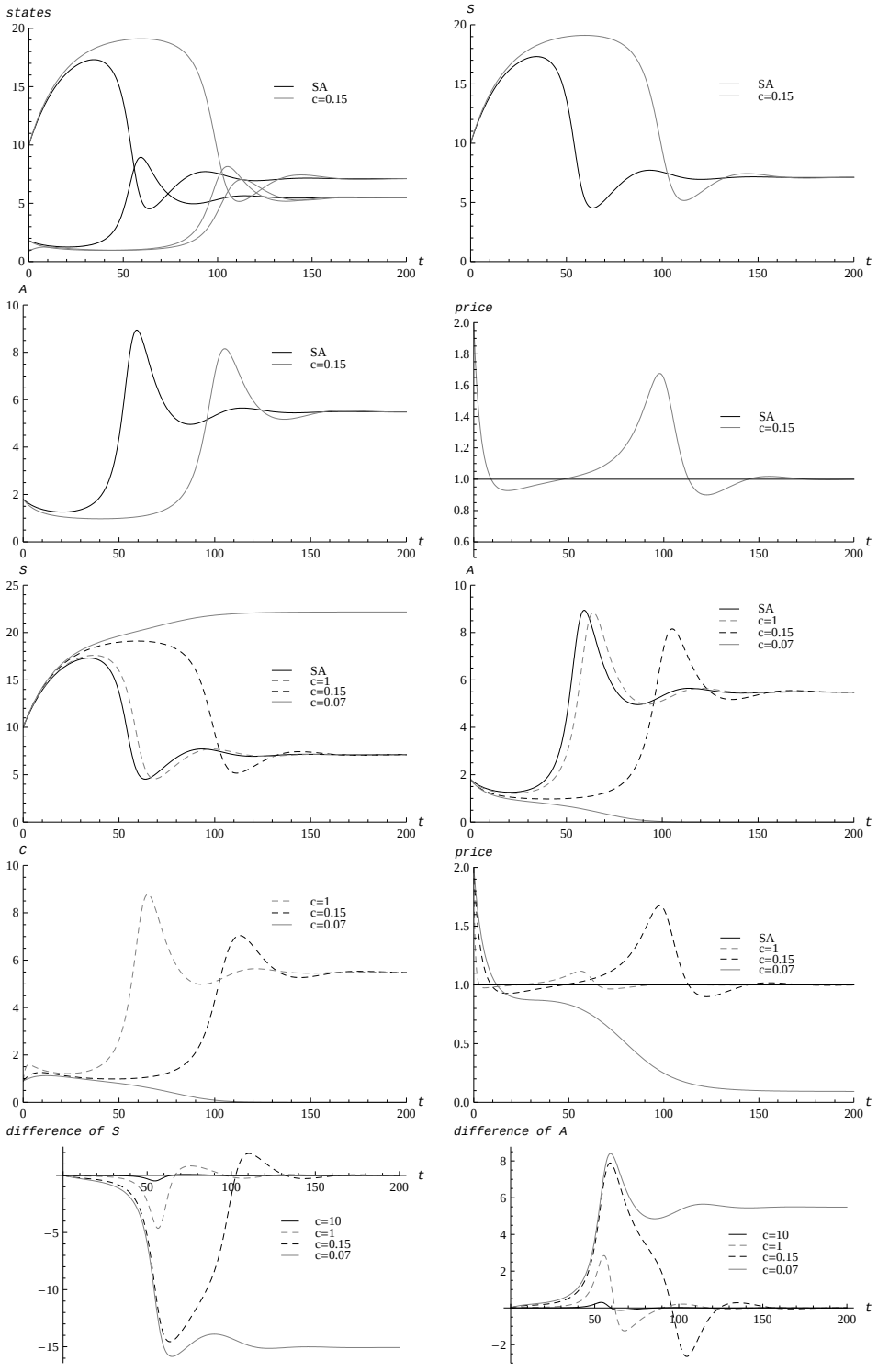

Figure 5.2: Comparison between the SAC and the SA model for the U.S. parameter set with $(S(0), A(0), C(0) = \frac{A(0)}{2}) = (10, 1.8, 0.9)$.

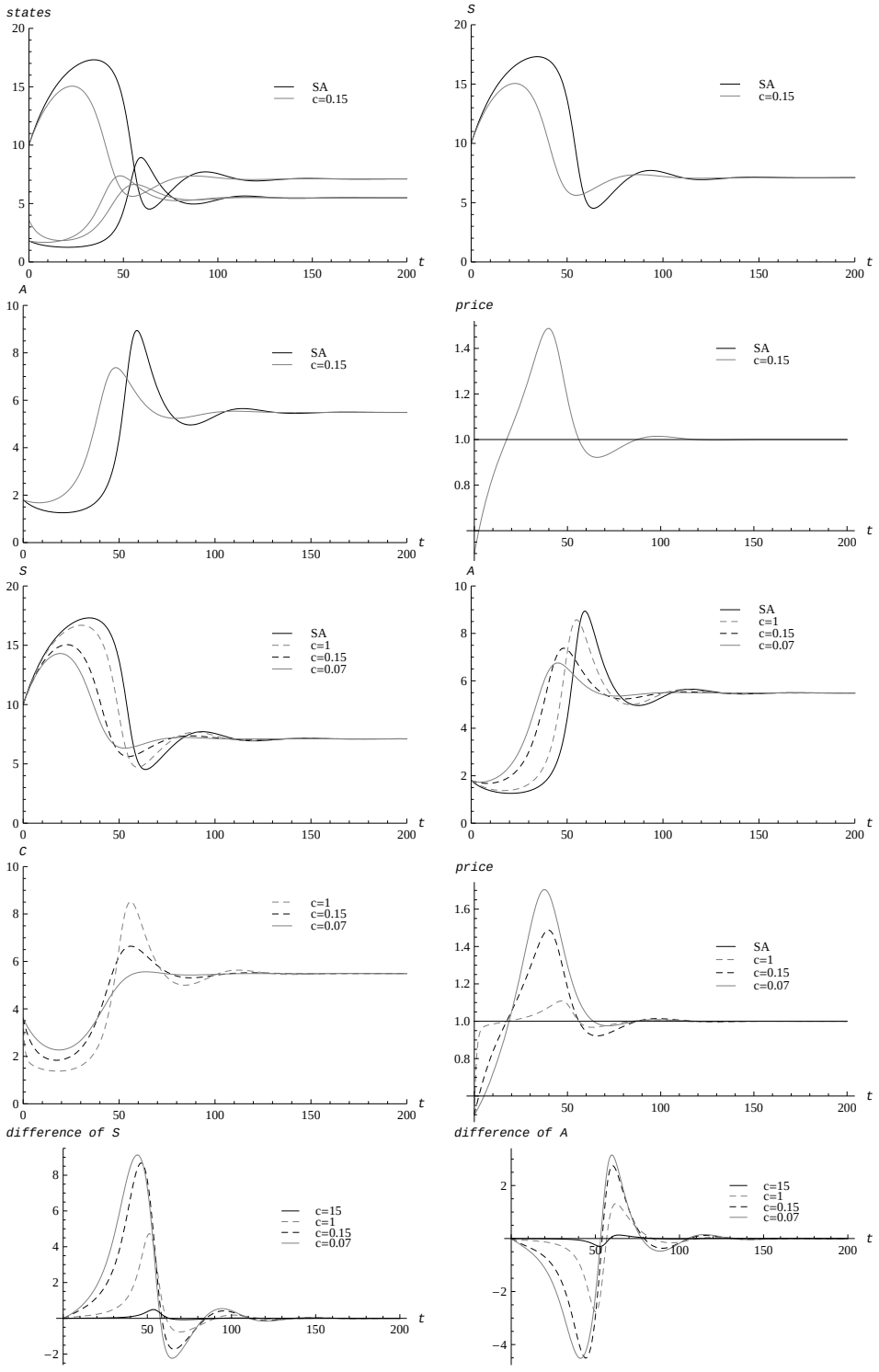

Figure 5.3: Comparison between the SAC and the SA model for the U.S. parameter set with $(S(0), A(0), C(0) = 2A(0)) = (10, 1.8, 3.6)$.

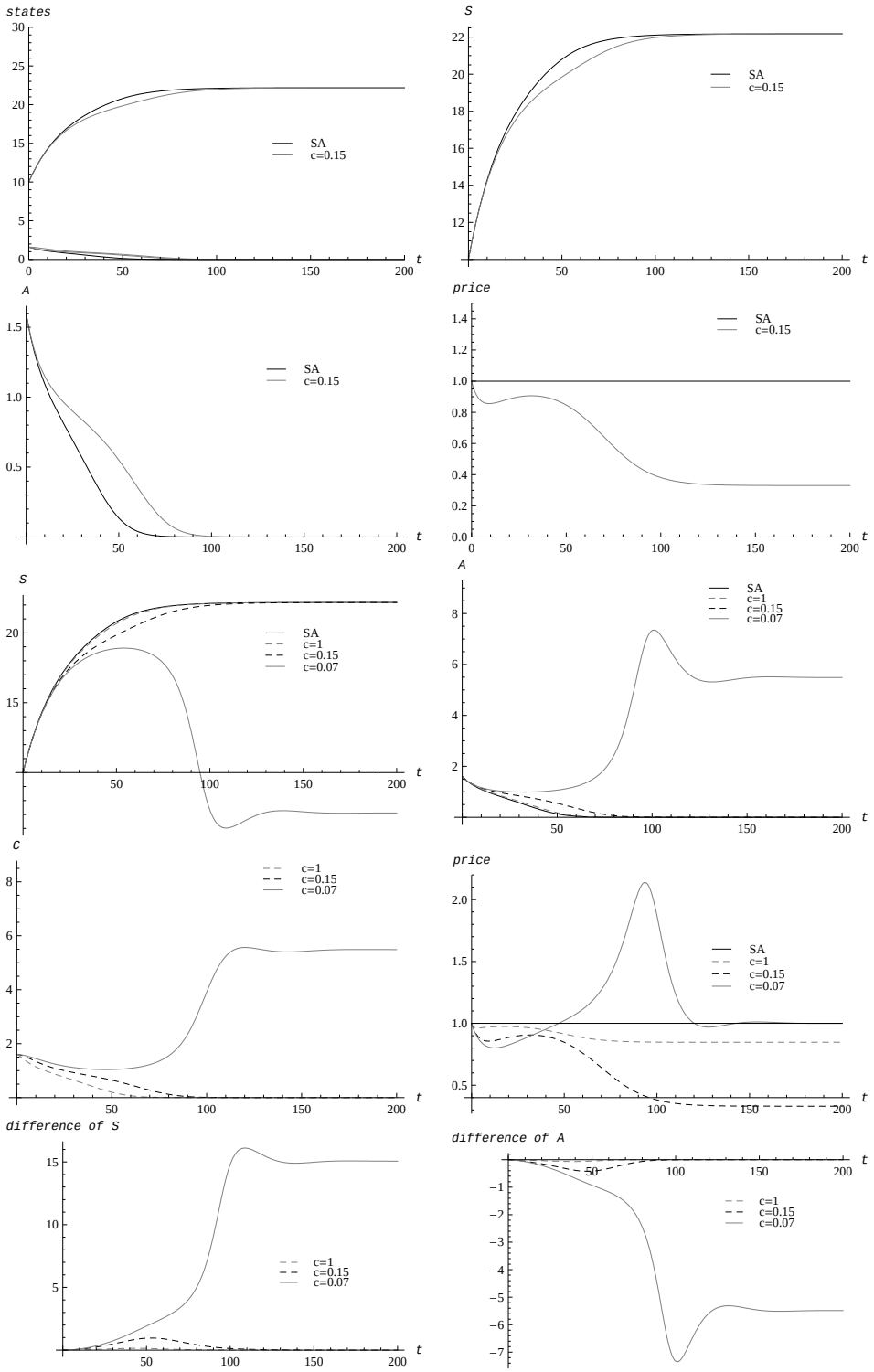

Figure 5.4: Comparison between the SAC and the SA model for the U.S. parameter set with $(S(0), A(0), C(0) = A(0)) = (10, 1.6, 1.6)$.

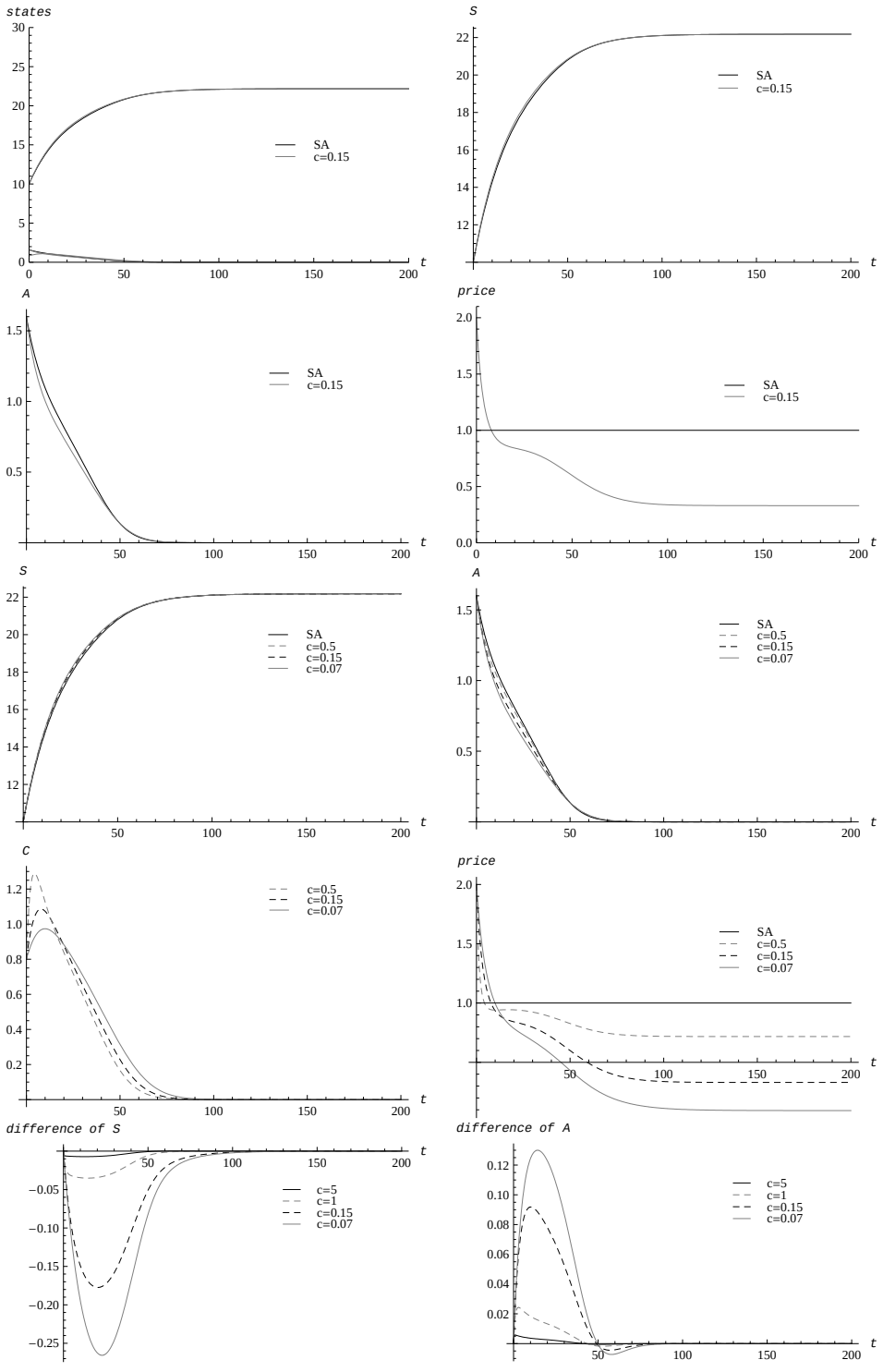

Figure 5.5: Comparison between the SAC and the SA model for the U.S. parameter set with $(S(0), A(0), C(0) = \frac{A(0)}{2}) = (10, 1.6, 0.8)$.

