From Pain Patient to Junkie: An Economic Theory of Painkiller Consumption and its Impact on Wellbeing and Longevity [*]

Holger Strulik[†]

Final Version. January 2021.

Abstract. In this paper, I propose a life cycle model of painkiller consumption that combines the theory of health deficit accumulation with the theory of addiction. Chronic pain is conceptualized as a persistent negative shock to lifetime utility that can be treated by pain relief medication. Individuals treated with opioid pain relievers (OPR) develop addiction, which increases their demand for opioids and reduces their welfare and life expectancy through side effects and potential overdose. I calibrate the model for a benchmark American and investigate the comparative dynamics of alternative drug characteristics, pain intensities, and ages of onsets of pain as well as their implications for welfare and life expectancy. Computational experiments are used to identify fully rational and imperfectly rational addiction behavior. Fully rational addicts reduce OPR use when new information about the addictive potential of these drugs arrives. Imperfectly rational addicts further develop their addiction and switch to illicit opioid use. Likewise, a discontinued prescription helps fully rational addicts to quit quickly, while it induces imperfectly rational individuals to take up heroin. I also discuss treatment of OPR addiction and the use of opioids in palliative care.

Keywords: pain, pain relief, addiction, opioid epidemic, health deficits, life expectancy, illicit drugs.

JEL: D15, D91, I10, I12.

[†] University of Goettingen, Department of Economics, Platz der Goettinger Sieben 3, 37073 Goettingen, Germany; email: holger.strulik@wiwi.uni-goettingen.de.

1. INTRODUCTION

Economists have developed theories on how health affects productivity (e.g. Grossman, 1972), the utility experienced from consumption (e.g. Finkelstein et al., 2013; Schuenemann et al., 2017a), the length of life (e.g. Ehrlich and Chuma, 1992; Dalgaard and Strulik, 2014), or the survival probability of individuals (e.g. Kuhn et al., 2015; Schuenemann et al., 2017b). This paper proposes a new theory that explores a separate channel through which health matters directly for wellbeing, life cycle choices, and longevity, namely through the experience of pain. This allows the discussion of a second form of treatment of illness, aside from investments in health maintenance and repair: the treatment of pain by analgesics (painkillers). Individuals are assumed to have alternative painkillers at their disposal that differ in price, efficacy, side effects on health, and the potential of addiction. Embedded in a life cycle theory of addiction, we then ask under which conditions individuals prefer pain treatment with opiod pain relievers (OPRs), quit using OPRs or switch to illicit opioid consumption, and whether OPR addiction can be rational. This discussion will take place in the context of the American opioid crisis (see Section 2).

The life cycle theory of addiction takes explicitly into account that life length is finite, endogenous, and affected by health and addiction behavior. When life is finite no steady state (of addiction) exists. This is so because a steady state is only reached asymptotically as time goes to infinity, which is impossible if life is finite. Formally, the boundary conditions for finite and infinite life are different such that lifetime behavior of individuals with finite and infinite life differs. That this distinction is relevant for OPR use is obvious when we consider opioid treatment in palliative care where remaining life expectancy is short and addiction is unlikely to fully develop compared to an opioid treatment of back pain in young age. In this sense, the proposed theory differs conceptually from most of the related addiction literature, where behavior is discussed at the steady state, and is thus (explicitly or implicitly) based on the assumption of an infinite life (e.g. Becker and Murphy, 1988; Orphanides and Zervos, 1995, 1998; Gruber and Koeszegi, 2001; see Strulik, 2018, for an exception). A life cycle theory allows to consider individuals who take into account the side effects of opioid use on health and the probability of premature death through overdose.

We discuss painkiller addiction under two different behavioral assumptions. According to the theory developed by Becker and Murphy (1988), individuals understand how their addiction

develops and they take this knowledge into account when they make their life cycle plans. This means that they optimally plan and control their addiction (if there is any). The consideration of fully rational addiction has been dubbed TORA (theory of rational addiction; Cawley and Ruhm, 2012). Alternatively, Strulik (2018) has proposed a theory of imperfectly rational addiction, in which otherwise fully rational individuals fail to optimally plan and control their addiction. We call this behavior imperfectly rational and abbreviate it analogously as TICA (theory of imperfectly controlled addiction).

This study shows analytically and with numerical experiments that TORA and TICA addicts respond fundamentally different to price and information shocks. An information shock assumes that an OPR, that formerly was thought to be non-addictive, is found to be addictive. As an optimal response to this information, TORA addicts are predicted to always reduce their consumption and in many cases the reduction leads eventually to an endogenous quitting of OPR use. TICA addicts, in contrast, do not respond with reduced consumption, but further develop their addiction, and consume even more OPRs. In a second computational experiment, it is shown that, after the termination of an OPR prescription, TORA addicts quit instantaneously while TICA addicts switch to the use of illicit opioids (heroin). These predictions can be used to identify whether painkiller addicts are fully rational and able to control their addiction. Clearly, drug policies (beyond information) are particularly in need if addiction is not fully rational. For the benchmark calibration it is estimated that an ideal methadone treatment would increase the value of life of an imperfectly rational OPR addict by about 13 percent and life expectancy at 20 by about 9 years.[1]

OPR consumption behavior predicted for TICA individuals could possibly be explained as well by other theories of addiction under bounded rationality, for example, based on assumptions of myopia or time-inconsistent decision making (although these theories are not yet available as life cycle theories). However, the assumptions needed to explain addiction would be much stronger because they would imply suboptimal (myopic or inconsistent) decision making also for all other domains of intertemporal choice. The assumption that individuals cannot perfectly control an addiction is minimal-invasive because it allows addicts to make rational decisions in other areas of daily life. It supports a benign view on painkiller addicts in the sense that their

---

[1]An ideal methadone treatment refers to a treatment with a methadone that is equally effective as heroin but not addictive.

only imperfection is their failure to control the addiction. This view seems to be particulary appropriate to address an opioid addiction problem that has hit individuals from all strata of society. It is policy-relevant since TICA individuals need support for quitting their addiction. Full disclosure of the addictive potential of OPRs is not sufficient for them to quit and the discontinuation of an OPR prescription pushes them to illicit opioid use and further deteriorates their life.

In order to investigate how the unintentional transition from pain patient to opioid addict affects health, longevity, and welfare, the theory of painkiller addiction is embedded in the life cycle model of health deficit accumulation by Dalgaard and Strulik (2014). The health deficit model implements the insight from medical science that individuals, as they grow older, develop health disorders, ranging from mild nuisances to serious conditions. Health deficits are measured by the so called frailty index (Mitnitski et al., 2002a). The frailty index provides the relative number of health deficits that an individual has from a long list of potential deficits. It has been shown that a quasi-exponential association between age and the frailty index exists and that the frailty index predicts death with high precision (Mitnitski et al., 2002b). Given the observable measure of health deficits, the health deficit model is straightforward to calibrate and it has a microfoundation in biology, based on reliability and redundancy of body cells (Gavrilov and Gavrilova, 1991).[2] Strulik (2018) uses the health deficit model to discuss addiction with respect to smoking. The study shows that, in a life cycle context, fully rational individuals increase consumption of addictive goods in old age and, in particular, shortly before death. Since actual cigarette consumption declines in old age, the theory questions whether TORA behavior appropriately captures cigarette addiction. Increasing end-of-life consumption is less suitable to exclude TORA behavior for opioids. The present paper thus proposes two new behavioral predictions to test for TORA addiction.

The theory of painkiller consumption assumes that pain is caused either instantaneously or gradually by the arrival of health deficits and is captured by a downward shift of the utility function. This feature makes pain observationally similar to depression as conceptualized in

---

[2]The health capital model (Grossman, 1972), in contrast, is based on a latent variable, health capital, which is unknown in the medical sciences. See Zweifel and Breyer (1997), Case and Deaton (2005), Almond and Currie (2011), and Dalgaard et al. (2020) for a critique of the health capital model. Other applications in economics using the health deficit approach include the Preston curve (Dalgaard and Strulik, 2014), the historical evolution of retirement (Dalgaard and Strulik, 2017), the role of adaptation for health behavior (Schünemann et al., 2017a), and the gender-gap in mortality (Schünemann et al., 2017b).

Strulik (2019) since both pain and depression reduce utility without apparent change in the fundamentals that typically enter the utility function. Indeed, it has been found that depression and pain share the same biological pathways and neurotransmitters and often respond to similar treatments (Bair et al., 2003; Verdu et al., 2008). The main difference between the present study and Strulik (2019) is the modeling and analysis of treatment. While in Strulik (2019) treatment is regarded to be always health- and welfare-improving we here explicitly consider its dark side. In particular, individuals may become addicted to the treatment, as increasing tolerance may motivate the consumption of higher doses of treatment. The cravings from addiction may generate pain that motivates to sustain treatment even when the original cause of pain is gone. Also, in contrast to Strulik (2019), treatment may have side-effects on health and bear the risk of death from overdose. Another distinguishing feature from Strulik (2019) is that individuals consider a vector of potential treatments with different prices, efficacy, and health repercussions.

The paper is organized as follows. To motivate the theory, the next section provides a brief overview of the literature on pain, pain treatment, and the opioid crisis. In Section 3, I integrate utility-reducing pain, treatment by alternative analgesics and illicit drugs, opioid addiction, and overdose death, into a life-cycle model of endogenous health and longevity. In Section 4, propositions on the determinants of painkiller use are proven. In Section 5, I parameterize and solve the full model and calibrate it for a benchmark U.S. American and three stylized painkillers. In Section 6, I present the main results on painkiller use and its effects on wellbeing and longevity. I show robustness of the conclusion that fully informed TORA individuals do not use OPRs for pain reduction and that a notable exception is opioid use in palliative care, i.e. when pain is combined with drastic life-shortening health shocks. In Section 7, I use computational experiments to identify the behavioral responses of fully rational and imperfectly rational OPR addicts to information and prescription shocks. I investigate the transition to non-medical OPR-use and heroin consumption and estimate the gains in terms of life expectancy and value of life from an ideal methadone treatment of OPR addiction. Section 8 concludes the paper.

2. Pain, Pain Treatment and the Opioid Crisis

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." (IASP Subcommittee on Taxonomy, 1979). One way to distinguish the experience of pain is by its duration. Acute pain

is usually transitory and lasts only until the causing health deficits are repaired or the cause of pain is removed. Here we focus on chronic pain that lasts longer, beyond any expected period of healing, and perhaps life-long. In this case, pain has lost its useful function as a warning signal of tissue damage, while pain management (rather than the repair of physical health deficits) is the main focus of treatment.

Chronic pain is widespread. According to estimates published by the Center for Disease Control and Prevention, 20.4% (50.0 million) of U.S. adults experienced chronic pain in the year 2016 (Dahlhamer et al., 2018). Chronic pain is highly prevalent in other developing and developed countries as well (Tsang et al., 2008), but in the U.S. the prevalence of chronic pain is significantly higher than anywhere else (Blanchflower and Oswald, 2019). Of those who reported chronic pain, 40% noted that they were constantly in pain (American Academy of Pain Management, 2003).[3] While it is obvious that the presence of pain reduces utility, it is more difficult to quantify its importance for wellbeing and life satisfaction. A recent study by Olafsdottir et al. (2020) provides such estimates based on the compensation variation method. It suggests that individuals who experience chronic pain would need to receive, on average, between 56 and 145 US Dollar per day in order to experience the same life satisfaction as in a (counterfactual) life without pain. Richer individuals and those who suffer from more severe pain exhibit a larger compensation value, i.e. a greater willingness to pay for a pain-free life.

Methods of pain relief differ in price, efficacy, and side effects. The WHO and other pain management guides recommend to treat mild to moderate pain with non-opioid pain relievers like Acetaminophen or non-steroidal anti-inflammatory drugs (NSAIDs) as, for example, panadol or ibuprofen. These analgesics are, however, of limited efficacy in relieving chronic pain (from, for example, osteoarthritis, back pain, or cancer). Treatment is usually regarded effective when it reduces pain by 50% and in many instances treatment is no more effective than a placebo.[4] Moreover, treatment with analgesics is subject to the so called ceiling effect. This means that there exists a limit as to how much pain can be reduced with a higher dosage. Above this level, increasing dosage does not relieve more pain but increases the risk of serious side effects.

---

[3]Pain is most prevalent among middle-aged Americans and this feature can be attributed to a period effect rather than to a mere age effect. Specifically, later born cohorts, at any age, experience more pain than earlier cohorts, an observation, which is unique to the USA and presumably originates from the opioid epidemic and its diffusion through society (Case et al., 2020).

[4]Since the response to analgesics is highly idiosyncratic, efficacy of pain relief is assessed by the number-needed-to-treat (NNT). This statistics is the number of persons who must be treated for one person to receive a certain effect. This effect is frequently calibrated as 50% pain relief (Katz et al., 2015).

More severe pain could be treated by weak opioids like codeine or strong opioid pain relievers (OPRs) such as morphine or oxycodone. Treatment with opioids may be more effective because it shuts off pain signals in the brain. However, with prolonged use of OPRs, the production of the body's endogenous opioids is inhibited and the opioid receptors' signaling mechanism adapts to the treatment. Tolerance occurs, which means that patients no longer respond to the treatment as strongly as they did initially and higher doses are required to achieve the same effect. Tolerance increases the risk of overdose, i.e. to apply the drug in larger quantities than recommended, which may result in a toxic state or death. Adaptation and reduced endogenous opioid production lead to withdrawal symptoms, i.e. craving and pain if the drug treatment is discontinued. In short, people become addicted (NIDA, 2018a).

That doctors and patients were aware of the threat of addiction certainly contributed to the fact that until the 1980s the use of prescription OPRs was mainly confined to treat acute pain and cancer pain (in palliative care). Then, a series of research papers argued that OPRs could be prescribed on a long-term basis with insignificant risk to addiction (e.g. Portenoy and Foley, 1986; Zenz et al., 1992), the pharmaceutical industry developed new slow-release OPRs (oxycontin) and convinced many physicians, in particular in the U.S., that OPRs can be prescribed safely and more freely, and an increasing share of OPRs were paid by insurance (Zhou et al., 2016). In 1997, the American Pain Society and the American Academy of Pain Medicine issued a consensus statement endorsing opioid use for chronic pain (Haddox et al., 1997).

As a result of these developments, opioid use in the U.S. began to accelerate rapidly in the mid 1990s. Opioid prescription quadrupled from 1990 to 2010, which can be attributed mainly to increasing OPRs treatment of chronic noncancer pain (CDC, 2017b). As a result, many patients developed an addiction and OPR-overdose deaths increased by about fivefold (CDC, 2017a). From 1999 to 2016, more than 630,000 people died from drug overdose. In 2016, more than 63,600 people died from drug overdoses, making it the leading cause of injury-related death in the United States (CDC, 2017a). While several other countries prescribed opioids more freely as well, the U.S. is exceptional in the sheer size of the phenomenon, which has been officially dubbed an epidemic. Americans contribute about 80% percent to the world-wide consumption of oxycontin (Volkow, 2014) and, on per capita terms, Americans consume about four times as much morphine equivalents as Europeans (CDC, 2017b). By the year 2016, about 2.0 million U.S. Americans had developed an addiction associated with prescription opioids (CDC, 2017b).

Since 2010, a second wave of opioid deaths developed. Prescriptions of OPRs stabilized and then decreased mildly (by about 20% from 2006 to 2017), leveling off at a level three times as high as 1999 (CDC, 2017b). Death from prescription OPR overdose also leveled off since about 2010, while overdose death from heroin rose sharply, by about factor 5 from 2010 to 2016 (CDC, 2017a). The surge of heroin consumption and the decline of OPR prescriptions are likely to be causally related. In the early 21st century it became obvious that prescription OPRs are chemically similar to heroin, act on the same brain systems, and are of similar addiction potential. In 2007, the developer of oxycontin pled guilty to criminal charges for misrepresenting the risk of addiction (van Zee, 2009). Health care providers gradually prescribed OPRs more reluctantly and the CDC re-reformed their recommendation of pain treatment (CDC, 2016). When prescription runs out, addicted users have an incentive to avoid withdrawal pain by switching to illicit opioids. Indeed, four out of five current heroin users report that their opioid use began with opioid pain relievers (Kolodny et al., 2015) and 94 percent of opioid users state to use heroin because prescription opioids are far more expensive and harder to obtain (Cicero, 2014). The new heroin wave thus fundamentally differs from the heroin waves in the 1950s and 60s. The typical heroin addicts are no longer poor residents of the inner city who started consumption for recreational purposes but white middle-class residents of the suburbs, who accidently became addicted to heroin by a generous OPR treatment of chronic pain. A study based on descriptions of decedents of OPR-overdose victims found that 87 percent of the deceased had used prescribed pain medication in the year before death (Johnson et al., 2013).[5]

The context of the opioid crisis relates this paper to a series of recent economic studies. Case and Deaton (2018) observed increasing mortality of middle-aged white Americans without a college degree and attributed it to increasing deaths from suicide, alcohol-related liver diseases, and drug overdoses. They dubbed this phenomenon as "deaths of despair" and hypothesized that it may be driven by declining wages, declining labor force participation, declining marriage rates, and more broadly by an increasing lack of opportunity for people without a college degree. Using county level data, Ruhm (2018) finds little support for the idea that deteriorating macroeconomic indicators fueled the opioid epidemic and argues instead that increasing overdose deaths are largely driven by higher availability and lower costs of opioids. Krueger (2017) argues that

---

[5]This paper focusses on the pathways from pain patient to opioid addict. The initiation of OPR use for recreational purposes could be easily integrated into the theory. See Volkow et al. (2018) for a review on the use and misuse of OPRs.

increasing opioid consumption might explain parts of the decline in labor force participation since the turn of the century. Currie et al. (2018) find that opioid prescription rates had no significant effect on male employment and a small positive effect on labor force participation of women. Schnell (2017) discusses a model of physician behavior, when OPRs can be obtained legally as well as illegally. Grossmann and Strulik (2018) propose a macroeconomic model to analyze the impact of deteriorating economic status and declining opioid prices and argue that both trends are necessary to motivate increasing opioid use of the middle class. Evans et al. (2018) argue that abuse-deterrent oxycontin, which entered the market in 2010, is associated with fewer OPR-related deaths and more heroin deaths with no effect on total deaths from overdose. Alpert et al. (2019) identify generous OPR prescription policies as a major determinant of opioid deaths across U.S. states. Strulik (2020) investigates the epidemic character of opioid addiction in a model of social contagion.

