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**FROM PAIN PATIENT TO JUNKIE: AN  
ECONOMIC THEORY OF PAINKILLER  
CONSUMPTION AND ITS IMPACT ON  
WELLBEING AND LONGEVITY**

**Revised Version May 2019**

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

Holger Strulik<sup>†</sup>

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**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 and their implications for welfare and life expectancy. Computational experiments are used to identify fully rational and imperfectly rational addiction behavior. Fully rational addicts quickly quit OPR use when new information about their addictive potential 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.

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## 1. INTRODUCTION

Economists 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 (eg. 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 to address a second form of treatment of illness, aside from investments in health maintenance and repair: the treatment of pain by analgesics (painkillers). 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). The experience of pain can be distinguished 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 and pain management (rather than the repair of physical health deficits) is the main focus of treatment.

Chronic pain is widespread. According to the most recent 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, 2018). Of those who reported chronic pain, 40% noted that they were constantly in pain (American Academy of Pain Management, 2003). 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. (2017) provides 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, however, are 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.<sup>1</sup> Moreover, treatment with analgesics is subject to the so called ceiling effect. This means there exists a limit as to how much pain can be reduced by increasing dosage. Above this level, increasing dosage does not relieve more pain but increases the risk of serious side effects.

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 do no longer respond to the treatment as strongly as they did initially and increasing doses are required to achieve the same effect. Tolerance increases the risk of overdose, i.e. to apply the drug in quantities greater 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).

The fact that doctors and patients were aware of the threat of addiction certainly contributed to 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 and this increase can be attributed mainly to increasing OPRs treatment of chronic noncancer pain (CDC, 2017b). As a result,

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<sup>1</sup>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).

many patients developed 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. American 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 than 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 increased sharply, by about factor 5 from 2010 to 2016 (CDC, 2017a). The surge of heroin consumption and the decline of OPR prescriptions are likely causally related. In the early 21st century it became increasingly 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, 4 out of 5 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 (or because of despair) but white middle-class residents of the suburbs who accidentally became addicted to heroin by a generous OPR treatment of chronic pain.

When a prescription is discontinued or the prescribed dose becomes insufficient due to increasing tolerance, addicted consumers may switch to illicit use of OPRs, obtained at a higher 'street' price, or substitute OPRs by other opioids, like heroin. 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. Recreational drug use, however, has been already covered by several economic theories of addiction such that it is omitted here to focus on the novel feature of initiation of addiction and illicit drug use through pain treatment (Kodolny et al., 2015). A study based on descriptions of decedents of OPR-overdose victims found that 87 percent of the deceased used prescribed pain medication in the year before death (Johnson et al., 2013).

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 get 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 there exists a quasi-exponential association between age and the frailty index 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).<sup>2</sup>

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

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<sup>2</sup>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 and Strulik (2015) 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).

explicitly consider its dark side. In particular, individuals may become addicted to the treatment, increasing tolerance may motivate the consumption of increasing doses of treatment, and the cravings from addiction may generate pain that motivates to sustain treatment 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. In particular, we investigate how, depending on prescription- and street-prices and the individual state of health, different types of pain treatment are preferred over the life cycle. Another distinguishing feature is that death is conceptualized as a stochastic event, which is necessary in order to include death from drug overdose in the analysis.

The paper discusses 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).<sup>3</sup>

The paper 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 optimal response to this information, TORA addicts are predicted to drastically reduce their consumption or completely quit using the drug. TICA addicts, in contrast, do not respond with reduced consumption, further develop their addiction, and consume even more OPRs. In a second computational experiment, it will be shown that, after the termination of an OPR

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<sup>3</sup>Both TORA and TICA addicts could be subject to other constraints on rationality like time-inconsistent decision making (Gruber and Koszegi, 2001) or self-control problems (Strulik, 2019). These assumptions are more severe in the sense that they imply suboptimal decision making also for all other life cycle choices. 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 takes a benign view on painkiller addicts in the sense that their only imperfection is their failure to control the addiction. While agents who fail to understand their time-inconsistent behavior have been called naive (Rabin, 1998), this attribute is perhaps too strong for pain patients who “only” fail to optimally control their addiction.



prescription, TORA addicts drastically reduce their consumption and eventually quit. TICA addicts, in contrast, switch to illicit OPR use or heroin consumption. 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 would increase the value of life of an imperfectly rational OPR addict by about 10 percent and life expectancy at 20 by about 9 years.