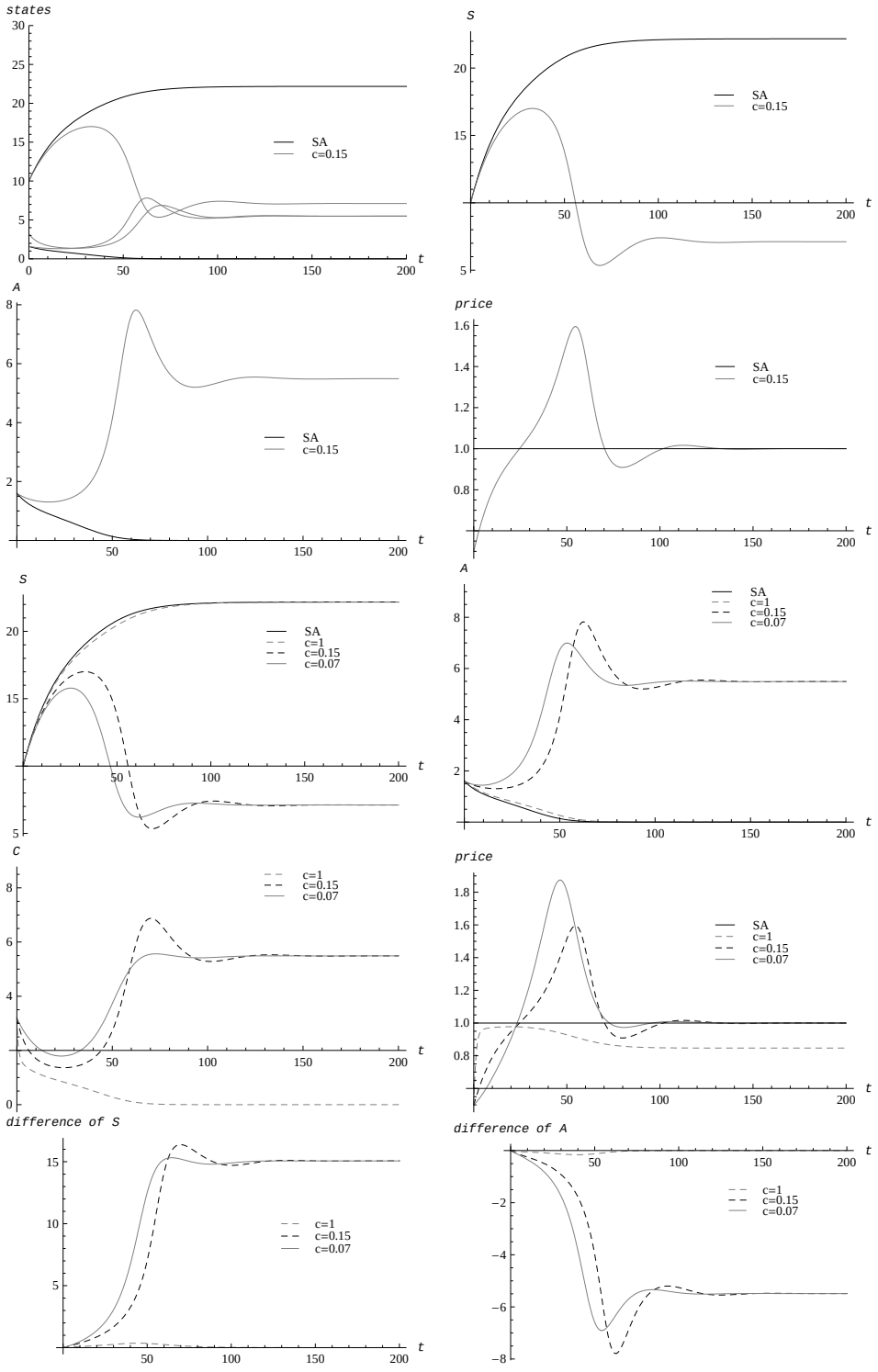

Figure 5.6: Comparison between the SAC and the SA model for the U.S. parameter set with $(S(0), A(0), C(0) = 2A(0)) = (10, 1.6, 3.2)$.

Chapter 6

The Relative Efficiency and Cost-Effectiveness of Treatment and Supply Shocks at Different Stages of the Epidemic

6.1 Treatment of U.S. Cocaine Use

This chapter deals with some strategic examinations, which can be seen as a preliminary stage to an optimal control model. First, we are interested in determining the relative efficiency of treatment at different stages of the drug epidemic. In this connection, we deal with the exit from active use, μ, because an increase of this parameter can be interpreted as resulting from successful treatment of users. This means that μ has actually the form $\mu + u(t)$ and in the uncontrolled model this control variable $u(t)$ is equal to zero.

However, the quitting rate will not be increased arbitrarily or in some optimal manner now. Rather, this will be the case only for one year and at different stages of the epidemic. Therefore, μ is increased in the first year, then in the second, and so on. These analyses are carried out for the first 50 years. Finally, we aim to determine that point in time where the effectiveness of treatment is the greatest. More precisely, we are looking for that time at

which most users quit because of increased investments in treatment. Our investigations are carried out on the basis of the objective functional

$$J = \int_0^\infty e^{-rt}\ A(t)\ dt,$$

which describes the discounted accumulation of users over an infinite planning horizon. We use a finite ($T = 200$) approximation to the infinite planning horizon

$$J = \int_0^T e^{-rt}\ A(t)\ dt + \int_T^\infty e^{-rt}\ \hat{A}\ dt,$$

where the integral

$$\int_T^\infty e^{-rt}\ \hat{A}\ dt = \hat{A} \int_T^\infty e^{-rt}\ dt = \hat{A} \lim_{N\to\infty} \int_T^N e^{-rt}\ dt = \hat{A}\ \frac{e^{-rT}}{r}$$

is added for a higher precision. Furthermore, we discount at an annual rate of $r = 0.04$. The approach used here is similar to examinations pertaining to prevention in another drug model as described in [Winkler et al., 2004].

The following analysis will be carried out for five different trajectories with the respective initial values given by $(S(0), A(0), C(0)) = (20, 2, 4)$, $(20, 2, 2)$, $(20, 2, 1)$, $(20, 4, 2)$, and $(5, 7, 5)$. In the first case there is an excess supply, while supply and demand coincide in the second case and $(S(0), A(0), C(0)) = (20, 2, 1)$ means an insufficient supply. The other two initial conditions serve for comparison purposes. Please note that all select trajectories converge to the high-use equilibrium $\hat{E}_2$ and therefore $\hat{A}$ is at the value 5.4888.

Every analysis requires two systems which are looked at: on the one hand that one, where μ is unchanged, and on the other hand that one, where the exit rate is increased. We will denote the corresponding trajectories by T_0 and T_1, respectively. Very first, the rise of μ is 1%, then 2%, then 5%, and finally 10%.

Before looking at the results, we still have to describe the computation of the cost functional associated with drug consumption. If we calculate the value of J where μ is not increased, then we get

$$J_0 = \int_0^T e^{-rt}\ A_{T_0}(t)\ dt + \int_T^\infty e^{-rt}\ \hat{A}\ dt.$$

If the control intervention treatment, however, is "switched on" in the first year, then J_1 calculates itself through

$$J_1 = \int_0^1 e^{-rt}\ A_{T_1}(t)\ dt + \int_0^T e^{-r(t+1)}\ A_{T_0}(t)\ dt + \int_{T+1}^{\infty} e^{-rt}\ \hat{A}\ dt.$$

Special attention should be paid to the second integral here. The values of the system T_0 are used there, but the initial conditions must be in accordance with the last values of the previous system, i.e. $A_{T_1}(1)$.

For the remaining years the calculation is given by

$$\begin{aligned} J_i &= \int_0^i e^{-rt}\ A_{T_0}(t)\ dt + \int_0^1 e^{-r(t+i)}\ A_{T_1}(t)\ dt + \\ &+ \int_0^T e^{-r(t+i+1)}\ A_{T_0}(t)\ dt + \int_{T+i+1}^{\infty} e^{-rt}\ \hat{A}\ dt \end{aligned}$$

with adjusted initial values.

The following figures finally show the results of our examinations. Since the illustrations are very similar, only those results are shown where the exit rate was increased by 1% and by 10%. Please note that the respective effects are expressed as percentage.

We see clearly that the first three initial values (Figures 6.1-6.3 and 6.6-6.8) as well as the 5th ones (Figures 6.5 and 6.10) yield the greatest effect in the first year. This means that an x% increase of μ within the first year leads to the lowest objective cost functional. This seems to be very plausible with regard to the discounting. With $(S(0), A(0), C(0)) = (20, 4, 2)$ it is different (Figures 6.4 and 6.9). Here, the greatest effect does not occur right at the beginning but only in the 8th year. This seems to be due to the higher value of $A(0)$. One possible conclusion is that treatment early in the epidemic is more valuable than later.

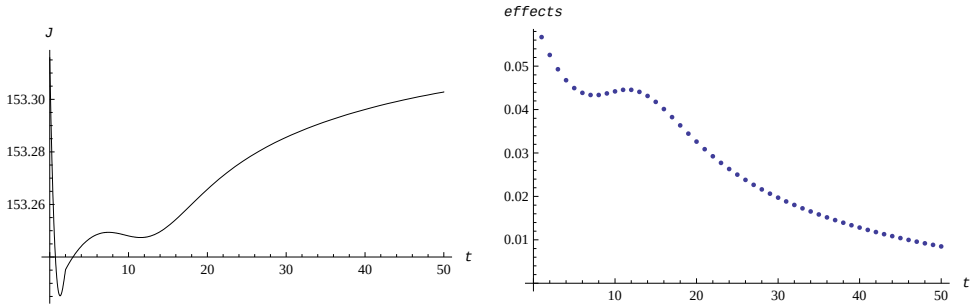

Figure 6.1: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 1%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 4)$.

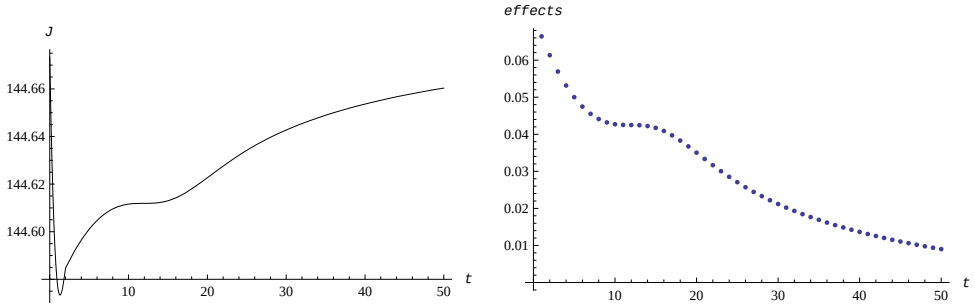

Figure 6.2: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 1%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 2)$.

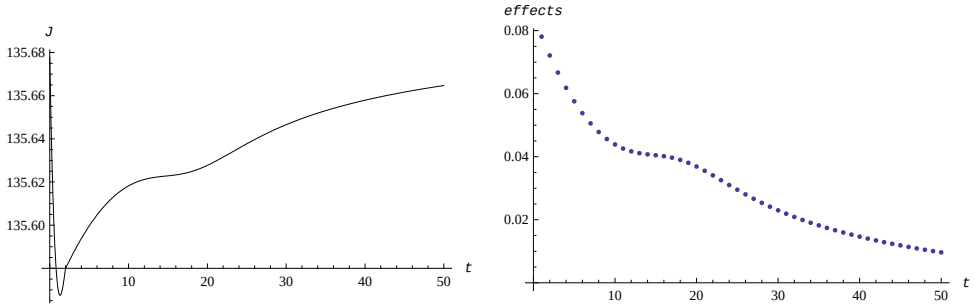

Figure 6.3: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 1%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 1)$.

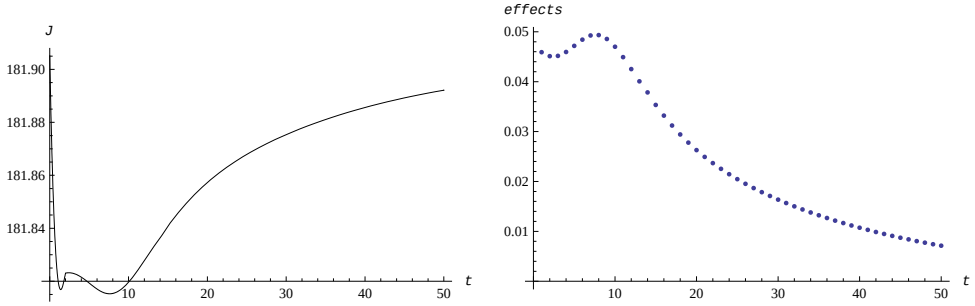

Figure 6.4: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 1%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 4, 2)$.