3. The Model

3.1. **Pain, Painkiller Consumption, and Addiction.** Pain is modeled as a health-related spontaneous or gradual downward shift of the utility function. The intensity of pain P depends non-negatively on the number of accumulated health deficits D and – if the individual is addicted – on the severity of addiction z such that $P(D, z)$ with $\partial P / \partial D \geq 0$, $\partial P / \partial z > 0$, and $P(0, 0) = 0$. Many aging-related health deficits are not associated with pain (e.g. shortsightedness, incontinence, dementia, or general weakness). Instead, a *specific* health deficit, such as sciatia, is the cause of pain and pain does not increase (much) by the arrival of other health deficits. The case of $\partial P / \partial D = 0$ is thus a useful benchmark in order to elaborate the main mechanisms of pain. As a robustness check, we investigate aging related pain, conceptualized as continuous $\partial P / \partial D > 0$ and verify that all main results are preserved. For the benchmark case, we furthermore assume that pain is chronic in the sense that it persists until death. In further applications we consider pain shocks at different ages of onset. We also investigate the case where pain is associated with a drastic increase in health deficits as, for example, malignant pain, and the implications for palliative care at the end of life.

Pain may be partially suppressed with painkillers. There exists a variety of painkiller drugs, indexed by $j = 1 \ldots J$. We assume that painkillers are substitutes such that, for every given state, individuals take either no painkiller or their most preferred painkiller. This assumption

allows individuals to change from one painkiller to another (and back) over time, while it requires that they take at most one painkiller at any instant of time. This minimal-invasive assumption of no simultaneous mixing allows for a closed-form solution of painkiller consumption. If several painkillers fulfil the first order condition for optimal use, individuals select the painkiller that provides the greatest net benefit. Net benefit is defined as the increase in utility through pain reduction minus the monetary and health costs of painkiller use (evaluated in utility units).

Let m^j denote the quantity consumed of painkiller j and let m denote the vector of quantities consumed of all painkillers, $m = \{m^1, \ldots, m^J\}$. Notice that, if at time t painkiller i is taken, $m^i(t) > 0$ and $m^j(t) = 0$ for all $j \neq i$. Taking painkillers reduces pain. Let $g(m)$ denote the fraction of pain that remains after taking painkillers such that $0 \leq g(m) \leq 1$ and $\partial g / \partial m < 0$. We assume declining marginal efficacy $\partial^2 g / \partial(m)^2 > 0$.

Let $u(c)$ denote the utility experienced from consumption, with $u' > 0$ and $u'' < 0$. Instantaneous utility is then given by

$$U = u(c) - P(D, z)g(m). \tag{1}$$

Throughout the paper, we assume that pain is mild enough for utility to stay positive and individuals have no reason to kill themselves intentionally. However, they may kill themselves unintentionally by overdose (see below).[6] The multiplicative coupling of P and g implies that painkiller consumption is completely ineffective when there is no pain (for $P = 0$). This means that we ignore the possibility of individuals consuming painkillers merely for the experience of pleasure. In the case of opioids, a recreational motive of drug use certainly exists. Here, however, we want to focus on experienced pain as the so far less explored pathway to addiction.[7]

The feature that utility is separable in goods consumption and pain implies that rich individuals (who consume more) do not experience pain differently (i.e. more or less severely) than poor individuals. It will be shown below that this assumption nevertheless captures the feature

---

[6]Empirically, it is difficult to determine whether overdose deaths are unintentional and some may be characterized as successful suicides (Cheatle, 2011; Madadi and Persaud, 2014).

[7]A recreational motive for painkiller use can easily be implemented in the current model by augmenting the utility function such that $U = u(c) - P(D, z)g(m) + f(m)$, in which $f(m)$ denotes the pleasure that pain-free individuals derive from painkiller consumption. The extension can additionally explain how pain-free individuals initiate an addiction, a phenomenon that has been widely discussed in the literature (e.g. Becker and Murphy, 1988; Cawley and Ruhm, 2012; Strulik, 2018). Notice that pain includes the cravings from addiction, which means that drug consumption of addicted individuals without physical pain is actually captured by the utility function in equation (1). For $f(m) = 0$, however, painkiller consumption was initiated by the past experience of physical pain in the life history of the individual. A recreational initiation of drug consumption could be included without loss of generality through $f(m) > 0$ but it would blur the focus of analysis.

that rich individuals experience pain differently in a relative sense such that they are willing to pay more to get rid of pain. The assumptions on $P(D, z)$ and $g(m)$ are sufficient such that the utility function fulfils the three defining features of addiction (Cawley and Ruhm, 2012): tolerance, $\partial U/\partial z < 0$, reinforcement $\partial^2 U/(\partial z \partial m) > 0$, and withdrawal, $\partial U/\partial m > 0$.

Following Becker and Murphy (1988), the strength of addiction is measured by the stock of addictive capital z. Addictive capital is always non-negative and strictly positive if the individual is addicted. The stock of addictive capital evolves according to

$$\dot{z} = \sum_{j=1}^{J} \alpha^j m^j - \psi z, \tag{2}$$

in which $\alpha^j \geq 0$ is the addictive power of drug j. For non addictive painkillers $\alpha^j = 0$. Like for all other parameters, we may think of α^j as being individual-specific such that some substances are addictive for some individuals but not for others. The parameter ψ measures the "depreciation rate" of addictive capital, i.e. the rate of disappearance of the physical and mental effects of past consumption of the painkiller.

3.2. **Health, Aging, and Life Cycle Behavior.** The model of painkiller consumption is embedded in the theory of health deficit accumulation. The theory is motivated by gerontological research showing that individuals, as they get older, develop new health deficits in a quasi-exponential way (Mitnitski et al., 2002a,b). The health deficit model of Dalgaard and Strulik (2014) considers that investments in health maintenance and repair h slow down the speed of health deficit accumulation. Here, we additionally consider that the consumption of (some) painkillers can be harmful for health. This means that new health deficits develop as

$$\dot{D} = \mu \left[D - Ah^\gamma + \sum_{j=1}^{J} B^j m^j - a \right], \tag{3}$$

in which μ is the "natural" force of aging, A and γ reflect the state of medical technology in health maintenance and repair, and a captures environmental effects (as in Dalgaard and Strulik, 2014). Additionally, B^j measures the unhealthiness of painkiller j. Examples for painkiller-specific side effects are discussed in conjunction with the calibration of the model.

Death is conceptualized as a stochastic event, which occurs with higher probability when many health deficits have been accumulated. Specifically, survival probability S is a negative function of health deficits D and the degree of addiction z. The latter captures the phenomenon of dying

incidentally (i.e. for any given state of health) from drug overdose. The mortality rate is given by $q = -\dot{S}/S$; it provides the probability to die at age t conditional on survival up to age t. There exists an upper limit of health deficits beyond which survival is impossible, $S(D, z) = 0$ for $D \geq \bar{D}$.[8]

Individuals receive a flow income w from work if working and from pensions when retired. Income is spent on consumption, saving, investments in health maintenance and repair and on reducing pain. This means that individual wealth k evolves according to

$$\dot{k} = w + (r + q)k - c - p\phi h - \sum_{j=1}^{J} \phi_m^j p_m^j m^j, \tag{4}$$

in which r is the interest rate, q is the mortality rate (i.e. we assume insurance by perfect annuities), p is the price of health investments, and ϕ is the out-of-pocket share of health expenditure for maintenance and repair. The price of painkiller drug j is denoted by p_m^j and the associated out-of-pocket share is ϕ_m^j. We allow for the existence of several types of painkillers that differ in side effects on health B^j, addictive power α^j, and efficacy in pain reduction, denoted by η^j (and introduced in detail later), as for example, mild analgesics like paracetamol (low η^j, low α^j, low B^j), more effective and potentially addictive prescription opioids like oxycontin (high η^j, high α^j, low ϕ_m^j), and illicit drugs like heroin (high η^j, high α^j, and high B^j, and high ϕ_m^j).

Life satisfaction is conceptualized as individual welfare and defined as expected discounted utility experienced over the course of life, $\int_0^T S(D, z)U e^{-\rho t}dt$, in which ρ denotes the rate of pure time preference and $S(D, z)$ denotes the survival probability. Individuals maximize expected lifetime utility by choosing consumption c, health investments h, and painkiller intake m^j. All state and control variables are non-negative. However, it will turn out that aside from painkiller consumption all control variables are always strictly positive such that we omit the respective non-negativity constraints.

We distinguish two types of individuals. According to the theory or rational addiction (TORA), individuals optimally plan and control their addiction (if there is any). The theory of imperfectly controlled addiction (TICA) in turn predicts, that otherwise fully rational

---

[8]In a more complex model one could also imagine that mortality risk from overdose depends directly on m. Here, we focus on the impact of addiction z on overdose. The feature that z is a state variable and cannot jump prevents an upward shift of S when individuals quit consuming addictive painkillers for all reasonable parameterizations of the model.

individuals fail to control how their addiction develops. Formally, this means that TICA individuals maximize expected lifetime utility taking into account the constraints (3) and (4) while TORA individuals additionally take into account constraint (2). The associated current-value Hamiltonian is

$$H = S(D, z) \left[u(c) - P(D, z) \cdot g(m) \right] + \lambda_k \left[w + (r + q)k - c - p\phi h - \sum_{j=1}^{J} \phi_m^j p_m^j m^j \right]$$

$$+ \lambda_D \mu \left[D - Ah^\gamma + \sum_{j=1}^{J} B^j m^j - a \right] + \Phi \lambda_z \left[\sum_{j=1}^{J} \alpha^j m^j - \psi z \right], \tag{5}$$

in which λ_k, λ_D, and λ_z are the costate variables for capital, health deficits, and addiction capital. Notice that health deficits and addiction reduce expected utility such that we expect $\lambda_D \leq 0$ and $\lambda_z \leq 0$. The parameter $\Phi \in \{0, \ 1\}$ in (5) is a toggle variable that distinguishes between the two methodological approaches to addiction. The TORA assumption of fully rational addiction is applied by $\Phi = 1$, which means that individuals take into account how their addiction develops in their life cycle planning. $\Phi = 0$ applies the TICA assumption such that individuals do not optimally plan their life-cycle trajectory of addiction. Notice that, aside from the view on addiction planning, both methods of obtaining individual behavior are identical. In particular, TICA individuals are not myopic or inconsistent. They fail to optimally control their addiction but otherwise try to optimally control their life, given this behavioral constraint.

Individuals maximize (5) given the initial values $D(0)$, $k(0)$, and $z(0)$ and the boundary conditions, $D(T) = \bar{D}$, $k(T) = \bar{k}$, and $\lambda_z(T) = 0$. This problem is known as a free-terminal time problem in optimal control theory. The maximum life span T is reached when the health deficit index reaches its maximum $\bar{D}$. However, facing stochastic survival $S(D, z)$, individuals expect to die earlier. Notice that there is no condition for addiction $z(T)$. Instead, the necessary transversality condition requires the shadow price of addiction to be $\lambda_z(T) = 0$ (see e.g. Theorem 7.1 in Acemoglu, 2009). The first order conditions for a maximum are:

$$S(D, z) \frac{\partial u(c)}{\partial c} = \lambda_k, \tag{6}$$

$$- \lambda_D \mu A \gamma h^{\gamma-1} = \lambda_k \phi p, \tag{7}$$

$$- S(D, z) P(D, z) \frac{\partial g(m)}{\partial m^j} \leq p_m^j \phi_m^j \lambda_k - \lambda_D \mu B^j - \Phi \lambda_z \alpha^j \quad \text{with} = \text{for } m^j > 0. \tag{8}$$

The left-hand sides of these first order conditions show the marginal benefits and the right-hand sides the marginal costs. Equation (6) equates the marginal utility from consumption with the marginal cost from consumption, which is one unit of savings evaluated with the shadow price of wealth λ_k. Equation (7) requires that the marginal benefit of health investments equals the marginal cost. The marginal cost consists of the monetary expenditure evaluated with the shadow price of wealth.

Equation (8) requires that the marginal benefit in terms of pain relief is not larger than the marginal cost of painkillers. If painkiller j is taken, marginal benefits and costs are equal. The marginal benefit consists of the marginal power of the painkiller in pain reduction times the experienced pain. The marginal cost consists of the monetary costs of one unit of painkiller, $p_m^j \phi_m^j$, evaluated with the shadow price of wealth, and the health costs, μB^j, evaluated with the shadow price of health deficits, λ_D. Individuals who rationally plan their addiction (i.e. for whom $\Phi = 1$) additionally take into account the increase in addiction α^j, caused by one unit of painkiller consumption evaluated at the shadow price of addiction λ_z. If several painkillers fulfil the first order condition (8) with equality, we assume that individuals choose the painkiller that provides the greatest net benefit. Net benefit of painkiller j is computed as

$$N^j = S(D,z)P(D,z)(1 - g(m^j)) - \lambda_k p_m^j \phi_m^j m^j + \lambda_D \mu B^j m^j + \Phi \lambda_z \alpha^j m^j.$$

It consists of the utility gains through pain reduction minus the monetary and health costs, which are converted into utility units by the shadow prices λ_k and λ_D. Fully rational individuals additionally take into account that painkiller use may cause future addiction and compute the anticipated utility costs of addiction with shadow price λ_z. Formally, the net benefit evaluates the total change of the objective function H when consumption of j increases from zero to m^j, where the quantity is determined by the partial change of the objective function, $\partial H / \partial m^j = 0$.

The costate equations associated with the optimal solution are given by

$$\lambda_k r = \lambda_k \rho - \dot{\lambda}_k, \tag{9}$$

$$\frac{\partial S(D,z)}{\partial D}\left[u(c) - P(D,z)g(m)\right] - S(D,z)\frac{\partial P(D,z)}{\partial D}g(m) + \lambda_D \mu = \lambda_D \rho - \dot{\lambda}_D, \quad \text{and} \tag{10}$$

$$-\frac{\partial P(D,z)}{\partial z}S(D,z)g(m) + \frac{\partial S(D,z)}{\partial z}\left[u(c) - P(D,z)\right]g(m) - \lambda_z \psi = \lambda_z \rho - \dot{\lambda}_z \quad \text{for } \Phi = 1, \tag{11}$$

and $\dot{\lambda}_z = \lambda_z = 0$ for $\Phi = 0$. Thus, individuals who rationally plan their addiction take the evolution of its shadow price λ_z into account. For imperfectly rational individuals the shadow price of addiction is zero.

4. Determinants of Painkiller Demand and Painkiller Choice

While the full model can be analyzed only numerically, some interesting insights into the determinants of painkiller demand and painkiller choice can be obtained by comparative static analysis. The comparative static analysis considers painkiller consumption for given values of the other control variables c and h.

LEMMA 1. *For fully rational (TORA) individuals, $\lambda_z(t) < 0$ for all $t < T$.*

To verify the claim, rewrite (11) as

$$\dot{\lambda}_z = \lambda_z(\rho + \psi) + \left\{ \frac{\partial P(D,z)}{\partial z} S(D,z) g(m) - \frac{\partial S(D,z)}{\partial z} \left[u(c) - P(D,z) \right] g(m) \right\}.$$

Notice that $\rho, \psi > 0$ and $0 \leq g(m) < 1$. Since $\partial P(D,z)/\partial z > 0$, $\partial S(D,z), \partial z < 0$, and $u(c) - P(D,z) > 0$, the term in curly brackets is positive. Thus, in order to reach the boundary condition $\lambda_z(T) = 0$, the shadow price of addiction has to be negative initially, $\lambda_z(0) < 0$, and converge to $\lambda_z(T) = 0$ from below. The result that the shadow price of addiction is negative throughout life is very intuitive since addiction is harmful for health and survival and thus reduces expected lifetime utility. Notice that the result does not require that addiction is actually present (it does not require $z > 0$). For non-addicted individuals λ_z captures the potential threat of addiction that is taken into account by TORA individuals when they consider taking a certain painkiller or not.