This paper is related to a series of recent economic studies investigating different aspects of the opioid crisis. 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 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) proposes 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 less OPR-related death and more heroin deaths with no effect on total deaths from overdose.

The paper is organized as follows. In the next section, I integrate utility-reducing pain, treatment by alternative analgesics and illicit drugs, opioid addiction, and overdose death, in a life-cycle model of endogenous health and longevity. In Section 3, I prove a couple of propositions on the determinants of painkiller use. In Section 4, I parameterize and solve the full model and calibrate it for a benchmark U.S. American and three stylized painkillers (ibuprofen, oxycontin,

heroin). In Section 5, I present the main results on painkiller use and its effects on wellbeing and longevity. Computational experiments identify the behavioral responses of fully rational and imperfectly rational OPR addicts after price and information shocks. A sensitivity analysis includes labor supply effects, different pain intensities and onsets, and the use of opioids in palliative care. 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 treatment of OPR addiction. Section 6 concludes the paper.

## 2. THE MODEL

**2.1. 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 sciatica, is the cause of pain and pain does not (much) increase 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  $j = 1 \dots J$ . We assume that painkillers are substitutes such that, for a 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. Let  $m^j$  denote the quantity consumed of painkiller  $j$  and let  $m$  denote the vector of quantities consumed of all painkillers,  $m = \{m^1, \dots, m^J\}$ . 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  $\partial g / \partial m^j < 0$ . We assume declining marginal efficacy  $\partial^2 g / \partial (m^j)^2 > 0$ . Some painkillers are potentially addictive and lead to increasing pain through the craving for painkiller treatment even in the absence of any pain from health deficits. This phenomenon is known as reinforcement in the theory of addiction.

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

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

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 that individuals consume painkillers merely for the experience of pleasure. In the case of opioids there exists certainly a recreational motive of drug use. Here, however, we want to focus on experienced pain as the so far less explored pathway to addiction.<sup>4</sup>

The feature that utility is separable in goods consumption and pain is a natural benchmark for the analysis. It implies that rich individuals (who consume more) do not experience pain differently (i.e. as more or less severely) than poor individuals. It will be shown below that this assumption nevertheless captures the feature 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  $P(D, h)$  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

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<sup>4</sup>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.

is addicted. The stock of addictive capital evolves according to

$$\dot{z} = \sum_{j=1}^n \alpha^j m^j - \psi z, \quad (2)$$

in which  $\alpha^j \geq 0$  is the addictive power of drug  $j$ . Addiction is thus conceptualized as a consumption habit with reinforcement, tolerance, and withdrawal characteristics. For non addictive painkillers  $\alpha^j = 0$ . Like for all other parameters, we may think of  $\alpha^j$  as being individual-specific such that some substances are addictive for some individuals but not for others. The parameter  $\psi$  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.

As motivated in the Introduction, we consider two different approaches to addiction. According to the theory of rational addiction (TORA), individuals optimally plan and control their addiction (if there is any). According to the theory of imperfectly controlled addiction (TICA), otherwise fully rational individuals fail to control how their addiction develops. Strulik (2018) showed that if life time is finite, TORA (or sophisticated addiction) necessarily implies that addictive goods consumption increases in old age and in particular before death. TICA (or naive addiction), in contrast, can explain falling or hump shaped lifetime paths of addictive consumption. Strulik (2018) argued that these predictions are more in line with actually observed age-consumption patterns when the addictive good is tobacco.

With painkiller consumption, however, it is a priori not clear that the TICA approach is superior to the TORA approach. In particular, with regards to opioids, complete abstention as well as increasing age-consumption profiles with the highest (over-) dose before death are frequently observed and these consumption trajectories can be explained by both theories. It would thus be useful to develop nother distinguishing features between the two approaches. Below, we consider an information shock. A painkiller that formerly was thought to be non-addictive is found to be addictive. After arrival of this information, TORA addicts are predicted to drastically reduce their consumption or to completely quit using the drug. TICA addicts, in contrast, do not respond with reduced consumption to the information shock. In a second computational experiment, it is shown below that, after a drastic price increase of prescription OPRs, TORA addicts drastically reduce their consumption and eventually quit. TICA addicts, in contrast, switch to illicit heroin consumption.