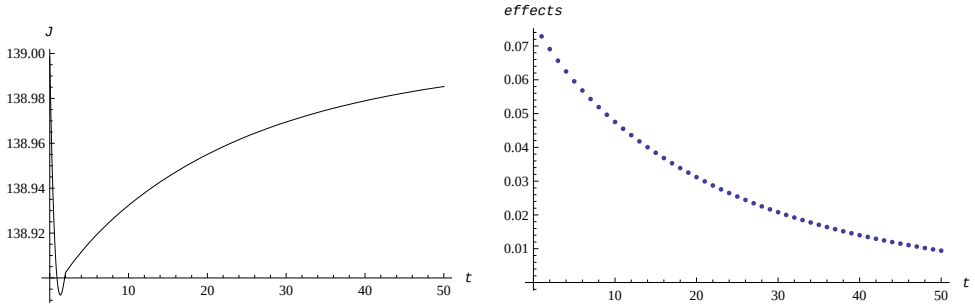

Figure 6.5: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 1%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (5, 7, 5)$.

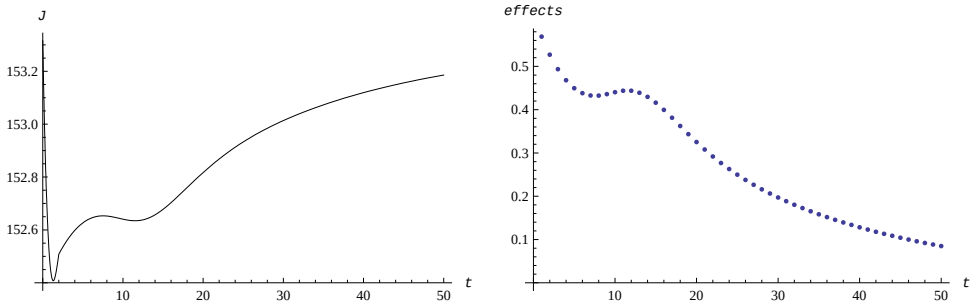

Figure 6.6: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 10%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 4)$.

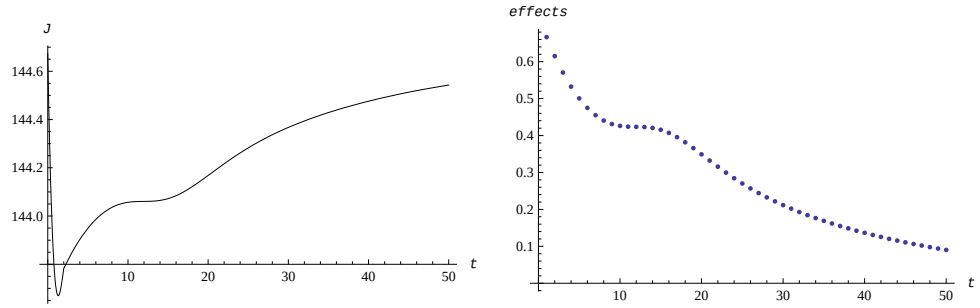

Figure 6.7: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 10%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 2)$.

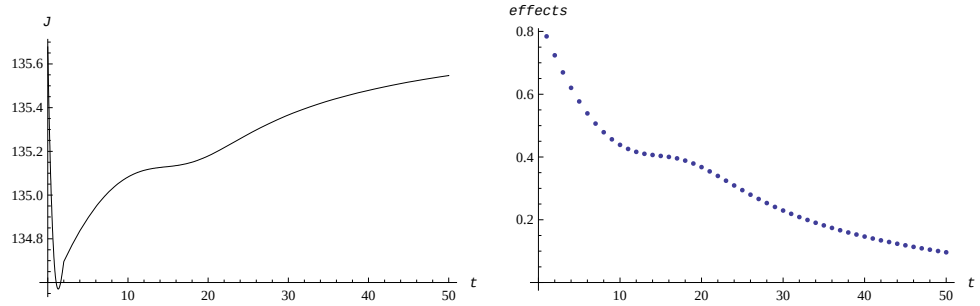

Figure 6.8: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 10%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 1)$.

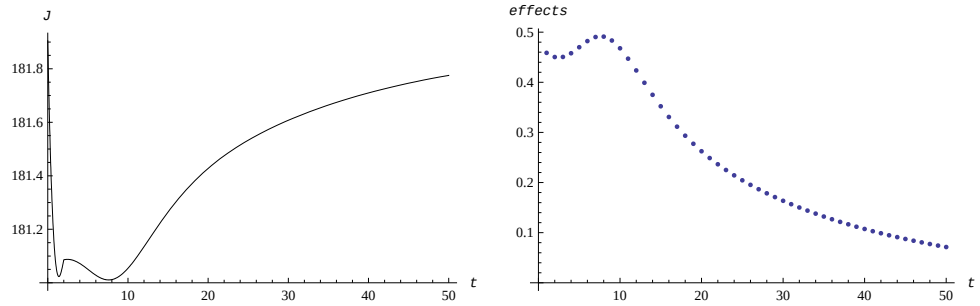

Figure 6.9: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 10%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 4, 2)$.

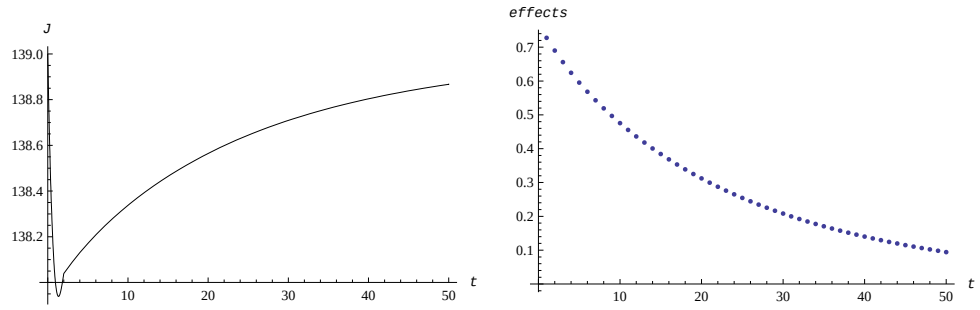

Figure 6.10: Plot of J and of the variation over time in the effectiveness of treatment when μ is increased by 10%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (5, 7, 5)$.

Digging somewhat deeper in the interpretations, something very interesting stands out. For the first initial values the decrease is not monotone. In order to examine this issue more closely, we look at the normalized effects and the normalized state variables within one graphic. Figure 6.11 shows this for the first three initial conditions in which μ was increased by 1%. The thick line describes the number of users. A connection with this state is obvious, since a higher number of users also leads to higher impacts of treatment, if the effects are assumed to be proportional and not in absolute terms. One can recognize that the effects start to climb shortly before the peak in $A(t)$ is reached.

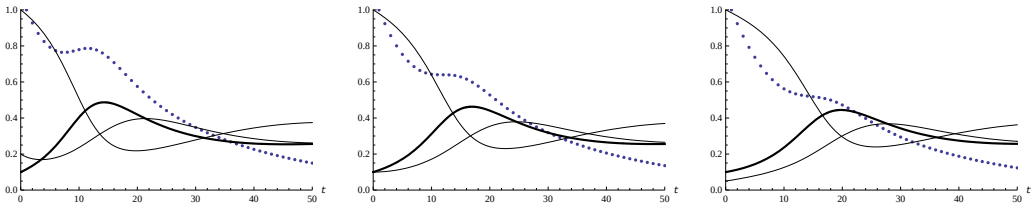

Figure 6.11: Normalized effects and state variables for $(S(0), A(0), C(0)) = (20, 2, 4)$, $(20, 2, 2)$, and $(20, 2, 1)$ when the exit from U.S. cocaine use is increased by 1%.

6.2 The Effects of a Supply Shock

Many illicit drug epidemics have experienced at least one significant supply shock. One prominent example is the recent Australian "heroin drought" (see, e.g., [Bultmann et al., 2008a,b], [Degenhardt et al., 2005], [Weatherburn et al., 2002]). In this section, we deal with such disruptions. In this connection this means that suddenly the supply could increase or drop at any time within the epidemic. One can interpret a change of C as follows: more/less is invested in the control of the supply side (dealers, etc.), so the drug price changes which in turn influences the consumers.

To simulate and investigate the effects of a supply shock, we project the model forward from the starting point. Then, we project it again from the same starting point except that $C(0)$ is cut or raised by, say, one-half. We repeat this exercise for various initial conditions corresponding to different stages of the epidemic. The examinations are conducted under the same preconditions and with the same initial values as in the previous section. Again, we want to determine the biggest effect. The effects are again measured by the discounted, aggregated number of users, i.e. the objective functional

$$J = \int_0^\infty e^{-rt}\, A(t)\, dt.$$

6.2.1 United States

At the beginning, we want to look at the consequences of a supply shock graphically. For this purpose, we consider a concrete scenario. The "throughput capacity" of the supply network is changed by a strong supply shock (50%) 10 years after the epidemic has started at the initial values $(S(0), A(0), C(0)) = (20, 2, 2)$. We start with the first case when C drops. By means of this change, the number of users also goes down, but to a lower extent. Consequently, the price increases what is plausible, since due to stronger controls on the part of the police, higher "costs" are caused to the dealers and this in turn affects the price. On the other hand, the higher price suppresses initiation. If C is increased, then we get opposite results. The evolution of the states, the price and the initiation are shown in Figures 6.12 and 6.13.

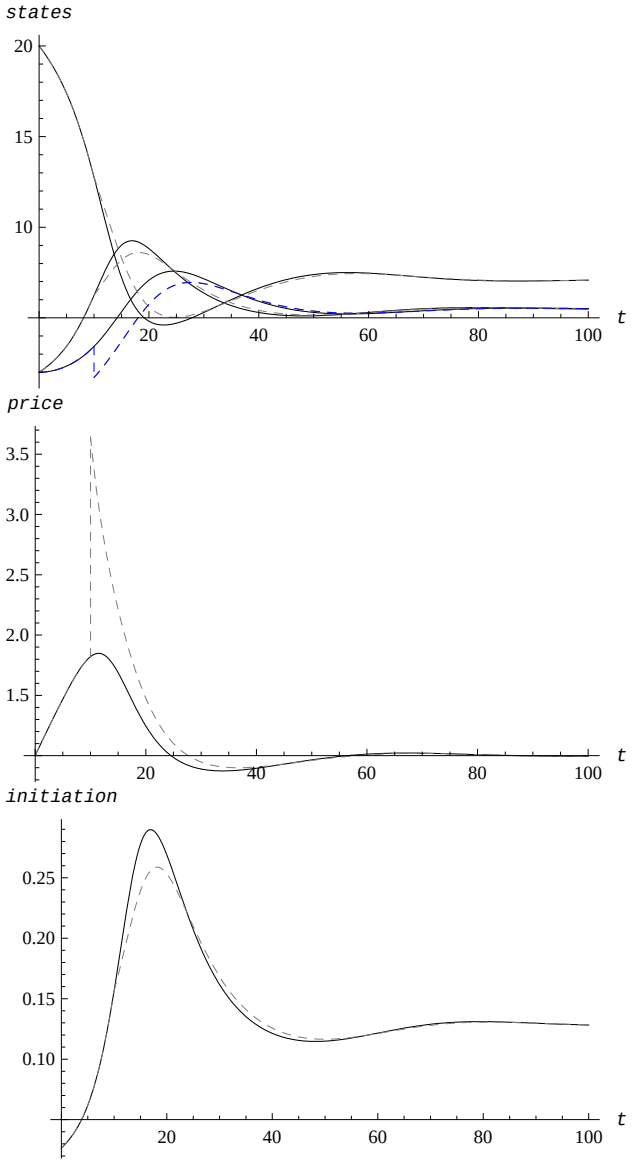

Figure 6.12: Evolution of the state variables, the price and initiation for $(S(0), A(0), C(0)) = (20, 2, 2)$, if the U.S. cocaine supply is cut by 50% after 10 years.

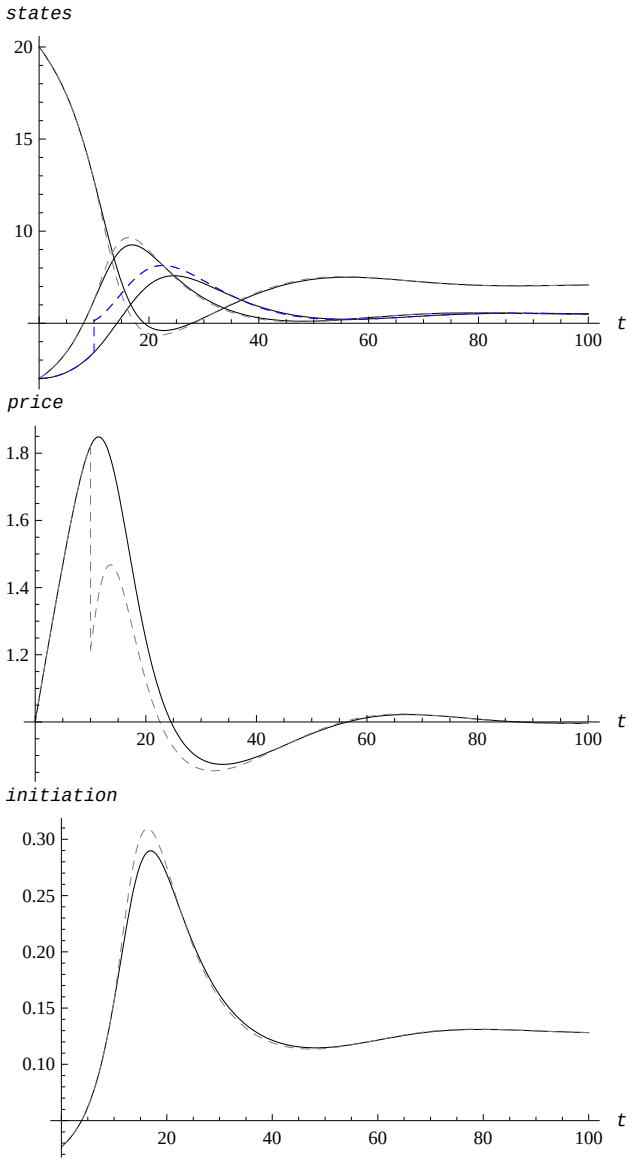

Figure 6.13: Evolution of the state variables, the price and initiation for $(S(0), A(0), C(0)) = (20, 2, 2)$, if the U.S. cocaine supply is increased by 50% after 10 years.

