TECHNISCHE UNIVERSITÄT WIEN

DIPLOMARBEIT

Prevention, Treatment, and Law Enforcement in a One-State Optimal Control Model of Illicit Drug Consumption

Ausgeführt am Institut für
Stochastik und Wirtschaftsmathematik
der Technischen Universität Wien

unter der Anleitung von

Ao.Prof. Dr. Gernot Tragler

durch

Dominique Wetter
In Gruben 15, 2120 Wolkersdorf

Wien, October 2016 Dominique Wetter

Ehrenwörtliche Erklärung

Ich erkläre ehrenwörtlich, dass ich die vorliegende Arbeit selbständig und ohne fremde Hilfe verfasst, andere als die angegebenen Quellen nicht benutzt und die den Quellen wörtlich oder inhaltlich entnommenen Stellen als solche kenntlich gemacht habe. Die Arbeit wurde bisher in gleicher oder ähnlicher Form keiner anderen inländischen oder ausländischen Prüfungsbehörde vorgelegt und auch noch nicht veröffentlicht. Die vorliegende Fassung entspricht der eingereichten elektronischen Version.

Danksagung

Ein großes Dankeschön geht an

... meinen Betreuer, Ao.Prof. Dr. Gernot Tragler, für die besonders tolle und kompetente Betreuung meiner Diplomarbeit, der Zweifel stets mit neuen Ideen beseitigen konnte und mit seinen Vorlesungen die besonders spannende Seite der Mathematik aufzeigte.

... Dieter Grass, für die Entwicklung von OCMat und der tollen Hilfestellung zu Matlab.

... meine Familie, für die jahrelange Unterstützung und Beistand.

... meine Studienkollegen, mit denen ich auf eine schöne gemeinsame Studienzeit zurückblicken darf.

... meine Professoren, die mir geholfen haben, meinen Horizont zu erweitern.

Abstract

A central question in drug policy is how control efforts should be divided among enforcement, treatment, and prevention. For this regard, Caulkins and Tragler [11] developed a model to study how the mix of control efforts should vary dynamically over the course of an epidemic. In this thesis, the initiation function of the model of Caulkins and Tragler [11] is changed to a logistic growth function and analysed for comparison. Due to some necessary adjustments of the parameters, a precise comparison turns out to be hardly possible. The results of the different sensitivity analyses regarding some parameters and their comparison between both models are presented.

Keywords: Dynamic programming/optimal control: applications to drug policy; Judicial/legal: crime and drug policy. Model comparison: power and logistic function.

Contents

1 Introduction

The war on drugs has always been prominent. A new drug law in Austria was released in June 2016, after realising that the one before (only to be in order since 01.01.2016) contained a loophole, which small dealers could use to avoid getting imprisoned with the implication that they were disturbed only minimally by the police while making their business (derStandard.at [13]). This caused more illegal activities, and the residents did not feel safe anymore and filed complaints. Even local pub and club owners were affected (wien.orf.at [54]) by an image loss, while at the same time tobacconists described a decline in sales (derStandard.at [14]). As a result, the state tried to quickly change this situation.

However, cities can never fully get rid of drugs, their users, and dealers, so some states try something different. Portugal, for example, tests the approach of decriminalizing drugs. Note that there is a great difference between legalisation and decriminalisation. In Portugal, this resulted in less new HIV-infections as well as less deaths caused by drugs, while the number of heroin-dependent users was reduced by about a half (derStandard.at [15]).

Rodrigo Duterte, the president of the Philippines, tries to eradicate drug use with brutal measures. He called out people to kill drug addicts (theguardian [43]) and also offered security officials bounties for the bodies of drug dealers, and he has repeatedly pledged to protect police from prosecution over the killings (theguardian [44]).

Illicit drugs impose enormous costs on society (Harwood et al. [22], UNODC [50]), and there is considerable debate over how policy makers should respond. While the measures above from Portugal and the Philippines are extreme, they show exemplarily that there are many different approaches of trying to get control over drug epidemics. In the US there are so called hawks, doves, and owls. Hawks try to eradicate the drug use by making the drugs more difficult to obtain. Doves want to deal more with the demand for drugs, that is, with the suffering of drug addicts, or potential addicts as such. The approach of owls emphasizes well-thought out research that is conducive to a peace treaty in the war on drugs, by reconstructing the perception of the

drug addict as a patient rather than as a criminal (Rogov [34]). Therefore, a central question concerns the relative roles of three broad strategies (i.e., enforcement, treatment, and prevention) with a focus on health and carrying them out with respect to human rights.

Prevention, e.g., not only means to prevent people from using drugs by showing them the possible harm of drugs but also can mean preventing users from getting harmful drugs. There are different programs for this matter. A prominent one in Austria is called "checkit!", where chemists come to events (parties, clubs, festivals, etc.) and offer a free analytical drug check for the ingredients, as drugs are often laced with other chemicals.

Enforcement here refers to actions taken against the drug supply chain that raise the cost of producing and distributing drugs and thereby increase retail prices (e.g., inhibiting drug trafficking) (Caulkins and Tragler [11]).

Treatment is meant to help drug dependent individuals to halt their consumption, prevent relapse, reduce their involvement in crime, change other dysfunctional behavior, and make a positive contribution to their family and community. There are many kinds of treatment, and they are of varying quality (Institute of Medicine [24],[25]). Hence, we model treatment somewhat abstractly as simply increasing the net quit rate and ignore the possibility that it might reduce the social damage per unit of consumption (Caulkins and Tragler [11]).

The key term of this present master thesis is the dynamic view of drug epidemics. For this purpose, we use a drug model developed by Caulkins and Tragler [11], change the initiation to a logistic growth function and compare the results.

2 Drugs and Addiction

2.1 Basic Definitions and Facts About Some Common Drugs

2.2 Addiction

Addiction can be described from different points of view like the medical, social, psychological, or other views. The definitions differ from each other but have one common ground - the aspect of compulsion (Uchtenhagen and Zieglgänsberger [49]).

Compulsion according to MediLexicon's Medical Dictionary [40]:
Uncontrollable thoughts or impulses to perform an act, often repetitively, as an unconscious mechanism to avoid unacceptable ideas and desires that, by themselves, arouse anxiety; the anxiety becomes fully manifest if performance of the compulsive act is prevented; may be associated with obsessive thoughts.

Some substance addictions can be caused by a physiological basis, as some substances become part of the cell metabolism so the withdrawal of the drug after short- or long-term chronic use can cause unpleasant physical symptoms [49]. The intensity of those depend on the inner and outer situation of the affected, which shows the great prominence of the psychological component of every addiction [49].
Besides substance addictions, there are also non-substance-related addictions like behavioral ones, which are forms of addiction that involve a compulsion to engage in a rewarding non-drug-related behavior - sometimes called a natural reward despite any negative consequences to the person's physical, mental, social, or financial well-being [55] (e.g., media-addiction).

2.3 Types of Drugs

A drug is any substance (with the exception of food and water) which, when taken into the body, alters the body's function either physically and/or psychologically. Drugs may be legal (e.g., alcohol, caffeine, or tobacco) or illegal (e.g., cannabis, ecstasy, cocaine, or heroin). Psychoactive drugs affect the central nervous system and alter a person's mood, thinking, and/or behavior. Psychoactive drugs may be divided into four categories [16]:

- Depressants: Drugs that decrease alertness by slowing down the activity of the central nervous system (e.g., heroin, alcohol, or analgesics).
- Stimulants: Drugs that increase the body's state of arousal by increasing the activity of the brain (e.g., caffeine, nicotine, or amphetamines).
- Hallucinogens: Drugs that alter perception and can cause hallucinations, such as seeing or hearing something that is not there (e.g., LSD and 'magic mushrooms').
- Other: Some drugs fall into the 'other' category, as they may have properties of more than one of the above categories (e.g., cannabis has depressive, hallucinogenic, and some stimulant properties).

2.4 Classification of Drugs by Possible Harm

Drugs can cause different levels of harm, like the possibility of an addiction but also the risk of a lethal dose. Mixing drugs more often leads to a fatal overdose, especially combining cocaine with heroin or morphine, which is called 'Speedballing' (Harm Reduction Coalition [21]).

The classification of risk per drug through evidence-based medicine is difficult (Wikipedia [56]). In 2010, members of the Independent Scientific Committee on Drugs, including two invited specialists, met in a 1-day interactive workshop to score 20 drugs based on 16 criteria: nine related to the harms that a drug produces in the individual and seven to the harms

caused to others. Drugs were scored out of 100 points, and the criteria were weighted to indicate their relative importance (Nutt et al. [31]).

The harms to the individual were (NHS Choices [2]):

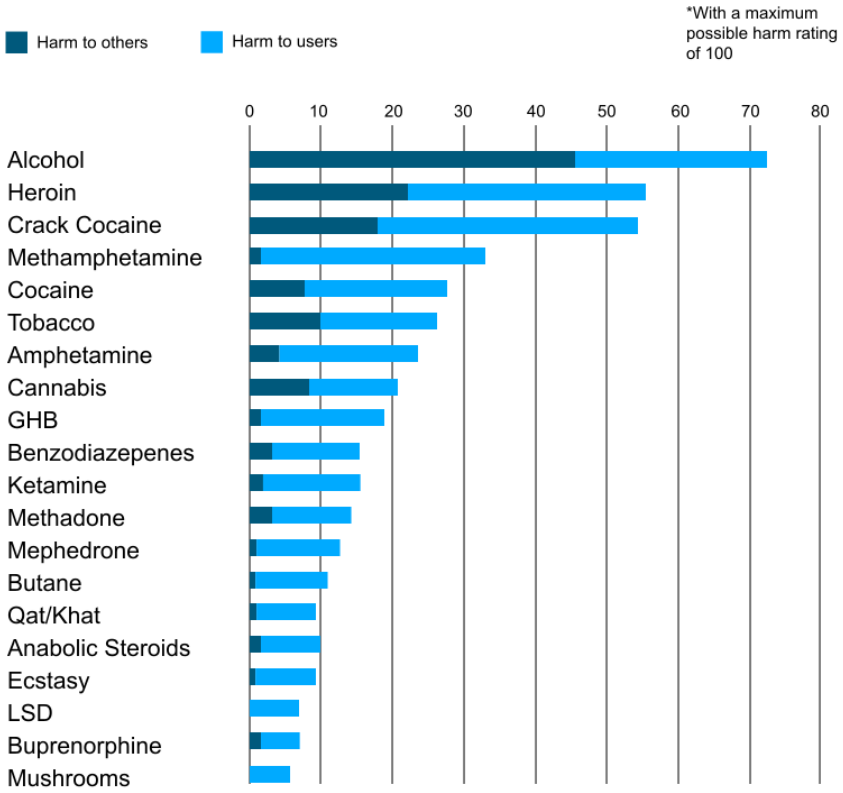
- drug-specific mortality
- drug-related mortality
- drug-specific damage
- drug-related damage
- dependence
- drug-specific impairment of mental functioning
- drug-related impairment of mental functioning
- loss of tangibles (income, housing, job, etc.)
- loss of relationships

The harms to others were (NHS Choices [2]):

- injury
- crime
- environmental damage
- family adversities
- international damage
- economic cost
- community

When the scores of individual harm and the harm to others were combined, alcohol was the most harmful drug, scoring 72 out of 100. This was followed by heroin (55) and crack cocaine (54). These were also the three most harmful drugs to others: alcohol (46), heroin (21), and crack cocaine (17). Crack cocaine (37), heroin (34), and metamphetamine (32) were considered the most harmful to individual users, in that order (NHS Choices [2]). The basic result of this study can be seen in Figure 1.

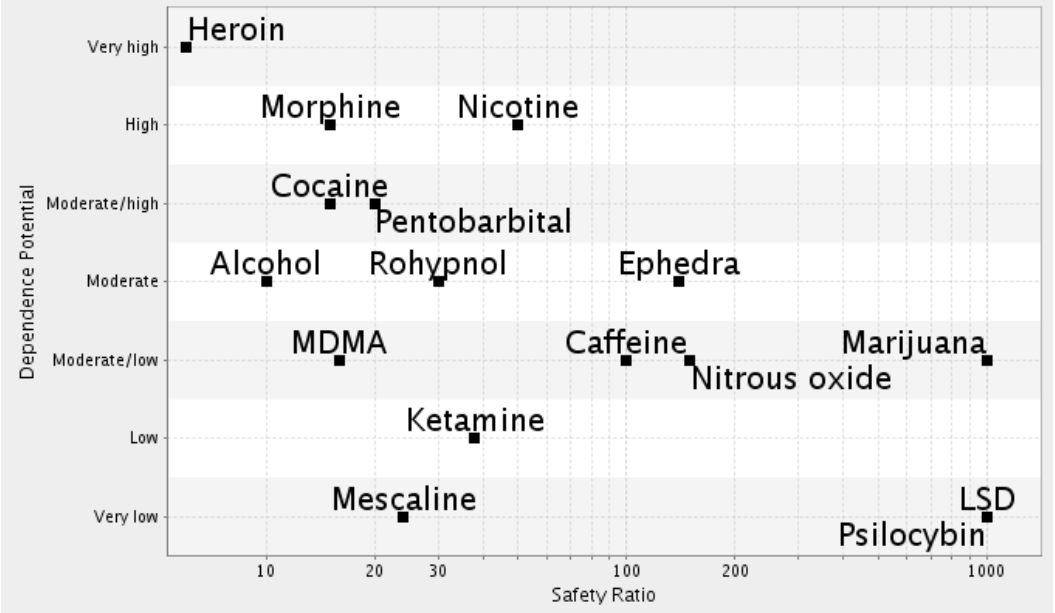
Figure 1: Harm Caused by Drugs



Source: Data by Nutt et al. [31], adapted by Tesseract2 [41].

Surprisingly, alcohol (despite being legal more often than the other drugs) is by far the most harmful; not only is it the most damaging to societies, it is also the fourth-most dangerous for the user. Most of the drugs were rated significantly less harmful than alcohol, with most of the harm befalling the user (Tesseract2 [41]). The high value of alcohol in the criteria harm to others can partly be explained by the high availability and the widespread tolerated consumption of alcohol by society. Other drugs with high overall harm rating have a much higher harm-to-user rating than alcohol, probably because of the higher dependence potential of drugs such as heroin, cocaine, and others, see Figure 2 (Wikipedia [56]) .

Figure 2: Safety Ratio and Dependence Potential of Psychoactive Drugs



Source: Gable [19], adapted by Thundermaker [46].

Note that the researchers highlighted the following limitations to their approach (NHS Choices [2]):

- They only considered harms, and say that some drugs do have benefits that may offset some harms (for example, the commercial benefits to society of the tobacco and alcohol industries).
- They note that their results may not be relevant to countries with different legal and cultural systems to the UK.
- They did not include prescription drugs.
- They did not investigate the harms associated with use of more than one drug or substance (for example, alcohol plus recreational drugs).

The bottom line about the harms of drug use of this research is the finding that heroin, crack, cocaine, and metamphetamines were most harmful to

individual users, but policy makers are interested in overall harms or the harms to society. The methods to quantify these will always have some element of subjectivity and will, therefore, always be controversial. The researchers have attempted to put numbers to the wider impact of different drugs, but the fact remains that alcohol tops the list of overall harms largely because it is a legal, widely used drug (NHS Choices [2]).