PROPOSITION 1. *Individuals are more inclined to use painkiller j, if they experience much pain (P is high), if the price p_m^j and the out-of-pocket ratio ϕ_m^j are low, and if the painkiller has low side-effects (B^j is low).*

For the proof, we insert (6) and (7) in (8) and obtain

$$G(m^j) \equiv -P(D,z) \frac{\partial g(m)}{\partial m^j} - \left(p_m^j \phi_m^j + \frac{p \phi B^j}{A \gamma h^{\gamma-1}} \right) \frac{\partial u(c)}{\partial c} + \frac{\Phi \lambda_z \alpha^j}{S(D,z)} \leq 0. \qquad (12)$$

The first term of G shows the benefit from painkiller use and the second term shows the cost in terms of marginal utility from consumption. Painkillers are not used when $G < 0$ and the

14

optimal dose of painkillers provides $G = 0$. Thus any change of a parameter or variable x that increases G, increases the propensity of painkiller use. Recalling that $\partial g / \partial (m^j) < 0$, we read off (11) that $\partial G / \partial P > 0$ and $\partial G / \partial x < 0$ for $x \in \left\{ p_m^j, \phi_m^j, p, \phi, B^j \right\}$. This means that, ceteris paribus, individuals in greater pain are more inclined to use painkillers. However, pain is endogenous. For TICA individuals (who ignore the last term in (12)), we can conclude that individuals who are heavily addicted to painkillers (for whom z is large) are more inclined to use painkillers. For TORA individuals this conclusion does not necessarily hold, since the derivative of the last term with respect to z is negative. In words, addicted TORA individuals know that if they consume even more painkillers, it would increase their future pain from addiction and reduce their survival probability. This feature allows TORA individuals to quit cold turkey, i.e. to reduce or terminate OPR consumption despite the pain from withdrawal.

At the intensive margin we obtain the following result:

PROPOSITION 2. *If painkiller j is used, the intensity of use is increasing in pain (P) and declining in the price p_m^j, the out-of-pocket ratio ϕ_m^j, and the severity of side-effects on health (B^j).*

The proof evaluates condition (11) when equality holds, applies the implicit function theorem, $\mathrm{d}m / \mathrm{d}x = -(\partial G / \partial x)/(\partial G / \partial m^j)$, and notes that $\partial G / \partial m^j = -P \partial^2 g / \partial (m^j)^2 < 0$.

PROPOSITION 3. *If painkiller j is known to be addictive, then it is less likely be used by fully rational (TORA) individuals (for whom $\Phi = 1$). If TORA individuals use a painkiller known to be addictive, they use it, ceteris paribus, less than TICA addicts who fail to control their addiction (for whom $\Phi = 0$).*

The proof is obvious from applying Lemma 1 to (12). Since the shadow price of addiction is negative for fully rational individuals ($\lambda_z < 0$), they face a higher marginal cost of addiction, which makes them less likely to use addictive substances and, if they use them, use them more cautiously than imperfectly rational individuals. Notice that TORA behavior differs from TICA behavior only when the substance is known to be addictive. These observations can be summarized as follows.

COROLLARY 1. *If painkiller j is not known to be addictive, it is consumed in equal quantities by TICA and TORA individuals. After the arrival of new information that the painkiller*

is addictive, TORA individuals (but not TICA individuals) reduce their consumption of the painkiller.

The first part of the proof inspects (12) for $\alpha = 0$. The second part of the proof inspects (12) for $\alpha > 0$ and applies Proposition 3. This result is potentially useful to identify the type of an individual by an information shock. To see this, assume that individuals decide upon OPR use believing that it is not addictive although it is addictive. This means that α enters with value zero in (12) albeit with a positive value in (2). Thus, if OPRs are used, TICA and TORA individuals use them in equal quantities, i.e. TICA and TORA behavior is observationally equivalent. The arrival of the information that the substance is actually addictive induces a behavioral change of TORA addicts, who now take the negative shadow price of addiction into account and consume less. For TICA addicts, in contrast, the new information does not cause a drop in drug consumption. How drastic the response of TORA individuals is and whether they respond with quitting depends on the characteristics of the OPR and the state of addiction when full information is disclosed. These features will be addressed in the numerical analysis of Section 7.
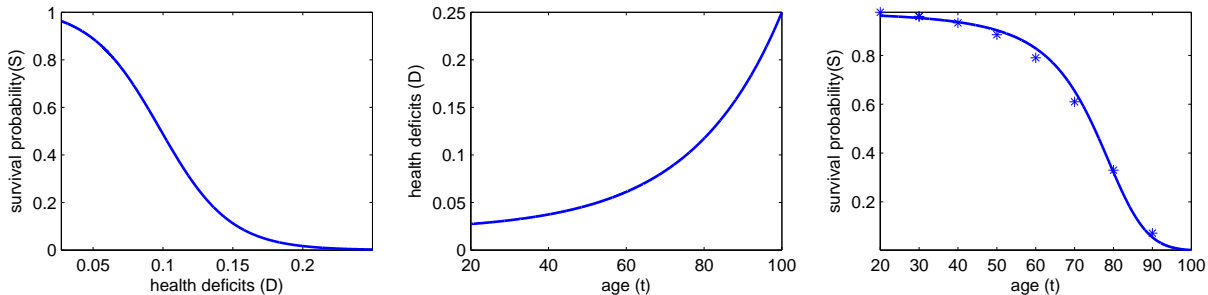
5. Solution of the Full Model

5.1. **Functional Forms, Euler Equations, and Painkiller Demand.** In order to analyze the comparative dynamics of the full model, we need to specify functional forms. We assume that utility from consumption is iso-elastic with an elasticity of intertemporal substitution of $1/\sigma$, $u = (c^{1-\sigma} - 1)/(1-\sigma)$. We assume that the survival probability is multiplicatively separable in its elements such that $S(D, z) = S_1(D)S_2(z)$. A parsimonious representation of the survival function $S_1(D)$ is given by the logistic function $S_1(D) = (1+\nu)/(1 + \nu e^{\xi D})$ for $D < \bar{D}$ and $S_1 = 0$ otherwise. The survival probability is unity at the state of best health ($D = 0$) and declines with first increasing and then decreasing rate as more health deficits are accumulated.

The panel on the left-hand side of Figure 1 shows the association between D and S implied by $S_1(D)$ for $\nu = 0.02$ and $\xi = 40$. The middle panel shows the association between age and accumulated deficits estimated by Mitnitski et al. (2002a) for 19-75 year-old Canadian men ($R^2 = 0.95$). When we feed these data into the $S_1(D(t))$ function, we get the "reduced form", $S_1(t)$, which shows survival as a function of age. The implied functional relationship is displayed on the right-hand side of Figure 1. Stars in the panel on the right-hand side indicate the survival

probability estimated from life tables for American men, taken from Strulik and Vollmer (2013). Death from overdose reduces the survival probability independently from health deficits such that $S_2 = e^{-\chi^j z}$. S_2 equals one for non-addicted individuals and declines exponentially in the degree of addiction z. The impact of addiction on overdose death is measured by the drug-specific parameter χ^j.

FIGURE 1: HEALTH-DEPENDENT SURVIVAL AND SURVIVAL BY AGE

Left: assumed function $S_1(D)$. Middle: Estimated association $D(t)$ (Mitnitski et al., 2002a). Right: Predicted (line) and estimated (stars) association between age and survival probability (estimate from Strulik and Vollmer, 2013).
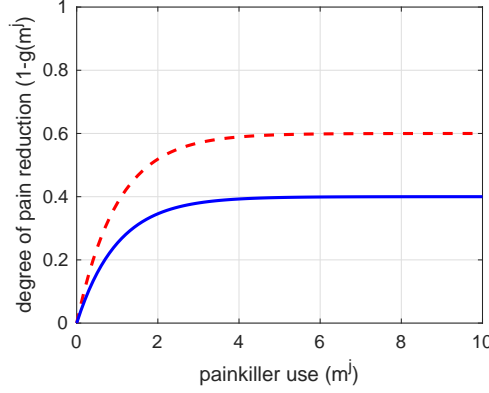
We assume that pain intensity is additively separable in health deficits and addictive capital, as well as linear in z, $P = \delta D^\omega + \zeta z$, in which δ and ω reflect the influence of health deficits on pain intensity and ζ reflects the influence of addiction; δ will be the key parameter to evaluate the intensity of pain.[9] To model painkiller efficacy, a function is needed that implements declining marginal efficacy and prevents simultaneous mixing, such that it provides a unique solution at any instant of time. Additionally, we want to capture the ceiling effect discussed in Section 2, i.e. the feature that for any painkiller a level beyond which pain cannot be reduced by further increasing drug intake exists. A parsimonious function that provides all these features is

$$g(m) = 1 - \max\left\{\eta^1\left(1 - e^{-m^1}\right), \ldots, \ \eta^J\left(1 - e^{-m^J}\right)\right\}. \tag{13}$$

The parameter η^j, $j \in \{1, \ J\}$ measures the maximum degree of pain that can be reduced by taking painkiller j. Figure 2 shows the degree of pain reduction when j is the preferred painkiller for $\eta^j = 0.4$ (solid lines) or $\eta^j = 0.6$ (dashed lines).

---

[9]For some individuals an opioid addiction may paradoxically increase pain sensitivity (Arout et al., 2015), a phenomenon that is not captured by the simple additive pain function. To discuss pain complementarity, I alternatively assume that aggregate pain is produced via a CES production function from chronic pain and pain from addiction: $P = [(\delta D^\omega)^{\tilde{\rho}} + (\zeta z)^{\tilde{\rho}}]^{\frac{1}{\tilde{\rho}}}$, in which $1/(1 - \tilde{\rho})$ is the elasticity of substitution. The additive model implicitly assumes $\tilde{\rho} = 1$ (infinite elasticity of substitution). It can be shown that the results presented below are robust to alternative assumptions on the elasticity of substitution when ζ is adjusted appropriately. (Results available upon request).

Given the functional forms, the Euler equations for consumption and health investments are obtained in (14) and (15), see the Appendix for details.

$$\frac{\dot{c}}{c} = \frac{r - \rho}{\sigma} \tag{14}$$

$$\frac{\dot{h}}{h} = \frac{r + q - \mu}{1 - \gamma} - \frac{\mu A \gamma h^{\gamma-1} c^\sigma}{\phi p (1 - \gamma)} \left\{ \frac{\nu \xi e^{\xi D}}{1 + \nu e^{\xi D}} \left[\frac{c^{1-\sigma} - 1}{1 - \sigma} - P(D, z) g(m) \right] - \delta \omega D^{\omega-1} g(m) \right\}. \tag{15}$$

If painkiller j is taken, its consumption level is obtained from (12) and (13) as

$$m^j = -\log R^j, \quad R^j \equiv \frac{\left(\phi_m^j p_m^j + \frac{p \phi B^j}{A \gamma h^{\gamma-1}} \right) c^{-\sigma} - \frac{\Phi \lambda_z \alpha^j}{S(\tilde{D}, z)}}{\eta^j P(D, z)}. \tag{16}$$

Notice that $R^j > 0$ since $\lambda_z = 0$ for imperfectly rational individuals and $\lambda_z < 0$ for fully rational individuals. The numerator of R^j summarizes the marginal costs (including health and addiction costs) form consuming painkiller j and the denominator reflects the marginal benefit in terms of pain reduction. If marginal costs exceed benefits, $R^j > 1$, and there is no interior solution solution for m^j. In this case, $m^j = 0$.

In Section 7 we perform a computational experiment, which is analytically motivated by the following observation.

PROPOSITION 4. *If, for an addictive painkiller j, the price p_m^j or the out-of-pocket ratio ϕ_m^j increases, then fully rational addicts are, ceteris paribus, more likely to respond with quitting than imperfectly rational addicts.*

The proof can be read off from (16) for an increase in p_m^j or ϕ_m^j. For $\lambda_z < 0$, R^j will exceed 1 for some given parameter values, while it stays below 1 when $\lambda_z = 0$.

5.2. **Calibration.** We begin with calibrating the life course of a pain-free individual, following Dalgaard and Strulik (2014). This means that we consider a 20-year-old American male in the year 2000. From Mitnitski et al. (2002a), we take the estimate of $\mu = 0.043$ for the force of aging. We set $r = 0.07$ as estimated by Jorda et al. (2019) for the long-run rate of return on equity and real estate. We normalize $p = 1$ and set $\phi = 0.28$ according to the average out-of-pocket share at all ages (Machlin and Carper, 2014). As explained above we calibrate the parameters of the survival function as $\nu = 0.02$ and $\xi = 40$. The individual earns an annual labor income of \$ 35,320 (BLS, 2011) until age 65, and afterwards a pension of $0.45 \cdot 35,320$ (with net replacement 0.45 according to OECD, 2016). We set the curvature parameter γ to 0.19 as in Dalgaard and Strulik (2014). We use the data generated by the model to compute the implied value of life (VOL) that the individual experiences along the optimal life cycle trajectory, $VOL = \int_{t_0}^{\tilde{T}} S(D)e^{-\rho\tau} u(c(\tau)\mathrm{d}\tau)/u'(c(0))$, in which $\tilde{T}$ fulfils $D(\tilde{T}) = \bar{D}$, and t_0 is the year of entry into economic life.

We calibrate the remaining parameters a, A, ρ, and σ such that (i) the reference American expects to die at age 75.5 (male life expectancy at 20 in the year 2000; NVSS, 2012), (ii) the age-weighted health expenditure is 13.3 percent of GDP as estimated for the US in the year 2000 (World Bank, 2015); whereby GDP per capita is computed as $w/(1-\alpha)$, assuming a labor share $1 - \alpha = 0.7$, (iii) health expenditure rises by about 2 percent per year (as in Dalgaard and Strulik, 2014), and (iv) the value of life at entry into economic life (at age 20) is \$ 6.3 million, as estimated by Murphy and Topel (2006). This leads to the estimates $a = 0.0185$, $A = 0.000325$, $\rho = 0.05$, and $\sigma = 1.06$. The estimate of σ is in line with recent studies suggesting that the "true" value is probably close to one (Chetty, 2006), or slightly above one (Layard et al., 2008).

The impact of pain on utility is calibrated using the study by Olafsdottir et al. (2020), which estimates the compensation variation for pain, i.e. the additional equivalized income needed to compensate an individual often suffering from pain for his loss in life satisfaction. Olafsdottir et al. (2020) estimate this compensation to lie between \$ 56 and \$ 145 per day, i.e. an annual extra income Δw between \$ 20,440 and \$ 52,925. Based on these findings, we estimate the value of $\delta > 0$ that pain, together with compensation Δw, provides the same expected lifetime utility as a pain-free life. For the benchmark pain scenario we set $\omega = 0$ such that the intensity of pain is independent from health deficits. We also set initial addiction $z = 0$. Assuming that chronic pain occurs at age 20 and continues until the end of life, this leads to the estimate $\delta = 0.26$

for $\Delta w = \$ \ 20{,}440$, which we define as the benchmark case of mild to moderate pain. We set $\zeta = 1.5$, which implies that, on average, the cravings from addiction generate an about four times higher desire for OPRs than chronic physical pain at the mild to moderate level.

As an out-of-sample prediction, we consider the needed compensation of the same pain shock (of $\delta = 0.26$) for an individual who is twice as rich as the benchmark individual. This is computed as $\$ \ 115$ per day. For an individual who is half as rich as the benchmark in turn, a compensation of $\$ \ 27$ is needed per day. In line with the methodology and results of Olafsdottir et al. (2020), the model thus predicts that richer people need more compensation to accept a certain intensity of pain because their marginal utility from consumption is low. In other words, richer people have a higher willingness to pay for pain avoidance.

For the benchmark run, individuals compare two painkillers, a light analgesic, assumed to be ibuprofen, and a prescription OPR, assumed to be oxycontin. For the benchmark run, we set $\phi_m^j = 0.19$ for all j according to the out-of-pocket share for prescription drugs (Stagniti, 2017). Ibuprofen is available for about $\$ \ 15$ per 500 tablets of 200mg. If j is ibuprofen, we assume $\eta^j = 0.4$, i.e. treatment reduces pain by 40 percent, and calibrate p_m^j such that total annual expenses are $\$ \ 130$, which corresponds to a dose of 600mg four times per day. Below we verify that the results are insensitive to alternative assumption on the price, the out-of-pocket share, and the efficacy for light painkillers. We assume that treatment has relatively low side-effects on health ($B^j = 10^{-5}$) and no addictive potential ($\alpha^j = 0$). Although it is possible to intentionally kill oneself using light analgesics, the probability of involuntary death from overdose is close to zero such that $\chi^j = 0$. Table 1 summarizes the painkiller characteristics.