Next, we want to deal with the case where we start from the equilibrium $\hat{E}_2 = (\hat{S}_2, \hat{A}_2, \hat{C}_2) = (7.1076, 5.4888, 5.4888)$. That means, we already are in the steady state when the 50% supply shock occurs. Figures 6.14 and 6.15 show the progression of the states, the price and the initiation. We observe that a supply shock of this magnitude disrupts the system for several decades.

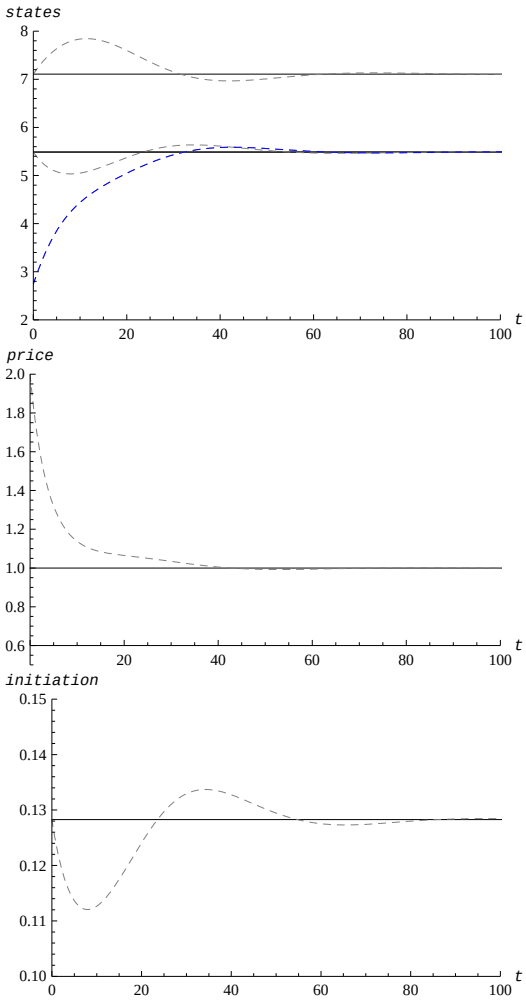

Figure 6.14: Evolution of the state variables, the price and initiation for $(S(0), A(0), C(0)) = \hat{E}_2 = (7.1076, 5.4888, 5.4888)$, if the U.S. cocaine supply is reduced by 50%.

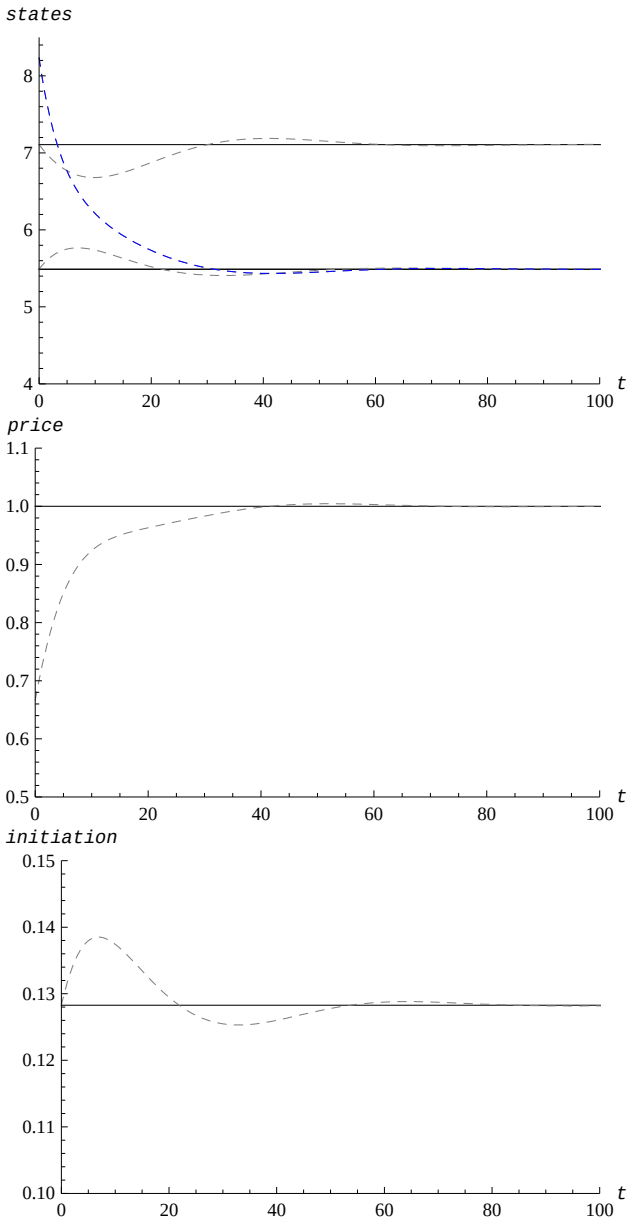

Figure 6.15: Evolution of the state variables, the price and initiation for $(S(0), A(0), C(0)) = \hat{E}_2 = (7.1076, 5.4888, 5.4888)$, if the U.S. cocaine supply is increased by 50%.

We now return to the question, when in an epidemic the effects of a supply shock are strongest. For that purpose, we will look at 5%, 10%, 25%, and finally 50% supply shocks in both directions. We will start with the same initial values as the ones used for the treatment section, and the objective functional J_0 is calculated as follows:

$$J_0 = \int_0^T e^{-rt}\ A(t)\ dt + \int_T^\infty e^{-rt}\ \hat{A}\ dt.$$

Thus, a finite (200-year) approximation to the infinite planning horizon is used with $\hat{A} = 5.4888$.

If we start at the same point, but increase or reduce $C(0)$, then we get

$$\tilde{J}_0 = \int_0^T e^{-rt}\ \tilde{A}(t)\ dt + \int_T^\infty e^{-rt}\ \hat{A}\ dt.$$

The calculation for supply shocks in the further years has one more stage. The trajectory is evaluated "normally" until time i, the last state values are then used as initial values in which C is additionally changed. So,

$$J_i = \int_0^i e^{-rt}\ A(t)\ dt + \int_0^T e^{-r(t+i)}\ \bar{A}(t)\ dt + \int_{T+i}^\infty e^{-rt}\ \hat{A}\ dt.$$

Finally, Figures 6.16-6.35 show our results. We can see that for all chosen initial values the effect is the greatest right in the 0th year. Since this applies to all analyses, only the 5% and the 50% supply shock is represented here.

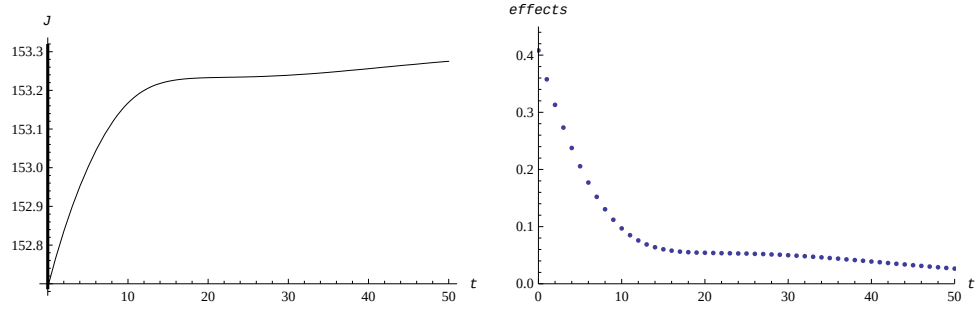

Figure 6.16: Illustration of J and of the effects (as percentage) when C is cut by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 4)$.

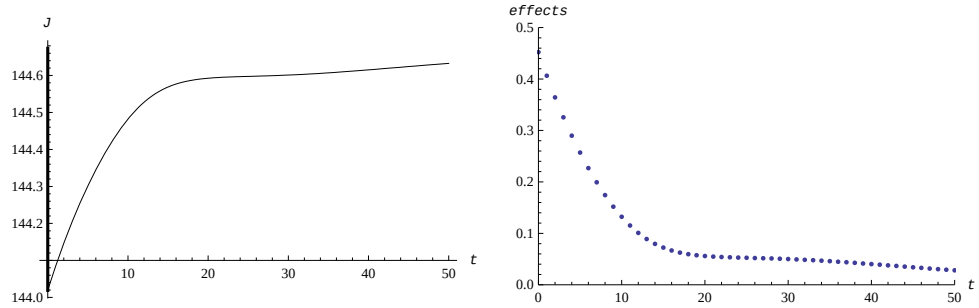

Figure 6.17: Illustration of J and of the effects (as percentage) when C is cut by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 2)$.

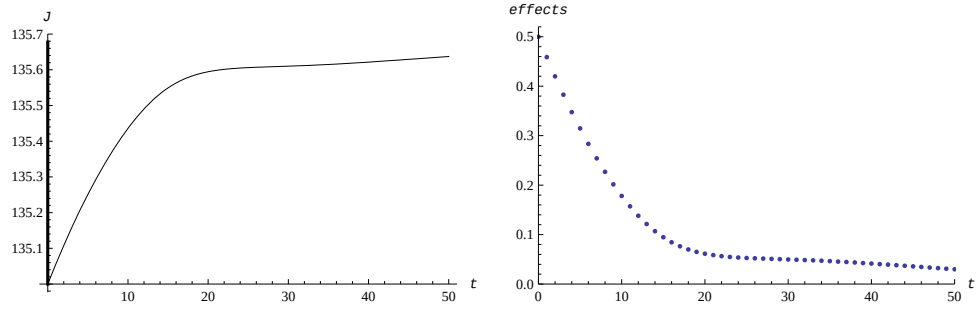

Figure 6.18: Illustration of J and of the effects (as percentage) when C is cut by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 1)$.

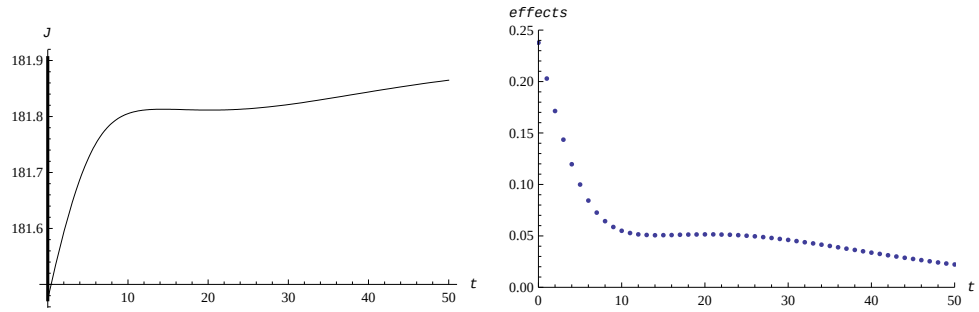

Figure 6.19: Illustration of J and of the effects (as percentage) when C is cut by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 4, 2)$.

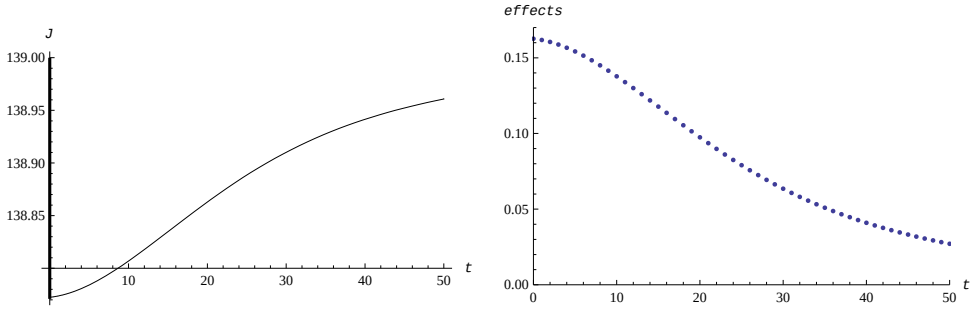

Figure 6.20: Illustration of J and of the effects (as percentage) when C is cut by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (5, 7, 5)$.

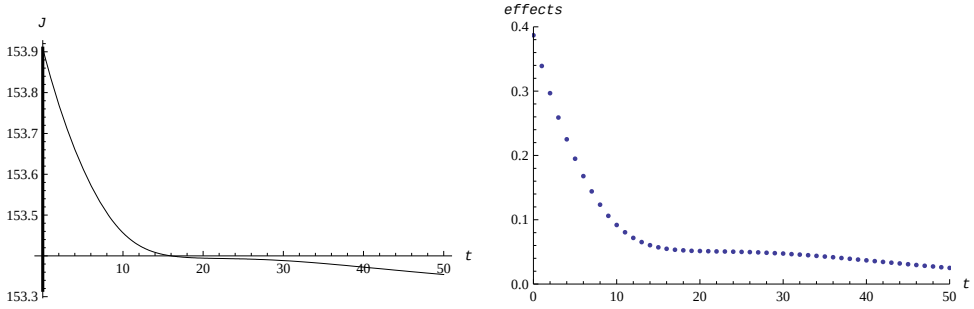

Figure 6.21: Illustration of J and of the effects (as percentage) when C is increased by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 4)$.

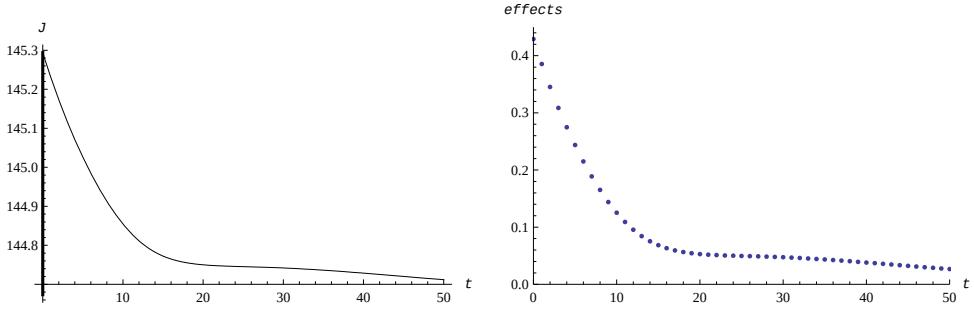

Figure 6.22: Illustration of J and of the effects (as percentage) when C is increased by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 2)$.