**2.2. 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., 2002). 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

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

in which  $\mu$  is the “natural” force of aging,  $A$  and  $\gamma$  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  $j$ .

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  $q = -\dot{S}/S$ . There exists an upper limit of health deficits beyond which survival is impossible,  $S(D, z) = 0$  for  $D \geq \bar{D}$ .

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

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

in which  $r$  is the interest rate,  $q$  is the conditional mortality rate (i.e. we assume insurance by perfect annuities),  $p$  is the price of health investment, and  $\phi$  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  $\phi_m^j$ . We allow for the existence of several types of painkillers comprising for example mild analgesics like paracetamol (low efficiency  $\eta^j$ , low harm  $B^j$ ), more effective and potentially addictive prescription opioids like oxycontin (high  $\eta^j$ , high  $\alpha^j$ , low  $\phi_m^j$ ), and illicit drugs like heroin (high  $\eta^j$ , high  $\alpha^j$ , high  $\phi_m^j$ , and high  $B^j$ ).

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  $\rho$  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  $m^j$ , taking into account the constraints (3) and (4). 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. The associated current-value Hamiltonian is

$$\begin{aligned}
H = & S(D, z) [u(c) - P(D, z) \cdot g(m)] \\
& + \lambda_k \left[ w + (r + q)k - c - p\phi h - \sum_{j=1}^J \phi_m^j p_m^j m^j \right] + \lambda_D \mu \left[ D - Ah^\gamma + \sum_{j=1}^J B^j m^j - a \right] \\
& + \Phi \lambda_z \left[ \sum_{j=1}^n \alpha^j m^j - \psi z \right], \tag{5}
\end{aligned}$$

in which  $\lambda_k$ ,  $\lambda_D$ , and  $\lambda_z$  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  $\Phi \in \{0, 1\}$  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 in their life plans how their addiction develops.  $\Phi = 0$  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 addicts are not myopic. 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 for the shadow price of addiction that  $\lambda_z(T) = 0$  (see e.g.

Theorem 7.1 in Acemoglu, 2009). The first order conditions for a maximum are:

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

$$- S(D, z) P(D, z) \frac{\partial g(m)}{\partial m^j} \leq p_m^j \phi_m^j \lambda_k - \lambda_D \mu B^j + \Phi \lambda_z \alpha^j \quad \text{with } = \text{ for } m^j > 0, \quad (7)$$

$$- \lambda_D \mu A \gamma h^{\gamma-1} = \lambda_k \phi p. \quad (8)$$

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  $\lambda_k$ . Equation (7) 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,  $\mu B^j$ , evaluated with the shadow price of health deficits,  $\lambda_D$ . Individuals who rationally plan their addiction (for whom  $\Phi = 1$ ) additionally take into account the increase in addiction  $\alpha^j$  caused by one unit of painkiller consumption evaluated at the shadow price of addiction  $\lambda_z$ . Equation (8) 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.

The costate equations associated with the optimal solution are given by

$$\lambda_k r = \lambda_k \rho - \dot{\lambda}_k, \quad (9)$$

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

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

and  $\dot{\lambda}_z = \lambda_z = 0$  for  $\Phi = 0$ . Thus, individuals who rationally plan their addiction take the evolution of its shadow price  $\lambda_z$  into account. For imperfectly rational individuals the shadow price of addiction is zero. Recall that this does not mean that their addiction does not influence their behavior. It means that they do not optimally plan and control their addiction.

### 3. 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.

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

To verify this, rewrite (11) as

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

Notice that  $\rho, \psi > 0$  and  $0 \leq g(m) < 1$ . The term in square brackets is positive since pain increases with addiction and survival declines with addiction. Thus, in order to reach the boundary condition  $\lambda(T) = 0$ , 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.<sup>5</sup> Notice that the result does not require that addiction is actually present (it does not require  $z > 0$ ). For non-addicted individuals  $\lambda_z$  captures the potential threat of addiction that is taken into account by TORA individuals when they consider whether they should take 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  $\phi_m^j$  are low, and if the painkiller has little side-effects ( $B^j$  is low).*

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

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

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 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

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<sup>5</sup>This result is a central element of the theory proposed in Strulik (2018) where it is derived and explained in more detail.