2.5 Cocaine

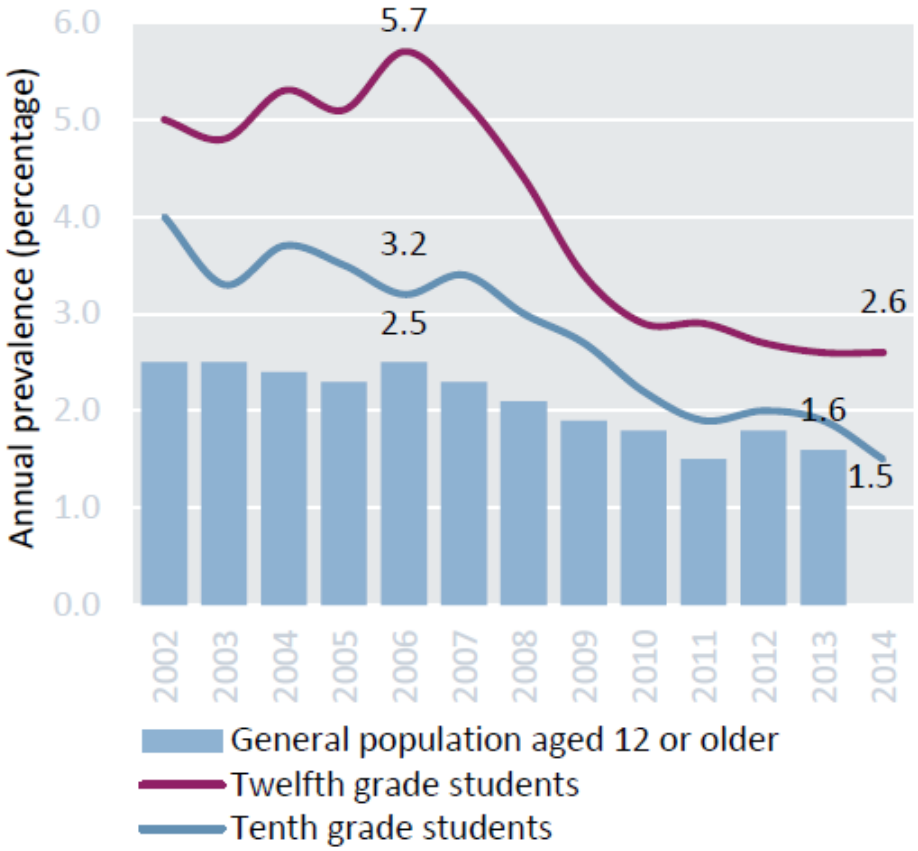
Cocaine, a natural alkaloid derived from the coca plant, is one of the most commonly abused illicit drugs. Cocaine is commonly abused by inhalation, nasal insufflation, and intravenous injection, resulting in many adverse effects. Cocaine can affect all body systems and the clinical presentation may primarily result from organ toxicity. Among the most severe complications are seizures, hemorrhagic and ischemic strokes, myocardial infarction, aortic dissection, rhabdomyolysis, mesenteric ischemia, acute renal injury, and multiple organ failure (Zimmerman [58]).
Dependent on the method with which it is used – e.g., smoked, snorted, or injected – cocaine can be acting quite rapidly. One of cocaine's effects in the brain is to increase dopamine release. Dopamine is a neurotransmitter that plays a role in the brain registering positive feelings, and "rewarding" the behaviors that led to those feelings to begin with. This increase of dopamine is, in part, what leads to the subjective "high" of cocaine use (DrugAbuse.com [17]).

The effect of cocaine is believed to have been recognized first by the South American indigenous people as they crossed the mountains. Their pack animals ate the coca leafs (Uchtenhagen and Zieglgänsberger [49]) and when chewed, coca acts as a mild stimulant and suppresses hunger, thirst, pain, and fatigue (TNI [45]).
Cocaine was first isolated (extracted from coca leaves) in 1859 by German chemist Albert Niemann, but it was not until the 1880s that it started to be popularized in the medical community. By 1905, it had become popular to snort cocaine and within five years, hospitals and medical literature had started reporting cases of nasal damage resulting from the use of this drug. In 1912, the United States government reported 5,000 cocaine-related deaths in one year and by 1922, the drug was officially banned. In the 1970s, cocaine emerged as the fashionable new drug for entertainers and business people. Cocaine seemed to be the perfect companion for a trip into the fast lane. It provided energy and helped people stay up. At some American universities,

the percentage of students who experimented with cocaine increased tenfold between 1970 and 1980. Traditionally, cocaine was a rich man's drug, due to the large expense of a cocaine habit. By the late 1980s, cocaine was no longer thought of as the drug of choice for the wealthy. By then, it had the reputation of America's most dangerous and addictive drug, linked with poverty, crime, and death [42].
As of 2014, cocaine is the third most trafficked drug in Europe, after herbal cannabis and cannabis resin. The global number of cocaine users estimates 17.0 million (UNODC [50]).

While there has been a decline in cocaine users over the years 2003 to 2013 in the United States (see Figure 3), the usage of cocaine in South America has increased. In 2013, the prevalence of cocaine use in the United States was estimated to be 1.6 per cent of the population aged 12 and older, and this has remained stable over the past few years, although it is still significantly lower than in 2006. Cocaine use among high-school students has been declining, with annual prevalence nearly halving since 2006, when it was reported to be 3.5 per cent, to 1.8 per cent in 2013. In South America, the annual prevalence of cocaine use was estimated to have increased from 0.7 per cent in 2010 (1.84 million users) to 1.2 per cent in 2012 (3.34 million users), three times the global estimated average level of consumption, and it remained at the same level in 2013 (UNODC [50]).

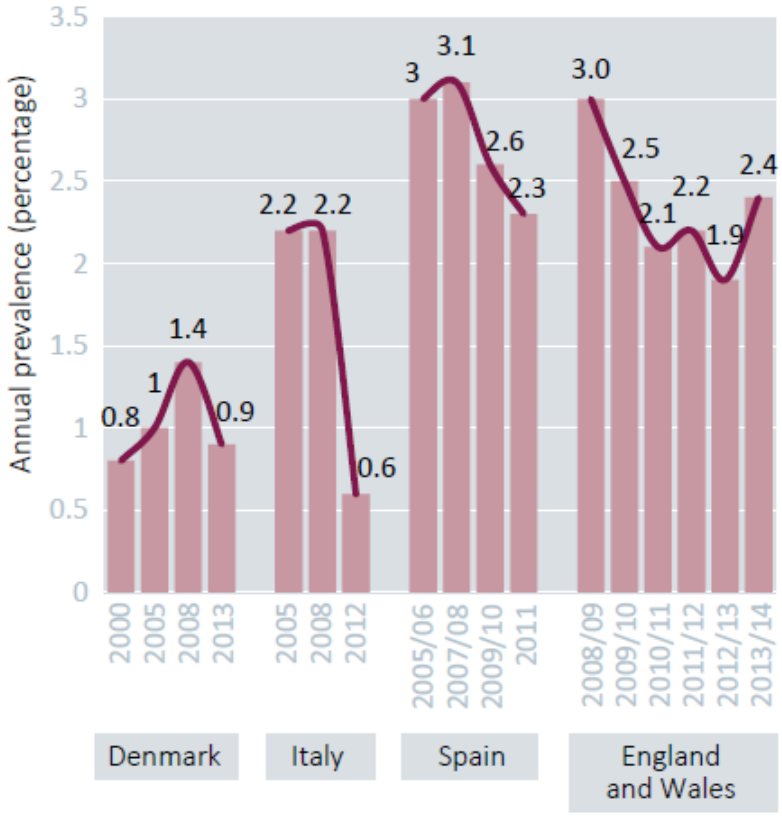
Figure 3: Cocaine Use in the United States, 2002-2013



Source: World Drug Report 2015 [50].

In Europe, the usage of cocaine remains high in the main markets of Western and Central Europe (around 1.0 per cent of the population aged 15-64). There are some signals, however, of a decreasing trend in countries with high levels of use, such as Denmark, Italy, and Spain, whereas the United Kingdom reports a rising trend in cocaine use in the past year (see Figure 4), and most of the remaining countries report stable or declining trends in cocaine use. There is also a declining trend in treatment demand for cocaine use, which may indicate a decline in the European cocaine market (UNODC [50]).

Figure 4: Trends in cocaine use in high prevalence countries in Europe



Source: World Drug Report 2015, [50].

3 Treatment, Enforcement, and Prevention

3.1 Treatment

For over four decades scientific research has shown that effective treatment for drug-use disorders has helped drug dependent individuals to halt their consumption, prevent relapse, reduce their involvement in crime, change other dysfunctional behavior, and make a positive contribution to their family and community. Effective treatment typically incorporates many components such as pharmacotherapy, behavioral therapy, and social support – each directed towards a particular aspect of the disorder and matching an individual's particular problems and needs (UNODC [50]).

With an estimated global average of one in six people who suffer from drug-use disorders or drug dependence receiving treatment each year, it is clear that the accessibility and availability of services for such conditions are limited in most countries. The fact that this figure is approximately 1 in 18 in Africa, compared with 1 in 5 in Western and Central Europe, however, points to large disparities between regions. Not included in these figures is the large proportion of drug users who may not be dependent but may still require interventions to prevent an escalation in their disability and comorbidity related to drug use (UNODC [50]).

Compared with the general population, people who inject drugs (PWID) are at an elevated risk of dying, primarily as a result of the transmission of infectious diseases, in particular HIV, and of fatal drug overdoses. A recent systematic review of cohort studies that followed PWID over time suggests that they experience a high mortality rate. The overall (pooled) mortality rate across the 65 cohort studies from 25 countries estimated a mortality rate of 2.35 deaths per 100 person-years (i.e., if 100 PWID were followed over one year, two to three deaths would be expected to occur among this group). This is a much higher level of mortality than among those of comparable age and gender among the general population (standardized mortality ratio = 14.68) (UNODC [50]). Continuity of treatment and the length of time spent in treatment can have an impact in reducing overdoses among PWID. Data from six studies showed a risk of dying some 2.5 times higher for PWID during off-treatment periods compared with in-treatment time periods (UNODC [50]).

Effective evidence-based interventions for prevention, treatment, and care of HIV for people who inject drugs include needle and syringe programmes, opioid substitution therapy, antiretroviral therapy (ART), and the availability of naloxone (UNODC [50]). ART are medications that treat HIV. The drugs do not kill or cure the virus. However, when taken in combination they can prevent the growth of the virus. When the virus is slowed down, so is HIV disease (aidsinfonet.org [1]).

The different treatment outcome domains identified as relevant to both the patient and to society include (UNODC [50]):

- Reduction in substance abuse; increase or improvement in personal health including physical and psychological improvements (including spiritual);
- Improvement in social functioning, including employment, family, and social relationships;
- Reductions in behaviors that are a threat to public health and safety or that are associated with the spread of infectious diseases or with personal and property crimes.

3.1.1 Long-Term Residential Treatment

Long-term residential treatment provides care 24 hours a day, generally in non-hospital settings. The best-known residential treatment model in the USA is the therapeutic community (TC), with planned lengths of stay of between 6 and 12 months. TCs focus on the "resocialization" of the individual and use the program's entire community – including other residents, staff, and the social context – as active components of treatment. Addiction is viewed in the context of an individual's social and psychological deficits, and treatment focuses on developing personal accountability and responsibility as well as socially productive lives. Treatment is highly structured and can be confrontational at times, with activities designed to help residents examine damaging beliefs, self-concepts, and destructive patterns of behavior and adopt new, more harmonious and constructive ways to interact with others. Many TCs offer comprehensive services, which can include employment training and other support services, onsite. Research shows that TCs can be modified to treat individuals with special needs, including adolescents, women, homeless individuals, people with severe mental disorders, and individuals in the criminal justice system (NIDA [29]).

3.1.2 Short-Term Residential Treatment

Short-term residential programs provide intensive but relatively brief treatment based on a modified 12-step approach. These programs were originally

designed to treat alcohol problems, but during the cocaine epidemic of the mid-1980s, many began to treat other types of substance use disorders. The original residential treatment model consisted of a 3- to 6-week hospital-based inpatient treatment phase followed by extended outpatient therapy and participation in a self-help group, such as AA. Following stays in residential treatment programs, it is important for individuals to remain engaged in outpatient treatment programs and/or aftercare programs. These programs help to reduce the risk of relapse once a patient leaves the residential setting (NIDA [29]).

3.1.3 Outpatient Treatment Programs

Outpatient treatment varies in the types and intensity of services offered. Such treatment costs less than residential or inpatient treatment and often is more suitable for people with jobs or extensive social supports. It should be noted, however, that low-intensity programs may offer little more than drug education. Other outpatient models, such as intensive day treatment, can be comparable to residential programs in services and effectiveness, depending on the individual patient's characteristics and needs. In many outpatient programs, group counselling can be a major component. Some outpatient programs are also designed to treat patients with medical or other mental health problems in addition to their drug disorders (NIDA [29]).

3.1.4 Individualized Drug Counselling

Individualized drug counselling not only focuses on reducing or stopping illicit drug or alcohol use; it also addresses related areas of impaired functioning such as employment status, illegal activity, and family/social relations as well as the content and structure of the patient's recovery program. Through its emphasis on short-term behavioral goals, individualized counselling helps the patient develop coping strategies and tools to abstain from drug use and maintain abstinence. The addiction counsellor encourages 12-step participation (at least one or two times per week) and makes referrals for needed supplemental medical, psychiatric, employment, and other services (NIDA

[29]).

3.1.5 Group Counselling

Many therapeutic settings use group therapy to capitalize on the social reinforcement offered by peer discussion and to help promote drug-free lifestyles. Research has shown that when group therapy either is offered in conjunction with individualized drug counselling or is formatted to reflect the principles of cognitive-behavioral therapy or contingency management, positive outcomes are achieved. Currently, researchers are testing conditions in which group therapy can be standardized and made more community-friendly (NIDA [29]).

3.2 Enforcement

Enforcement here refers to actions taken against the drug supply chain that raise the cost of producing and distributing drugs and thereby increase retail prices (e.g., inhibiting drug trafficking) (Caulkins and Tragler [11]).

3.3 Prevention

Substance abuse prevention, also known as drug abuse prevention, is a process that attempts to prevent the onset of substance use or limit the development of problems associated with using psychoactive substances. Prevention efforts may focus on the individual or their surroundings. A concept known as "environmental prevention" focuses on changing community conditions or policies so that the availability of substances is reduced as well as the demand.

Substance abuse prevention efforts typically focus on minors – children and teens. Substances typically targeted by preventive efforts include alcohol (including binge drinking, drunkenness, and driving under the influence), tobacco (including cigarettes and various forms of smokeless tobacco), marijuana, inhalants (volatile solvents including among other things glue, gasoline, aerosols, ether, fumes from correction fluid and marking pens), co-

caine, methamphetamine, steroids, club drugs (such as MDMA), and opioids (Wikipedia [57]).

3.3.1 Structural Prevention

The environment of teenagers influences the probability to get in contact with psychoactive substances and so to try and later consume them frequently. Therefore different measures are used to create an environment that protects the teenagers and also minimizes the probability to consume psychoactive substances. These activities are called Structural Prevention and include actions to reduce the availability of substances, special measures of youth protection and health promotion, and measures in other areas that increase the living conditions and so indirectly the health of teenagers (Weigl et al. [53]).

3.3.2 Universal Addiction Prevention

Universal prevention measures are aimed at people who, as an overall group, display an average risk of later substance abuse (e.g., the general population, school classes). Parental training and family programmes, in particular with regard to alcohol use, are to be recommended as an effective universal approach in the family setting. Proven universal school-based prevention programmes to prevent alcohol misuse include alcohol-specific, behavioral interventions as well as specific life skills programmes and a classroom-based behavior management programme (Bühler et al. [6]). A gender-specific approach is especially used in eating disorder prevention programs but also for other forms of addiction (Weigl et al. [53]). Specific effective universal approaches in the leisure/recreational setting (e.g., sports clubs, nightlife, peer, and mentoring programmes) have still not been identified. In this regard, relatively general reference must still be made to high-quality programmes to improve personal and social skills, implemented in a non-school setting. With regard to (mass) media interventions, there is now evidence for the effectiveness of internet- and computer-based universal prevention programmes. Systematic cooperation between community stakeholders and

the implementation of local alcohol regulations could increase effectiveness in this area. Studies published since 2004 suggest that tobacco and alcohol control strategies that raise prices for alcohol and tobacco products; lead to increased controls and sanctions on sales of tobacco and alcohol to minors; impose restrictions on alcohol advertising; or curtail opportunities to smoke through smoking bans are effective (Bühler et al. [6]).

3.3.3 Selective Addiction Prevention

Selective measures are aimed at people who, as a group, display an above-average risk of later substance abuse (e.g., children from families affected by addiction, children with behavioral problems, students, patients in hospitals). Some measures (Weigl et al. [53]):

- In the family: supervision and assistance of first-time parents by midwives; life skills training for children displaying problem behaviors and their parents; family programmes for families affected by addiction (alcohol).
- In schools: life skills programmes with additional indicated elements for older adolescents (16-20 years of age, alcohol) who have an individual high risk of illicit drug use.
- In colleges: personal, brief interventions, online and computer-based feedback and normative feedback, web-based programmes, gender-specific expectancy-challenge interventions, multicomponent approaches consisting of providing information, motivational interviewing and feedback (alcohol).
- In leisure/community settings: mentoring programmes with teenagers (alcohol), multicomponent projects in the family and leisure settings with case manager (alcohol, illicit drugs).
- In healthcare settings: personal, brief interventions in the hospital setting (alcohol, cannabis).