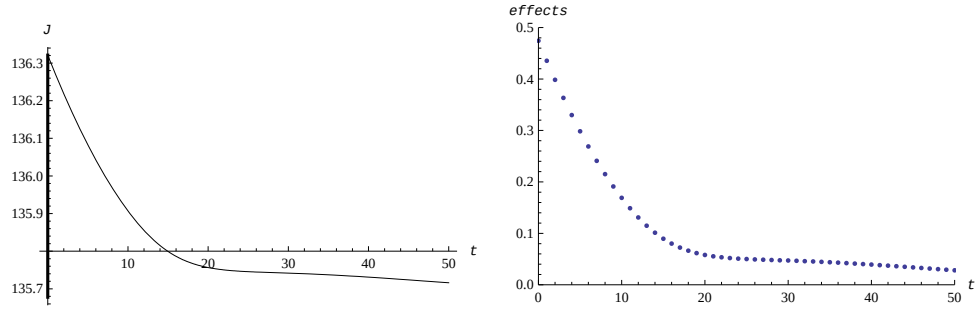

Figure 6.23: Illustration of J and of the effects (as percentage) when C is increased by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 1)$.

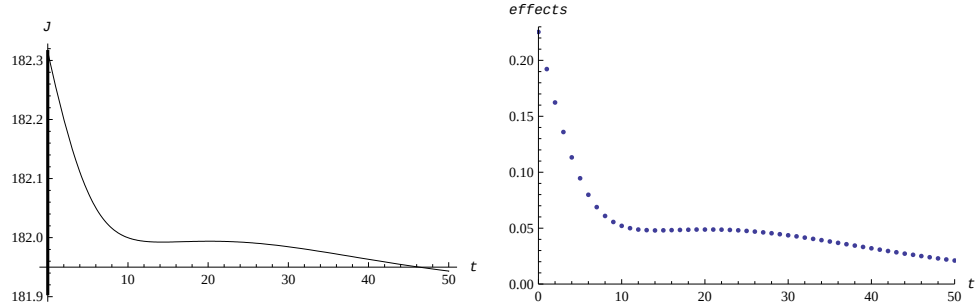

Figure 6.24: Illustration of J and of the effects (as percentage) when C is increased by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 4, 2)$.

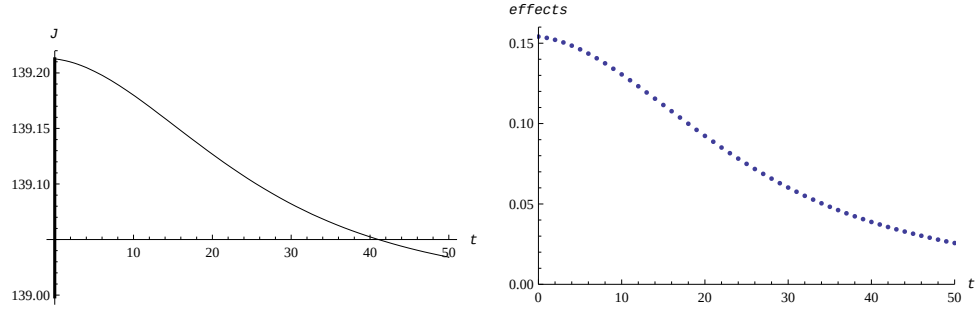

Figure 6.25: Illustration of J and of the effects (as percentage) when C is increased by 5%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (5, 7, 5)$.

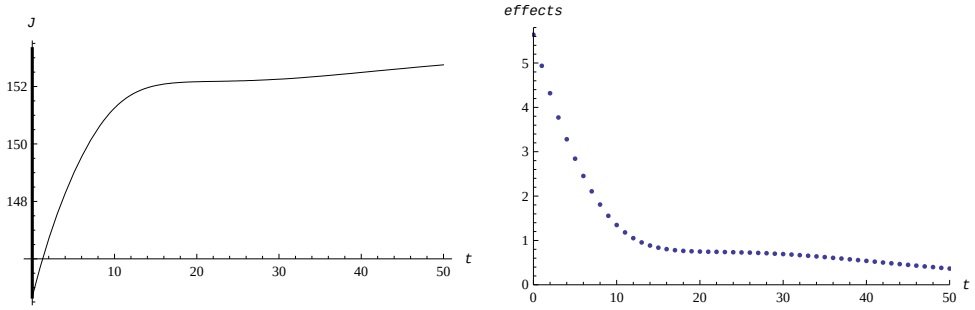

Figure 6.26: Illustration of J and of the effects (as percentage) when C is cut by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 4)$.

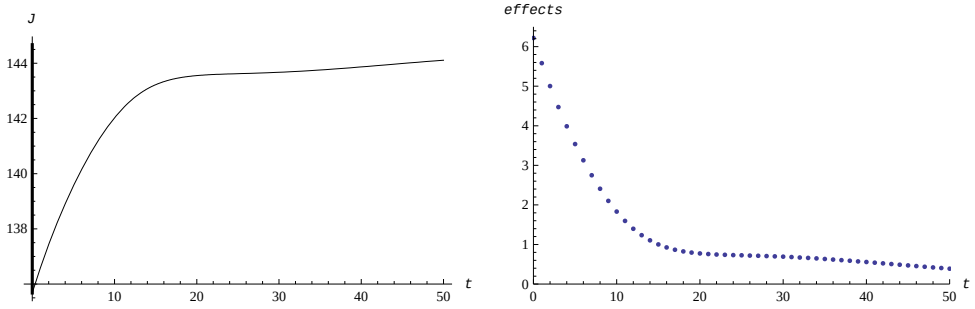

Figure 6.27: Illustration of J and of the effects (as percentage) when C is cut by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 2)$.

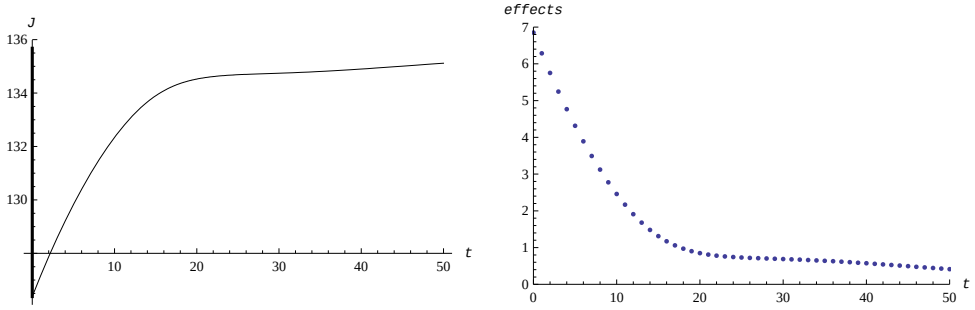

Figure 6.28: Illustration of J and of the effects (as percentage) when C is cut by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 1)$.

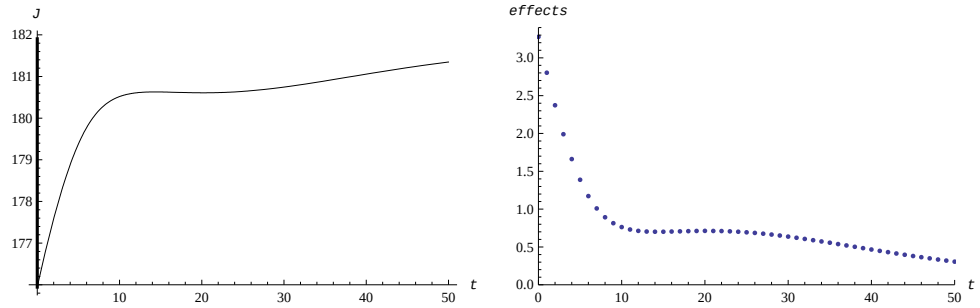

Figure 6.29: Illustration of J and of the effects (as percentage) when C is cut by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 4, 2)$.

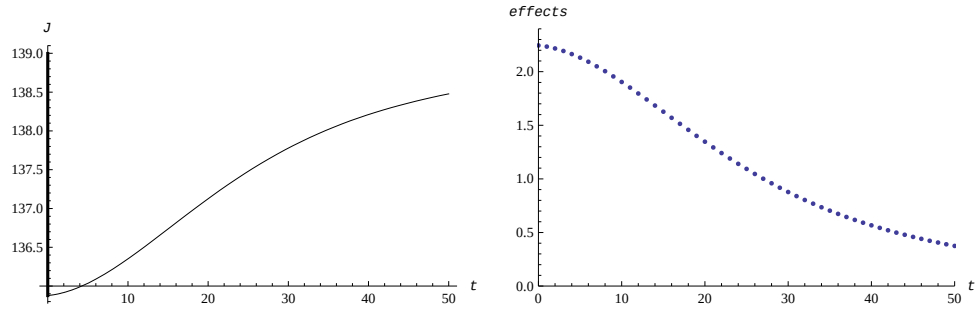

Figure 6.30: Illustration of J and of the effects (as percentage) when C is cut by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (5, 7, 5)$.

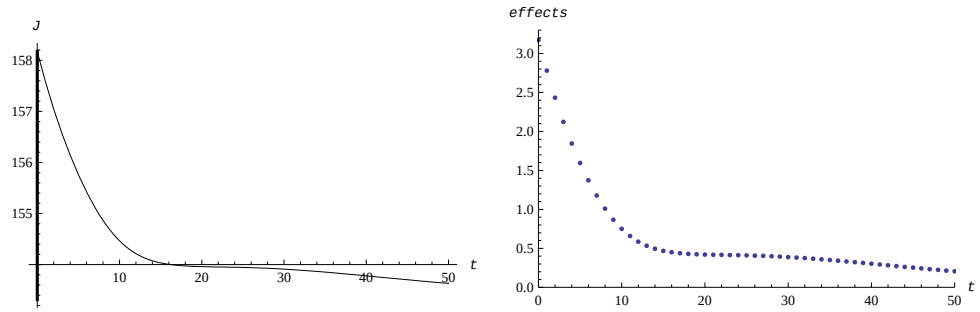

Figure 6.31: Illustration of J and of the effects (as percentage) when C is increased by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 4)$.

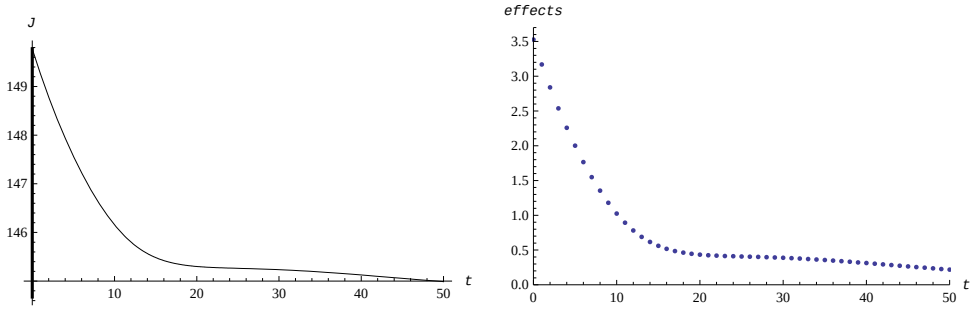

Figure 6.32: Illustration of J and of the effects (as percentage) when C is increased by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 2)$.

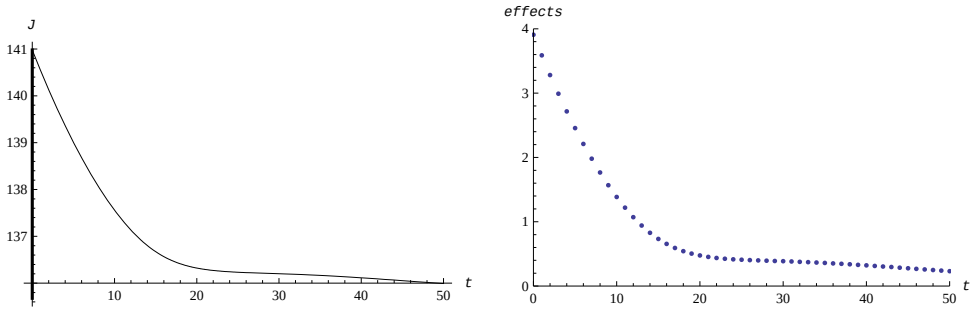

Figure 6.33: Illustration of J and of the effects (as percentage) when C is increased by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 2, 1)$.

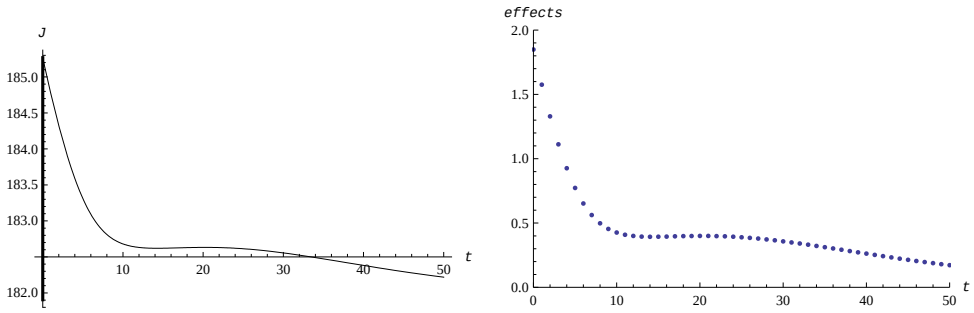

Figure 6.34: Illustration of J and of the effects (as percentage) when C is increased by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (20, 4, 2)$.

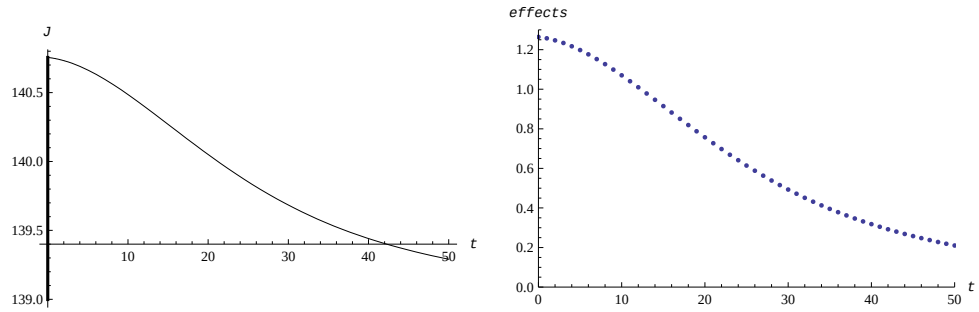

Figure 6.35: Illustration of J and of the effects (as percentage) when C is increased by 50%. The initial conditions for the system with the U.S. parameter values are $(S(0), A(0), C(0)) = (5, 7, 5)$.

Admittedly, these results are not really significant, since only five initial values were considered. For this reason, we want to expand our examinations a little. In particular, we look at the (A, S)-plane and form a grid consisting of 40×40 points, which are now the initial values. For the supply at the initial state, we use $C(0) = A(0)$ and $C(0) = \frac{A(0)}{2}$. Then, a 50% supply shock is simulated for each of these initial conditions. This means that the supply is cut by one-half and also at different stages of the epidemic. For the purpose of reducing the computing time, we only consider the first 15 years for the U.S. cocaine use. Furthermore, there are now trajectories which converge to $\hat{A} = 0$, so in this case the last integral drops out of the calculation.