(11) that  $\partial G/\partial P > 0$  and  $\partial G/\partial x < 0$  for  $x \in \{p_m^j, \phi_m^j, p, \phi, A, B^j\}$ . As a corollary we observe that individuals who are heavily addicted to painkillers (for whom  $z$  is large) are more inclined to use painkillers. 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  $\phi_m^j$ , and the severity of side-effects on health ( $B^j$ ).*

The proof evaluates condition (11) when equality holds, applies the implicit function theorem,  $dm/dx = -(\partial G/\partial x)/(\partial G/\partial m^j)$ , 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  $\Phi = 0$ ).*

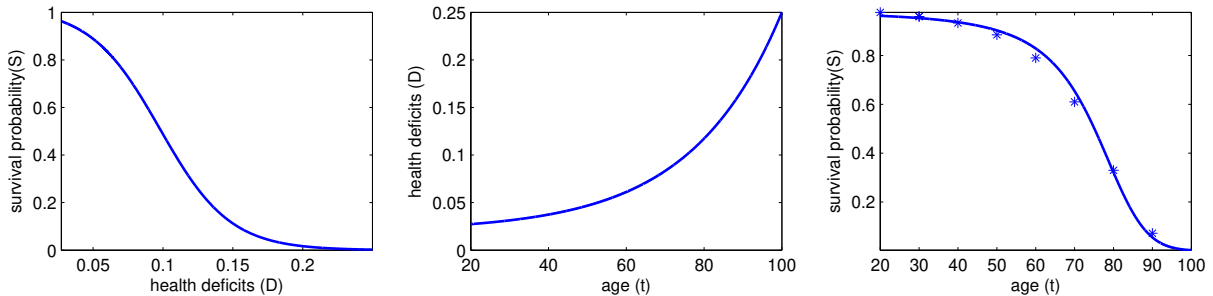
The proof is obvious from applying Lemma 1 to (11). Since the shadow price of addiction is negative for fully rational individuals ( $\lambda_z < 0$ ), they face a higher marginal cost of addiction, which makes them less likely to use addictive substances and, if they use them, they use them more cautiously than imperfectly rational individuals. Notice that both types of individuals would use an addictive substance in equal amounts if they wrongly believe that it is not addictive (i.e. if they believe  $\alpha^j = 0$ ). As shown below, this observation can be exploited for a computational experiment that identifies TORA and TICA behavior.

#### 4. SOLUTION OF THE FULL MODEL

**4.1. 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 shown 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 U.S. American men, taken from Strulik and Vollmer (2013). Death from overdose reduces the survival probability independently from health deficits such that  $S_2 = e^{-\chi^j z t}$ .  $S_2$  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  $\chi^j$ .

FIGURE 1: HEALTH-DEPENDENT SURVIVAL AND SURVIVAL BY AGE



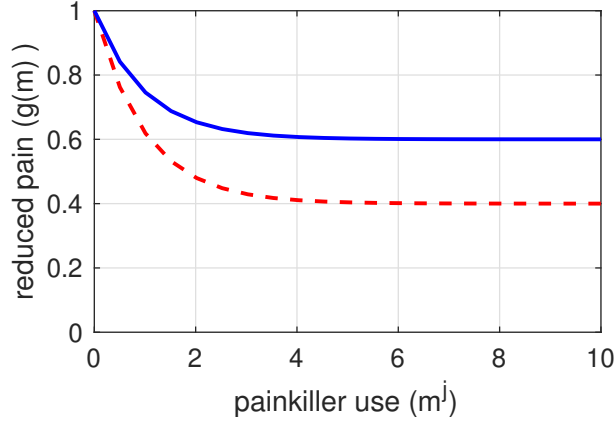
Left: assumed function  $S_1(D)$ . Middle: Estimated association  $D(t)$  (Mitnitski et al., 2002a). Right: Predicted (line) and estimated (stars) association between age and survival probability (estimate from Strulik and Vollmer, 2013).