People with a migration background are more prone for an addiction, as the migration itself can be a traumatizing event which can lead to addictive behavior. For the purpose of selective addiction prevention, especially the migrants whose social factors lead to great vulnerability and cannot be reached sufficiently with universal prevention, should be the target group (Weigl et al. [53]).

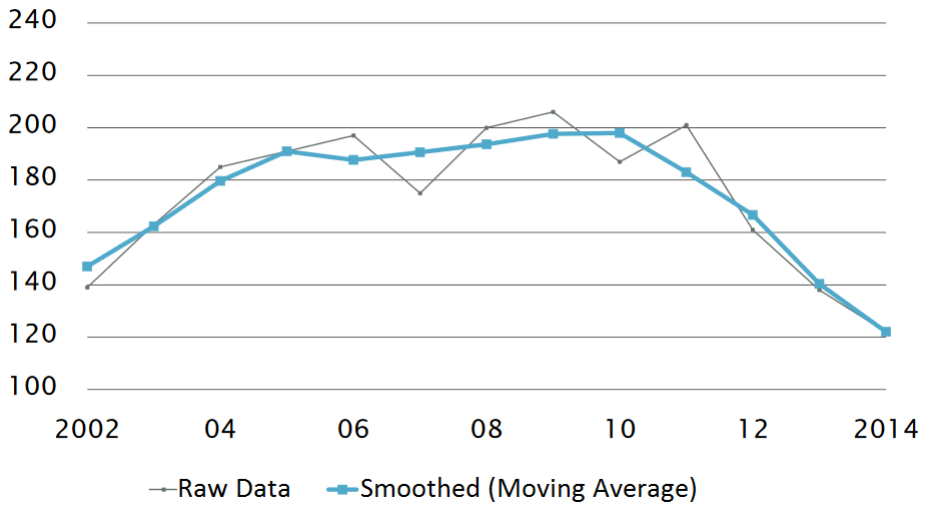
3.3.4 Indicated Addiction Prevention

Indicated addiction prevention has the goal to prevent the transition from drug use to drug abuse, where alcohol consuming teenagers are the most common target groups. Its goal is to early detect substance use, before it shows signs of addiction, and start intervening (Weigl et al [53]). In Austria, one of the programs for indicated addiction prevention in schools is called 'Step by Step', which was developed in cooperation with addiction prevention offices from Switzerland, Vorarlberg (Austrian province) and Liechtenstein. Its motto is 'Helfen statt Strafen', which means 'help instead of punishment' (Österreichische Arbeitsgemeinschaft Suchtvorbeugung [33]).

4 Situation in Austria

The drug and addiction policy in Austria is defined through laws and regulations and also strategies and concepts by the provinces. The goal is to minimize addiction in society, which is perceived as a disease. The drug/addiction strategies of the provinces mostly follow the path of differing the forms of addiction of different drugs. The total amount of public expenditures for drugs and addiction in Austria is unknown, as they are often not labelled as such (Weigl et al. [53]).

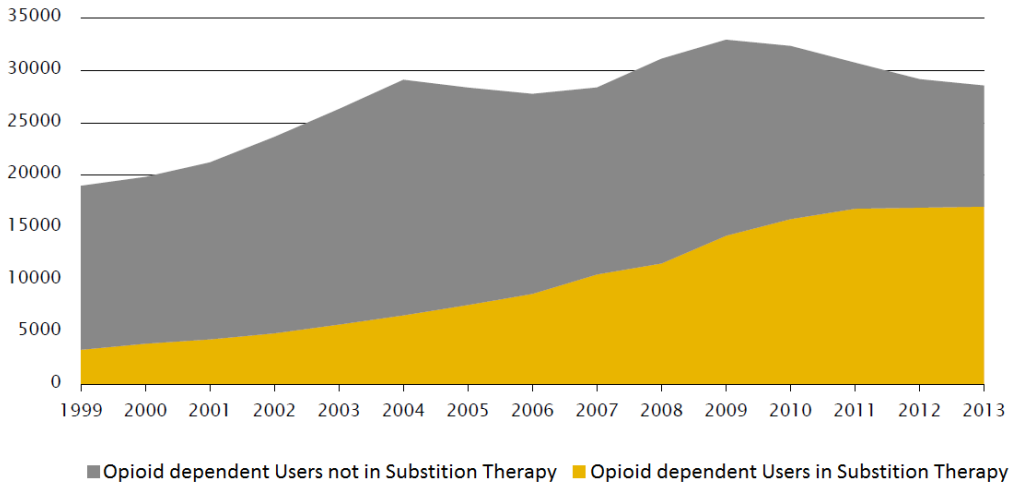
Figure 5: Drug-related Deaths in Austria, 2002-2014



Source: Bericht zur Drogensituation 2015, [53].

In 2014, there have been 122 drug-related deaths due to overdose in Austria (Figure 5). 83% of the deaths have been the result of mixing opiates with alcohol, psychotropics, or other substances. In only 10% of the cases, exclusively opiates were detected in the autopsy. So by far the most drug-related deaths were due to combined drug intoxication with opiates in the mix (Weigl et al. [53]).

Figure 6: Opioid Dependent Users (in Substitution Therapy) in Austria, 1999-2013

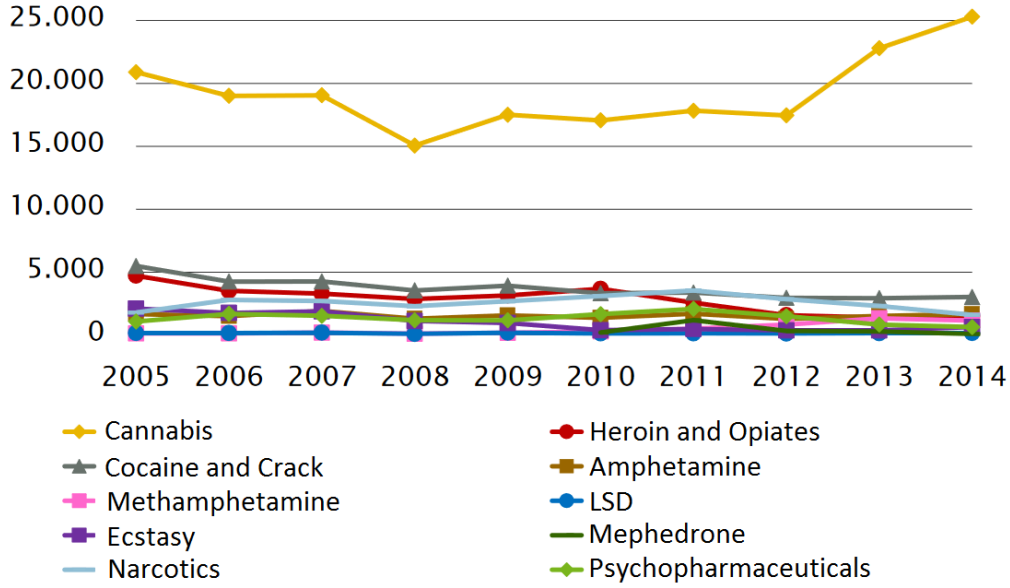


Source: Busch et al., [4].

About two-thirds of the approximately 28,000 to 29,000 opioid users in 2013 received treatment (Figure 6). The percentage of opioid users in substitution therapy differs per region and is about 60 percent in Austria overall. Substitution therapy is by far the most common treatment in austria for opioid users. Figure 6 shows that the amount of people receiving treatment has grown very fastly. Since the year 2000 the estimated opioid users only grew by about 50%, while the number of people in substitution therapy since then has grown to 5 times the amount up to 17,272 people in 2014 (Busch et al. [4]).

Figure 7 shows the trend of drug law violations per drug or drug type. While there was a continuous decline from 2005 to 2013 for most drugs, the number of drug law violations significantly increased from 2013 to 2014. For cannabis there was already a big rise in numbers from 2012 to 2013. The most striking growth from 2013-2014 had ecstasy with +70%, while cannabis, heroin and opiates, and amphetamines grew by about ten percent.

Figure 7: Drug Law Violations in Austria, 2005-2014



Source: Bericht zur Drogensituation 2015, [53].

The growth of ecstasy broke the year-long trend of decreasing numbers of drug law violations, which is because of the high number of police checks in 2014. Cannabis holds about 80% of all drug law violations in 2014 (Weigl et al. [53]).

Summary of the drug situation in Austria for 2014 (Busch and Weigl [5]):

- Most of the users in treatment are opioid-dependent users, like it has been in the past.
- The rate of opioid users in substitution therapy has grown continuously over the past years.
- The number of opioid users is decreasing continuously (especially the number of young adults).
- There is no sign of a future change in the trend from opioid to methamphetamine, mephedrone, or new psychopharmaceuticals.

- The number of drug-related deaths is continuously decreasing.
- The infection rate of users with intravenous drug use is very high for Hepatitis C but low for HIV.

5 The Models and Their Mathematical Formulation

5.1 The Base Model

As base model we use the one developed by Caulkins and Tragler [11] as described in what follows.

If we let $u(t)$, $v(t)$, and $w(t)$ denote treatment, enforcement, and prevention spending, respectively, then the discussion in Caulkins and Tragler [11] suggests the following formulation:

$$\min_{\{u(t),v(t),w(t)\}} J = \int_0^\infty e^{-rt} \left(\kappa\theta A(t) p(A(t), v(t))^{-\omega} + u(t) + v(t) + w(t)\right) dt$$

subject to

$$\dot{A}(t) = kA(t)^\alpha p(A(t), v(t))^{-a} \Psi(w(t)) - c\beta(A(t), u(t))A(t) - \\ -\mu p(A(t), v(t))^b A(t)$$

and the non-negativity constraints

$$u(t) \geq 0, v(t) \geq 0, w(t) \geq 0,$$

where

J = the discounted weighted sum of the costs of drug use and control,
r = the time discount rate,
κ = the social cost per unit of consumption,
θ = per capita rate of consumption at baseline prices,
$A(t)$ = the number of users at time t,
$p(A(t), v(t))$ = the retail price,
ω = absolute value of the short-run price elasticity of demand,
k = constant governing the rate of initiation,
α = exponent governing concavity of contagious aspect of initiation,
a = absolute value of the elasticity of initiation with respect to price,
$\Psi(w(t))$ = proportion of initiation remaining after prevention,
c = treatment efficiency proportionality constant,

$\beta(A(t), u(t))$ = outflow rate due to treatment,
μ = baseline per capita rate at which users quit without treatment, and
b = elasticity of desistance with respect to price.

As in Tragler et al. [48], treatment's increment to the per capita outflow rate is assumed to be proportional to treatment spending per capita raised to an exponent (z) that reflects diminishing returns, with a small constant in the denominator (δ) to prevent division by zero:

$$\beta(A(t), u(t)) = \left(\frac{u(t)}{A(t) + \delta}\right)^z.$$

We model enforcement's effect on price as in Caulkins et al. [7] and Tragler et al. [48]:

$$p(A(t), v(t)) = d + e\frac{v(t)}{A(t) + \epsilon},$$

where d and e are positive constants and ϵ is an arbitrarily small constant that avoids division by zero.

Following Behrens et al. [3], we model prevention as reducing initiation by a certain proportion. That proportion increases with prevention spending but at a decreasing rate because of diminishing returns. Specifically, we model

$$\Psi(w(t)) = h + (1 - h)e^{-mw(t)}$$

for positive constants h and m.

5.1.1 Pontryagin's Maximum Principle

The following application of Pontryagin's Maximum Principle for this model is taken from Caulkins and Tragler [11] with some small details changed or information added by the author.

The model cannot be solved analytically completely, but we will describe the derivation of the necessary optimality conditions according to Pontryagin's maximum principle (cf. Grass et al. [20], Feichtinger and Hartl

[18], Leonard and Long [26]). For simplicity, the time argument t will mostly be omitted from now on and we abbreviate the functions $\beta(A(t), u(t))$, $p(A(t), v(t))$, and $\Psi(w(t))$ by β, p, and Ψ, respectively.

The current value Hamiltonian H for the problem is given by

$$H = -(\kappa\theta A p^{-\omega} + u + v + w) + \lambda(kA^{\alpha}p^{-a}\Psi - c\beta A - \mu p^{b}A),$$

where λ describes the current-value costate variable.

It is not necessary to formulate the maximum principle for the Lagrangean, which incorporates the non-negativity constraints for the controls, since u, v, and w all turn out to be positive in the analysis described in Caulkins and Tragler [11].

According to Pontryagin's maximum principle we have the following three necessary optimality conditions:

$$u = \arg\max_{u} H,$$

$$v = \arg\max_{v} H,$$

and

$$w = \arg\max_{w} H.$$

A sufficient condition for H to be concave with respect to the controls (u, v, w) is the negativity of the costate variable, i.e.:

$$\lambda < 0 \Rightarrow H_{uu}, H_{vv}, H_{ww} < 0$$

(for proof see A.1). Due to the concavity of the Hamiltonian with respect to all three controls (u, v, w), setting the first-order partial derivatives equal to zero leads to the unrestricted extremum. These equations allow one to describe u and w as functions of v and A, so the solutions are described in terms of phase portraits in the A-v plane.

$$H_u = 0 \Rightarrow \qquad \lambda = \frac{-1}{c\beta_u A}, \tag{1}$$

$$H_w = 0 \Rightarrow \qquad \lambda = \frac{1}{kp^{-a}A^{\alpha}\Psi_w}, \tag{2}$$

$$H_v = 0 \Rightarrow \lambda = \frac{1 - \kappa\theta\omega p^{-\omega-1}p_v A}{-akp^{-a-1}p_v A^{\alpha}\Psi - \mu bp^{b-1}p_v A}. \tag{3}$$

Note that subscripts denote the derivatives w.r.t. the corresponding variables.

The concavity of the maximized Hamiltonian with respect to the state variable, however, cannot be guaranteed, so the usual sufficiency conditions are not satisfied.

With Equations (1)-(3) we can describe u, w, and λ as functions of A and v as follows:

$$\begin{aligned} \lambda(A,v) &:= \frac{\frac{p_v}{p}\left(\frac{a}{m} + \kappa\theta\omega p^{-\omega}A\right) - 1}{ahkp^{-a-1}p_v A^{\alpha} + \mu b p^{b-1} p_v A}, \\ u(A,v) &:= \left(\frac{-(A+\delta)^z}{czA\lambda(A,v)}\right)^{\frac{1}{z-1}}, \\ w(A,v) &:= \frac{1}{m}\ln\big((h-1)kmp^{-a}A^{\alpha}\lambda(A,v)\big). \end{aligned} \tag{4}$$

Due to this simplification we can concentrate on the two variables A and v. To gain an equation for $\dot{v}$ we differentiate λ with respect to time:

$$\dot{\lambda} = \lambda_A \dot{A} + \lambda_v \dot{v}. \tag{5}$$

Setting (5) equal to the costate equation

$$\dot{\lambda} = r\lambda - H_A,$$

yields:

$$\dot{v} = \frac{r\lambda - H_A - \lambda_A \dot{A}}{\lambda_v}. \tag{6}$$

where we insert $\lambda(A,v)$ from (4) and the corresponding derivatives λ_A and λ_v as well as H_A given by

$$\begin{aligned} H_A = &-\kappa\theta p^{-\omega-1}(p - \omega p_A A) + \lambda[kp^{-a-1}\Psi(\alpha A^{\alpha-1}p - aA^{\alpha}p_A) - \\ &-c(\beta_A A + \beta) - \mu p^{b-1}(bp_A A + p)]. \end{aligned} \tag{7}$$

5.1.2 Parameters

The following parameters and their derivation is taken from Caulkins and Tragler [11].

Tragler et al. [47] describe in detail how parameters are derived from the literature. The parameters are summarized by Caulkins and Tragler [11] as follows. Briefly, the price elasticity parameters (a, b, and ω) collectively generate a long term price elasticity of demand of -1 (Caulkins et al. [7]), Caulkins and Reuter [8]), half coming from reduced consumption by current users (ω) and half from changes in the number of users, with the latter divided equally between impacts on initiation (a) and quitting (b).

For consistency with Rydell and Everingham [35] and Tragler et al. [48], we take the baseline price to be \$106.73 per pure gram and choose as initiation parameters $\alpha = 0.3$ and $k = 5,167$ to make initiation $1,000,000$ per year when the number of users $A = 6,500,000$ in base conditions. They estimate total baseline consumption as 291 (pure) metric tons, so we set $\theta = 14.6259$ (since $14.6259 \times 0.10673^{-0.5} = 291,000,000/6,500,000$ and price is expressed in thousands of dollars).