The main reason to prefer opioids over light painkillsers in treatment of chronic pain is their higher efficacy in pain relief. If j is oxycontin, we set $\eta^j = 0.6$ such that the OPR reduces 60 percent of untreated pain. We show below that the main results do not depend on the absolute values of opioid efficacy. However, efficacy needs to be higher for OPRs than for light painkillers because otherwise not even TICA individuals would ever start taking them, given the much lower price of ibuprofen.[10] Prescribed oycontin is available at a price of about $\$ \ 1.25$ for a 10mg tablet. For the benchmark run we assume that the opioid is prescribed and expenses are covered

---

[10]The efficacy parameters η^j can be thought of as being individual-specific. Hence, efficacy of OPRs may be below that of light painkillers for some individuals and perhaps even for the majority of society. We ignore these intellectually uninteresting cases and focus on individuals for whom OPR efficacy is greater and the question whether greater efficacy is sufficient to motivate OPR consumption.

up to an out-of-pocket share of 0.19 by insurance. We calibrate p_m^j such that treatment begins with a relatively mild dose since initially there is no demand for pain from addiction. This leads to the estimate $p_m^j = 500$. We assume that total annual expenditure is \$ 1825, corresponding to an intake of 20mg oxycontin twice per day. If OPRs are bought on the black market, however, their price increases by about factor 8 (DEA, 2015; Gupta, 2016), implying $p_m^j = 4,000$.

Prescription opioid treatment may lead to severe side effects on health (respiratory depression, constipation, liver damage, brain damage), which are however still low compared to those caused by illicit opioids. To capture these effects we set $B^j = 5 \cdot 10^{-5}$, which is half of the impact assumed for heroin (see below). Since oxycontin is based on the same active substance as heroin, we assume that the addictive potential is also similar. For the benchmark run we set $\alpha^j = 0.03$ and $\psi = 0.1$, implying that an addicted individual demands about twice as much oxycontin as a non-addicted individual. Since α and ψ are unobserved and potentially individual-specific, we meet the involved parameter uncertainty with a sensitivity analysis (Section 6.2). The greatest health risk for addicted individuals originates from overdose. This risk, however, is lower than the overdose risk from illicit heroin intake because the purity and dosage of prescription drugs can be better controlled. For the benchmark run we assume $\chi^j = 0.002$, implying a mortality rate from overdose that is half of that of heroin (calibrated below).

Table 1: Three Types of Painkillers

	ibuprofen	oxycontin	heroin
efficacy	low ($\eta = 0.4$)	high ($\eta = 0.6$)	high ($\eta = 0.6$)
price	low ($p_m = 30$)	high ($p_m = 500$ or 4000)	medium ($p_m = 400$)
out-of-pocket	low ($\phi_m = 0.19$)	low ($\phi_m = 0.19$) or high ($\phi_m = 1$)	high ($\phi_m = 1$)
side effects	low ($B = 10^{-5}$)	moderate ($B = 5 \cdot 10^{-5}$)	high ($B = 10^{-4}$)
addictive	low ($\alpha = 0$)	high ($\alpha = 0.03$)	high ($\alpha = 0.03$)
overdose	low ($\chi = 0$)	high ($\chi = 0.002$)	very high ($\chi = 0.004$)

The table shows calibrated values for prescription oxycontin. If the opioid is bought on the black market (for non-medical use) the calibrated price changes from 500 to 4000 (and ϕ_m changes to 1). This is the price with which consumers compare the heroin price.

As a form of non-prescribed and illicit pain treatment we consider heroin consumption. We assume that heroin has the same efficacy and the same addictive potential as prescription opioids, but a lower market price, a 100% out-of-pocket share, more side-effects, and a higher overdose probability. In terms of morphine equivalents, heroin is available at about one-tenth of the market price of opioid pain medication (DEA, 2015; Gupta, 2016). This implies the estimate
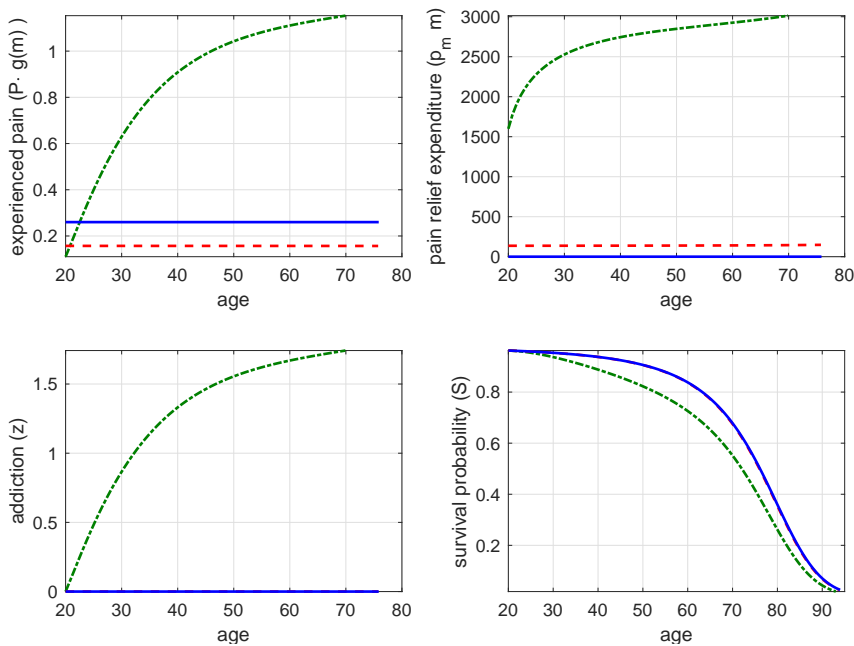
$p_m^j = 400$. To calibrate death from overdose, we take the crude mortality rate for death from drug overdose of people who inject drugs from Mathers et al. (2013) as 0.62 percent. From this we estimate $\chi = 0.004$ for heroin. In addition to the general health consequences from opioid use (described above), heroin use leads to a faster deterioration of health due to HIV and other blood-borne viruses transmitted through shared needles and syringes. We capture this fact by setting $B^j = 10^{-4}$. Taking by itself, the increased aging due to infections accounts for 1.0 year lost in life expectancy at 20.

6. Pain, Painkiller Use, Wellbeing and Longevity: Results

6.1. **Benchmark Results.** We solve the life cycle decision problem with the relaxation algorithm of Trimborn et al. (2008). The method provides the exact constrained-optimal life cycle trajectories, up to a user-specified approximation error (which is set to 10^{-5}). Figure 3 shows the life cycle trajectories for pain, pain relief expenditure, addiction, and survival probability at any age. Blue (solid) lines show the trajectories for untreated chronic pain at benchmark level of $\delta = 0.26$. For better comparisons, the pain and addiction trajectories end at the expected age of death, which is at the age of 75.5 years. The trajectory for survival probability, however, shows the predicted survival rate at any age. Red (dashed) lines represent the trajectories for light pain treatment (ibuprofen).

For the benchmark parameters, TICA individuals, who do not optimally control their addiction, prefer OPR treatment over light painkillers, whereas TORA individuals, who understand the addiction potential of OPRs and take it into account in the pain treatment calculus, prefer light painkillers. The life cycle dynamics for (TICA-) OPR use are shown by green (dash-dotted) lines in Figure 3. We see that treatment is initially more effective in removing pain than light painkillers (upper left panel). However, as the individual becomes addicted (lower left panel), additional pain is created from increasing tolerance. This in turn leads the individual to increase its demand for pain treatment, which leads to a rising pain relief expenditure. Over the course of life, pain relief expenditure almost doubles compared to initial treatment (upper right panel). Increasing addiction and higher dosage of opioids also raise mortality, mainly through increasing

FIGURE 3: PAIN, PAIN TREATMENT, AND HEALTH OUTCOMES

Blue (solid) lines: Reference American with untreated moderate pain ($\delta = 0.26$). Red (dashed) lines: common analgesic ($\eta = 0.4$, $\alpha = 0$). Green (dash-dotted) lines: prescription opioid ($\eta = 0.6$, $\alpha = 0.03$).

risk of overdose, shifting the survival curve inwards (lower right panel). Overall, life expectancy declines by 5.4 years.[11]

These results are summarized in the three first rows of Table 2 using two aggregate indicators of wellbeing, lifetime utility and life expectancy, both measured as deviations from the potential values of the same individual if it were pain-free and not addicted. Lifetime utility is measured in terms of relative deviation (in percent). In relative terms, the loss in lifetime utility is equal to the loss in the value of live, evaluated at age 20. The loss in life expectancy is more usefully measured in absolute terms (in years). Thus, the first row in Table 2 shows that the life-long experience of mild to moderate chronic pain without treatment reduces lifetime utility by 3.3 percent and life expectancy by an insignificant amount of 0.06 years. Life expectancy declines because life extension is less desirable when there is pain such that individuals invest somewhat

---

[11]There is some suggestive evidence that the order of magnitude of the model's predictions are well aligned with the scarce empirical evidence available. Chang et al. (2018) estimate that U.S. Americans who were in opioid-dependence treatment at some point in the period 2006–2009 lost 7.7 years in life expectancy, of which about half is explained by accidental overdose. The study, however, does not attribute the loss to a particular type of opioid addiction and is thus likely to contain also heroin addicts, for whom excess mortality is estimated to be much greater than for prescription opioids (Smyth et al., 2008).

less in their health. The use of light painkillers (case 2) improves wellbeing compared to no treatment, while lifetime utility falls short of that of a pain-free individual by 2 percent.

Table 2: Comparative Dynamics and Sensitivity Analysis: Effects on Wellbeing and Longevity

case	Δ V/V	ΔLE
1) moderate pain, untreated ($\delta = 0.26$)	-3.35	-0.06
2) light painkiller ($\eta = 0.4$)	-2.08	-0.16
3) prescription opioid ($\eta = 0.6$)	-12.8	-5.38
4) ...and switch to light painkiller at age 30 ($\Phi = 1$)	-10.8	-1.17

$\Delta V/V$ is the change in expected life-time utility (the value of life) in percent. ΔLE is the change in life expectancy at age 20 in years.

The use of prescription opioids (case 3) reduces lifetime utility by 12.8 percent and life expectancy by 5.4 years. The loss of life satisfaction comes through three channels: (i) a shorter life expectancy due to faster deteriorating health and the probability of overdose; (ii) the expenditure for opioids reducing the funds available for consumption, savings, and health investments, (iii) an increasing tolerance and cravings for opioids reducing instantaneous utility and thus the desire for a long life. This causes addicted individuals to save less for future health expenditure, which in turn speeds up health deficit accumulation and increases the probability of death.

6.2. **Sensitivity Analysis and Comparative Dynamics.** In this section, I briefly summarize the results of an extensive sensitivity analysis of OPR use, which is described in detail in the Appendix. In order to save space, we do not compare whole life histories for different parameters, but condense the results of comparative dynamics in two aggregate measures of the quality and quantity of life, $\Delta V/V$ and ΔLE. We observe that TICA individuals consume more OPRs and lose more compared to the benchmark in terms of life expectancy and value of life if the addictive power of OPRs (α^j) is greater, if tolerance (ζ) is higher, if the depreciation rate of addiction ψ is lower, if the efficacy of the OPR opposed to conventional painkillers is greater, and if the price (p_m^j) or the out-of-pocket ratio ϕ_m^j are lower. Results are similar to the benchmark results when chronic pain is aging related and increases with the accumulation of health deficits. In all these situations, TICA individuals prefer OPR use, while TORA individuals prefer light painkillers. This general finding turns out to be robust against different assumptions about the age at onset of chronic pain and its duration, an extension of the model towards labor supply effects of pain, and for pain intensities that increase the calibrated level of moderate pain by up to factor 5.

24

These observations raise the question under which conditions OPR could become the preferred treatment of fully rational individuals. The next two sections address this question with respect to conditions of the painkiller and conditions of the pain patient.

6.3. **Conditions for Rational OPR Use: Efficacy and Addictive Power.** In this section we investigate conditions of efficacy and addictive power with respect to developing an addiction and maintaining an addiction that need to be fulfilled for OPR addiction to be fully rational. These experiments are interesting for at least two reasons: (i) to assess the robustness of the central result that, given full information, only TICA individuals consume OPRs and become addicted; (ii) to assess whether and how life cycle trajectories of rational addiction differ from those of TICA addiction. Specifically, we determine the level below which addictive power of the drug j has to decline such that fully rational individuals prefer OPRs over light painkillers, considering the calibrated Reference American with chronic pain and alternative values of OPR efficacy η^j and the depreciation rate of addiction ψ.

Results of these experiments are shown in Table 3, which is read as follows. For $\eta^j = 0.6$ and $\psi = 0.1$, a value of α^j below 0.001 is needed to motivate fully rational individuals to take OPRs. The general takeaway from the results in the table is that only when efficacy is very large and addiction depreciates quickly, addiction becomes rational for a level of addictive power (α^j) that is significantly larger than zero. The intuition for the results is as follows. If ψ is large relative to α^j, addiction converges to an almost stationary value. Under these conditions, rational individuals prefer OPRs if the power in pain reduction is very large. If pain reduction is not sufficiently large, even the option of a quasi-stationary addiction level does not motivate rational OPR use. Due to the larger negative health effects and monetary costs, the welfare gain from OPRs use is still below that from use of light painkillers.

The life cycle trajectories for a typical rational addict are shown in Figure 4. The parameter values for this example are $\eta^j = 0.8$, $\psi = 0.5$, and $\alpha^j = 0.1$ such that OPR use is rational. The other parameters are as in the benchmark calibration. We see that rational addiction stabilizes quickly at a low level at which the addictive power from current OPR use is balanced by the depreciation of past use (formally, $\alpha^j m^j \approx \psi z$ in eq. 2). This means that pain also stabilizes at a low level, at about half of that with use of light painkillers. This effective and large pain reduction motivates the use of OPRs even though the medication is more expensive and has side-effects on health (life expectancy declines by 0.6 years compared to light painkiller use). In contrast,
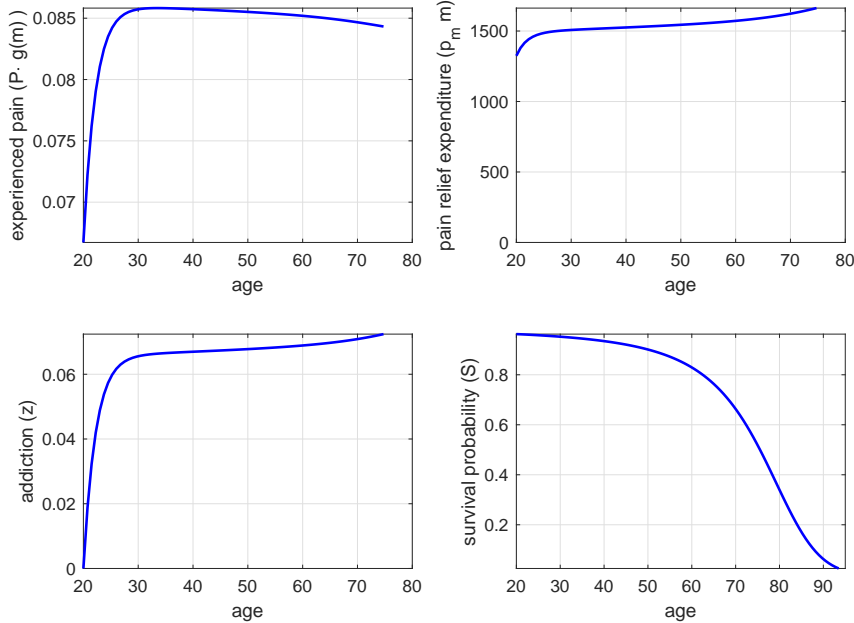
Table 3: Rational OPR Use: Cutoffs

η^j	ψ	α^j
0.6	0.1	0.001
0.8	0.1	0.003
0.9	0.1	0.005
0.6	0.5	0.002
0.6	1.0	0.009
0.8	0.5	0.011
0.9	0.5	0.038

The table shows for different levels of efficacy of OPRs (η^j) and depreciation of addiction ψ the cutoff level of addictive power α^j below which rational individuals prefer OPRs against light painkillers. All other parameters are as in the benchmark calibration.

for TICA individuals, OPR use is effective only for a short period after first consumption, as shown by the dash-dotted life cycle trajectories in Figure 3. With prolonged OPR use, addiction continuously increases as TICA individuals become more tolerant and consume larger quantities of OPRs. Correctly anticipating these feedback effects, fully rational individuals would not initiate an OPR addiction. In summary, effective pain treatment and a quasi-stationary, non-increasing time profile of OPR consumption are identified as characteristics of rational OPR addiction.