After calculating the objective functionals, the year with the strongest effect is assigned to every initial value. The corresponding results are represented in Figures 6.36 and 6.37. If supply and demand coincide, then there are initial states where the strongest effect of a supply shock occurs only after 10 years. For $C(0) = \frac{A(0)}{2}$ the maximum effect is after 8 years.

In the case where $C(0) = A(0)$, we go for another analysis. We consider two concrete initial values: one within the white area (highest efficiency early) and one within the black area (highest efficiency late) and both close together. For that purpose, we choose $(S(0), A(0), C(0)) = (4.97533, 10.9776, 10.9776)$ and $(S(0), A(0), C(0)) = (4.26457, 10.9776, 10.9776)$. While in the first case the effect is the biggest already in the 0th year, the second initial conditions show their strongest effect only in the 10th year. If one compares the

respective state paths (see Figure 6.38), then something interesting stands out. Primarily the evolution of the numbers of users is very similar over time, but the control-recommendation is completely different. It is reasonable to assume that in an optimal control formulation, this model will exhibit so-called DNSS thresholds ([Grass et al., 2008]).

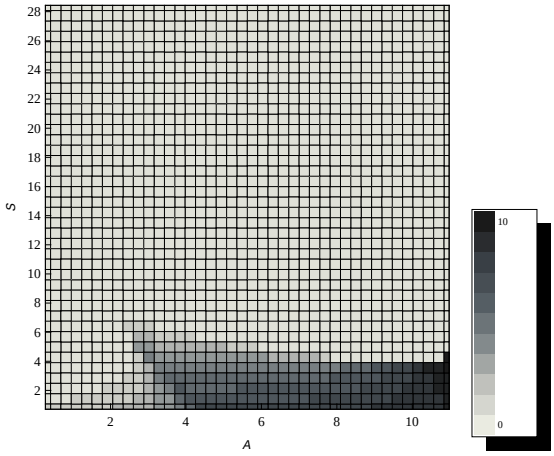

Figure 6.36: Years, where the consequence of a shock is the biggest when the U.S. cocaine supply is reduced by 50% and $C(0) = A(0)$.

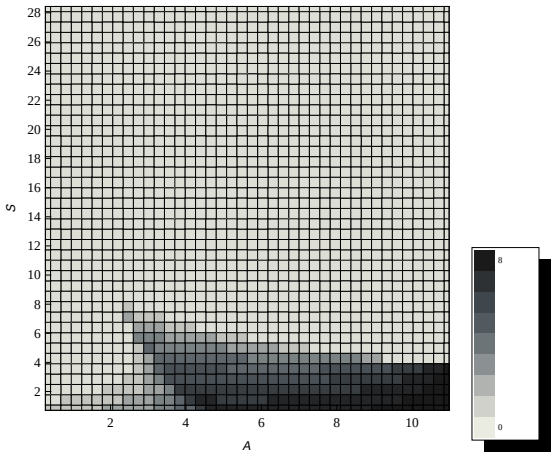

Figure 6.37: Years, where the consequence of a shock is the biggest when the U.S. cocaine supply is reduced by 50% and $C(0) = \frac{A(0)}{2}$.

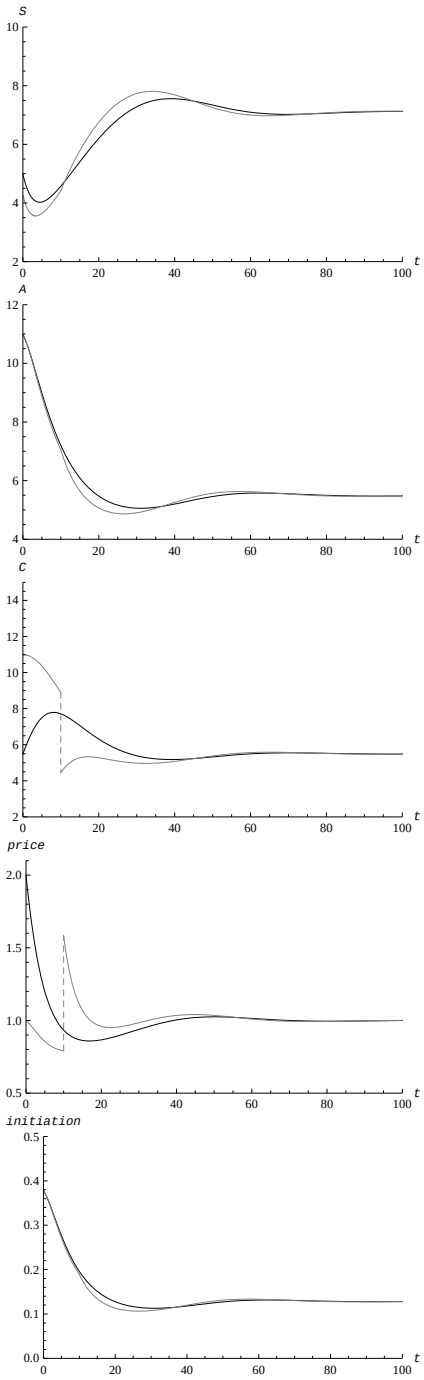

Figure 6.38: Comparison of the states, the price and the initiation for $(S(0), A(0), C(0)) = (4.97533, 10.9776, 10.9776)$ with a 50% supply shock at the beginning of the U.S. cocaine epidemic and for $(S(0), A(0), C(0)) = (4.26457, 10.9776, 10.9776)$ with a 50% supply shock after 10 years.

In the next and last examination $C(0)$ shall not be reduced by a certain percentage, but rather by an absolute value λ for the purpose of allowing some kind of "cost-effectiveness" analysis. Again, we deal with 40×40 initial points in the (A,S)-plane in which $C(0)$ and $A(0)$ are assumed to be equal. For determining the effects, the trajectory emanating from $(S(0),A(0),C(0))$ is used first and then that one which originates from $(S(0),A(0),C(0)-\lambda)$. We will denote the corresponding cost functionals by J and $\tilde{J}$ and subsequently calculate the relation $\frac{J-\tilde{J}}{\lambda}$. The bigger this value is, the bigger is the benefit or the damage per unit of capacity destroyed (depending on whether the values are positive or negative, respectively). However, here we are not using only the number of users as a measure, but we also look at three other objective functional forms:

$$\begin{aligned} J1 &= \int_0^\infty e^{-rt}\ A(t)\ dt \\ J2 &= \int_0^\infty e^{-rt}\ C(t)\ dt \\ J3 &= \int_0^\infty e^{-rt}\ p^{-0.5}\ A(t)\ dt \\ J4 &= \int_0^\infty e^{-rt}\ p^{0.5}\ A(t)\ dt. \end{aligned}$$

This means that also some proxy for the number of drug sellers ($J2$), the quantity of drugs consumed ($J3$), and the amount spent on drugs ($J4$) are added. Furthermore, we consider three different scenarios: $\lambda=1$, $\lambda=2$, and finally $\lambda=4$.

Figures 6.39-6.50 show the results. For ease of exposition and comparison, a suitable scaling was used. All values were divided by the minimum of all values and then the logarithm to the base 2 was applied so that all values start from 0.

The black curves in these graphs correspond to the isocline $\dot{A}=0$ whereas the black dashed lines indicate the isocline $\dot{S}=0$. In each case, the steady state $\hat{E}_2$ is depicted as a black dot. Besides, for $J4$ the values $\frac{J-\tilde{J}}{\lambda}$ are always negative and for this reason the absolute value was taken here and so no benefit but a damage is represented.

It is interesting to see that the policy recommendations are fairly similar for $J1$, $J2$, and $J3$, while for $J4$, everything is different. In other words, policy makers who care about the amount of money spent for drugs should act differently than those who neglect this part of social costs. Another obvious finding is that the larger the reduction λ, the smaller is the benefit per unit reduction, which is less surprising.

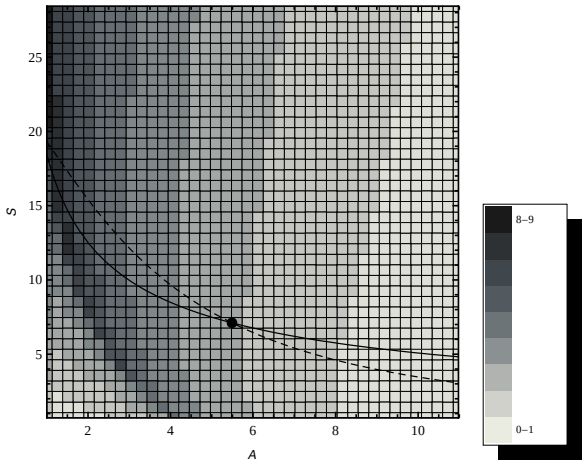

Figure 6.39: Illustration of the benefit, measured in terms of $J1$ and in which the U.S. cocaine supply is reduced by $\lambda = 1$.

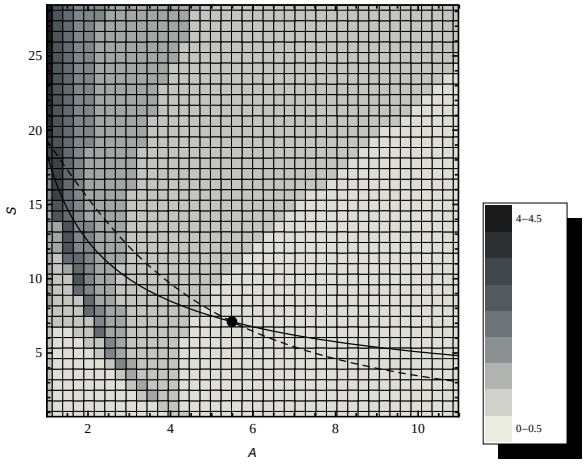

Figure 6.40: Illustration of the benefit, measured in terms of $J2$ and in which the U.S. cocaine supply is reduced by $\lambda = 1$.

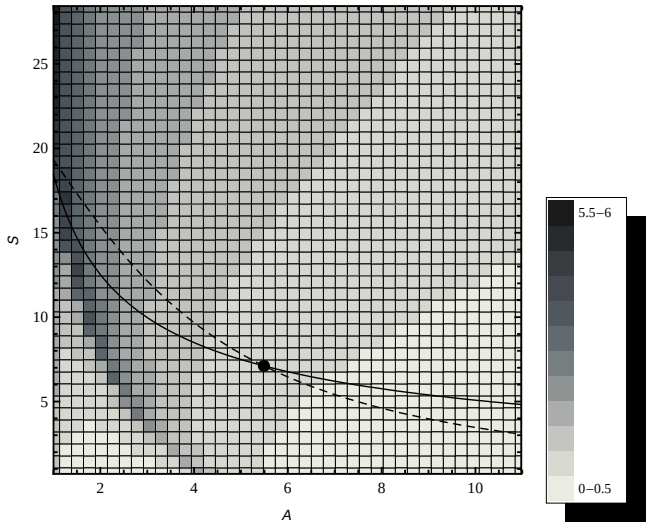

Figure 6.41: Illustration of the benefit, measured in terms of $J3$ and in which the U.S. cocaine supply is reduced by $\lambda = 1$.

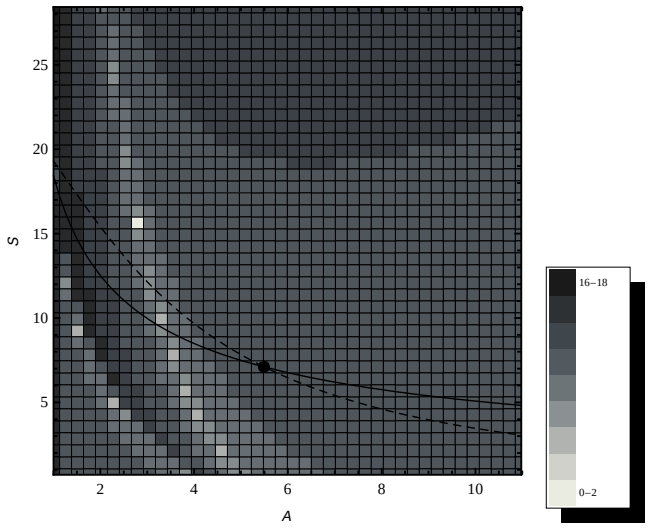

Figure 6.42: Illustration of the damage, measured in terms of $J4$ and in which the U.S. cocaine supply is reduced by $\lambda = 1$.

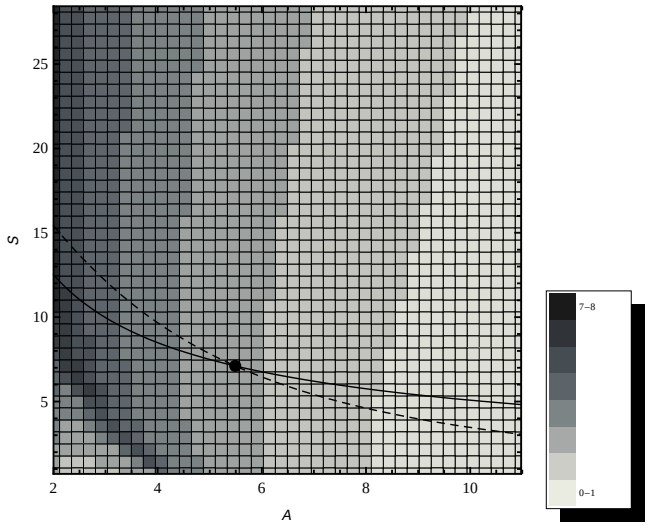

Figure 6.43: Illustration of the benefit, measured in terms of $J1$ and in which the U.S. cocaine supply is reduced by $\lambda = 2$.

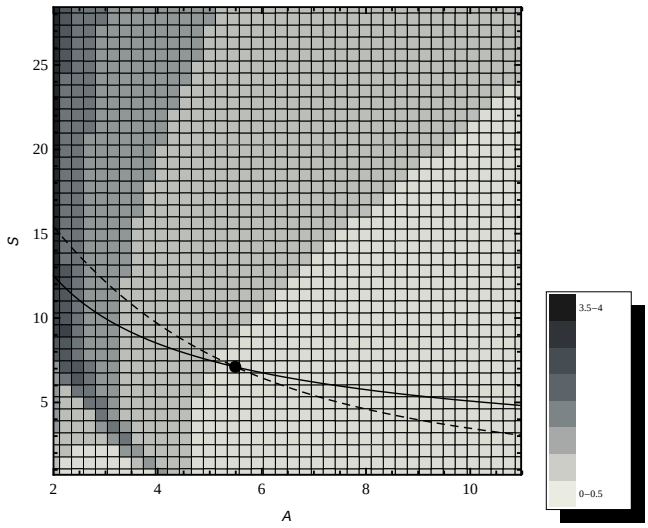

Figure 6.44: Illustration of the benefit, measured in terms of $J2$ and in which the U.S. cocaine supply is reduced by $\lambda = 2$.