Rydell and Everingham [35] (p. 38) report cocaine-related health and productivity costs of \$19.68B for cocaine in 1992, dividing by 291 metric tons of consumption implies an average social cost per gram of \$67.6/gram (in 1992 dollars). These figures do not include crime-related costs, so in light of Miller et al. [27], we take \$100/gram as our base value ($\kappa = 0.1$ since dollars are measured in thousands). In view of Caulkins et al. [9] we also consider larger values in the sensitivity analysis.

The price function parameters ($d = 0.06792$ and $e = 0.02655$) reflect a price of \$106.73 per gram under base case enforcement spending and an elasticity of price with respect to enforcement spending of 0.3636 as in Caulkins et al. [7].

As in Tragler et al. [48] we assume $c = 0.04323$ and $z = 0.6$. These values reflect Rydell and Everingham's [35] estimates that spending an average of \$1,700 - \$2,000 per admission to treatment provides a 13% chance of ending heavy use, over and above baseline exit rates.

We adopt Behrens et al.'s [3] value of $h = 0.84$, but modify their value of m slightly (1.93×10^{-6} vs. 2.37×10^{-6}) to reflect better the size of the birth cohorts on whom prevention is targetted.

The outflow parameter $\mu = 0.18841$ was selected to make the outflow be

700,000 users per year at base prices, which reflects the observed population change (ONDCP [32]) net of initiation and treatment during the recent years of relative stability. The discount rate is set at $r = 0.04$ as in Rydell et al. [36] and Caulkins et al. [7].

These values are summarized in Table 1. Two values are given for parameters d, e, k, κ, μ, and θ. The values in brackets are the ones just described. For analytical convenience, we adjust d, e, k, and μ so that $\kappa = 1$ and $\theta = 1$, yielding the second set of values for those parameters.

Table 1: Base case parameter values

Parameter	Value	Description
a	0.25	absolute value of the elasticity of initiation with respect to price
α	0.3	exponent reflecting contagiousness of initiation
b	0.25	elasticity of desistance with respect to price
c	0.04323	treatment efficiency proportionality constant
d	0.03175 [0.06792]	price with minimal enforcement (in thousands of \$)
δ	0.001	constant to avoid division by zero
e	0.01241 [0.02655]	enforcement efficiency proportionality constant
ϵ	0.001	constant to avoid division by zero
h	0.84	one minus maximum proportion of baseline initiation prevention can avert with full implementation
k	$4,272$ $[5,167]$	initiation constant
κ	1 [0.1]	social cost per gram consumed (in thousands of \$)
m	1.93×10^{-6}	prevention efficiency proportionality constant
μ	0.22786 [0.18841]	natural outflow rate from use
ω	0.5	absolute value of the short run elasticity of demand
θ	1 [14.6259]	per capita consumption constant
r	0.04	annual discount rate (time preference rate)
z	0.6	$1 - z$ reflects treatment's diminishing returns

5.2 The Base Model without Controls

$$J = \int_0^\infty e^{-rt} \left(\kappa\theta A(t) p(A(t), v(t))^{-\omega}\right) dt$$

subject to

$$\dot{A}(t) = kA(t)^\alpha p(A(t), v(t))^{-a} \Psi(w(t)) - c\beta(A(t), u(t))A(t) - \mu p(A(t), v(t))^b A(t).$$

With the functions $u(t)$, $v(t)$, and $w(t)$ being set to 0:

$$\beta(A(t), 0) = 0,$$

$$p(A(t), 0) = d,$$

$$\Psi(0) = 1,$$

this gives us:

$$\dot{A}(t) = kA(t)^\alpha d^{-a} - \mu d^b A.$$

Setting $\dot{A}$ equal to zero, this will give us two steady states. The first one obviously is at $A = 0$, and the second one at $A = 14,942,254$.

5.3 The Model with Logistic Growth

Now we change the initiation term from a power function to a logistic growth function:

$$kA^{\alpha} \rightarrow \bar{k}A(\bar{A} - A).$$

Besides that change, we use the same functions as defined in Section 5.1.

The logistic growth function was first developed by the Belgian mathematician Verhulst in 1838 [51], who studied it in the context of population growth and later named it so in 1845 [52]. Logistic functions are defined by the growth rate, which is $\bar{k}$ in our model, and secondly by a carrying capacity, which is the number of maximum drug users $\bar{A}$ here. We derive the values for $\bar{k}$ and $\bar{A}$ from Caulkins et al. [10] by making use of the parameters for the inflow into the state S describing the number of *susceptibles* (k there) and the exit rate from state S (δ there). This gives us the maximum possible number of susceptibles to drug use ($\hat{S}$), which we will use as carrying capacity $\bar{A}$. Note that a change of magnitude is necessary, because the units for S in Caulkins et al. [10] are in millions.

$$\hat{S} = \frac{k}{\delta} = \frac{1.3417}{0.0605} = 22.1768595 \Rightarrow \bar{A} = 22,176,860$$

$$kA^{\alpha} = \bar{k}A(\bar{A} - A) \Rightarrow \bar{k} = \frac{kA^{\alpha}}{A(\bar{A} - A)}$$

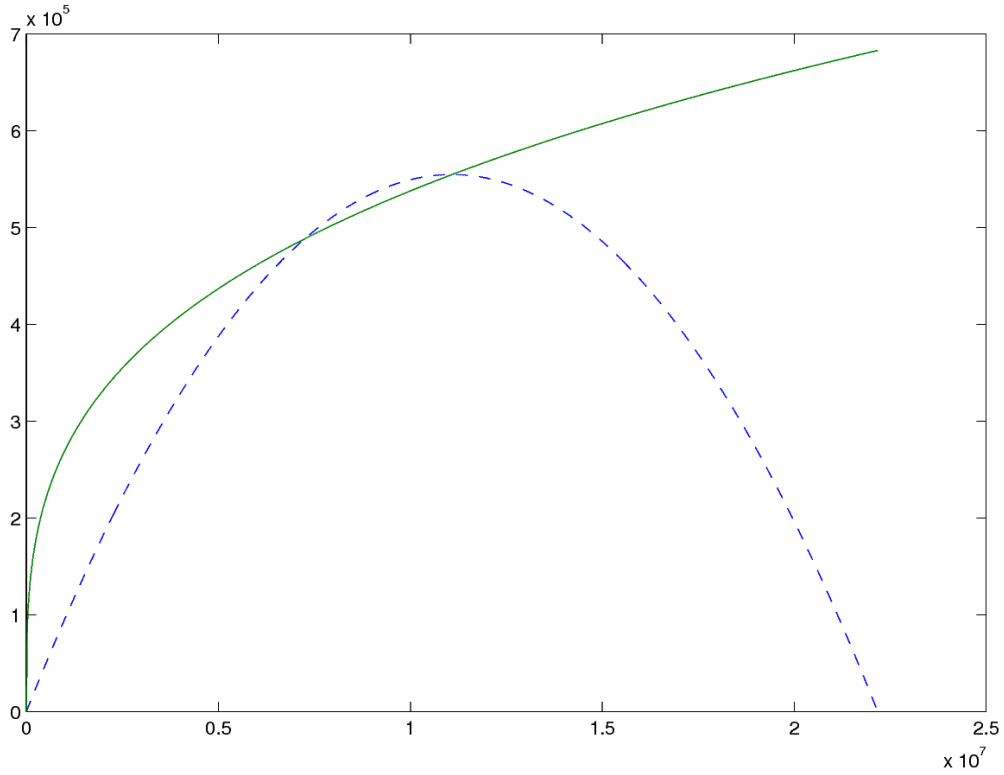
Now we use two values for A: ($A = \frac{\bar{A}}{2}$ and the base case value $A = 6,500,000$) and correspondingly get for each a value for $\bar{k}$. The average of both of these values is $\bar{k} = 4.57492 \times 10^{-9}$, which we are going to use in this thesis.

In Figure 8 we can see the initiation term for both functions with the respective parameters over the users A.

This change does not affect the utility functional J but it does change $\dot{A}$:

$$\min_{\{u(t),v(t),w(t)\}} J = \int_0^{\infty} e^{-rt} \left(\kappa\theta A(t)p(A(t), v(t))^{-\omega} + u(t) + v(t) + w(t)\right) dt$$

Figure 8: Initiation with logistic growth function (dashed) and with power function (continuous).



subject to

$$\dot{A}(t) = \bar{k}A(\bar{A} - A)p(A(t), v(t))^{-a}\Psi(w(t)) - c\beta(A(t), u(t))A(t) - \\ -\mu p(A(t), v(t))^{b}A(t)$$

and the non-negativity constraints

$$u(t) \geq 0, v(t) \geq 0, w(t) \geq 0.$$

5.3.1 Pontryagin's Maximum Principle

The new Hamiltonian H for the problem is given by

$$H = -(\kappa\theta A p^{-\omega} + u + v + w) + \lambda(kA(\bar{A} - A)p^{-a}\Psi - c\beta A - \mu p^{b}A).$$

According to Pontryagin's maximum principle we again have the following three necessary optimality conditions:

$$u = \arg\max_u H,$$

$$v = \arg\max_v H,$$

and

$$w = \arg\max_w H.$$

A sufficient condition for H to be concave with respect to the controls (u, v, w) is the negativity of the costate variable, i.e.:

$$\lambda < 0 \Rightarrow H_{uu}, H_{vv}, H_{ww} < 0$$

(for a proof see Appendix A.2). Due to the concavity of the Hamiltonian H with respect to (u, v, w), setting the first order partial derivatives equal to zero leads to the unrestricted extremum, and we get the following expressions for the costate λ:

$$H_u = 0 \Rightarrow \qquad \lambda = \frac{-1}{c\beta_u A}, \tag{8}$$

$$H_w = 0 \Rightarrow \qquad \lambda = \frac{1}{\bar{k}p^{-a}A(\bar{A}-A)\Psi_w}, \tag{9}$$

$$H_v = 0 \Rightarrow \lambda = \frac{1-\kappa\omega p^{-\omega-1}p_v A}{-a\bar{k}p^{-a-1}p_v A(\bar{A}-A)\Psi - \mu b p^{b-1}p_v A}. \tag{10}$$

The concavity of the maximized Hamiltonian with respect to the state variable, however, cannot be guaranteed, so the usual sufficiency conditions are not satisfied.

With Equations (8)-(10) we can describe u and w as functions of v and A as follows:

$$\lambda(A,v) := \frac{\frac{p_v}{p}\left(\frac{a}{m} + \kappa\theta\omega p^{-\omega}A\right) - 1}{ah\bar{k}p^{-a-1}p_v A(\bar{A}-A) + \mu b p^{b-1}p_v A}, \tag{11}$$

$$u(A,v) := \left(\frac{-(A+\delta)^z}{czA\lambda(A,v)}\right)^{\frac{1}{z-1}},$$

$$w(A,v) := \frac{1}{m}\ln\big((h-1)\bar{k}mp^{-a}A(\bar{A}-A)\lambda(A,v)\big).$$

Due to this simplification we can concentrate on the two variables A and v.

To gain an equation for $\dot{v}$ we differentiate λ with respect to time:

$$\dot{\lambda} = \lambda_A \dot{A} + \lambda_v \dot{v}. \tag{12}$$

Setting (12) equal to the costate equation

$$\dot{\lambda} = r\lambda - H_A,$$

yields:

$$\dot{v} = \frac{r\lambda - H_A - \lambda_A \dot{A}}{\lambda_v}. \tag{13}$$

where we insert $\lambda(A, v)$ from (12) and the corresponding derivatives λ_A and λ_v as well as H_A given by

$$\begin{aligned} H_A = &-\kappa\theta p^{-\omega-1}(p - \omega p_A A) + \lambda[\bar{k}p^{-a-1}\Psi((\bar{A} - A)p - Ap - aA(\bar{A} - A)p_A) - \\ &-c(\beta_A A + \beta) - \mu p^{b-1}(bp_A A + p)]. \end{aligned} \tag{14}$$

5.3.2 Parameters

We use the same parameters as defined in Section 5.1, except for the ones summarized in Table 2.

Table 2: Base case parameter values in the model with logistic growth

Parameter	Value	Description
κ	0.34	social cost per gram consumed (in thousands of \$)
$\bar{k}$	4.57492×10^{-9}	initiation constant
$\bar{A}$	$22,176,860$	maximum number of drug users

5.4 The Model with Logistic Growth without Controls

$$J = \int_0^\infty e^{-rt} \left(\kappa\theta A(t) p(A(t), v(t))^{-\omega}\right) dt$$

subject to

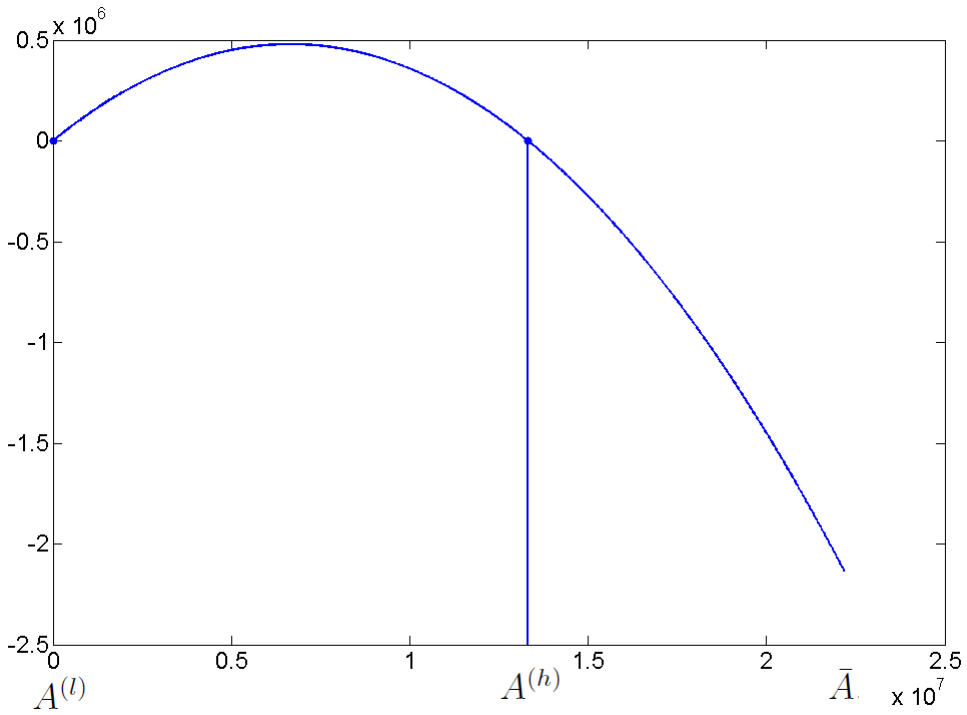
$$\dot{A}(t) = \bar{k}A(\bar{A} - A)d^{-a} - \mu d^b A.$$

Setting $\dot{A}$ equal to zero leads to:

$$\dot{A} \stackrel{!}{=} 0 \Rightarrow A^{(h)} = \frac{-\mu d^b}{\bar{k}d^{-a}} + \bar{A} \text{ or } A^{(l)} = 0.$$

In this model, we leave the users to their fate and just watch the system evolved. For a number of users near zero (but greater than zero) this will lead to a rapid growth at start, and then an increasingly moderate growth until A reaches the number of 13.302.103 users, which is the high equilibrium point $A^{(h)}$. We defined the maximum number of drug users $\bar{A}$, so there is no higher number of users possible, and between the high equilibrium point and $\bar{A}$ there will always be a decline of users, causing a convergence to the high equilibrium point $A^{(h)}$. For zero drug users as starting value, there will be no change to the number of users, so zero is our low equilibrium point $A^{(l)}$. This is illustrated in Figure 9.