Figure 4: Quasi-Stationarity of Addiction and Rational OPR Use

Reference American with moderate pain and fully rational OPR addiction. Parameters: $\eta = 0.8$, $\psi = 0.5$, $\alpha = 0.1$ for OPR use. Other parameters as for benchmark case (Figure 3 in the paper).

It could be argued that the life cycle pattern of rational OPR use does not qualify as an addiction in its original sense. Although the underlying utility function still fulfils the defining formal characteristics of addiction, the phenomenon of tolerance is no longer visible for rational addiction. Tolerance would require that greater levels of drug consumption are needed to maintain the same level of utility (or pain). Here, in contrast, rationally addicted individuals maintain an almost constant level of pain with constant OPR consumption.[12]

6.4. **OPRs in Palliative Care.** We next consider whether OPR use can be rational when severe pain arises in conjunction with drastic health shocks. This is a kind-of out-of-sample application since the benchmark model considers mild to medium chronic pain (such as, e.g. back pain). The new scenario describes palliative care in end-of-life situations (such as cancer pain). A distinctive feature of palliative care is the short remaining life expectancy, which makes it less likely that an addiction fully develops. In order to stress this feature, we return to the benchmark calibration, which means that the addictive power of OPRs is sufficiently strong such that fully rational individuals do not use OPRs for treatment of mild or moderate chronic pain. As shown in the Appendix, rational individuals neither prefer OPRs in treatment of severe pain if pain does not occur in conjunction with drastic life-length reducing health shocks (see results from Figure A.3 and Table A.2).

In order to check whether the model supports opioid pain treatment in end-of-life situations with drastic health shocks, we first consider a severe pain shock ($\delta = 1$) at age 70, which is accompanied by a spontaneous increase in the health deficits index D from 8 to 13 percent ($\Delta D = 0.05$). As a result, life expectancy at age 70 declines from 11.1 to 4.7 years. As shown in case 1 of Table 4, severe pain causes an additional loss of welfare of 12 percent. Notice that, in deviation to the previous results, the value of life is measured from the start of the illness (in the example, age 70) and $\Delta V/V$ and ΔLE are measured relative to an individual of the same age with the same health shock but without pain. As shown in case 3, OPR treatment is no longer significantly worse than no treatment although it is still outperformed by light painkillers and thus not preferred by fully rational individuals.

---

[12]A further characteristic of rational OPR use is that it increases in old age. This feature is a general phenomenon of rational addiction when life is finite (see Strulik, 2018). The explanation is that rational individuals anticipate that they are likely dead before the addictive capital stock has fully increased in response to the higher consumption in old age and thus consuming addictive goods is less costly from a lifetime perspective. Formally, the shadow price of addiction (λ_z) declines in old age. In Figure 4, this feature is not very pronounced because of the low addictive power and large depreciation rate of addictive capital.

Table 4: Pain and Severely Life-Shortening Health Shocks

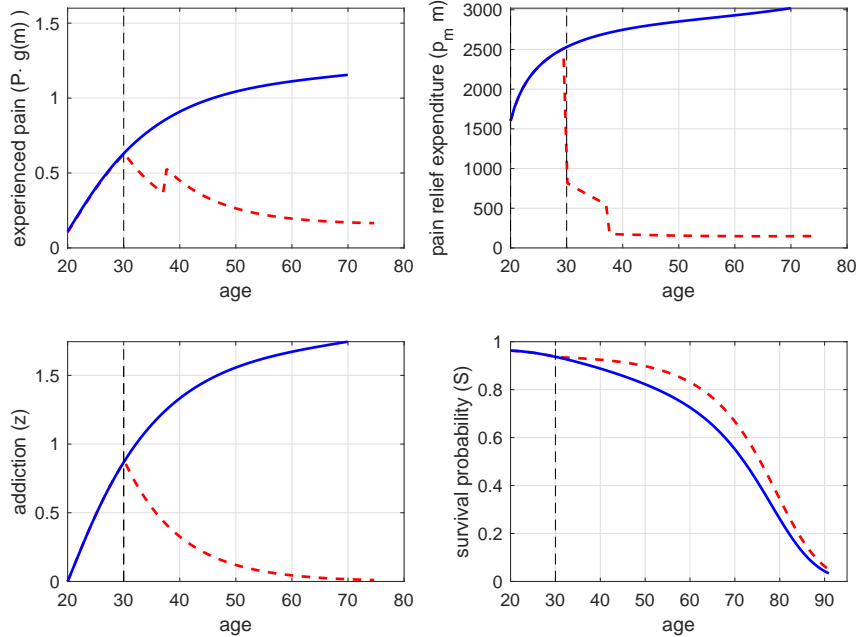| case	$\Delta V/V$	ΔLE
Pain onset at age 70, severe pain ($\delta = 1$) and $\Delta D = 0.05$.		
1) no treatment	-12.0	-0.00
2) light painkiller	-7.30	-0.00
3) OPR treatment	-12.3	-0.02
4) OPR treatment ($\eta = 0.8$)	-5.73	-0.03
5) ... and TORA addiction	-4.91	-0.02
Pain onset at age 40, severe pain ($\delta = 1.0$) and $\Delta D = 0.1$.		
6) no treatment	-12.1	-0.00
7) light painkiller	-7.34	-0.00
8) OPR treatment	-9.87	-0.02
9) OPR treatment ($\eta = 0.8$)	-6.21	-0.03
10) ... and TORA addiction	-5.23	-0.02

Expected lifetime utility V is measured from the age when the shock hits (the remaining value of life) and measured relative to a pain-free individual with the same health shock.

These results change when the efficacy of OPRs rises. Considering the fact that malignant pain may be associated with anxiety and depression (see e.g. Massie, 2004; Wilson et al., 2007), which are also addressed by opioids (Bair et al., 2003; Verdu et al., 2008), it is plausible that efficacy of OPRs (but not of light painkillers) rises in malignant pain treatment and in palliative care. In case 4 of Table 4 we see that when efficacy rises to 0.8 OPR treatment outperforms treatment with light painkillers. In this case also TORA individuals prefer OPR treatment, although they would use it somewhat more sparingly, as shown in case 5.

These conclusions do not rest on the onset of severe pain in old age but rather on their appearance in conjunction with drastic, life-shortening shocks in physical health. In order to verify this statement, we next consider a 40-year-old individual who experiences severe pain together with a drastic increase of health deficits from 3.7 to 13.7 percent such that life expectancy at 40 reduces to 4.7 years. The effect of severe pain on wellbeing is similar; OPR treatment dominates no treatment, and, if its efficacy rises to 0.8, it dominates treatment by light painkillers and becomes the preferred treatment of fully rational individuals. We expect that OPR use can be rational also at lower efficacy levels, if health shocks are even more drastic and further reduce remaining life expectancy. With very short remaining life expectancy, however, it becomes increasingly difficult to numerically solve the model. The main conclusion is that the model supports rational OPR use in end-of-life scenarios if efficacy is sufficiently strong. This result is not based on special assumptions about low addictive power of OPRs (as for the case of OPRs

use in chronic pain treatment discussed in the previous section). OPR use in palliative care is thus found to be consistent with rationality.

FIGURE 5: NEW INFORMATION AT AGE 30: OPRS ARE ADDICTIVE

Blue (solid) lines: Imperfectly controlled addiction (TICA); red (dashed) lines: fully rational addiction (TORA).

7. INFORMATION AND PRESCRIPTION SHOCKS

7.1. **A Benchmark Case.** It has been shown that, with full information, fully rational individuals prefer light pain killers for a wide range of parameter levels. However, even if we observe that they take OPR pain relievers, it would be hard to argue in favor of policy intervention, given that this was a deliberate choice with perfect control of the addiction. Imperfectly controlled addiction, in contrast, calls for policy intervention since individuals suffer losses in wellbeing and health due to their inability to control the addiction. In the benchmark calibration, TICA individuals (who prefer OPRs) lose 10.7 percent in value of life and 5.2 years in life expectancy compared to TORA individuals. In order to motivate and design policy interventions it is thus essential to know whether actual behavior is better described by rational or imperfectly controlled addiction.

The type of an individual can be identified by the response to the disclosure of the addictive power of an OPR: TORA individuals (and only TORA individuals) will always respond by using OPRs less (Corollary 1). For this distinct response to be useful for practical identification purposes, however, the response needs to be clearly observable, i.e. it needs to be sufficiently large. In this and the next we explore information shocks and the conditions for a drastic

reduction that eventually leads to quitting of OPR use. We begin with the benchmark calibration and assume that initially individuals believe that OPRs are not addictive such that chronic pain patients develop an addiction irrespective of their type. After ten years, when the benchmark individual is 30, it is revealed that the substance is actually addictive (with strength $\alpha^j = 0.03$). This setup can be related to the American opioid crisis by recalling that the American Pain Society endorsed the use of opioids for chronic pain in 1997 and that ten years later, in 2007, the developer of oxycontin pled guilty to criminal charges for misrepresenting the risk of addiction (Haddox et al, 1997; van Zee, 2009).

Figure 5 shows the predicted behavior. For TICA individuals there is no behavioral change. The blue (solid) lines reiterate the life cycle behavior from the green (dashed-dotted) lines in Figure 3. For TORA individuals, the arrival of the new information causes quite drastic changes. As shown by the red (dashed) lines, individuals who optimally control their addiction drastically reduce their consumption. Due to reduced consumption, addiction and pain gradually recede. After about 7 years, when pain is sufficiently low, TORA individuals endogenously quit OPR intake and switch to light painkillers. As a result, they experience a large gain in terms of survival probability and in terms of material wellbeing. As shown in row 4 of Table 2, the behavioral response provides a gain of more than 4 years in life expectancy (compared to TICA behavior from row 3).

7.2. **Sensitivity Analysis: Optimal Quitting.** The response of TORA individuals to reduce consumption when the addictive power of a substance is universal (see Corollary 1). Whether they completely abandon OPR use, however, depends on the characteristics of the drug and the timing of the information shock. This is shown by the results presented in Table 5, which shows for alternative addictive power α^j, depreciation of addiction ψ, and efficacy in pain reduction η^j of the OPR the elapsed time from the information shock to the complete abandoning of OPR use, depending on the timing of the information shock. The first row reiterates the benchmark case from Figure 4. The information shock is experienced at age 30, i.e. after the addiction developed for 10 years, and the individual completely abandons OPR use after 7.6 years, i.e. at age 37.6.

With cases 1) to 3) we consider alternative depreciation rates of addictive capital ψ. Depreciation affects the size of the addictive stock at the time of the information shock as well as the speed of decline of addiction and the pain from withdrawal when OPR use is reduced or

abandoned. We observe a non-monotonous response of quitting time. For increases of ψ at low levels, individuals quit faster because, apparently, the withdrawal effect dominates. For increases of ψ at high levels, individuals quit more slowly because the stock-size effect dominates. For example, for $\eta = 0.5$, z is by factor 4 lower at shock time than in the benchmark case. This implies that it is less costly in terms of income and life-expectancy to maintain the addiction at a low level. For $\psi = 1.0$, never quitting is the optimal response, which is represented by the "–" symbol in Table 5. Instead, TORA individuals reduces their addiction by about 20 percent and maintain it constant at that low level until the end of life. It has already been discussed in Section 6.3, whether this behavior describes addiction in a strict sense because the phenomenon of tolerance is no longer visible when individuals maintain a constant low level of addiction.

Table 5: OPR Characteristics, Age at Info Shock, and Quitting Time

case	α^j	ψ	η^j	age at shock	time to quit
benchmark	0.03	0.1	0.6	30	7.6
1)	0.03	0.2	0.6	30	5.8
2)	0.03	0.5	0.6	30	7.0
3)	0.03	1.0	0.6	30	–
4)	0.01	0.1	0.6	30	22.1
5)	0.01	0.2	0.6	30	–
6)	0.09	0.1	0.6	30	7.5
7)	0.09	0.2	0.6	30	4.4
8)	0.09	0.5	0.6	30	2.6
9)	0.09	1.0	0.6	30	2.0
10)	0.03	0.1	0.5	30	0.1
11)	0.03	0.1	0.7	30	14.5
12)	0.03	0.1	0.8	30	–
13)	0.03	0.1	0.6	25	1.4
14)	0.03	0.1	0.6	21	0.0
15)	0.03	0.1	0.6	35	10.5
16)	0.03	0.1	0.6	40	12.8
17)	0.03	0.5	0.6	40	7.7
18)	0.03	1.0	0.6	40	–

The table shows for alternative OPR characteristics of addictive power α^j, depreciation of addiction ψ, efficacy η^j, and different age at arrival of the information that OPRs are addictive, the time in years needed by TORA individuals to quit OPR use completely. The "–" symbol means that individuals never quit. All other parameters are as in the benchmark calibration.

Cases 4) and 5) show a similar behavior when the addictive power of the OPR is low, which is explained by a similar mechanism. For $\alpha = 0.01$, the addiction at shock time is small and thus it is not costly to maintain it for a while or forever. For ψ, individuals abandon OPR use after 22 years whereas for $\psi = 0.2$ they maintain it forever. Cases 6) to 9) consider cases of greater

addictive power ($\alpha^j = 0.09$). In contrast to cases 1) to 3), quitting declines monotonously ψ, for all considered depreciation rates. The reason is that addictive power of the OPR is so high that addiction cannot be controlled and maintained at a low level by reducing consumption. A high depreciation rate allows for fast quitting with relatively low pain from withdrawal.

Cases 10) to 12) show the large sensitivity of the optimal response with respect to efficacy of the OPR. If efficacy is low and not much higher than that of light painkillers ($\eta^j = 0.5$), TORA individuals abandon OPR use immediately. On the other hand if efficacy is very high, TORA individuals maintain OPR use, as shown in case 12) for $\eta^j = 0.8$.
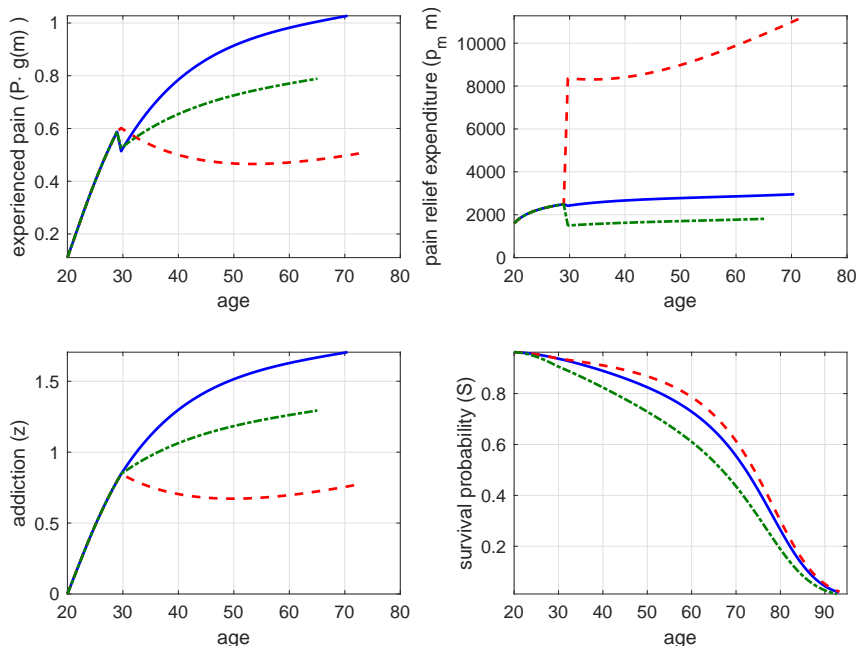
Finally, we analyze sensitivity with respect to the timing of the information. The longer the misinformation period the more time individuals have to build up addiction stocks and the harder it will be to reduce and abandon the addiction after full information. This intuition is confirmed in cases 13) to 18) of Table 5. If under benchmark conditions, the correct information about addictive power arrives after 5 years (instead of 10), the time to quit declines to less than a year and a half. If the information arrives after one year, TORA individuals quit immediately. On the other hand, if the information arrives later, it takes longer to quit and, if addictive capital depreciates sufficiently slowly, there is no quitting.

It is interesting to recall from Section 6.3 that, for the parameter constellations of the cases 3), 5), 12) and 18) in Table 5, TORA individuals would not have initiated OPR use under full information. However, after having developed an addiction due to misinformation, they prefer to keep it and continue to use OPRs despite the disclosure of their addictive power. These are perhaps the cases where the loss in term of health and lifetime caused by misinformation is most clearly visible for TORA individuals. Summarizing, we conclude that TORA individuals always respond to the disclosure of the addictive power of OPRs by using less, a behavior which distinguishes them from TICA individuals. In many but not all situations, full information is also sufficient for TORA individuals to induce quitting of OPR use without further need of policy interventions.