For simplicity 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  $\delta$  and  $\omega$  reflect the influence of health deficits on pain intensity and  $\zeta$  reflects the influence of addiction;  $\delta$  will be the key parameter to evaluate the intensity of pain. For painkiller efficacy we need a function that implements declining marginal efficacy and prevents simultaneous mixing such that it provides at any instant a unique solution. Additionally, we want to capture the ceiling effect discussed in the Introduction, i.e. the feature that for any painkiller there exists a level beyond which pain cannot be reduced by further increasing drug intake. A parsimonious function that provides all these features is

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

The parameter  $\eta^j$ ,  $j \in \{1, J\}$  measures the maximum degree of pain that can be reduced by taking painkiller  $j$ . Figure 2 shows the painkiller function when  $j$  is the preferred painkiller and  $\eta^j = 0.4$  (solid lines) or  $\eta^j = 0.6$  (dashed lines). The exponential function (13) captures well the ceiling effect, i.e. that there exists a maximum level of pain reduction ( $\eta^j$ ) which is quickly approached with increasing painkiller consumption.

FIGURE 2: PAINKILLER USE AND EFFICACY



Blue (solid)line:  $\eta^j = 0.4$ ; red (dashed) line:  $\eta^j = 0.6$ .

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

$$\frac{\dot{c}}{c} = \frac{r - \rho}{\sigma} \quad (14)$$

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

If painkiller  $j$  is taken, its consumption level is obtained from (10) and (13) as

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

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) from 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 for  $m^j$ . Hence  $m^j = 0$ .

In Section 5 we perform a computational experiment, which can be analytically motivated by the following observation.

PROPOSITION 4. *If, for an addictive painkiller  $j$ , the price  $p_m^j$  or the out-of-pocket  $\phi_m^j$  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  $\phi_m^j$ . For  $\lambda_z < 0$ ,  $R^j$  will exceed 1 for some given parameter values for which it stays below 1 when  $\lambda_z = 0$ .

If the first order conditions provide an interior solution for several painkillers, the unique optimal solution is found by applying (13). This means that for equal (net) marginal efficacy of painkillers, individuals take the one with the greatest absolute efficacy, i.e. the one that provides the greatest reduction in pain.

**4.2. 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. (2017) 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  $\gamma$  to 0.19 as in Dalgaard and Strulik (2014) and close to the estimate of 0.2 by Hall and Jones (2007). We use the data generated by the model to compute the implied value of life ( $VOL$ ) that the individual experiences along the the optimal life cycle trajectory,  $VOL = \int_0^{\tilde{T}} S(D)e^{-\rho\tau} u(c(\tau))d\tau / u'(c(t))$ , in which  $\tilde{T}$  fulfils  $D(\tilde{T}) = \bar{D}$ .

We calibrate the remaining parameters  $a$ ,  $A$ ,  $\rho$ , and  $\sigma$  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 according to 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 is \$ 6.3 million, as estimated by Murphy and Topel (2005). This leads to the estimates  $a = 0.0185$ ,  $A = 0.000325$ ,  $\rho = 0.05$ , and  $\sigma = 1.06$ . The

estimate of  $\sigma$  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. (2017) which estimates the compensation variation for pain, i.e. the additional equivalized income needed to compensate an individual who often suffers from pain for his loss in life satisfaction. Olafsdottir et al. (2017) estimate this compensation to lie between \$56 and \$ 145 per day, i.e. an annual extra income  $\Delta w$  between \$ 20,440 and \$ 52,925. Based on these findings, we estimate the value of  $\delta > 0$  that provides with compensation  $\Delta w$  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. We provide a sensitivity analysis for a pain parameters.

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 benchmark, we compute a compensation of \$ 27 per day. In line with the methodology and results of Olafsdottir et al. (2017), 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). As a light painkiller we consider a stylized treatment with ibuprofen. This analgesic 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 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

( $B^j = 10^{-15}$ ) 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 painkillers 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.<sup>6</sup> Prescribed oxycontin 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 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  $\psi^j = 0.1$ , implying that an addicted individual demands about twice as much oxycontin as a non-addicted individual. Since  $\alpha$  and  $\psi$  are unobserved and potentially individual-specific, we meet the involved parameter uncertainty with a sensitivity analysis. 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 the prescription drug 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).

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<sup>6</sup>The efficacy parameters  $\eta^j$  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.