Figure 9: The growth rate of A for the logistic-growth-function model without controls: $\dot{A}$ over A



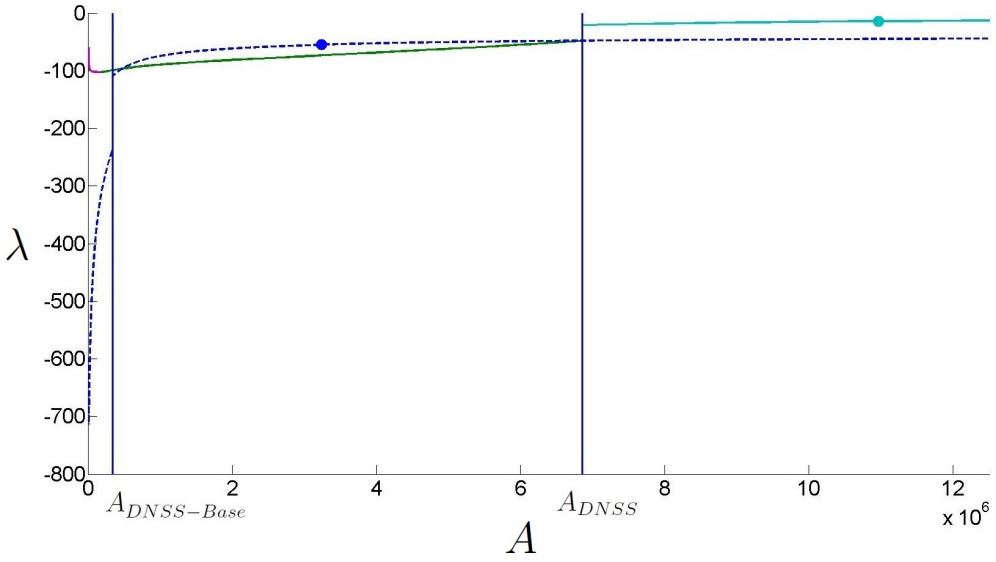
6 Sensitivity Analysis

With parameter κ being 1 like in Caulkins and Tragler [11], no inner equilibrium could be found, so we varied this parameter and an admissible equilibrium was found for κ lower than 0.4, so we settled for $\kappa = 0.34$ as the new base case value. This means that the social cost per gram consumed dropped to about a third, compared to the original base value, which is rather radical, so we presume it will have a big impact on the results. We will later go more into detail about this in the sensitivity analysis of κ. Note that enforcement is zero in all of the following analysis.

One can see in Figure 10 that the number of users in the saddle point equilibrium got higher rather drastically. While in the base model $A = 3.24 \times 10^6$ at the saddle point, in the logistic growth model the number of

users got up to 1.096×10^7, which is about 3.38 times as much. Figure 10 also shows a jump discontinuity for each model, which is where there is a so-called Dechert-Nishimura-Sethi-Skiba (DNSS) point (Dechert and Nishimura [12], Sethi [37],[38], Skiba [39], cf. Grass et al. [20]) that defines two basins of attraction according to whether the optimal policy is to push it to the lower limit equilibrium or to just approach the high volume saddle point equilibrium. For the base case parameter values, that point is $A_{DNSS} = 6,858,614$ users.

Figure 10: Saddle point and DNSS threshold of the base model (dashed) and the logistic-growth-function model (continuous) in their base case compared with each other in the $A - \lambda$ plane. $A_{DNSS-Base}$ marks the DNSS threshold of the base model, A_{DNSS} the DNSS threshold of the logistic-growth-function model. The round markers show the saddle points of the base model (blue) and the logistic-growth-function model (teal).



We also assume that there is some lower limit, $\underline{A}$, on the number of users (e.g., $\underline{A} = 10,000$) below which control efforts cannot drive the problem

(e.g., because these residual users cannot be detected), then the point $(\underline{A}, \underline{\lambda})$ becomes another equilibrium.

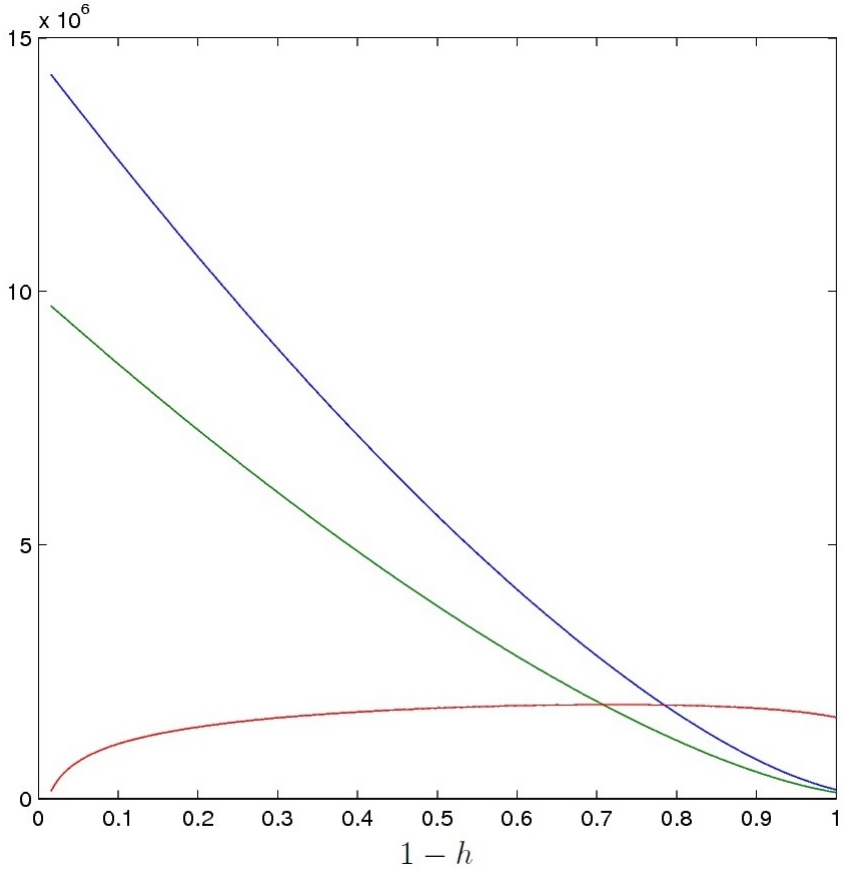
6.1 Sensitivity Analysis with Respect to the Strength of Prevention

As pointed out in Caulkins and Tragler [11], there is a reasonably strong basis for believing that current, model primary prevention technologies can reduce initiation by about $1-h = 16\%$, but sensitivity analysis with respect to parameter h is still of interest for some reasons. First, many programs that are actually being used are not model programs, so the effectiveness of prevention today may be smaller (higher h). Second, better prevention technologies may be available in the future. For example, immunotherapies being developed to treat cocaine addiction might conceivably be used for primary prevention (Hardwood and Myers [23]). There are plausible circumstances under which such vaccinations could be highly cost-ineffective for prophylactic purposes, but the very existence of such research suggests that prevention technology is not static (Caulkins and Tragler [11]).

Moving from left to right in Figure 11 corresponds to prevention in the base model becoming more powerful (reducing h, increasing $1-h$). Spending on prevention increases as it becomes more effective, while spending both on treatment and enforcement decrease with reducing h. Turning to the logistic-growth-function model (Figure 12), moving from right to left corresponds to prevention becoming more powerful (reducing h). Spending on prevention again increases as it becomes more effective, but surprisingly spending on treatment also increases with reducing h.

While spending on prevention is a concave function of h, treatment is convex and there exist two points of intersection. In Caulkins and Tragler [11] there was only one intersection point where prevention was very effective (h between 0.15 and 0.3) and substituted for the other much more costly interventions (cf. Figure 11). The functions of u and v were convex in h and w also concave. Note that there exist no admissible equilibria in our model

Figure 11: The levels of optimal control spending (treatment u (blue), enforcement v (green), prevention w (red)) as functions of $1-h$ at $\hat{A}^{(h)}$ for the base model.

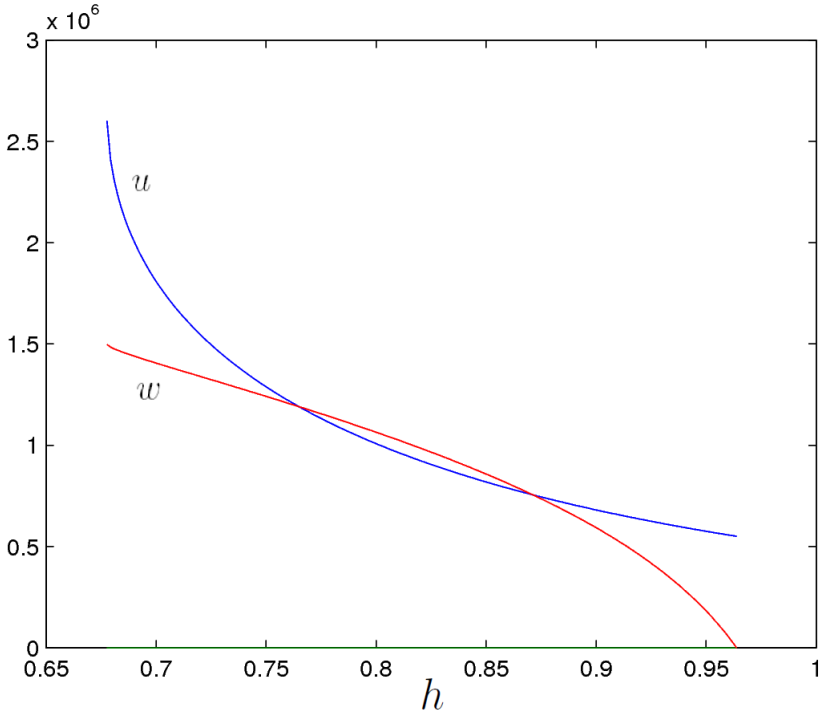


for h greater than about 0.96 and lower than about 0.67, while in the model from Caulkins and Tragler [11] there is an admissible equilibrium for h from about 0 to 1.

When we take a further look, one can see in Figure 13 that the saddle point moves to the left with drastically reduced drug users. While reducing h from about 0.9638 to 0.6777, which is a relative reduction of nearly 30%, the number of users are decreased from 1.2694×10^7 to 5.9154×10^6, which is about 53.4%. This is not surprising as the overall spending on interventions at the far right of h is much lower than on the far left. If one could choose

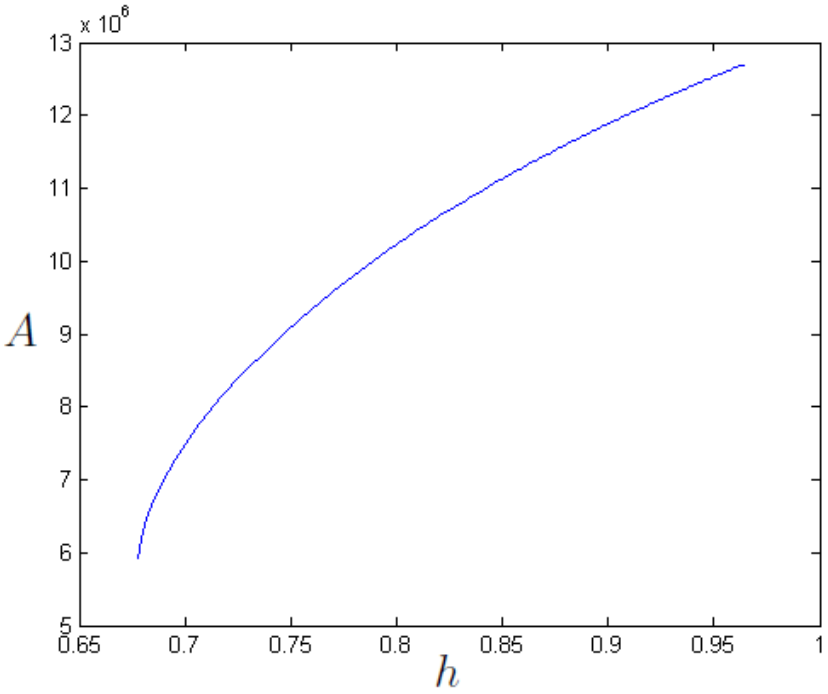
the effectiveness of prevention for this model, the choice should be near the left intersection point to get maximum cost efficiency.

Figure 12: The levels of optimal control spending in equilibrium as functions of h for the logistic-growth-function model. Treatment spending v (green) is zero.



There is a heteroclinic connection for h being 0.7287, so for smaller values of h only the path to $\underline{A}$ is admissible, see Figure 14. At h=0.9637 the high equilibrium is no longer admissible, and for values higher than 0.9637 there exists an equilibrium with enforcement v and prevention spending w being both zero. Between 0.9637 and 1 there exists a DNSS threshold, see Figure 15.

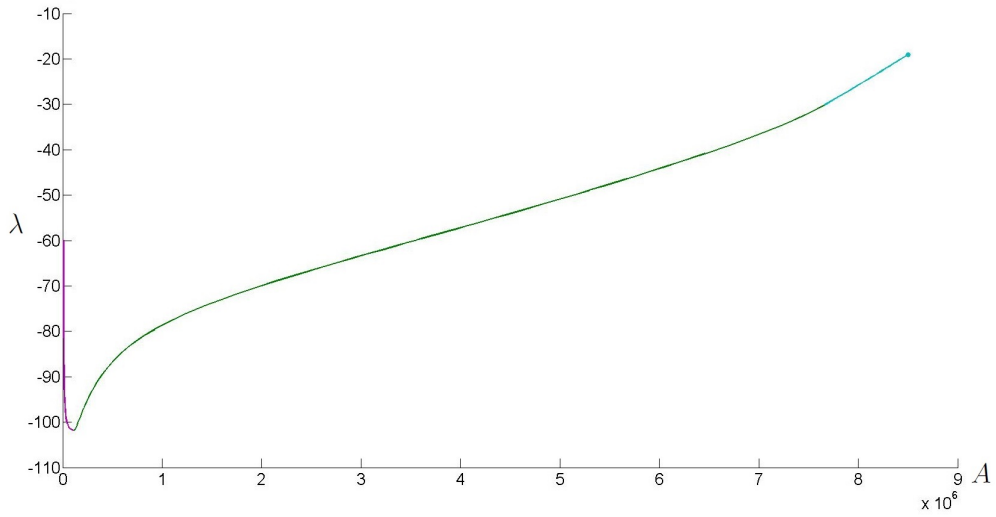
Figure 13: The saddle point movement as function of h for the logistic-growth-function model.



6.2 Sensitivity Analysis with Respect to Treatment Effectiveness

As pointed out in UNODC's World Drug Report 2015 [50], drug treatment is often perceived as ineffective. There it is said that one major difference in the perception of their ineffectiveness is that drug dependence treatments are not provided and evaluated under the same assumptions that pertain to other chronic illnesses. Particularly important in this regard is that drug dependence treatments are rarely delivered under a continuing care model that would be appropriate for a chronic health problem. Indeed, with the exception of methadone maintenance and the 12-step approach, most contemporary treatments for drug dependence are acute care episodes (UNODC [50]). However, for cocaine addictions behavioral treatments have proven to be effective in both residential and outpatient settings and also scientists found out that cocaine induces changes in the brain related to other neuro-

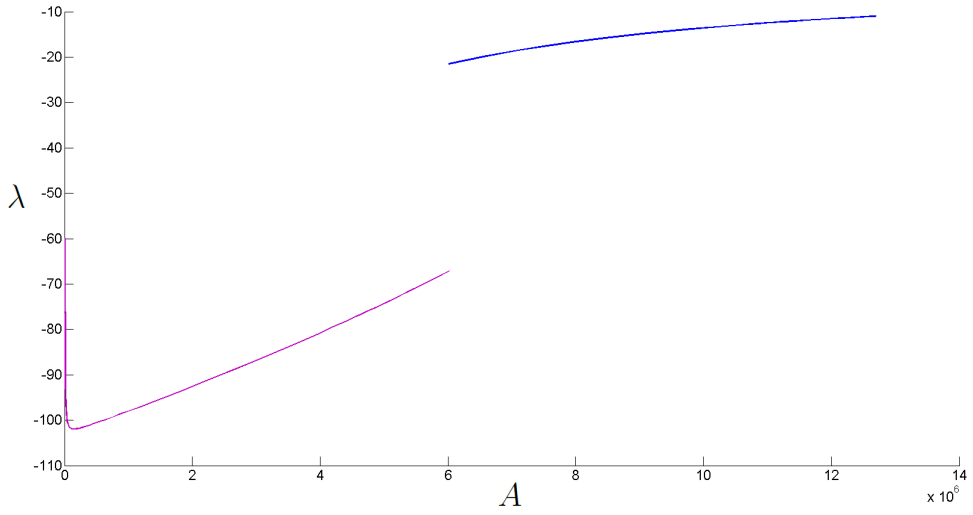
Figure 14: Heteroclinic connection for h=0.7287 in the $A - \lambda$ plane for the logistic-growth-function model.



transmitters than dopamine. They are currently working on new medications and vaccines that let your immune-system create cocaine-specific antibodies that bind to cocaine, preventing it from getting into the brain (NIDA [30]). So in the future the effectiveness of treatment for cocaine could drastically change and therefore the parameter c for treatment effectiveness is an appropriate object of sensitivity analysis.