7.3. Prescription Shocks, OPR Abuse and Illicit Drug Consumption.
In this section we investigate the behavioral changes induced by a discontinuation of the OPR prescription and consider the transition from pain patient to junkie. We consider a scenario where the original source of pain, say back pain, disappears such that any non-addict would discontinue pain treatment. Suppose that chronic pain of benchmark strength ($\delta = 0.26$) appears at age

20 and disappears at age 30. In the first case, reflected by solid (blue) lines in Figure 6, we assume that the individual receives prescription opioids and support from health insurance such that p_m and ϕ_m stay at their original values. As shown in the upper left panel of Figure 6, the loss in pain due to absent back pain causes a mere wrinkle in the trajectory of life-cycle pain of the addicted. Soon, additional cravings from addiction compensate for the temporary liberation from back pain and the life cycle trajectories for pain, pain relief expenditure, and life expectancy follow the original (benchmark) path. Case 1 in Table 6 shows the implied losses of wellbeing and life expectancy, which hardly differ from those for OPR treatment and lifelong chronic pain (case 3 in Table 2).

FIGURE 6: OPR ABUSE AND ILLICIT DRUG CONSUMPTION

All lines: Original pain ($\delta = 0.26$) terminates at age 30. Blue (solid) lines: continued prescription OPR use. Red (dashed lines): black market OPR use. Green (dash-dotted) lines: heroin use.

It is conceivable, however, that without a diagnosis of physical pain, the prescription of OPRs is terminated. One option is then to satisfy an addiction through purchases from the black market. Referring to the calibration from Section 4, such a change is captured by an eightfold price increase as well as by an increase of the out-of-pocket share to 100 percent. Keeping everything else as in the benchmark calibration, red lines in Figure 6 show the implied life cycle trajectories. Facing the higher price, the individual responds with reduced demand. Average lifetime OPR use declines by almost 100% when opioids are obtained on the market (average m declines from 6.0 to 3.3). Initially, addiction declines (lower left panel) but then it rises

slowly again. Over time, the individual returns to about the level of addiction developed when prescription was terminated. As a result of lower addiction, the survival probability improves compared to prescription-fueled addiction (lower right panel). As shown in case 2 of Table 6, the loss in wellbeing and the loss in life expectancy are lower than under continued prescription. This outcome is accompanied by a drastic increase in drug expenditure (upper right panel in Figure 6) and a short-run increase in pain, which recedes quickly (upper left panel) due to the lower level of addiction (lower left panel).

Alternatively, individuals may consider to move to heroin use, which, given the calibration from above, is available at one-tenth of the price of black market OPR but bears additional health risks and an elevated risk of overdose. Applying Proposition 2 to the calibrated values, we find indeed that TICA addicts prefer heroin consumption over black market OPR use. The implied life cycle trajectories are shown by dash-dotted (green) lines in Figure 6. The levels of consumption, expenditure, and addiction fall short of those under prescription OPR use, but the survival probability declines due to increased side effects and a higher probability of overdose. As a result, life expectancy declines by 10.6 years and lifetime utility declines by 16.9 percent (case 3 in Table 6).

In order to understand why addicted individuals prefer heroin over black market oxycontin even though the implied lifetime utility is lower, recall that TICA individuals fail to predict how their drug habit develops. This relatively mild form of bounded rationality is sufficient to explain the observable behavior of moving from black market OPR use to heroin because of its lower price (per morphine equivalent). A TORA individual would neither start using prescription OPRs (other than in palliative care), nor would he continue OPR use after pain terminated, nor would he switch from OPR use to heroin because all of these transitions reduce lifetime utility.

These features are next illustrated with another computational experiment that identifies TORA and TICA behavior. As in the experiment of Section 7.1, all individuals start out at age 20 believing that OPRs are not addictive and after 10 years, at age 30, it is revealed that OPRs are actually addictive. Additionally, now doctors abandon the supply of prescription opioids. At age 30, both types of individuals are equally addicted, as in Section 7.1, but now they face other options. They could obtain illicit OPRs from the street at much higher price, or switch to heroin, or quit addictive goods consumption (cold turkey). As shown above, TICA individuals prefer to

Table 6: OPR Abuse, Illicit Drug Consumption, and Treatment

case	Δ V/V	ΔLE
1) continued prescription OPR	-12.6	-5.16
2) black market oxycontin	-9.77	-2.80
3) heroin	-16.9	-10.6
4) ... and information shock (TORA behavior)	-4.48	-0.75
5) ideal therapy	-3.87	-0.96
6) as 4) and $\psi = 1$	-1.85	-0.35
7) as 5) and $\psi = 1$	-1.77	-0.38

The experiment eliminates chronic pain and (from case 2 on) the prescription of OPRs at age 30. The ideal therapy maintains efficacy of OPRs and eliminates addiction. It reduces the drug price to 1 Dollar (ideal methadon).
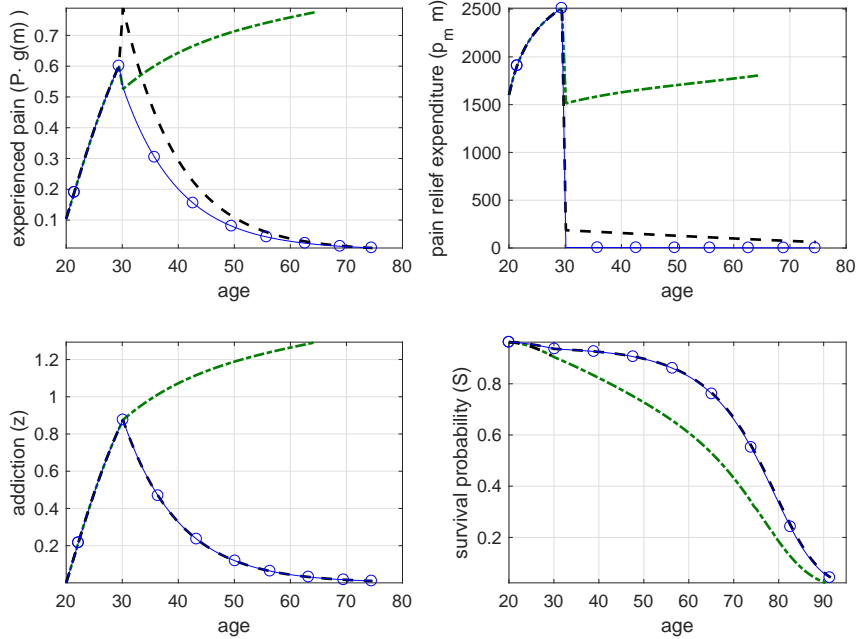
switch to heroin. The implied lifetime trajectories are shown again by green dash-dotted lines in Figure 7.

TORA individuals, in contrast, respond to the shocks by quitting instantaneously. The lifetime trajectories are shown by dashed black lines in Figure 7. Pain rises after withdrawal due to cravings from addiction and then declines asymptotically to zero. Compared to the scenario of Section 7.1, quitting occurs instantaneously because the information about addictive power of OPRs is now accompanied by a drastic increase of price and out-of-pocket ratio for OPRs. Illicit opioids are not an option for fully informed rational individuals. Since quitting occurs at young age, individuals almost reach the life expectancy of a pain-free, never addicted individual (life expectancy declines by 0.75 years). The value of life also improves greatly but there remain significant losses due to pain from addiction (before and after quitting).

In summary, a discontinued OPR prescription policy has opposing effects on TORA and TICA individuals. TORA individuals quit instantaneously while TICA individuals take up heroin. This insight has important policy implications. For TORA individuals full information and discontinued prescription are sufficient to induce rapid quitting. The same policy, however, drives TICA individuals towards consumption of illicit and even more dangerous consumption of black market opioids.

In contrast to TORA individuals, TICA individuals would benefit greatly from additional policy measures that support them in quitting opioids. To illustrate this proposition, we finally consider a stylized ideal treatment of addiction. The opioid replacement therapy offers a drug that relieves the narcotic cravings without contributing to addiction, like methadone. This "ideal methadone" provides the same efficacy as heroin ($\eta^j = 0.6$) with zero impact on addiction

All lines: chronic pain ($\delta = 0.26$) terminates at age 30 and prescription OPR is discontinued ($p_m^j = 4000$, $\phi_m^j = 1$). Green (dash-dotted) lines: heroin use. Black (dashed) lines: optimal withdrawal of TORA addicts. Blue (circled) lines: ideal therapy for TICA addicts. See text for details.

($\alpha^j = 0$) and entails similar health effects as controlled prescription OPR use. It is administered at close to zero costs ($p_m^j = 1$ dollar). We also assume that the provision of the ideal methadone is controlled in the sense that individuals receive the optimal quantity for pain reduction but they are not allowed to buy or sell the drug on markets or to combine it with other drugs.

The implied life cycle trajectories under these assumptions are shown by blue circled lines in Figure 7. The ideal methadone meliorates cravings from addiction but does not contribute to the stock of addiction capital. It allows TICA addicts to withdraw gradually with minimum pain and close to zero costs. Interestingly, this generates roughly the same improvements in health and life expectancy that TORA addicts achieve without any help from drug policy. This can be seen by the almost perfect overlap of the black and blue survival curves in the lower right panel of Figure 7. Effects on wellbeing are summarized in Table 6. The gain in value of life (compared to heroin use) is larger for the ideal therapy than for TORA behavior because there is no pain from cold withdrawal. The gain in terms of life expectancy (compared to heroin use) is smaller than for TORA withdrawal due to the side effects of the heroin replacement.

It could be argued that withdrawal symptoms decline too slowly in the calibration. For sensitivity analysis, we thus set $\psi = 1$. This implies that the half-life at which addiction recedes

declines from about 7 years to about 0.7 years and the loss in lifetime utility is further reduced (in absolute values), see case 6 and 7 of Table 6. Rational quitting and ideal methadone therapy are now almost indistinguishable in their improvement of length and value of life.

8. Conclusion

This study provides a first attempt to develop a theory of pain, painkiller use, and addiction and to integrate it in a life-cycle model of endogenous health and longevity. Individuals are conceptualized as forward-looking maximizers of their lifetime wellbeing. Two types are distinguished. TORA individuals perfectly plan and control their addiction (if they have any), while TICA individuals fail to control their addiction. Individuals suffering from mild to moderate chronic pain may use light painkillers or prescription OPRs for pain relief. If information on the addictive power of OPRs is available, TORA individuals would not initiate an addiction for plausible assumptions on OPR efficacy and addictive power. The small refinement towards imperfectly controlled addiction allows the conceptualization of prescription OPR use by chronic pain patients as well as the transition to illicit OPR use. Pain patients who fail to optimally control their addiction experience drastic reductions in wellbeing and life expectancy as unintended consequences. This qualitative result is robust to alternative assumptions on the efficacy of painkillers, drug prices, out-of-pocket ratios, and the addictive characteristics of OPRs. As an exception, it can be fully rational to use opioids in palliative care, i.e. when pain is experienced in conjunction with a severely lifetime reducing deterioration of health.

If individuals wrongly believe that OPRs are not addictive, TORA and TICA addiction is observationally identical. However, if new information arrives, the model predicts drastically different changes in behavior. When it is revealed that OPRs are addictive, TORA individuals always reduce the use of OPRs and, for a wide range of parameter values, eventually quit taking them. TICA individuals in turn do not reduce their consumption, further develop their addiction, and consume even more. Numerical experiments also show that the parameter range for which TORA individuals would not initiate OPR use is wider than the parameter range for which they terminate OPR use when full information about addiction is revealed. In other words, there is a range of parameter values for which even TORA individuals may end up with permanent addiction and OPR use due to temporary misinformation about the addictive power of these painkillers.

Another computational experiment considers the termination of prescription OPRs. Faced with much higher street prices and a 100 percent out-of-pocket ratio, TICA individuals are predicted to turn to cheaper heroin consumption. TORA individuals are predicted to terminate any drug consumption. Hence, if individuals were fully rational in the Becker-Murphy (1988) sense, there would be no pressing need for policy interventions and addiction therapy. TICA individuals, in contrast, could greatly benefit from addiction treatment in terms of longevity and wellbeing. For the benchmark individual, an ideal treatment of OPR addiction would increase the value of life by about 13 percent and life expectancy by about 9 years.

Alternative, non-pharmacologic treatments of pain and addiction such as yoga and cognitive behavioral therapy are perhaps more difficult to capture by the current framework. Other forms of opioid use, however, could be easily integrated in the model. For example, misuse of prescription opioids (e.g. crushing tablets and injecting the substance) could be introduced as an intermediate step in the transition to heroin or the transition to an even more powerful and deadly opioid such as fentanyl. Other extensions of the theory could consider the joint use of several prescription painkillers or the supplement of prescription OPRs with street purchases. A mild reformulation of the utility function could capture recreational OPR use. These features fell prey to Occam's razor in order to constrain the length of the paper.

A limitation of the model is its focus on the pain patient. The supply side is taken exogenously by considering alternative prescription and street prices, out-of-pocket ratios, and information policy. By highlighting individual decision processes and their consequences on wellbeing and life expectancy, the analysis neglects to explain the behavior of health providers and the pharmaceutical industry as well as macroeconomic context and social dynamics, which all play a role for a full understanding of the opioid epidemic. Also the welfare analysis is constrained to the individual level and neglects, for example, intergenerational welfare effects of more restrictive OPR prescription rules.

REFERENCES

Acemoglu, D. (2009). *Introduction to Modern Economic Growth.* Princeton University Press.

Almond, D., and Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives* 25(3), 153-72.

Alpert, A. E., Evans, W. N., Lieber, E. M., and Powell, D. (2019). Origins of the Opioid Crisis and Its Enduring Impacts. NBER Working Paper No. w26500.

American Academy of Pain Management (2003). Proceedings of the 2003 Annual Meeting of the American Academy of Pain Management, Denver, CO.

Arout, C. A., Edens, E., Petrakis, I. L., and Sofuoglu, M. (2015). Targeting opioid-induced hyperalgesia in clinical treatment: neurobiological considerations. *CNS Drugs* 29(6), 465-486.

Blanchflower, D.G., and Oswald, A. (2019). Unhappiness and Pain in Modern America: A Review Essay, and Further Evidence, on Carol Graham's Happiness for All? *Journal of Economic Literature* 57(2), 385-402.

Becker, G.S., and Murphy, K.M. (1988). A theory of rational addiction. *Journal of Political Economy* 96(4), 675-700.

Bair, M.J., Robinson, R.L., Katon, W., and Kroenke, K. (2003). Depression and pain comorbidity: a literature review. *Archives of Internal Medicine* 163(20), 2433-2445.

Busse, J.W., Wang, L., Kamaleldin, M., et al. (2018). Opioids for Chronic Noncancer Pain: A Systematic Review and Meta-analysis. *JAMA* 320(23): 2448-2460.

Case, A., and Deaton, A.S. (2005). Broken down by work and sex: How our health declines. In: *Analyses in the Economics of Aging*, University of Chicago Press, 185-212.

Case, A., and Deaton, A. (2017). Mortality and morbidity in the 21st century. *Brookings Papers on Economic Activity*, Spring 2017, 397-467.

Case, A., Deaton, A., and Stone, A. A. (2020). Decoding the mystery of American pain reveals a warning for the future. *Proceedings of the National Academy of Sciences*, forthcoming.

Cawley, J., and Ruhm, C. (2012). The economics of risky health behaviors, in: in Mark V. Pauly, Thomas G. McGuire, and Pedro P. Barros (eds.) *Handbook of Health Economics* Volume 2. New York: Elsevier, 2012, 95-199.

CDC (2016). CDC Guideline for Prescribing Opioids for Chronic Pain. Centers for Disease Control and Prevention. `https://www.cdc.gov/mmwr/volumes/65/rr/rr6501e1.htm`

CDC (2017). Understanding the Epidemic. Centers for Disease Control and Prevention. `https://www.cdc.gov/drugoverdose/epidemic/index.html`

CDC (2017b) Vital Signs: Changes in Opioid Prescribing in the United States, 2006–2015. Centers for Disease Control and Prevention. `https://www.cdc.gov/mmwr/volumes/66/wr/mm6626a4.htm`.

Chang, K. C., Wang, J. D., Saxon, A., Matthews, A. G., Woody, G., and Hser, Y. I. (2017). Causes of death and expected years of life lost among treated opioid-dependent individuals in the United States and Taiwan. *International Journal of Drug Policy* 43, 1-6.

Cheatle, M. D. (2011). Depression, chronic pain, and suicide by overdose: on the edge. *Pain Medicine* 12, S43-S48.

Chetty, R. (2006). A new method of estimating risk aversion. *American Economic Review* 96, 1821-1834.

Chou, R., Qaseem, A., Snow, V., Casey, D., Cross, J. T., Shekelle, P., and Owens, D. K. (2007). Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. *Annals of Internal Medicine* 147(7), 478-491.

Cicero, T.J, Ellis, M.S, Surratt, H.L., Kurtz, S.P. (2014) The changing face of heroin use in the United States: a retrospective analysis of the past 50 years. *JAMA Psychiatry* 71(7), 821-826.