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 ( $\chi = 0.004$ )

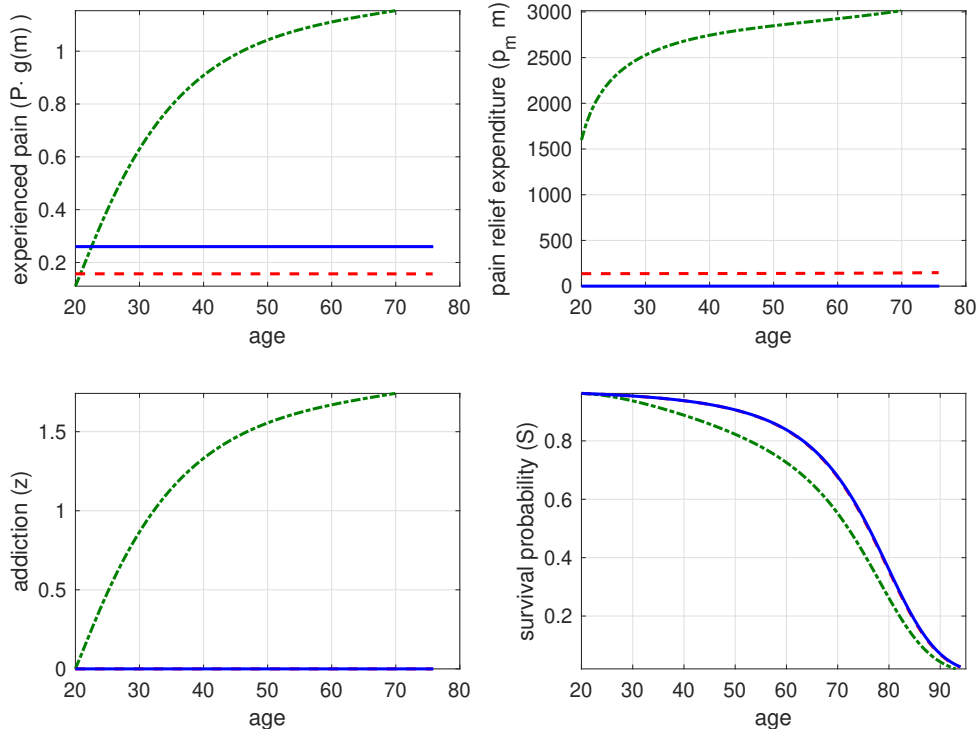
The table shows calibrated values for prescription oxycontin. If the opioid is bought on the black market (for non-medical use) the calibrated price changes from 500 to 4000 (and  $\phi_m$  changes to 1). This is the price with which consumers compare the heroin price.

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, which is sold at street price of about 8 times the prescription price of about \$ 1 per mg (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 in people who inject drugs from Mathers et al. (2013) as 0.62 percent. From this we estimate  $\chi = 0.004$  for heroin. Heroin use also leads to faster deterioration of general health due HIV and other blood-borne viruses transmitted through shared needles and syringes. We capture this fact by setting to  $B^j = 10^{-4}$ . Taking for itself, the increased aging due to infections accounts for 1.0 year lost in life expectancy at 20.

## 5. PAIN, PAINKILLER USE, WELLBEING AND LONGEVITY: RESULTS

**5.1. 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 age 75.5 years. The trajectory for survival probability, however, shows the predicted survival rate at any age. Red (dashed) lines show the trajectories for light pain treatment (ibuprofen).

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 ( $\eta = 0.6$ ,  $\alpha = 0.03$ ).

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. The individual responses to increasing pain by increasing demand for pain treatment and pain relief expenditure rises. 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 increase mortality, mainly through increasing risk of overdose, and the survival curve shifts inwards (lower right panel). Overall, life expectancy declines by 5.4 years.

These results are summarized in the three first rows of Table 2 using two aggregate indicators of wellbeing, life time utility and life expectancy, both measured as deviations from the potential



values achieved by the same individual if it were pain-free and not addicted. Life time utility is measured in terms of relative deviation (in percent), since relative numbers are more informative. In relative terms, the loss in lifetime utility is equal to the loss in the value of life. 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 less in their health. The use of light painkillers (case 2) improves wellbeing compared to no treatment, lifetime utility falls short of that of a pain-free individual by 2 percent.