Varying this parameter affects the saddle-point equilibrium in the following way. The more effective treatment is, the greater its share of control spending in steady state, and the fewer users there are in steady state. In particular, if treatment were 1% more effective, it would be optimal in steady state to spend about 3% more on treatment but also 0.02% more on prevention . While in Caulkins and Tragler [11] treatment spending increases while enforcement and prevention spending decline (+0.97%, -0.86%, and -0.22%, respectively, to be precise), the decline in the number of users in the saddle-point equilibrium is even greater (-1.65%). In our model, there is also a decline of users, but it is a very small one compared to the 3.12% more spending on treatment and 0.2% on prevention. So while the overall spending on controls increases by about 1.62%, the number of users only declines

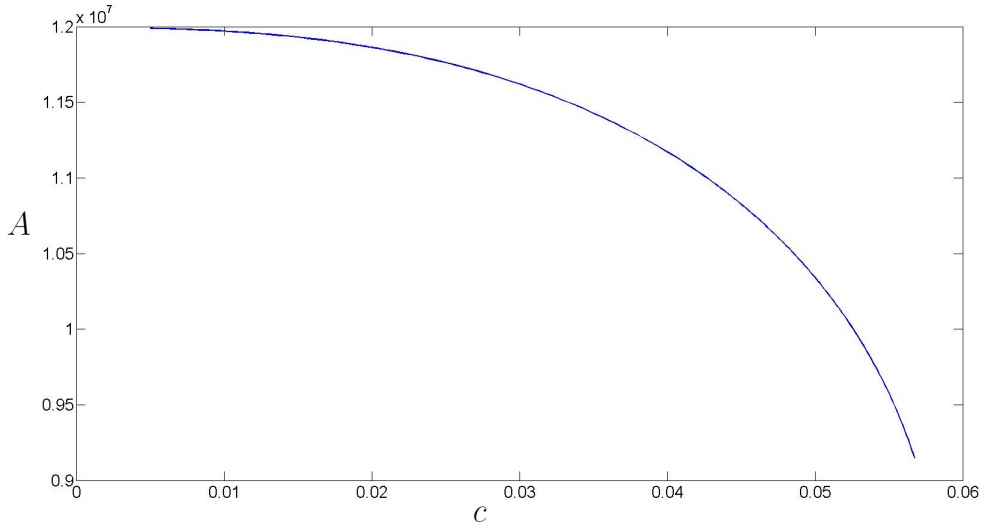
Figure 15: DNSS Point at h=1 in the $A - \lambda$ plane for the logistic-growth-function model.



by about -0.29%. This can be seen in Figure 16. Increasing the effectiveness of treatment by 1% also shifts the DNSS point to the right by about 1.97%.

Approximately at c being 0.005, treatment spending u becomes zero. For values of c greater 0.0567, no admissible saddle-point equilibria exist. Figure 17 shows that prevention w is barely affected by c, while treatment u is greatly affected, and both are convex functions of c.

Figure 16: The saddle point movement as function of c for the logistic-growth-function model.

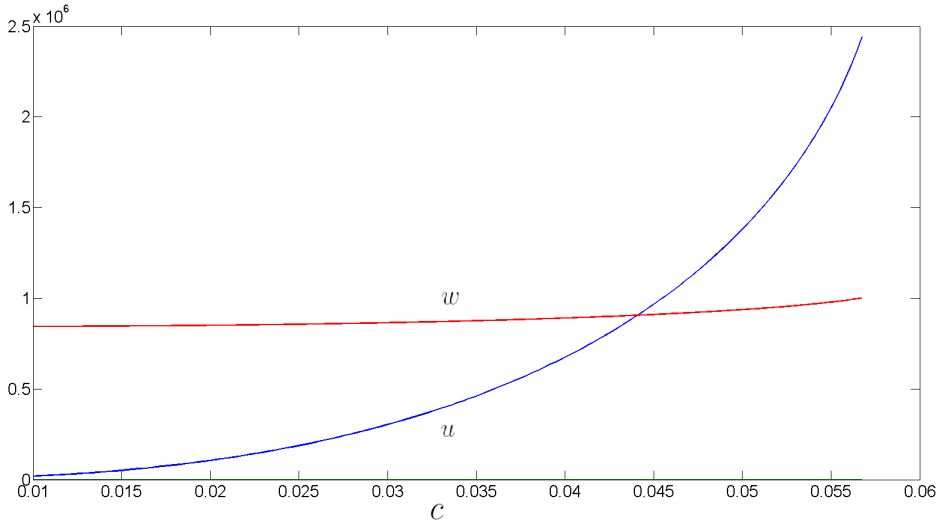


6.3 Sensitivity Analysis with Respect to the Social Cost per Gram Consumed

The level of spending substantially depends on the presumed social cost per gram of cocaine consumed, but determining this value is not easy, because there are data limitations and different approaches as there is no general rule what costs should be included as social costs (Caulkins and Tragler [11]. Moreover, social costs can not always be linked to particular types of drugs, as many users do not exclusively use only one drug but more (Moore [28]). For comparison, we opted to go for keeping the same parameters as in the base model, but there were no admissible equilibria to be found, so we changed the base case value for κ from 1 ($100/gram) to 0.34 ($34/gram).

If the social cost per gram were believed to be 10% higher, then the optimal level of drug control spending at the saddle equilibrium would be 19.7% higher. Likewise, if κ were 20% lower, the optimal steady state spending would be 16.7% lower. In both cases, the changes are more dramatic for treatment (+33.11% and -26.5%) and less dramatic for prevention (+7% and -7.5%).

Figure 17: The levels of optimal control spending in equilibrium as functions of c for the logistic-growth-function model. Law enforcement spending is zero throughout.



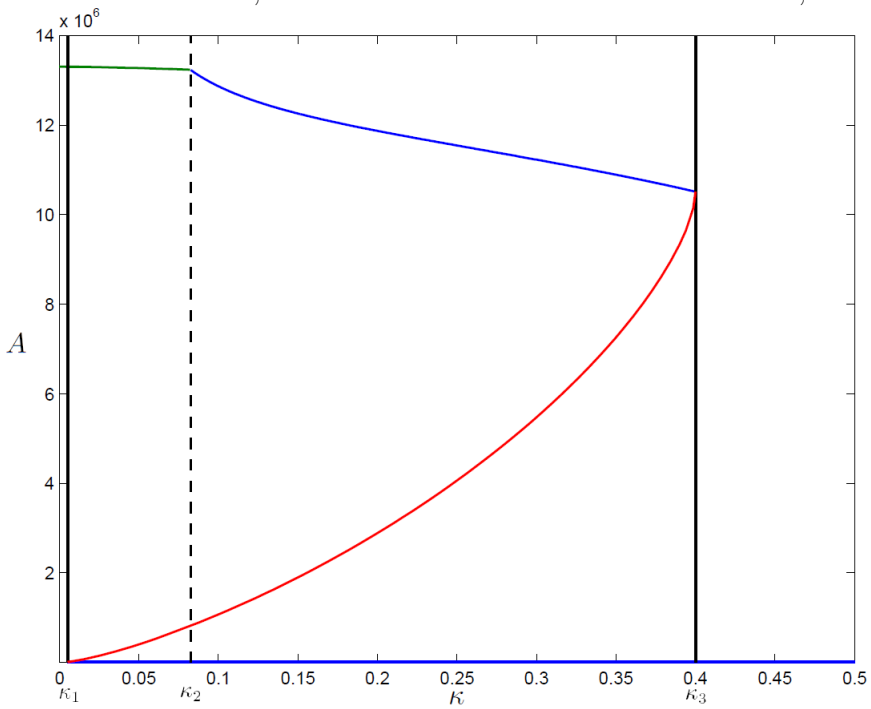
Reducing κ shifts the DNSS threshold to the left, making it disappear when κ drops to 0.0053. The DNSS threshold shifts to the right as κ increases, merging with the saddle point equilibrium when $\kappa = 0.4$ (see Figure 18).

This is the same behavior as in Caulkins and Tragler [11], although there the sensitivity analysis of κ only included the range of κ between 0.5 and 2.5, while in our model there was no admissible equilibrium for κ higher than 0.4. So for comparison we analyse the movement of the equilibria for κ lower than 0.5, but shortly after reducing κ, the non-negativity constraint was not fulfilled by the implicit control v anymore. This happens for κ being about 0.461, see Figure 19. It was also described that the DNSS threshold disappeared for κ lower than 0.7.

At κ_1 (0.0053) and κ_3 (0.4) we find heteroclinic bifurcations (see Figure 18), which is where a heteroclinic connection between two equilibria emerges at the criticial value of the parameter (cf. Grass et al. [20]). These points are threshold points but not indifference points.

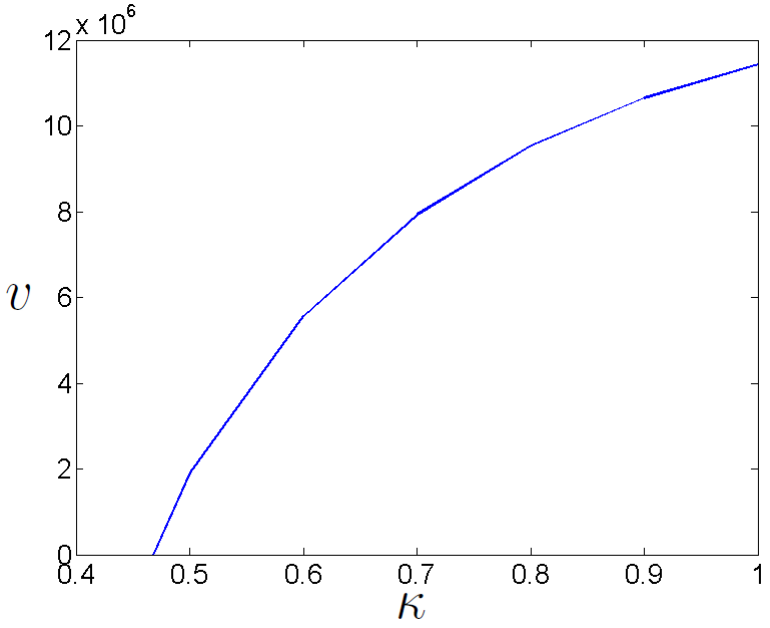
Between zero and κ_1 only the high equilibrium is optimal. $\underline{A}$ of course still exists, but it is not an optimal solution. To the right side of κ_3, the high

Figure 18: The influence of κ on the equilibrium values and the DNSS threshold. The relation between κ and the high equilibrium is displayed in the upper blue branch. This is were enforcement spending v is zero. The horizontal green line at the top left between κ_1 and κ_2 is where enforcement v and prevention spending w are both zero. The red curve between κ_1 and κ_3 bending upwards represents the level of the DNSS threshold. The horizontal blue line at the very bottom is the lower limit at $\underline{A}$, even though it looks like zero because of the scale, this is where the number of users is 10,000.



equilibrium still exists for some values, but only $\underline{A}$ is optimal.

Figure 19: Steady-state value of law enforcement spending v as a function of κ in the base model.



6.4 Sensitivity Analysis with Respect to the Maximum Number of Drug Users

The maximum number of drug users for sure is dynamic and changes over the years, as also the population in the US varies. So a sensitivity analysis with respect to $\bar{A}$ is of interest and it turns out that it has a great effect on the results. Raising $\bar{A}$ by 10% leads to shifting the DNSS threshold by 3.25% to the right but increases the number of users in the saddle point equilibrium by +22.1%. This is not surprising, because we did not also vary $\bar{k}$ to keep the rate of initiation under base case conditions constant per year. Spending on control is also affected significantly, but as treatment spending decreases strongly, the spending on prevention increases only by a small margin (-18.21% and +1.21%, respectively, to be precise; see Figure 20).

Reducing $\bar{A}$ by 10% lets the DNSS threshold shift to the left by only about 0.01%, but again the position of the saddle point is greatly affected (-23.55%). Treatment spending increases by 30% and prevention decreases by 1.39%.

Admissible equilibria exist for values of $\bar{A}$ between about 1.79×10^7 and about 2.69×10^7. See Figure 21 for the number of users at the saddle-point equilibrium and Figure 20 for optimal control spending as functions of $\bar{A}$.

Figure 20: The levels of optimal control spending as functions of $\bar{A}$ for the logistic-growth-function model. Enforcement spending v is again zero.

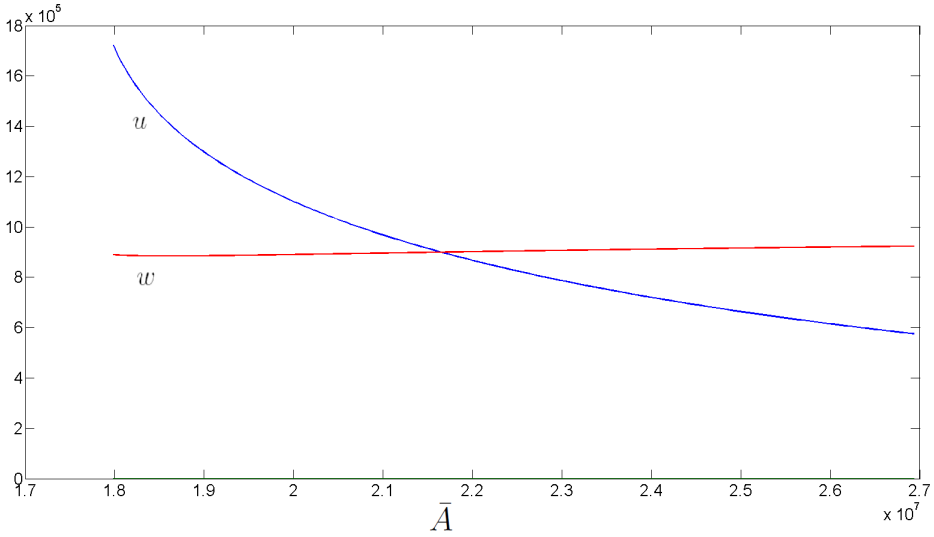
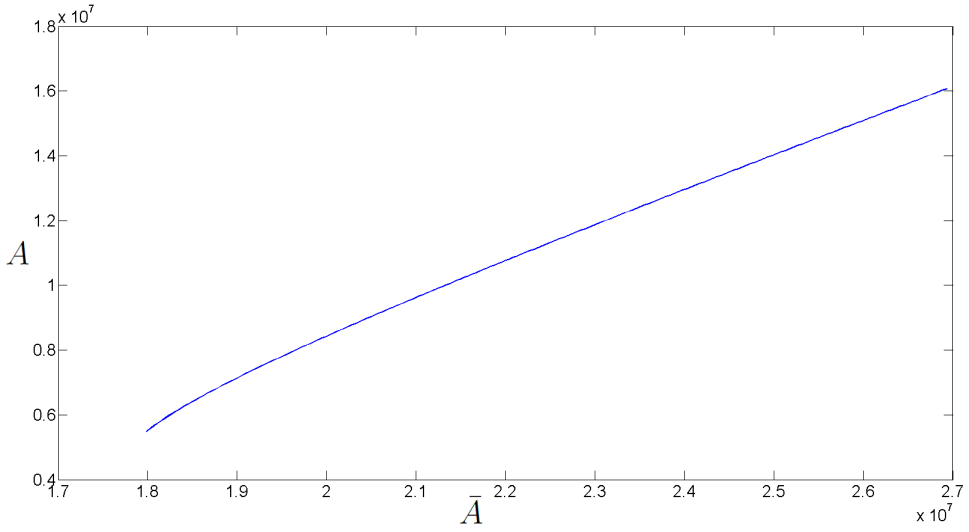


Figure 21: The saddle point movement as function of $\bar{A}$ for the logistic-growth-function model.