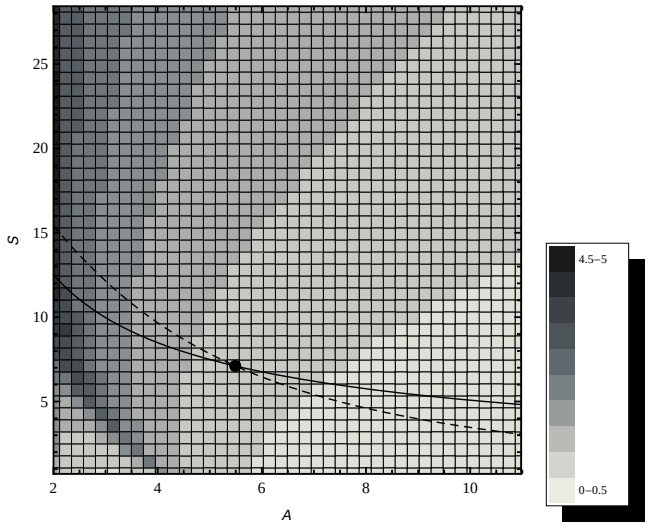

Figure 6.45: Illustration of the benefit, measured in terms of $J3$ and in which the U.S. cocaine supply is reduced by $\lambda = 2$.

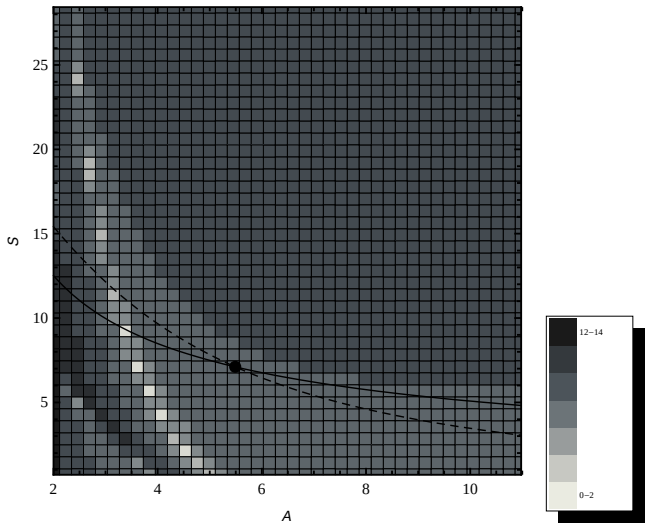

Figure 6.46: Illustration of the damage, measured in terms of $J4$ and in which the U.S. cocaine supply is reduced by $\lambda = 2$.

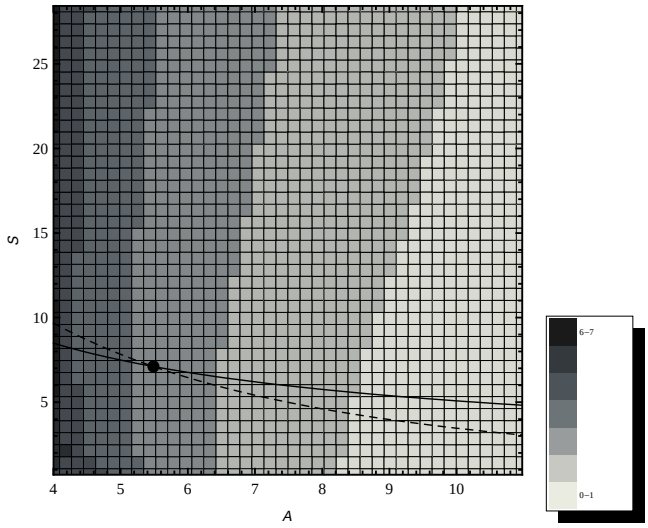

Figure 6.47: Illustration of the benefit, measured in terms of $J1$ and in which the U.S. cocaine supply is reduced by $\lambda = 4$.

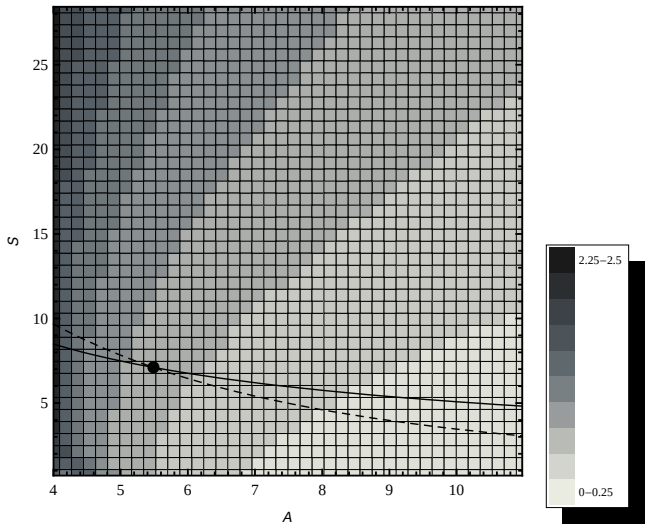

Figure 6.48: Illustration of the benefit, measured in terms of $J2$ and in which the U.S. cocaine supply is reduced by $\lambda = 4$.

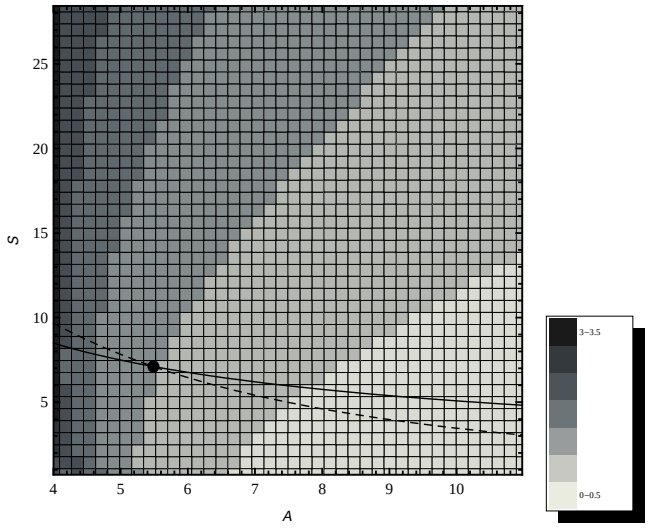

Figure 6.49: Illustration of the benefit, measured in terms of $J3$ and in which the U.S. cocaine supply is reduced by $\lambda = 4$.

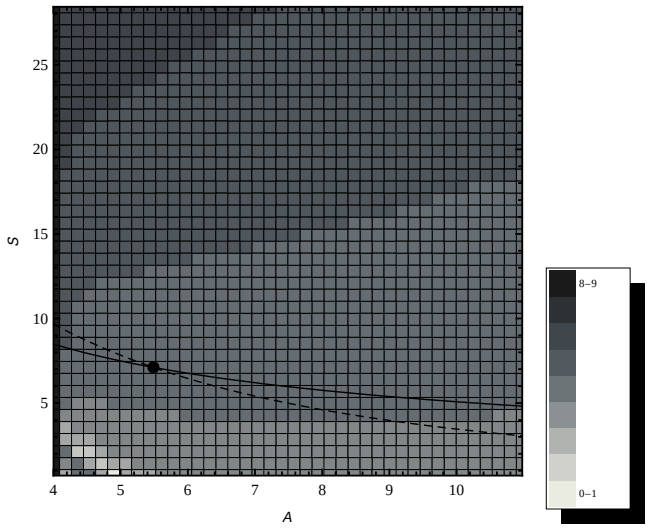

Figure 6.50: Illustration of the damage, measured in terms of $J4$ and in which the U.S. cocaine supply is reduced by $\lambda = 4$.

6.2.2 Australia

For the examinations of this chapter, so far, only the U.S. parameter values were substituted into our base model. The last two analyses, however, were carried out also for the Australian IDU epidemic. Figures 6.51-6.64 summarize the results for the cases $\lambda = 0.05$, $\lambda = 0.1$, and $\lambda = 0.2$. The conclusions we can draw are qualitatively the same as those for the U.S. cocaine epidemic.

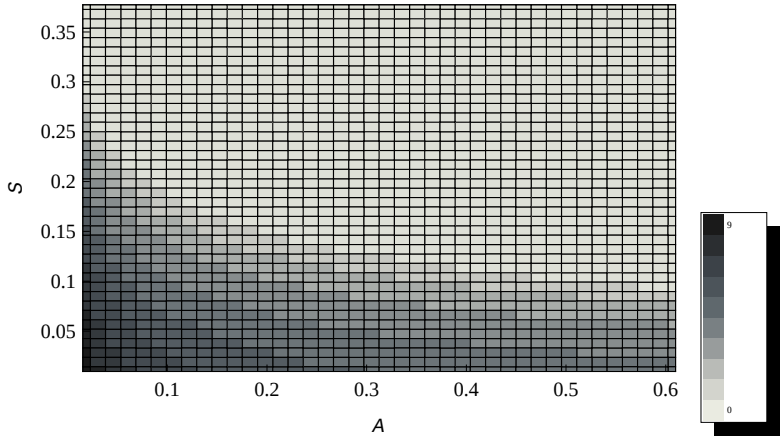

Figure 6.51: Years with the strongest effect, when the Australian ID-supply is reduced by 50% and $C(0) = A(0)$.

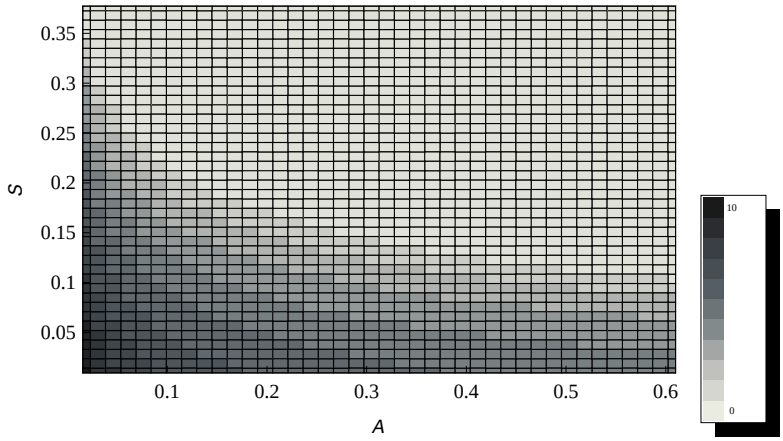

Figure 6.52: Years with the strongest effect, when the Australian ID-supply is reduced by 50% and $C(0) = \frac{A(0)}{2}$.

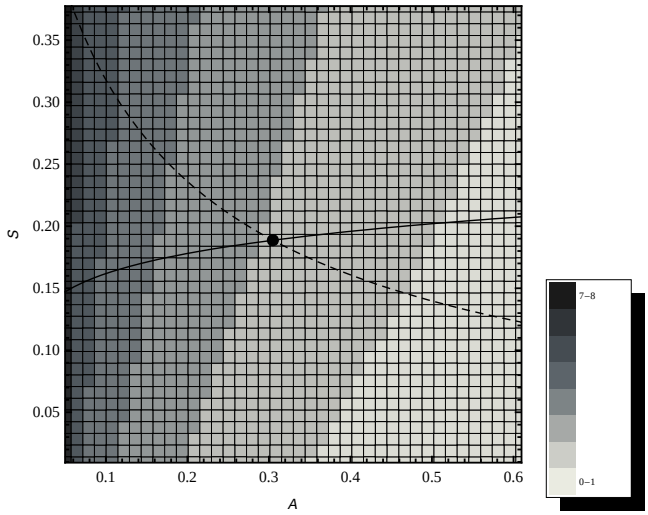

Figure 6.53: Illustration of the benefit, measured in terms of $J1$ and in which the Australian ID-supply is reduced by $\lambda = 0.05$.

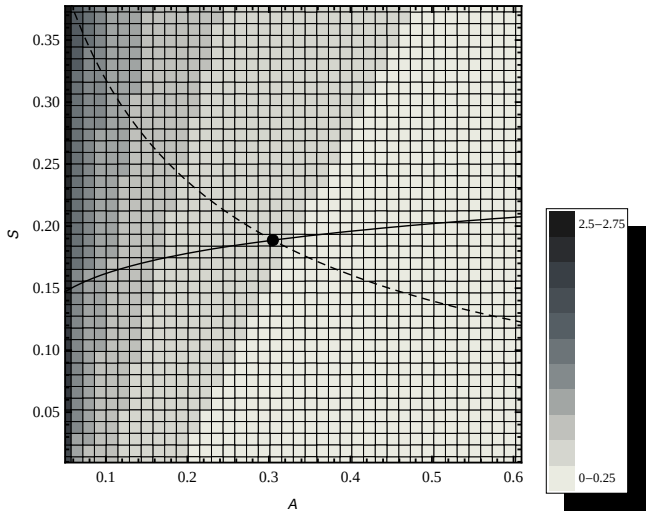

Figure 6.54: Illustration of the benefit, measured in terms of $J2$ and in which the Australian ID-supply is reduced by $\lambda = 0.05$.

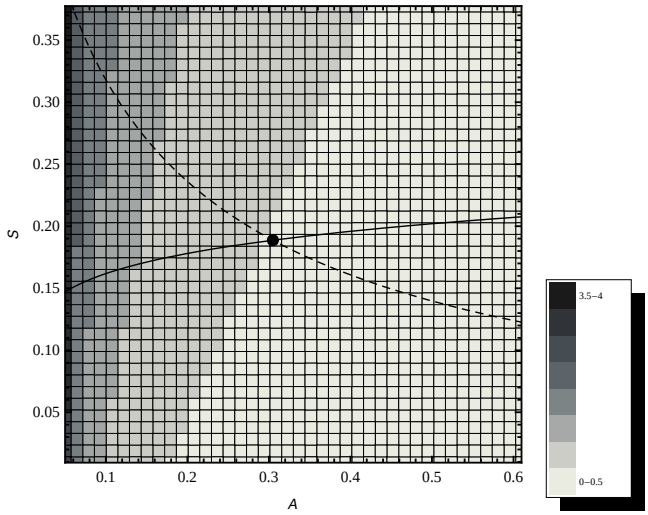

Figure 6.55: Illustration of the benefit, measured in terms of $J3$ and in which the Australian ID-supply is reduced by $\lambda = 0.05$.