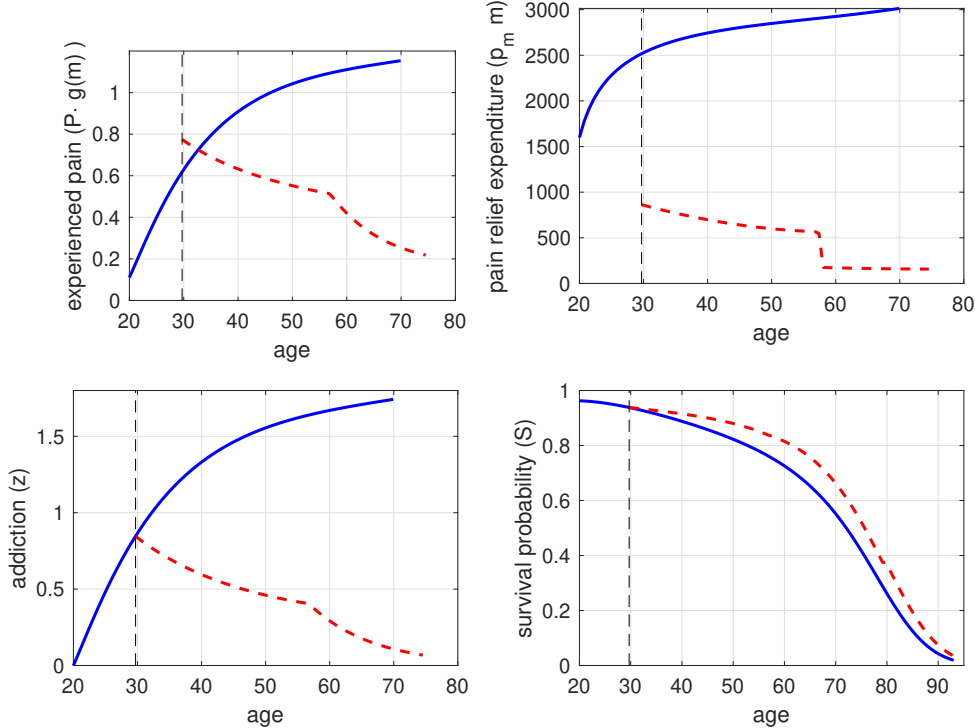
The use of prescription opioids (case 3) reduces lifetime utility by 12.8 percent and life expectancy by 5.4 years. The loss of life satisfaction comes through three channels: (i) through shorter life expectancy due to faster deteriorating health and the probability of overdose; (ii) because the expenditure for opioids reduces the funds available for consumptions, savings, and health investments, (iii) because increasing tolerance and increasing cravings for opioids reduce expected 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.

**5.2. Is Painkiller Addiction Rational?** For the benchmark calibration, fully rational individuals prefer light pain killers. However, even if they took OPR pain relievers, it would be hard to argue for 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 because of their inability to control the addiction. For the benchmark calibration, TICA individuals (who prefer OPRs) lose 12.1 percent in value of life and 5.2 percent in life expectancy compared to TORA individuals. In order to motivate and design policy interventions it would thus be of interest to know whether actual behavior is better described by rational or imperfectly controlled addiction.

The following computational experiment identifies TORA and TICA behavior by exploiting the idea of initially imperfect information and the later arrival of full information. Assume that initially individuals wrongly believe that OPRs are not addictive although they actually are. This can be captured by setting  $\Phi = 0$  for both types of individuals. According to this refinement,

TORA individuals take the shadow price of addiction only into account if they believe that the substance is addictive. If they wrongly believe that the substance is not addictive, TORA and TICA behavior is observationally equivalent. Both types of individuals prefer OPR pain relievers. However, 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.