Compton, W. M., Jones, C. M., Baldwin, G. T. (2016). Relationship between nonmedical prescription-opioid use and heroin use. New England Journal of Medicine, 374(2), 154-163.

Currie, J., Jin, J.Y., Schnell, M. (2018). U.S. employment and opioids: is there a connection? NBER Working Paper 24440.

Dahlhamer J., Lucas J., Zelaya, C., et al. (2018). Prevalence of Chronic Pain and High-Impact Chronic Pain Among Adults – United States, 2016. MMWR Morbity and Mortality Weakly Report 2018; 67, 1001–1006. `http://dx.doi.org/10.15585/mmwr.mm6736a2`

Dalgaard, C-J. and Strulik, H. (2014). Optimal aging and death: Understanding the Preston Curve, *Journal of the European Economic Association* 12, 672-701.

Dalgaard, C-J. and Strulik, H. (2017). The genesis of the golden age: Accounting for the rise in health and leisure. *Review of Economic Dynamics* 24, 132-151.

Dalgard, C.-J., Hansen, C.W. and Strulik, H. (2020). Fetal Origins – A Life Cycle Model of Health and Aging from Conception to Death, cege Discussion Paper 400, University of Goettingen.

Dasgupta, N., Funk, M.J., Proescholdbell, S., Hirsch, A., Ribisl, K.M., and Marshall, S. (2016). Cohort study of the impact of high-dose opioid analgesics on overdose mortality. *Pain Medicine* 17(1), 85-98.

DEA (2015). National Heroin Threat Assessment Summary. DEA Intelligence Report, April 2015. `https://www.dea.gov/documents/2015/04/01/2015-national-heroin-threat-assessment-summary`

Ehrlich, I. and Chuma, H. (1990). A Model of the demand for longevity and the value of life extension. *Journal of Political Economy* 98, 761–82.

Evans, W., Lieber, E., Power, P. (2018). How the reformulation of oxycontin ignited the heroin epidemic. NBER Working Paper 24475.

Finkelstein, A., Luttmer, E.F., and Notowidigdo, M.J. (2013). What good is wealth without health? The effect of health on the marginal utility of consumption. *Journal of the European Economic Association* 11(1), 221-258.

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., and Turk, D. C. (2007). The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychological bulletin, 133(4), 581.

Gavrilov, L.A., and Gavrilova, N.S. (1991). *The Biology of Human Life Span: A Quantitative Approach.* Harwood Academic Publishers, London.

Greene, M. S., and Chambers, R. A. (2015). Pseudoaddiction: fact or fiction? An investigation of the medical literature. Current addiction reports, 2(4), 310-317.

Grossman, M. (1972). On the concept of health capital and the demand for health. *Journal of Political Economy* 80, 223-255.

Grossmann, V. and Strulik, H. (2018). Illicit Drugs and the Decline of the Middle Class. University of Goettingen, cege Discussion Paper 353.

Gruber, J., and Koszegi, B. (2001). Is addiction rational? Theory and evidence. *Quarterly Journal of Economics* 116(4), 1261-1303.

Gupta, S. (2016) Unintended consequences: Why painkiller addicts turn to heroin, CNN International, June 2, 2016. `https://edition.cnn.com/2014/08/29/health/gupta-unintended-consequences/index.html`

Haddox, J. D., Joranson, D., Angarola, R. T., Brady, A., Carr, D. B., Blonsky, E. R., ... and Simon, D. (1997). The use of opioids for the treatment of chronic pain. *Clinical Journal of Pain* 13(1), 6-8

Hughes P.H., and Rieche O. (1995). Heroin epidemics revisited. Epidemiologic Reviews 17(1), 66-73.

IASP Subcommittee on Taxonomy (1979). The need of a taxonomy. *Pain* 6, 249-252.

Johnson, E.M., Lanier, W.A., Merrill, R.M., Crook, J., Porucznik, C.A., Rolfs, R.T., and Sauer, B. (2013). Unintentional prescription opioid-related overdose deaths: description of decedents by next of kin or best contact, Utah, 2008–2009. *Journal of General Internal Medicine* 28(4), 522-529.

Jorda, O., Knoll, K., Kuvshinov, D., Schularick, M., and Taylor, A.M. (2019). The Rate of Return on Everything, 1870–2015. *Quarterly Journal of Economics* 134(3), 1225-1298.

Katz, N., Paillard, F.C., and Van Inwegen, R. (2015). A review of the use of the number needed to treat to evaluate the efficacy of analgesics. *The Journal of Pain* 16(2), 116-123.

Kolodny, A., Courtwright, D.T., Hwang, C.S., Kreiner, P., Eadie, J. L., Clark, T.W., and Alexander, G.C. (2015). The prescription opioid and heroin crisis: a public health approach to an epidemic of addiction. *Annual Review of Public Health* 36, 559-574.

Krueger, A.B. (2017). Where have all the workers gone? *Brookings Papers on Economic Activity*, Fall 2017, 1-87.

Kuehn, B. M. (2014). Driven by prescription drug abuse, heroin use increases among suburban and rural whites. *Jama* 312(2), 118-119.

Kuhn, M., Wrzaczek, S., Prskawetz, A., and Feichtinger, G. (2015). Optimal choice of health and retirement in a life-cycle model. *Journal of Economic Theory* 158, 186-212.

Layard, R., Mayraz, G., and Nickell, S. (2008). The marginal utility of income. *Journal of Public Economics* 92, 1846-1857.

Machlin, S. and Carper, K. (2014) Out-of-Pocket Health Care Expenses by Age and Insurance Coverage, United States, 2011. Statistical Brief 441. June 2014. Agency for Healthcare Research and Quality. Rockville, MD.

Madadi, P., and Persaud, N. (2014). Suicide by means of opioid overdose in patients with chronic pain. *Current Pain and Headache Reports* 18(11), 460.

Magni, G., and Merskey, H. (1987). A simple examination of the relationships between pain, organic lesions and psychiatric illness. Pain, 29(3), 295-300.

Massie, M. J. (2004). Prevalence of depression in patients with cancer. *JNCI Monographs* 2004(32), 57-71.

Mathers, B.M., Degenhardt, L., Bucello, C., Lemon, J., Wiessing, L., and Hickman, M. (2013). Mortality among people who inject drugs: a systematic review and meta-analysis. *Bulletin of the World Health Organization* 91(2), 102-123.

McNamee, P., and Mendolia, S. (2014). The effect of chronic pain on life satisfaction: evidence from Australian data. Social science and medicine, 121, 65-73.

Meldrum, M.L. (2016). The ongoing opioid prescription epidemic: historical context. *American Journal of Public Health* 106(8), 1365-1366.

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002a). The accumulation of deficits with age and possible invariants of aging. *Scientific World* 2, 1816-1822.

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002b). The mortality rate as a function of accumulated deficits in a frailty index. *Mechanisms of Ageing and Development* 123, 1457-1460.

Muhuri, P.K., Gfroerer, J.C., Davies, M.C. (2013). Substance Abuse and Mental Health Services Administration. Associations of nonmedical pain reliever use and initiation of heroin use in the United States. CBHSQ Data Review, Center for Behavioral Health Statistics and Quality, August 2013.

Murphy, K.M., and Topel, R. H. (2006). The value of health and longevity. *Journal of Political Economy* 114(5), 871-904.

NIDA (2018a). What are prescription opioids? National Institute on Drug Abuse; National Institutes of Health; U.S. Department of Health and Human Services. `https://www.drugabuse.gov/publications/drugfacts/prescription-opioids`

NIDA (2018b). Prescription Opioids and Heroin. Research Report Series, National Instituts on Drug Abuse; U.S. Department of Health and Human Services.

Olafsdottir, T., Asgeirsdottir, T.L., and Norton, E.C. (2020). Valuing Pain using the Subjective Well-being Method. *Economics and Human Biology* 37, 100827.

Orphanides, A., and Zervos, D. (1995). Rational addiction with learning and regret. *Journal of Political Economy* 103(4), 739-758.

Orphanides, A., and Zervos, D. (1998). Myopia and addictive behaviour. ıEconomic Journal 108(446), 75-91.

Pavarin, R. M. (2015). Mortality risk among heroin abusers: Clients and non-clients of public treatment centers for drug addiction. Substance use and misuse, 50(13), 1690-1696.

Portenoy, R.K., and Foley, K.M. (1986). Chronic use of opioid analgesics in non-malignant pain: report of 38 cases. *Pain* 25(2), 171-186.

Rabin, M. (1998). Psychology and economics. *Journal of Economic Literature* 36(1), 11.

Ruhm, Christopher J. (2018). Death of Despair or Drug Problems. NBER Working Paper No. 24188.

Schnell, M. (2017). Physician behavior in the presence of a secondary market: The case of prescription opioids. Mimeo, University of Princeton.

Schünemann,, J., Strulik, H., and Trimborn, T. (2017a). Going from bad to worse: Adaptation to poor health, health spending, longevity, and the value of life. *Journal of Economic Behavior and Organization* 140, 130-146.

Schünemann, J., Strulik, H., Trimborn, T. (2017b). Sex Differences in Morbidity and Mortality: How Much Is Explained by Behavior?. *Journal of Health Economics* 54, 79-90.

Schurer, S., Shields, M. A., and Jones, A. M. (2014). Socio-economic inequalities in bodily pain over the life cycle: longitudinal evidence from Australia, Britain and Germany. Journal of the Royal Statistical Society: Series A (Statistics in Society), 177(4), 783-806.

Smyth, B., Hoffman, V., Fan, J., and Hser, Y. I. (2007). Years of potential life lost among heroin addicts 33 years after treatment. *Preventive Medicine* 44(4), 369-374.

Stagnitti, M.N. (2017). Total Expenses, Total Utilization, Distribution by Source of Payment and Average Total and Out-of-Pocket Payments for Outpatient Prescription Opioids in the U.S. Adult Civilian Noninstitutionalized Population, 2015. Statistical Brief 505. November 2017. Agency for Healthcare Research and Quality, Rockville, MD.

Strulik, H. (2018). Smoking kills: An economic theory of addiction, health deficit accumulation, and longevity. *Journal of Health Economics* 62, 1-12.

Strulik, H. (2019). An Economic Theory of Depression and its Impact on Health Behavior and Longevity, *Journal of Economic Behavior and Organization* 158, 269-287.

Strulik, H. (2019b). Limited self-control and longevity. *Health Economics* 28, 57-64.

Strulik, H. (2020). Opioid epidemics. *Economics and Human Biology* 37, 100835.

Strulik, H., and Vollmer, S., (2013). Long-run trends of human aging and longevity. *Journal of Population Economics* 26(4), 1303-1323.

Trimborn, T., Koch, K.-J. and Steger, T.M. (2008). Multidimensional transitional dynamics: A simple numerical procedure. *Macroeconomic Dynamics* 12, 301–319.

Tsang, A., Von Korff, M., Lee, S., Alonso, J., Karam, E., Angermeyer, M. C., ... and Gureje, O. (2008). Common chronic pain conditions in developed and developing countries: gender and age differences and comorbidity with depression-anxiety disorders. *The Journal of Pain* 9(10), 883-891.

Van Zee, A. (2009). The promotion and marketing of oxycontin: commercial triumph, public health tragedy. *American Journal of Public Health* 99(2), 221-227.

Verdu, B., Decosterd, I., Buclin, T., Stiefel, F., and Berney, A. (2008). Antidepressants for the treatment of chronic pain. Drugs, 68(18), 2611-2632.

Volkow, N.D. (2014) America's Addiction to Opioids: Heroin and Prescription Drug Abuse. National Institute on Drug Abuse.

Volkow, N., Benveniste, H., and McLellan, A. T. (2018). Use and misuse of opioids in chronic pain. *Annual Review of Medicine* 69, 451-465.

Vowles, K.E., McEntee, M.L., Julnes, P.S., Frohe, T., Ney, J.P., and van der Goes, D. N. (2015). Rates of opioid misuse, abuse, and addiction in chronic pain: a systematic review and data synthesis. *Pain* 156(4), 569-576.

Williams, C.M., Maher, C.G., Latimer, J., McLachlan, A. J., Hancock, M.J., Day, R.O., and Lin, C W.C. (2014). Efficacy of paracetamol for acute low-back pain: a double-blind, randomised controlled trial. *The Lancet* 384(9954), 1586-1596.

Wilson, K. G., Chochinov, H. M., Skirko, M. G., Allard, P., Chary, S., Gagnon, P. R., ... and Fainsinger, R. L. (2007). Depression and anxiety disorders in palliative cancer care. *Journal of Pain and Symptom Management* 33(2), 118-129.

Zenz, M., Strumpf, M., and Tryba, M. (1992). Long-term oral opioid therapy in patients with chronic nonmalignant pain. *Journal of Pain and Symptom Management* 7(2), 69-77.

Zhou, C., Florence, C.S., and Dowell, D. (2016). Payments For Opioids Shifted Substantially To Public And Private Insurers While Consumer Spending Declined, 1999–2012. *Health Affairs* 35(5), 824-831.

Zweifel, P. and Breyer, F. (1997). *Health Economics*, Oxford University Press, Oxford.

Appendix A: Derivation of the Euler Equations

Obtain $\dot{\lambda}_k$ from differentiation of (6) with respect to age and substitute λ_k and $\dot{\lambda}_k$ in (9) to obtain (14). Log-differentiate (7) with respect to age and insert (9) to obtain:

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left(\frac{\dot{\lambda}_D}{\lambda_D} + r - \rho\right) \tag{A.1}$$

Use (6) and (7) to replace λ_k and λ_D in (10) and obtain

$$\frac{\dot{\lambda}_D}{\lambda_D} = \rho - \mu - \frac{\mu A \gamma h^{\gamma-1} c^\sigma}{\phi p}\left\{\frac{\partial S/\partial D}{S(D,z)}\left[\frac{c^{1-\sigma}-1}{1-\sigma} - P(D,z)g(m)\right] - \delta\omega D^{\omega-1}g(m)\right\}. \tag{A.2}$$

Insert $\frac{\partial S/\partial D}{S(D,z)}$ obtained from the function parameterized in Section 4.1 and then insert (A.2) in (A.1) to obtain (15). Insert $\partial g/\partial m^j = -\eta^j \exp(-m^j)$ and the parameterized pain function in (12) and solve $G(m^j) = 0$ for m^j to obtain (16).
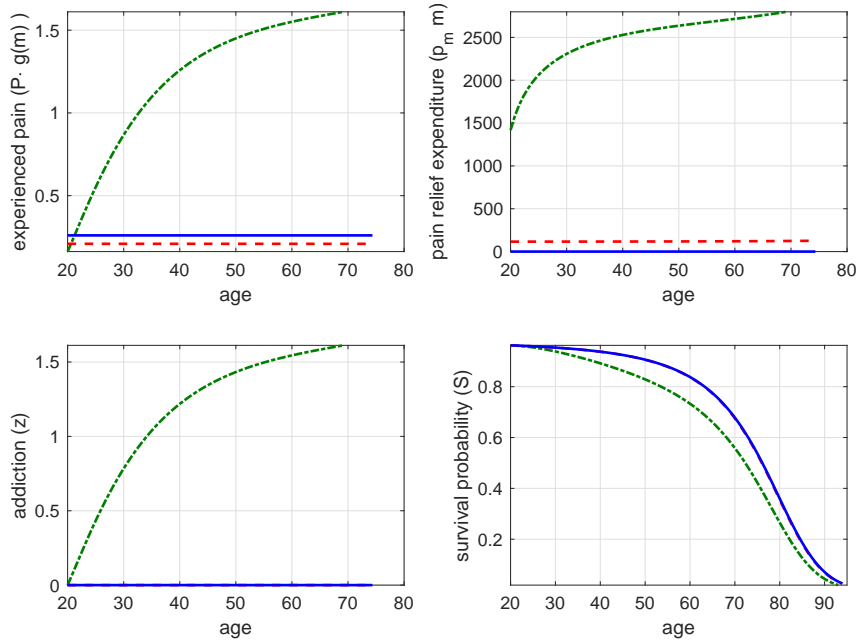
Appendix B: Sensitivity Analysis and Comparative Dynamics

Results of the Sensitivity Analysis are shown in Table A.1, which extends the benchmark results shown in Table 1. To alleviate comparisons, Table A.1 reiterates the benchmark case and the benchmark experiment as case 1 to 4. We first consider lower efficacy of pain treatment. A recent literature has argued that the power of OPRs in reducing chronic pain has been overrated (see Busse et al., 2018 for a systematic review). We thus consider $\eta^j = 0.4$ if j is oxycontin and $\eta^j = 0.2$ if j is ibuprofen. As explained above, a case where efficacy of OPRs falls below that of light painkillers may be observable at the individual level but is intellectually uninteresting because there would be no OPR use. Hence, we consider the efficacy of both painkillers to be lower than for the benchmark run. As shown for case 5 to 6 in Table A.1, the implications of these changes on welfare are relatively small for the case of light painkillers and substantial for OPRs. Qualitatively, we observe again that TORA individuals prefer light painkillers and TICA individuals prefer OPRs. Figure A.1 shows the predicted lifetime trajectories.