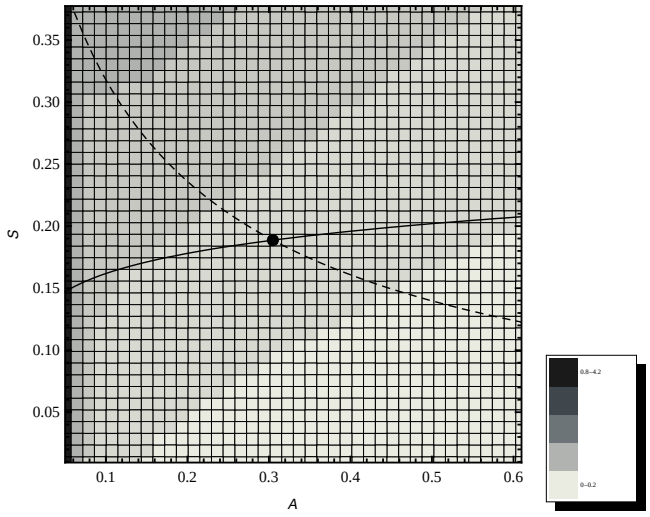

Figure 6.56: Illustration of the damage, measured in terms of $J4$ and in which the Australian ID-supply is reduced by $\lambda = 0.05$.

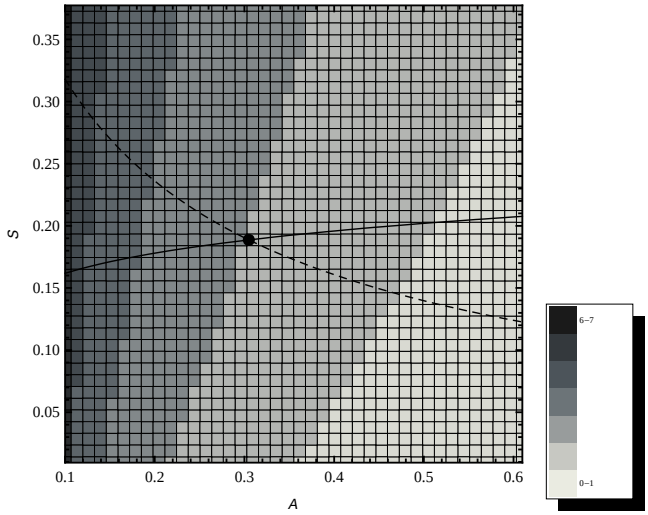

Figure 6.57: Illustration of the benefit, measured in terms of $J1$ and in which the Australian ID-supply is reduced by $\lambda = 0.1$.

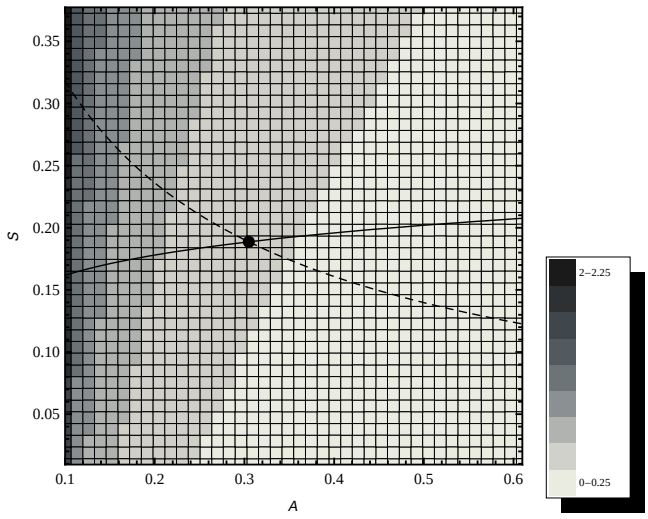

Figure 6.58: Illustration of the benefit, measured in terms of $J2$ and in which the Australian ID-supply is reduced by $\lambda = 0.1$.

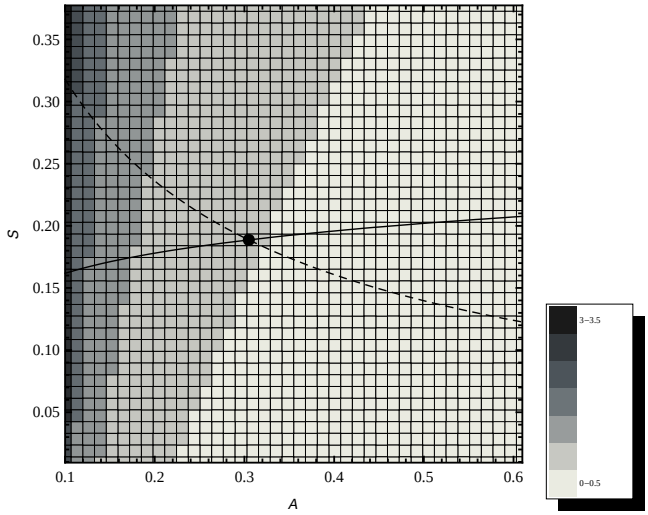

Figure 6.59: Illustration of the benefit, measured in terms of $J3$ and in which the Australian ID-supply is reduced by $\lambda = 0.1$.

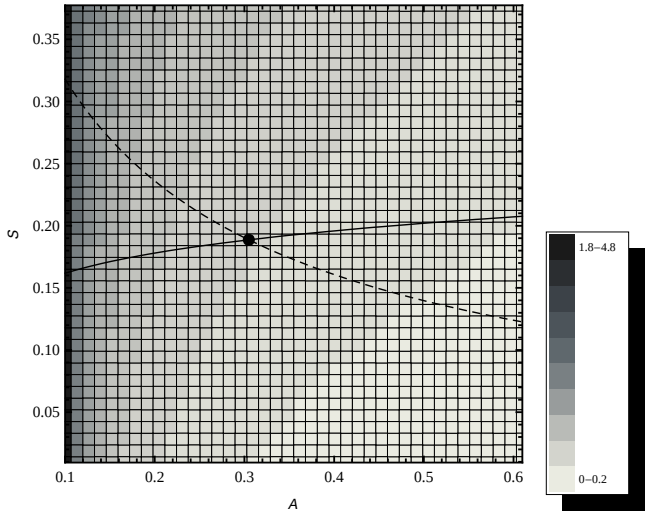

Figure 6.60: Illustration of the damage, measured in terms of $J4$ and in which the Australian ID-supply is reduced by $\lambda = 0.1$.

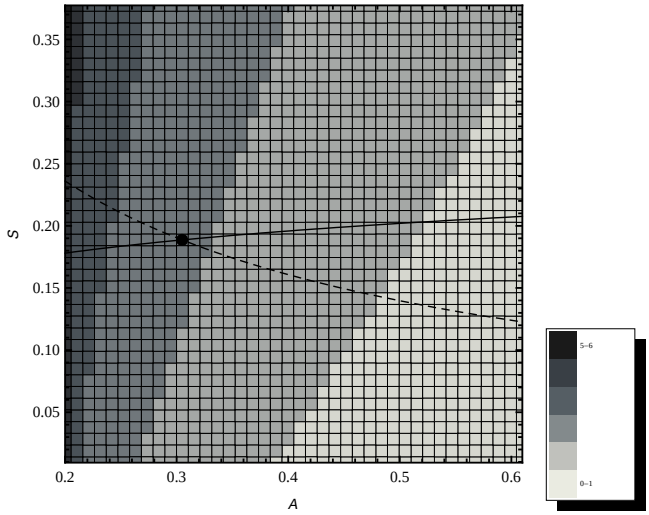

Figure 6.61: Illustration of the benefit, measured in terms of $J1$ and in which the Australian ID-supply is reduced by $\lambda = 0.2$.

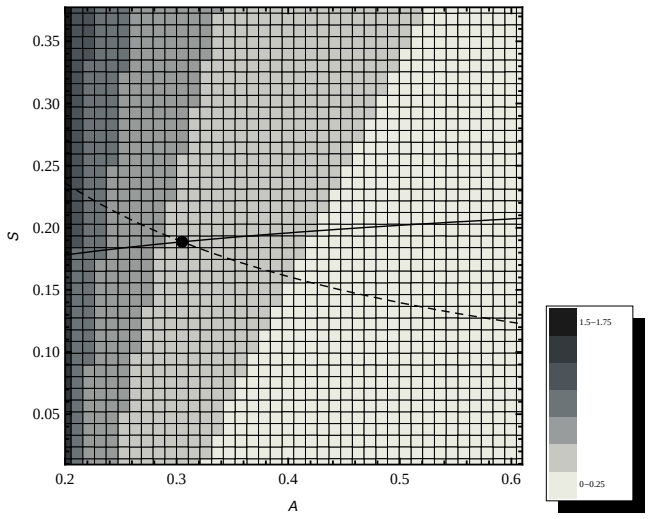

Figure 6.62: Illustration of the benefit, measured in terms of $J2$ and in which the Australian ID-supply is reduced by $\lambda = 0.2$.

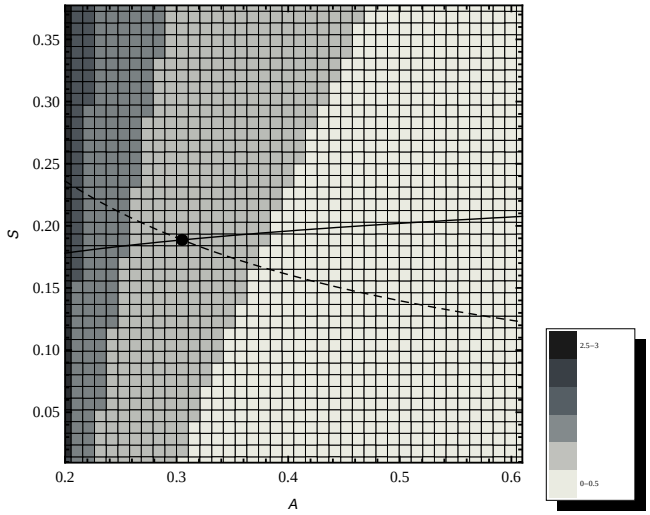

Figure 6.63: Illustration of the benefit, measured in terms of $J3$ and in which the Australian ID-supply is reduced by $\lambda = 0.2$.

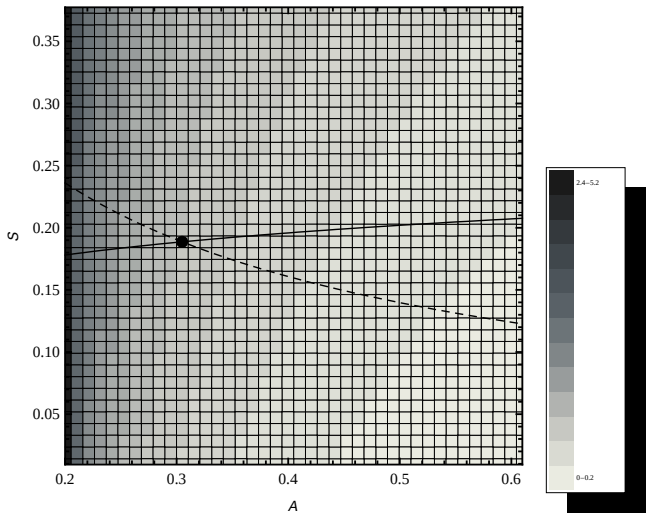

Figure 6.64: Illustration of the damage, measured in terms of $J4$ and in which the Australian ID-supply is reduced by $\lambda = 0.2$.

Chapter 7

Conclusions and Suggestions for Extensions

This thesis was devoted to a dynamic three-state model of drug epidemics derived from the well-known two-state SA model. In addition to susceptible non-users and users of a drug, also the "throughput capacity" of the drug supply network was integrated. Hence, drug supply, price, and demand got connected with each other. Examinations were carried out for two different drug epidemics in two different countries, i.e. the U.S. cocaine use and the Australian injection drug use.

The main purpose of the first part was to analyze the uncontrolled model. This means that the steady states were determined and afterwards their stability behavior was analyzed. A sensitivity and bifurcation analysis was conducted in order to deal with the problem that such base parameterizations can never be exact. Furthermore, the SAC and SA dynamics was compared with each other, which yielded some very interesting results. Having mastered this, we focused on some strategic examinations as a preliminary stage to the optimal control framework. We have interpreted an increase of the parameter μ as a successful method of treatment through which more users quit, and then the relative efficiency of treatment at different stages of a drug epidemic was investigated. Subsequently, we have also dealt with supply shocks. We looked at shocks of different size at different stages of

the epidemic, and these analyses have represented one main emphasis of this thesis. Altogether, the U.S. parameterization has provided the more interesting and more insightful results. This perhaps can be explained by $\beta > 1$.

We want to conclude this thesis by pointing to some possible extensions that may be taken into consideration in future work.

- Probably, the most important extension would be to look at the optimal control model, in which optimal drug control strategies can be derived. Our preliminary results suggest that we may expect DNSS "tipping" thresholds in an optimal control formulation. It will be particularly interesting to see how the policy recommendations change depending on which of the cost components (i.e., number of users, size of throughput capacity, amount of drugs used, amount of money spent on drug use) are considered in the objective functional and/or how they are weighted.

- Since in our approach the drug price p effects prevalence only through initiation, the parameter a should probably be increased from 0.25 to 0.5. Another possibility is to let also the exit from active use to depend on price.

- Further sensitivity analyses for the exponent in the initiation function, β, should be carried out. Some other hypothetical parameter scenarios (e.g., $\beta = 1.25$ or $\beta = 1$) could be considered.

- We chose the logarithm as one specific functional form in the equation for the throughput capacity. Other functional forms might be considered.

- Chapter 5 leaves a lot of room for further examinations. For instance, what is the closest to base case situation for which SAC trajectories look very different than SA trajectories? How small could a supply shock be so that SAC still looks different than SA? What parameter values tend to make the C-state more important in the sense of making SAC look different from SA?

- An extension of the cost-effectiveness-analyses in Chapter 6 seems worthwhile. Here, the results could also be shown in terms of impact per person removed by treatment. This means that the number of users would be reduced by an absolute value λ instead of the current throughput capacity of the supply network.

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List of Figures

List of Tables

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