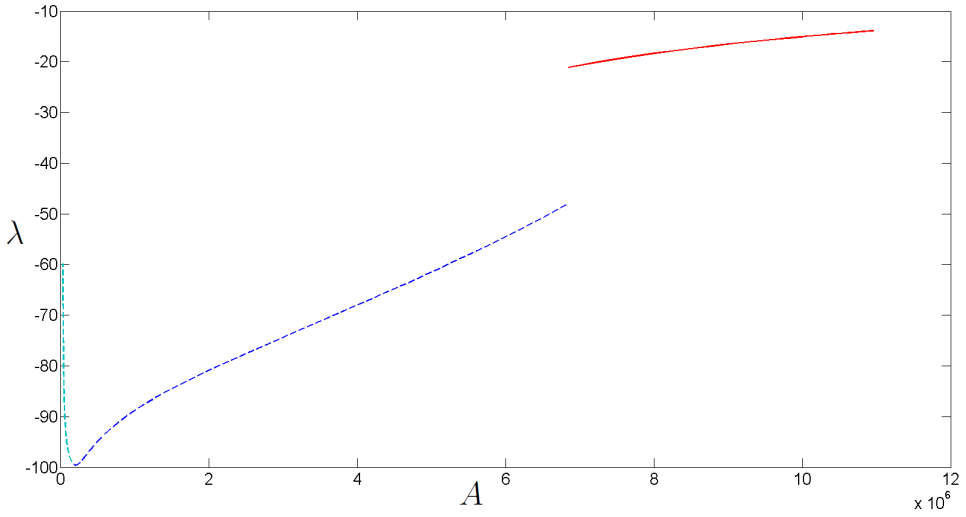
6.5 Sensitivity Analysis with Respect to the Lower Limit on the Number of Users

The larger the lower limit, $\underline{A}$, below which control cannot drive the number of users, the smaller the DNSS point. In Caulkins and Tragler [11], doubling $\underline{A}$ from 10,000 to 20,000 reduces the DNSS point by two thirds (reduces it from 334,339 to 128,268). So both models share the same effect, but not nearly to the same extent. In our model, doubling $\underline{A}$ from 10,000 to 20,000 users reduces the DNSS point only by about 0.0538%. The movement of the DNSS point for more values of $\underline{A}$ can be seen in Table 3. For values between 100 and 100,000 for $\underline{A}$, the location of the saddle-point equilibrium basically does not change at all in our model, so control spending is also barely affected. Figure 22 exemplarily displays the case of a DNSS threshold for $\underline{A}$=40,000.

Table 3: The effects of varying $\underline{A}$ on the DNSS Point.

$\underline{A}$	DNSS
100	$6,861,185.274$
$1,000$	$6,861,057.793$
$5,000$	$6,860,132.113$
$10,000$	$6,858,613.643$
$20,000$	$6,854,927.325$
$40,000$	$6,845,994.929$
$100,000$	$6,812,434.011$

Figure 22: DNSS threshold in the $A - \lambda$ plane for $\underline{A} = 40,000$.



7 OCMat & Matlab Source Codes

For some parts of the analysis, the OCMat-Toolbox[2] was used. Here are some of the Matlab source codes which were used to calculate and plot data presented in this master thesis.

Init-File for Toolbox OCMat for the base model:

```
Type
standardmodel

Description

Modelname
drugmodelII

Variable
state::A
control::u,v,w

Statedynamics
ode::DA=k*A^alpha*(d+e*v/(A+epsilon))^(-a)*(h+(1-h)*exp(-m*...
    w))-c*(u/(A+delta))^z*A-mu*(d+e*v/(A+epsilon))^b*A

Objective
int::-kappa*theta*A*(d+e*v/(A+epsilon))^(-omega)-u-v-w

Controlconstraint % identifier has to contain an alphabetic...
     character
CC1::ineq::u>=ulow
CC2::ineq::v>=vlow
CC3::ineq::w>=wlow

ArcDefinition
0::[]

```

[2]OCMat is a collection of MATLAB files developed to analyze optimal control models developed by ORCOS, Vienna University of Technology.

```
Control
0::*::implicit

Parameter
r::0.04
a::0.25
alpha::0.3
b::0.25
c::0.04323
d::0.03175
delta::0.001
e::0.01241
epsilon::0.001
h::0.84
k::4272
kappa::1
m::1.93e-6
mu::0.22786
omega::0.5
theta::1
z::0.6
ulow::0
vlow::0
wlow::0
Alow::10000
```

Initialising Model and Equilibria of the base model:

```
m=stdocmodel('drugmodelII');
par=parametervalue(m);
opt=optimset('TolFun',1e-13,'MaxFunEvals',15000,'MaxIter'...
    ,15000,'TolX',1e-10);
x=fsolve(@drugEquilibrium,[3.24e6;1.14e7],opt,par);
J=drugCanonicalSystemJacobian([],x,par);
x2=fsolve(@drugEquilibrium,[0.2e6;1.04e7],opt,par);
J2=drugCanonicalSystemJacobian([],x2,par);
```

Plot h and $1-h$ for the base model:

```
par(21)=10000 % Alow = 10,000
par(22)=7e6;
xinit=x(:,end);
h=linspace(0,1,251);x0=xinit;ctrl=[];for ii=1:length(h);par...
    (10)=h(ii);xnew=fsolve(@drugEquilibrium,x0,opt,par);x...
    (:,ii)=xnew;x0=xnew;ctrl(:,ii)=drugOptimalControl(0,x0,...
    par);end;index=find(ctrl(3,:)<0);x(:,index)=[];ctrl(:,...
    index)=[];h(index)=[];
plot(h,[x(2,:);ctrl(2:3,:)])
plot(1-h,[x(2,:);ctrl(2:3,:)])
```

Plot of saddle-point equilibrium and DNSS-point for both models (comparison):

```
par(1)=0.04;
par(2)=0.25;
par(3)=0.3;
par(4)=0.25;
par(5)=0.04323;
par(6)=0.03175;
par(7)=0.001;
par(8)=0.01241;
par(9)=0.001;
par(10)=0.84;
par(11)=4272;
par(12)=1;
par(13)=1.93e-6;
par(14)=0.22786;
par(15)=0.5;
par(16)=1;
```

```
17 par(17)=0.6;
18
19 opt=optimset('TolFun',1e-13,'MaxFunEvals',15000,'MaxIter'...
       ,15000,'TolX',1e-10);
20 x=fsolve(@drugEquilibrium,[3.24e6;1.14e7],opt,par);
21 J=drugCanonicalSystemJacobian([],x,par);
22 [e1 e2]=eig(J)
23 x2=fsolve(@drugEquilibrium,[0.2e6;1.04e7],opt,par);
24 J2=drugCanonicalSystemJacobian([],x2,par);
25 [e3 e4]=eig(J2);
26
27 opt=odeset('abstol',1e-13,'reltol',1e-13)
28 trj=ode45(@drugCanonicalSystem,[0,-1000],x+1e-3*e1(:,1),opt...
       ,par)    %continuation
29
30 %Path to Amin
31 %calc v-value (dynamic of canonical system A. set to 0 zero...
       and => v numerical)
32
33 opt=optimset('TolFun',1e-13,'MaxFunEvals',15000,'MaxIter'...
       ,15000,'TolX',1e-10);
34 v=fzero(@drugvequation,1e7,opt,1e4,par);
35 trj2=ode45(@drugCanonicalSystem,[0,-10],[1e4;v],opt,par);
36 H=drugHamiltonian([],trj.y,par);
37 H2=drugHamiltonian([],trj2.y,par);
38 %Intersection Point of both Hamiltonians => DNSS Point
39
40 [v0,o,idx1,idx2] = intersections(trj.y(1,:),H,trj2.y(1,:),...
       H2);
41 global USERDRUG;USERDRUG.Amin=1e4;USERDRUG.Amax=1e8;...
       USERDRUG.Askiba=v0;
42 [Askiba,o,idx1,idx2] = intersections(trj.y(1,:),H,trj2.y...
       (1,:),H2);
43 global USERDRUG;USERDRUG.Amin=1e4;USERDRUG.Amax=1e8;...
       USERDRUG.Askiba=Askiba;
44
45 opt=odeset('abstol',1e-13,'reltol',1e-13,'event',@drugEvent...
       )
46 trj=ode45(@drugCanonicalSystem,[0,-1000],x+1e-3*e1(:,1),opt...
```

```
    ,par)
trj2=ode45(@drugCanonicalSystem,[0,-10],[1e4;v],opt,par)
global USERDRUG;USERDRUG.Amin=1e4;USERDRUG.Amax=2e7;...
    USERDRUG.Askiba=Askiba;
trj3=ode45(@drugCanonicalSystem,[0,-1000],x-1e-3*e1(:,1),...
    opt,par)
h=plot(trj.y(1,:),trj.y(2,:),trj2.y(1,:),trj2.y(2,:),trj3.y...
    (1,:),trj3.y(2,:)),hold on;plot(x(1),x(2),'marker','.',...
    'markersize',20),figure(gcf); set(gca,'xlim',[0 5e6])
set(h,'color',[0 0 1])

%DNSS Point @jump discontinuity & saddle-point @mark - A-v ...
    plane.
%now IN A-lambda-plane:

l1=drugOptimalControl([],trj.y,par);l2=drugOptimalControl...
    ([],trj2.y,par);l3=drugOptimalControl([],trj3.y,par);
lhat=drugOptimalControl([],x,par)
clf;plot(trj.y(1,:),l1(1,:),trj2.y(1,:),l2(1,:),trj3.y(1,:)...
    ,l3(1,:)),hold on;plot(x(1),lhat(1),'marker','.','...
    markersize',20),figure(gcf); set(gca,'xlim',[0 5e6])


%now lets to this for comparison for the log model:

opt=setocoptions('OCCONTARG','MaxStepWidth',3e6,'...
    InitStepWidth',1e3,'MinStepWidth',1e1, ...
'SBVPOC','BCJacobian',0,'FJacobian',1,'MeshAdaptAbsTol',1e...
    -4,'MeshAdaptRelTol',1e-5,'NMax',10000, ...
'GENERAL','AdmissibleTolerance',1e-1,'TrivialArcMeshNum'...
    ,10, ...
'NEWTON','MaxNewtonIters',15,'MaxProbes',10,'RelTol',1e-2,'...
    AbsTol',5e-3);
ocEP=equilibrium(m);
eigval=real(eig(ocEP{1}));eigval(eigval>-1e-5)=[];T=20/min(...
    abs(eigval));
sol=initocmat_AE_EP(m,ocEP{1},1,1e7,[],'TruncationTime',T,'...
    pathtype','sc');
c=bvpcont('extremal2ep',sol,[],opt);   %starts Boundary ...
```

```
      value problem
71
72 store(m,'extremal2ep');
73 save(m,[],[],'BaseCase')
74
75 clf;xcoord=1;ycoord=1;xvar='state';yvar='costate';plotcont(...
      m,xvar,xcoord,yvar,ycoord,'contclass','...
      indifferencesolution4ae2ftae','contfield','...
      ExtremalSolution','Index',[]);hold on;plotcont(m,xvar,...
      xcoord,yvar,ycoord,'contclass','extremal2ep','contfield...
      ','ExtremalSolution','Index',8);plotlimitset(m,xvar,...
      xcoord,yvar,ycoord,'Index',1,'Marker','.','MarkerSize'...
      ,20);figure(gcf);
76
77 delete(m,'Continuation',8)
78 sol=initocmat_AE_EP(m,ocEP{1},1,1e8,[],'TruncationTime',T,'...
      pathtype','sc');
79 c=bvpcont('extremal2ep',sol,[],opt);
80
81 store(m,'extremal2ep');
82 save(m,[],[],'BaseCase')
83
84 clf;xcoord=1;ycoord=1;xvar='state';yvar='costate';plotcont(...
      m,xvar,xcoord,yvar,ycoord,'contclass','...
      indifferencesolution4ae2ftae','contfield','...
      ExtremalSolution','Index',[]);hold on;plotcont(m,xvar,...
      xcoord,yvar,ycoord,'contclass','extremal2ep','contfield...
      ','ExtremalSolution','Index',8);plotlimitset(m,xvar,...
      xcoord,yvar,ycoord,'Index',1,'Marker','.','MarkerSize'...
      ,20);figure(gcf);
85 global USERDRUG;USERDRUG.Amin=1e4;USERDRUG.Amax=1e8;...
      USERDRUG.Askiba=Askiba;
86 opt=odeset('abstol',1e-13,'reltol',1e-13,'event',@drugEvent...
      )
87 trj3=ode45(@drugCanonicalSystem,[0,-1000],x-1e-3*e1(:,1),...
      opt,par)
88 l1=drugOptimalControl([],trj.y,par);l2=drugOptimalControl...
      ([],trj2.y,par);l3=drugOptimalControl([],trj3.y,par);
89 hold on;plot(trj.y(1,:),l1(1,:),trj2.y(1,:),l2(1,:),trj3.y...
```

```
    (1,:),l3(1,:)),hold on;plot(x(1),lhat(1),'marker','.','...
    markersize',20),figure(gcf); set(gca,'xlim',[0 2e7])
```

8 Conclusions & Discussion

The main objective of this master thesis was the comparison of two different drug models. The base model was developed by Caulkins and Tragler [11] with a power function as initiation, while the second model instead used a logistic growth function as initiation. The goal was to use the same base case parameters to see if this change of initiation has a critical effect on the results. Unfortunately, no inner equilibrium could be found for the base case parameters for the model with logistic growth, so the value of the social cost parameter κ was lowered down to 0.34 (from 1) as new base case value for the logistic growth model. Note that the base case value of κ for the base model is still 1. With this parameter change, an admissible equilibrium was found, but one of the three controls, enforcement v, was zero throughout the whole analysis, which was not the case for the base model. Due to this, and the radical parameter value change of κ, a precise comparison of the two models is hardly possible. Nevertheless, the results are still of interest.

8.1 Both Models in Their Base Case Condition

With base case conditions for each model, the number of users in the saddle point of the model with logistic growth is more than three times as big as for the base model. At the same time, the sum of control spendings for the base model is more than ten times higher. As κ was lowered to about a third for the logistic growth model, which is the social cost per unit of consumption, the logistic growth model is expected to have less control spending for given the same number of users. But the ratio of the increase of the number and users and the decline of the sum of control spendings comes as a surprise, so this is a rather radical difference between the two models. The DNSS threshold of the base model was somewhere near $\underline{A}$, at least in comparison to the DNSS threshold of the logistic-growth-model, where the DNSS threshold is far more to the right. But the distances between the DNSS threshold and the saddle point of both models are very similar.

8.2 Results from Sensitivity Analyses with Respect to some Parameters

The levels of optimal control spending in equilibrium differed significantly between the two models. While decreasing the strength of prevention h, the saddle point of the base model moved to the left and also control spending decreased. The saddle point of the logistic-growth-function model also moved to the left, but at the same time the level of control spending went up, which is a great difference in comparison. For the model with logistic growth, the optimal spending on prevention was least affected by varying the given parameters. Only for the sensitivity analysis of the strength of prevention, prevention w was affected significantly by causing a convergence to zero by increasing h, until it hit zero for a high value of h. In comparison, in the base model, prevention behaved the same way, but never actually hit the value of zero for values of h between 0 and 1.

When varying the effectiveness of treatment c, both models showed the same behavior, but not to the same extent. More effective treatment lead to higher spending on treatment and also the numbers of users in the steady state were fewer. But compared to the base model, the effect on the location of the saddle-point equilibrium was rather low. Prevention w again was not significantly affected by varying c for both models, but for the base model this lead to less spending on enforcement v, which was zero throughout all sensitivity analyses of the model with logistic growth.

Lowering κ from the respective base case value lead to lowering the DNSS threshold for both models. While each model has a different base case value of κ, the disappearance of the DNSS threshold takes place for about the same amount of lowering κ from the base case value. For both models, the DNSS threshold merged with the saddle-point equilibrium for higher values of κ than the base value. For the logistic growth model, this already happened by raising κ only by a little bit, while for the base model the DNSS threshold was very low compared to the logistic-growth-function model, so it was necessary to raise κ by a whole margin, until the DNSS threshold merged with the saddle-point equilibrium.

Increasing the maximum number of drug users $\bar{A}$ lead to a significant raise of the number of users in the saddle-point equilibrium and also shifted the DNSS threshold to the right. These results are not surprising, as $\bar{A}$ is one of the two parameters of the initiation function and the other one, $\bar{k}$, was not varied, so it was expected that a higher initiation rate would lead to a higher number of users in the steady state. The parameter $\bar{A}$ does not exist for the base model, but varying the initiation exponent α in the base model lead to qualitatively the same result. Note that Caulkins and Tragler [11] kept the rate of initiation under base case conditions constant by also varying k, so a direct comparison is again not possible.