For all robustness checks shown in Table A.1, light painkiller use is the preferred treatment of fully informed TORA individuals if OPRs are addictive, while OPR use is the preferred treatment of TICA individuals. Case 7 and 8 show that results are insignificantly affected if light painkillers are much cheaper (half of benchmark price) or bought over the counter with 100 percent out-of-pocket share. The reason is that the impact of light painkillers use on lifetime behavior is small anyway. Painkiller expenditure makes up an insignificant amount of total expenditure, pain reduction is small, and there exists the ceiling effect which prevents excessive consumption of light painkillers.

In case 9 we briefly consider the case of OPRs not being addictive ($\alpha^j = 0$), i.e. we consider an individual that is otherwise identical to the benchmark individual but physiologically unable to develop an OPR addiction. Naturally, OPR treatment is now preferred regardless of the behavioral assumption on addiction control. As seen by comparison with ibuprofen treatment from case 2, addiction-free OPR treatment leads to a lower loss in welfare through pain (due to

Efficacy of painkillers: $\eta^j = 0.4$ if j is oxycontin, $\eta^j = 0.2$ if j is ibuprofen. All other parameters as for the benchmark run from Figure 3. Blue (solid) lines: Reference American with untreated moderate pain ($\delta = 0.26$). Red (dashed) lines: common analgesic ($\eta = 0.4$, $\alpha = 0$). Green (dash-dotted) lines: prescription opioid ($\eta = 0.6$, $\alpha = 0.03$).

its greater efficacy) although it leads to a greater loss in life expectancy (due to its more severe side-effects).[13]

If addiction is stronger than in the benchmark case, the negative effects of OPR use increase. In case 10 in Table 1 we see that doubling the strength of addiction (to $\alpha = 0.06$) entails an almost proportional reduction of wellbeing (to $\Delta V/V = -25.7$) and life expectancy ($\Delta LE = -10.7$ years). Case 11 shows similar results for a doubling of pain from addiction (ζ increases from 1.5 to 3.0), capturing an individual with higher negative tolerance and thus faster adaptation to opioid use. Case 12 reports similar effects for a reduction of the depreciation rate of addiction capital by factor 2 (to 0.05), a second channel that makes withdrawal more difficult.

In case 13 we consider an individual who finances OPRs completely out of pocket. Compared to subsidized OPR use (case 3), the negative consequences on wellbeing and life expectancy are somewhat smaller. The effects are small because pain reduction requires a certain dose of OPR and a pronounced reduction in demand would lead to a strong loss of pain relief (see Figure 2). As a result, OPR demand and thus addiction are not much affected by the fact that individuals cover all costs privately. This feature becomes even more evident for case 15 that abandons access to prescription opioids such that OPRs are bought on the black market

---

[13]According to the meta study of Vowles et al. (2015), between 8 and 23% of chronic pain patients treated with OPRs develop an addiction. It may well be that some pain patients treated with OPRs do not develop an addiction because $\alpha^j = 0$. However, these cases are less interesting for the present study. The more interesting case is that addiction does not develop although $\alpha^j > 0$ because patients rationally control OPR intake, see Section 6.2.

Table A.1: Comparative Dynamics and Sensitivity Analysis: Effects on Wellbeing and Longevity

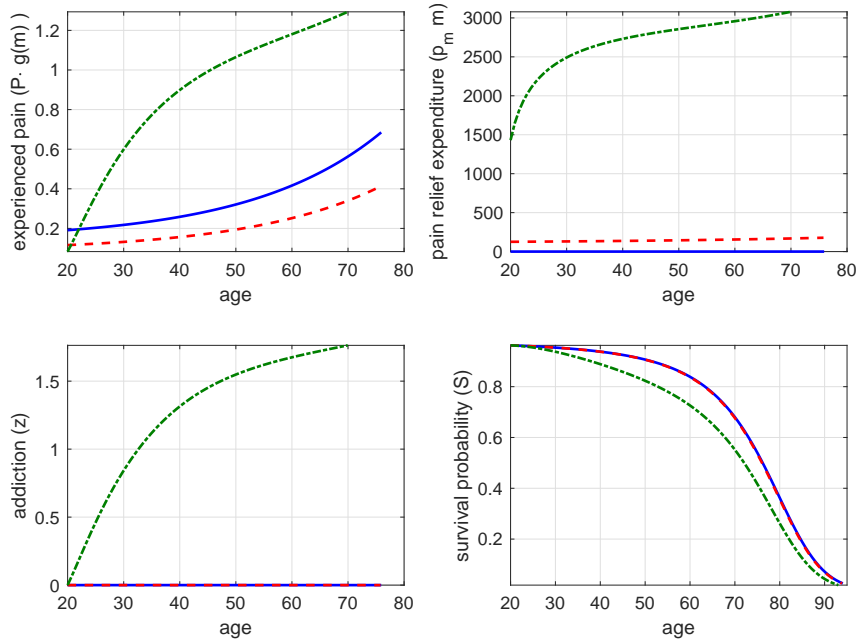| case	$\Delta V/V$	ΔLE
1) moderate pain, untreated ($\delta = 0.26$)	-3.35	-0.06
2) light painkiller ($\eta = 0.4$)	-2.08	-0.16
3) prescription opioid ($\eta = 0.6$)	-12.8	-5.38
4) ...and switch to light painkiller at age 30 ($\Phi = 1$)	-10.8	-1.17
5) light painkiller ($\eta = 0.2$)	-2.74	-0.16
6) prescription opioid ($\eta = 0.4$)	-15.7	-6.37
light painkiller ($\eta = 0.4$) and...		
7) low price ($p_m = 15$)	-2.08	-0.16
8) full out-of-pocket ($\phi = 1$)	-2.11	-0.17
prescription opioid ($\eta = 0.6$) and...		
9) no addiction ($\alpha = 0$)	-1.68	-0.51
10) strong addiction ($\alpha = 0.06$)	-25.7	-10.7
11) higher tolerance ($\zeta = 3$)	-22.6	-6.35
12) lower depreciation ($\psi = 0.05$)	-18.5	-8.86
13) high out-of-pocket ($\phi = 1$)	-11.2	-4.65
14) black market ($p_m = 5000$, $\phi_m = 1$)	-8.6	-3.0
aging-related pain ($\omega = 1$) and...		
15) moderate pain, untreated ($\delta = 0.7$)	-3.39	-0.00
16) light painkiller ($\eta = 0.4$)	-2.11	-0.14
17) prescription opioid ($\eta = 0.6$)	-12.7	-5.37
labor market effects for moderate pain ($\delta = 0.26$), $\beta = 20$, and...		
18) no treatment	-3.42	-0.07
19) prescription opioid ($\eta = 0.6$)	-13.3	-5.44

$\Delta V/V$ is the change in expected life-time utility (the value of life) in percent. ΔLE is the change in life expectancy at age 20 in years.

at an eightfold higher price. As a result of the drastic increase, individuals reduce demand and the overdose probability as well as the health repercussions imply a loss of life expectancy of "only" 3.0 years compared to 5.4 years for subsidized prescription OPR use. Lower OPR consumption affects wellbeing positively through reduced health effects but negatively through more unfulfilled cravings due to addiction.

We next consider aging-related pain, i.e. pain that increases in conjunction with the development of health deficits. For that purpose, we set $\omega = 1$ and re-calibrate δ such that pain requires the same compensation (of \$ 56 per day) as in the benchmark case. This leads to the estimate $\delta = 7$. The resulting life cycle trajectories are shown in Figure A.2. Despite the different evolution of pain, the implications for wellbeing and life expectancy are very similar to the benchmark run, as shown in case 15-17 in Table A.1 (compared to case 1-3).

Labor Supply Effects. In an extension of the model we next take into account that pain and addiction may affect labor market supply. This feature can most conveniently be implemented by considering early retirement. According to the setup of the life cycle model, the permanent income hypothesis applies and all effects of reduced labor supply run through reductions in

FIGURE A.2: AGING-RELATED PAIN, PAIN TREATMENT, AND HEALTH OUTCOMES

Blue (solid) lines: Reference American with untreated moderate pain ($\delta = 0.26$). Red (dashed) lines: common analgesic ($\eta = 0.4$, $\alpha = 0$). Green (dash-dotted) lines: prescription opioid ($\eta = 0.6$, $\alpha = 0.03$).
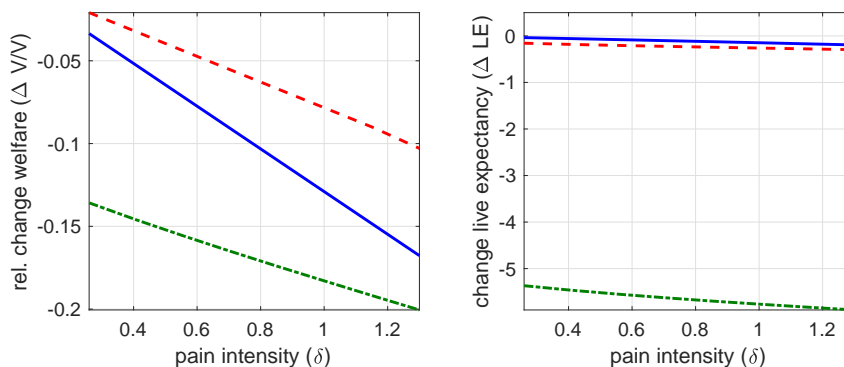
lifetime income. Changes in labor supply at the intensive and extensive margin thus have the same effects on health and wellbeing as long as they result in the same change of discounted lifetime income. Suppose that retirement age R is reached when $R = \bar{R} - \beta P(D(R), z(R))$. Without pain, individuals retire at $\bar{R} = 45$, i.e. at age 65 as in the benchmark case. We consider a drastic reduction in labor supply through pain by setting $\beta = 20$. This means that untreated pain leads to 5.2 years earlier retirement and a loss of lifetime income of more than \$ 180,000. The study of Garthwaite (2012) considers the labor supply effects of specific non-OPR painkillers and estimates that the removal of Vioxx, a cox-2 inhibitor developed to treat chronic joint pain, has caused a 54 percent reduction of the probability of working for individuals with joint conditions. Given that these individuals were 55 to 75 years old, they had a maximum of around 10 years of potential labor supply ahead and thus experienced a maximum loss of about 5 years income. The average income of the sampled individuals with joint pain is \$ 37,530 and thus similar to that of the Reference American. The numerical experiment thus captures the upper end of Garthwaite's (2012) estimates of the labor supply and income effects for untreated joint pain.[14] As shown in case 18 and 19 in Table A.1 (when compared to case 1 and 3), this leads to only marginal changes in the impact of pain and pain treatment on wellbeing and life expectancy. The reason is that the income effects from reduced labor supply are dwarfed by the value of pain. Pain evaluated at \$ 56 per day accumulates to a compensation value of \$1,134,420

---

[14]It seems conceivable that opioid addiction exerts an independent influence on unemployment. Krueger (2017) provides evidence in favor of a large negative impact of the opioid crisis on labor force participation. Currie et al. (2018), however, cannot confirm this result and provide evidence in favor of a mild positive effect of opioid consumption on female employment and no effect for male employment.

per lifetime. Thus even labor supply effects that could be considered implausibly large in light of the empirical evidence (see Currie et al., 2018) entail only small changes in the benchmark results.

Pain Intensity. We next return to the basic model and investigage the comparative dynamics with respect to pain intensity δ. Results are condensed in Figure A.3. Solid (blue) lines show the change in wellbeing and life expectancy (compared to the pain-free benchmark) for alternative untreated pain intensities. Dashed (red) lines show the same outcomes when pain is treated with a light painkiller (ibuprofen) and dash-dotted (green) lines show the outcomes for OPR treatment. All lines originate at benchmark pain ($\delta = 0.26$) and end at fivefold benchmark pain ($5 \cdot 0.26 = 1.3$). At $\delta = 0.51$, pain intensity is at the upper bound of the estimates based on Olafsdottir et al. (2020), reflecting a compensation value of pain of \$ 145 per day. However, this value refers to average pain in a sample of individuals who mostly experienced mild to moderate pain and in which only 19% experienced severe pain. It is thus reasonable to consider also much more severe pain in order to cover the whole distribution of potential states of pain. The (arbitrary) cut off at $\delta = 1.3$ implies a compensation value of pain of more than \$ 350,000 per year, which is about ten times the annual income of the reference American.

FIGURE A.3: PAIN INTENSITY, WELLBEING, AND LIFE EXPECTANCY

Blue (solid) lines: Reference American with untreated moderate pain. Red (dashed lines): common analgesic ($\eta = 0.4$, $\alpha = 0$). Green (dash-dotted) lines: prescription opioid ($\eta = 0.6$, $\alpha = 0.03$).

Figure A.3 shows that lifetime utility declines steeply if pain remains untreated. The loss in lifetime utility (value of life) declines almost linearly in pain intensity from -3 percent to -17 percent. When pain is treated, lifetime utility declines less steeply. The flattest slope is obtained for OPR treatment because chronic pain is a smaller component of the lifetime utility of addicts. Given the different slopes, the lines could in principle intersect. This is, however, not the case, which means that, for all pain intensities, light painkiller treatment is preferred over OPR treatment by TORA individuals. These results are not robust to drastic improvements in efficacy of OPRs. If, for example, efficacy of OPRs were higher than 0.9 and the pain intensity exceeded 1.2, lifetime utility would improve by use of OPRs, indicating that individuals suffering from great pain benefit from highly efficient OPR treatment even when the negative effects on increasing tolerance, addiction, and overdose possibility are taken into account. The panel on

the right hand side of Figure A.3 shows that life expectancy declines mildly in pain intensity because an extension of a painful life is less desirable such that individuals reduce savings and health investments. The impact of chronic pain on life expectancy, however, is dwarfed by the impact of side effects and overdose due to addiction.

Onset of Pain. In this section we abandon the assumption that chronic pain is always present and consider instead pain shocks. Particularly interesting is the case of chronic pain occurring for the first time in old age. With increasing age, it becomes more likely that the individual dies before addiction unfolds completely, a fact that could make OPR treatment more desirable. For better comparison, we now compute V as the expected *remaining* lifetime utility from the moment of occurrence of the pain shock. We begin with a case of moderate pain ($\delta = 0.26$) occurring at age 60. At this age, the expected value of remaining lifetime utility without pain is 70.2 and the remaining life expectancy without pain is 13.3 years.

Results are summarized in case 1-4 of Table A.2. If pain remains untreated, the individual experiences about the same *relative* loss in lifetime utility of 3.2 percent as it was obtained for chronic pain from age 20 onwards. The impact on life expectancy is smaller not only in absolute terms but also in relative terms. Through OPR treatment the individual loses 0.55 years, i.e. 4 percent of remaining life expectancy, whereas chronic pain and OPR use from age 20 on caused a loss of 5.38 years, i.e. 7 percent of remaining life expectancy, in the benchmark run. The reason is that addiction does not fully develop and the negative side-effects do not fully unfold if the shock hits in old age. A greater part of the negative impact of OPR treatment stems from cravings of addiction and less from its health effects. Case 4 shows that an individual who develops addiction at half of benchmark speed ($\alpha = 0.015$) still experiences lower remaining lifetime utility with OPR use than with light painkiller use. The use of light painkillers is still preferred over OPRs by TORA individuals.

The picture changes somewhat when we consider the results for a severe pain shock at age 60 ($\delta = 1.0$) in case 5-8 of Table A.2. Now, if addiction develops slowly (for $\alpha = 0.015$), OPR treatment improves lifetime utility compared to no treatment although treatment with light painkillers still outperforms OPR treatment. Qualitatively, these results are preserved when the severe pain shock hits at age 70 (as shown in case 9 to 12). This means that severe pain is not sufficient to motivate OPR use of fully rational elderly individuals as long as light painkillers are available.

Table A.2: Pain Onset in Old Age: Effects on Wellbeing and Longevity

case	Δ V/V	ΔLE
Pain onset at age 60, moderate pain ($\delta = 0.26$).		
1) no treatment	-3.23	-0.00
2) light painkiller	-2.00	-0.01
3) OPR treatment	-13.5	-0.55
4) OPR, slow addiction ($\alpha = 0.015$)	-7.11	-0.29
Pain onset at age 60, severe pain ($\delta = 1.0$).		
5) no treatment	-12.4	-0.02
6) light painkiller	-7.50	-0.03
7) OPR treatment	-18.4	-0.62
8) OPR, slow addiction ($\alpha = 0.015$)	-11.7	-0.33
Pain onset at age 70, severe pain ($\delta = 1.0$).		
9) no treatment	-12.4	-0.01
10) light painkiller	-7.40	-0.01
11) OPR treatment	-16.7	-0.23
12) OPR, slow addiction ($\alpha = 0.015$)	-10.9	-0.13

Expected lifetime utility V is measured from the age when the shock hits (the remaining value of life).