Increasing the lower limit on the number of users $\underline{A}$ lead to the same effect of the DNSS threshold for both models, but not nearly to the same extent. While for the base model increasing $\underline{A}$ lead to a massive shift of the DNSS point to the left, increasing $\underline{A}$ for the model with logistic growth only lead to a very small and insignificant shift to the right of the DNSS point.

To conclude, while to some extent both models behaved the same way, there were some rather drastical differences, especially for the levels of control spending.

A Appendix

A.1 Proof of the Concavity with Respect to u, v, and w of the Hamiltonian in the Optimally Controlled Allocation Problem for $\lambda < 0$ for the Base Model

The first order derivatives of the Hamiltonian H with respect to u, v, and w are given by

$$\begin{aligned} H_u &= -1 + \lambda(-c\beta_u A), \\ H_w &= -1 + \lambda(kA^\alpha p^{-a}\Psi_w), \\ H_v &= -(\kappa\theta A(p^{-\omega})_v + 1) + \lambda(kA^\alpha(p^{-a})_v\Psi - \mu(p^b)_v A), \end{aligned}$$

which yield

$$\begin{aligned} H_{uu} &= -\lambda c\beta_{uu}A, \\ H_{ww} &= \lambda(kA^\alpha p^{-a}\Psi_{ww}), \\ H_{vv} &= -(\kappa\theta A(p^{-\omega})_{vv}) + \lambda(kA^\alpha(p^{-a})_{vv}\Psi - \mu(p^b)_{vv}A), \end{aligned}$$

for the second derivatives, with the derivatives of β, p, and Ψ being

$$\beta_u = z\left(\frac{u}{A+\delta}\right)^{z-1}\frac{1}{A+\delta}, \tag{15}$$

$$\beta_{uu} = z(z-1)\left(\frac{u}{A+\delta}\right)^{z-2}\left(\frac{1}{A+\delta}\right)^2, \tag{16}$$

$$p_v = \frac{e}{A+\epsilon}, \tag{17}$$

$$(p^b)_v = b\left(d + e\frac{v}{A+\epsilon}\right)^{b-1}\frac{e}{A+\epsilon} = bp^{b-1}p_v, \tag{18}$$

$$(p^b)_{vv} = b(b-1)(p_v)^2p^{b-2}, \tag{19}$$

$$(p^{-\omega})_v = -\omega\left(d + e\frac{v}{A+\epsilon}\right)^{-\omega-1}\frac{e}{A+\epsilon} = -\omega p^{-\omega-1}p_v, \tag{20}$$

$$(p^{-\omega})_{vv} = \omega(\omega+1)(p_v)^2p^{-\omega-2}, \tag{21}$$

$$(p^{-a})_v = -a\left(d + e\frac{v}{A+\epsilon}\right)^{-a-1}\frac{e}{A+\epsilon} = -ap^{-a-1}p_v, \tag{22}$$

$$(p^{-a})_{vv} = a(a+1)(p_v)^2p^{-a-2}, \tag{23}$$

$$\Psi_w = (h-1)me^{-mw}, \tag{24}$$

$$\Psi_{ww} = (1-h)m^2e^{-mw}, \tag{25}$$

so H_{uu}, H_{vv}, and H_{ww} are negative, if $\lambda < 0$ holds.

A.2 Proof of the Concavity with Respect to u, v, and w of the Hamiltonian in the Optimally Controlled Allocation Problem for $\lambda < 0$ for the Model with Logistic Growth

The first order derivatives of the Hamiltonian H with respect to u, v, and w are given by

$$H_u = -1 + \lambda(-c\beta_u A),$$

$$H_w = -1 + \lambda(kA(\bar{A}-A)p^{-a}\Psi_w),$$

$$H_v = -(\kappa\theta A(p^{-\omega})_v + 1) + \lambda(kA(\bar{A}-A)(p^{-a})_v\Psi - \mu(p^b)_vA),$$

which yield

$$\begin{aligned} H_{uu} &= -\lambda c \beta_{uu} A, \\ H_{ww} &= \lambda(kA(\bar{A} - A)p^{-a}\Psi_{ww}), \\ H_{vv} &= -(\kappa\theta A(p^{-\omega})_{vv}) + \lambda(kA(\bar{A} - A)(p^{-a})_{vv}\Psi - \mu(p^b)_{vv}A), \end{aligned}$$

for the second derivatives. As (15)-(25) are the same, so H_{uu}, H_{vv}, and H_{ww} are negative, if $\lambda < 0$ holds.

References

[1] AIDS InfoNet, A Project of the International Association of Providers of AIDS Care. What Is Antiretroviral Therapy (ART)? Fact Sheet Number 403. http://www.aidsinfonet.org/fact_sheets/view/403 (03.08.2016).

[2] Analysis by Bazian, Edited by NHS Choices. November 1 2010. Study compares drug harms. http://www.bazian.com/ & http://www.nhs.uk/news/2010/11November/Pages/study-compares-drug-harms.aspx (10.10.2016).

[3] Behrens, D.A., Caulkins, J.P., Tragler, G., Haunschmied, J., and Feichtinger, G. 2000. Optimal control of drug epidemics: Prevent and treat – but not at the same time. Management Science. 46(3) 333-347.

[4] Busch, M., Anzenberger, J., Grabenhofer-Eggerth, A., Kellner, K., Kerschbaum, H., Klein, C., and Türscherl, E. 2015. Gesundheit Österreich GmbH. Epidemiologiebericht Drogen 2015.

[5] Busch M., and Weigl M. 2015. Drogensituation in Österreich - Kontinuität und Veränderung. Tagung Sucht: Epidemiologie und Strategie, 10. Mai 2016.

[6] Bühler, Anneke, and Thrul, Johannes. 2015. Prevention of addictive behaviours. European Monitoring Centre for Drugs and Drug Addiction. ISBN 978-92-9168-824-1.

[7] Caulkins, J.P., Rydell, C.P., Schwabe, W.L., and Chiesa, J. 1997. Mandatory Minimum Drug Sentences: Throwing Away the Key or the Taxpayers' Money? RAND, Santa Monica, CA.

[8] Caulkins, J.P., and Reuter, P. 1998. What Price Data Tell Us About Drug Markets. Journal of Drug Issues, 28(3), 593-612.

[9] Caulkins, J.P., Paddock, S., Pacula, R., and Chiesa, J. 2002. School-based drug prevention: What kind of drug use does it prevent? RAND, Santa Monica, CA.

[10] Caulkins, J.P., Feichtinger, G., Tragler, G., and Wallner, D. 2010. When in a drug epidemic should the policy objective switch from use reduction to harm reduction? European Journal of Operational Research 201, 308-318.

[11] Caulkins, J.P., and G. Tragler. 2016. Dynamic Drug Policy: Optimally Varying the Mix of Treatment, Price-Raising Enforcement, and Primary Prevention Over Time. In: H. Dawid et al. (eds.), Dynamic Perspectives on Managerial Decision Making, Dynamic Modeling and Econometrics in Economics and Finance 22, DOI 10.1007/978-3-319-39120-5_2. Springer International Publishing Switzerland. Pp. 11-35.

[12] Dechert, W.D., and Nishimura, K. 1983. A Complete Characterization of Optimal Growth Paths in an Aggregated Model with a Non-Concave Production Function. Journal of Economic Theory 31(2), 332-354.

[13] derStandard.at. 2016. Ab Juni sollen Drogendeals nicht mehr sichtbar sein (31.05.2016). http://derstandard.at/2000037909790/Ab-Juni-sollen-Drogendeals-nicht-mehr-sichtbar-sein (20.10.2016).

[14] derStandard.at. 2016. Polizeipräsenz gegen Drogendeals auf Wiens Straßen (01.06.2016). http://derstandard.at/2000038054256/Polizeipraesenz-gegen-Drogendeals-auf-Wiens-Strassen (20.10.2016).

[15] derStandard.at. 2016. Was passiert, wenn ein Land alle Drogen legalisiert (16.08.2016). http://derstandard.at/2000042539163/Was-passiert-wenn-ein-Land-alle-Drogen-legalisiert (20.10.2016).

[16] Drug & Alcohol Recovery and Education Centre. Drug Information. http://watershed.org.au/drug-information.html (01.08.2016).

[17] DrugAbuse.com. The Effects of Cocaine Use. http://drugabuse.com/library/the-effects-of-cocaine-use/ (10.06.2016).

[18] Feichtinger, G., and Hartl, R.F. 1986. Optimale Kontrolle Ökonomischer Prozesse – Anwendungen des Maximumprinzips in den Wirtschaftswissenschaften. De Gruyter, Berlin.

[19] Gable, R.S. 2006. Acute toxicity of drugs versus regulatory status. J. M. Fish (Ed.), Drugs and Society: U.S. Public Policy, pp.149-162. Lanham, MD: Rowman & Littlefield Publishers.

[20] Grass, D., Caulkins, J.P., Feichtinger, G., Tragler, and G., Behrens, D.A. 2008. Optimal Control of Nonlinear Processes: With Applications in Drugs, Corruption and Terror. Springer, Berlin.

[21] Harm Reduction Coalition. Mixing Drugs. http://harmreduction.org/issues/overdose-prevention/overview/ overdose-basics/opioid-od-risks-prevention/mixing-drugs/ (10.06.2016).

[22] Harwood, H., D. Fountain, and G. Livermore. 1998. The Economic Costs of Alcohol and Drug Abuse in the United States, 1992. Bethesda, MD: National Institute on Drug Abuse and National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health.

[23] Harwood, H.J., and Myers, T.G. (eds.). 2004. New Treatments for Addictions. Washington, DC: National Academy Press.

[24] Institute of Medicine (IOM). 1990. Treating Drug Problems. National Academy Press, Washington DC.

[25] Institute of Medicine (IOM). 1996. Pathways of Addiction: Opportunities in Drug Abuse Research. National Academy Press, Washington DC.

[26] Léonard, D., and Long, N.V. 1992. Optimal Control Theory and Static Optimization in Economics. Cambridge University Press, Cambridge.

[27] Miller, T.R., Cohen, M.A., and Wiersema, B. 1996. Victim Costs and Consequences: A New Look. National Institute of Justice, Washington, DC.

[28] Moore, T. 2007. Monograph No. 14: Working estimates of the social costs per gram and per user for cannabis, cocaine, opiates and amphetamines. DPMP Monograph Series. Sydney: National Drug and Alcohol Research Centre.

[29] National Institute on Drug Abuse (NIDA), National Institutes of Health, and U.S. Department of Health and Human Services. 1999. Principles of Drug Addiction Treatment - A Research-Based Guide. NIH Publication No. 12-4180.

[30] National Institute on Drug Abuse (NIDA), National Institutes of Health, and U.S. Department of Health and Human Services. 2016. Cocaine. https://www.drugabuse.gov/publications/research-reports/cocaine/what-treatments-are-effective-cocaine-abusers (12.10.2016).

[31] Nutt, David J. et al. 2010. Drug harms in the UK: a multicriteria decision analysis. The Lancet, Volume 376, Issue 9752, 1558 - 1565.

[32] Office of National Drug Control Strategy. 1996. The National Drug Control Strategy: 1996. The White House, Washington, DC.

[33] Österreichische Arbeitsgemeinschaft Suchtvorbeugung. https://www.suchtvorbeugung.net/stepbystep/pages/home.php (26.09.2016).

[34] Rogov, P. 2014. The War On Drugs: Hawks, Doves and Owls (05.06.2014). Global Research. http://www.globalresearch.ca/the-war-on-drugs-hawks-doves-and-owls/5385498 (20.10.2016).

[35] Rydell, C.P., and Everingham, S.S. 1994. Controlling Cocaine. Supply Versus Demand Programs. RAND, Santa Monica, CA.

[36] Rydell, C.P., Caulkins, J.P., and Everingham, S.S. 1996. Enforcement or Treatment? Modeling the Relative Efficacy of Alternatives for Controlling Cocaine. Operations Research 44, 1-9.

[37] Sethi, S.P. 1977. Nearest feasible paths in optimal control problems: Theory, examples, and counterexamples. Journal of Optimization Theory and Applications 23(4), 563-579.

[38] Sethi, S.P. 1979. Optimal advertising policy with the contagion model. Journal of Optimization Theory and Applications 29(4), 615-627.

[39] Skiba, A.K. 1978. Optional Growth with a Convex-Concave Production Function, Econometrica 46, 527-539.

[40] Stedman's Medical Dictionary © Wolters Kluwer Health. Online Lexicon, MediLexicon International Ltd ©2004-2016. http://www.medilexicon.com/ (12.06.2016).

[41] Tesseract2. "Scoring drugs", The Economist, data from "Drug harms in the UK: a multi-criteria decision analysis", by David Nutt, Leslie King and Lawrence Phillips, on behalf of the Independent Scientific Committee on Drugs. The Lancet. 2010 Nov 6. doi:10.1016/S0140-6736(10)61462-6. https://commons.wikimedia.org/w/index.php?curid=17243892 (28.07.2016).

[42] The Foundation for a Drug-Free World. Cocaine: A Short History. http://www.drugfreeworld.org/drugfacts/cocaine/a-short-history.html (31.07.2016).

[43] theguardian. 2016. Philippines president Rodrigo Duterte urges people to kill drug addicts (01.07.2016). https://www.theguardian.com/world/2016/jul/01/philippines-president-rodrigo-duterte-urges-people-to-kill-drug-addicts (20.10.2016).

[44] theguardian. 2016. Philippine president Rodrigo Duterte to extend drug war as 'cannot kill them all' (19.09.2016). https://www.theguardian.com/world/2016/sep/19/philippine-president-rodrigo-duterte-extend-drug-war-kill-them-all (20.10.2016).

[45] The Transnational Institute (TNI). Coca leaf: Myths and Reality. https://www.tni.org/en/primer/coca-leaf-myths-and-reality (12.06.2016).

[46] Thundermaker (Own work). 2010. Drug safety and dependence. GFDL or CC BY-SA 4.0-3.0-2.5-2.0-1.0. Wikimedia Commons. https://upload.wikimedia.org/wikipedia/commons/c/c9/Drug_safety_and_dependence.png (10.10.2016).

[47] Tragler, G., Caulkins, J.P., and Feichtinger, G. 1997. The Impact of Enforcement and Treatment on Illicit Drug Consumption. Working Paper 212, Department of Operations Research and Systems Theory, Vienna University of Technology, Vienna.

[48] Tragler, G., Caulkins, J.P., and Feichtinger, G. 2001. Optimal dynamic allocation of treatment and enforcement in illicit drug control. Operations Research 49(3), 352-362.

[49] Uchtenhagen, A., and Zieglgänsberger, A. 2000. Suchtmedizin. München/Jena. Urban und Fischer Verlag, ISBN-10: 3437217801, ISBN-13: 978-3437217807.

[50] United Nations Office on Drugs and Crime (UNODC). 2015 World Drug Report. United Nations publication, Sales No. E.15.XI.6. ISBN: 978-92-1-148282-9.

[51] Verhulst, P.F. 1838. Notice sur la loi que la population suit dans son accroissement. Correspondence mathématique et physique. 10, 113-121.

[52] Verhulst, P.F. 1845. Recherches mathématiques sur la loi d'accroissement de la population. Nouveaux Mémoires de l'Académie Royale des Sciences et Belles-Lettres de Bruxelles. 18, 1-42.

[53] Weigl, M., Anzenberger, J., Busch, M., Horvath, I., and Türscherl, E. 2015. Gesundheit Österreich GmbH. Bericht zur Drogensituation 2015. ISBN 978-3-85159-194-1.

[54] wien.orf.at. 2016. U6-Drogenszene: Problem für Lokale (21.04.2016). http://wien.orf.at/news/stories/2769493/ (20.10.2016).

[55] Wikipedia Contributors. 2016, July 8. Bevahorial Addiction. https://en.wikipedia.org/w/index.php?title=Behavioral_addiction&oldid=728970047 (13.07.2016).

[56] Wikipedia Contributors. 2016, October 2. Droge. https://de.wikipedia.org/w/index.php?title=Droge&oldid=158388049 (10.10.2016).

[57] Wikipedia Contributors. 2016, September 3. Substance abuse prevention. https://en.wikipedia.org/w/index.php?title=Substance_abuse_prevention& oldid=737528228 (26.09.2016).

[58] Zimmerman, Janice L. 2012. Cocaine Intoxication. Critical Care Clinics, Volume 28, Issue 4, 517-526.