FIGURE 4: NEW INFORMATION AT AGE 30: OPRs ARE ADDICTIVE



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

To illustrate these considerations with the benchmark model we 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  $\alpha^j = 0.03$ ). Figure 4 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 addiction, the arrival of the new information causes quite drastic changes. As shown by red (dashed) lines, individuals who optimally control their addiction drastically reduce their consumption such that

experienced pain increases in the short run. Due to reduced consumption, addiction gradually recedes and thus pain declines in the medium run. After a while 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). The gain in value of life is smaller because of the induced pain from withdrawal but it is still substantial. The loss of lifetime utility is 2 percent smaller than in the case of unchanged behavior.<sup>7</sup> Depending on parameter specification, the drop in consumption and the convergence speed to an addiction-free life may be slower or faster. Figure A.2 in the Appendix shows that behavior is qualitatively similar if treatment has less power in pain reduction but that TORA addicts are induced to quit much earlier. In Section 5.8 it is shown that quitting occurs also much faster if the arrival of new information on addiction is accompanied by an increase in the price of OPRs. The point is thus of qualitative nature: Only TORA addicts respond to information about the addiction properties of painkillers by reducing their consumption.

**5.3. Sensitivity Analysis Comparative Dynamics.** We next consider the robustness of results by summarizing the comparative dynamics of OPR treatment using the two aggregate measures of the quality and quantity of life,  $\Delta V/V$  and  $\Delta LE$ . We first consider lower efficacy of pain treatment. A recent literature has argued that the power in reducing chronic pain has been overrated for OPRs (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 in Table 2, the implications of these changes on welfare are relatively small for the case light painkillers and substantial for OPRs. Qualitatively, we observe again that TORA individuals prefer light painkillers and TICA individuals prefer OPRs. Figure A.1 in the Appendix shows the predicted lifetime trajectories. Row 7) shows the results from the information arrival experiment for the case of lower OPR efficacy. In this case, TORA individuals quit using OPRs much earlier (see Figure A.2 in the

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<sup>7</sup>For all experiments in Table 2, lifetime utility is computed at the initial age 20. If lifetime utility were computed at age 30 (at the age of the information arrival) the gain from optimally controlling an addiction would be much higher.

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

case	$\Delta V/V$	$\Delta LE$
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
7) ...and switch to light painkiller at age 30 ( $\Phi = 1$ )	-8.68	-0.64
light painkiller ( $\eta = 0.4$ ) and...		
8) low price ( $p_m = 15$ )	-2.08	-0.16
9) full out-of-pocket ( $\phi = 1$ )	-2.11	-0.17
prescription opioid ( $\eta = 0.6$ ) and...		
10) no addiction ( $\alpha = 0$ )	-1.68	-0.51
11) strong addiction ( $\alpha = 0.06$ )	-25.7	-10.7
12) higher tolerance ( $\zeta = 3$ )	-22.6	-6.35
13) lower depreciation ( $\psi = 0.05$ )	-18.5	-8.86
14) high out-of-pocket ( $\phi = 1$ )	-11.2	-4.65
15) black market ( $p_m = 5000, \phi_m = 1$ )	-8.6	-3.0
aging-related pain ( $\omega = 1$ ) and...		
16) moderate pain, untreated ( $\delta = 0.7$ )	-3.39	-0.00
17) light painkiller ( $\eta = 0.4$ )	-2.11	-0.14
18) prescription opioid ( $\eta = 0.6$ )	-12.7	-5.37
labor market effects for moderate pain ( $\delta = 0.26$ ), $\beta = 20$ , and...		
19) no treatment	-3.42	-0.07
20) 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.  $\Delta LE$  is the change in life expectancy at age 20 in years.

Appendix). As a result the loss in life expectancy and of value of life is lower (as compared to case 4) due to the lower health damage during a much shorter addiction period.

For all robustness checks shown in Table 2, 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. Row 8) and 9) 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 anyway small. 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 row 10) we briefly consider the case that OPRs are not 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 its greater efficacy) although it leads to a greater loss in life expectancy (due to its more severe side-effects).<sup>8</sup>

If addiction is stronger than in the benchmark case, the negative effects of OPR use increase. In row 11) 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). Row 12) shows similar results for a doubling of pain from addiction ( $\zeta$  increases from 1.5 to 3.0), capturing an individual with higher negative tolerance and thus faster adaptation to opioid use. Row 13) 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 14 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 inefficiency of the painkiller (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 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. If available, individuals suffering from an addiction control problem may thus prefer to switch to a less expensive opioid like heroin, a scenario that we discuss in detail below.

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<sup>8</sup>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 5.2.

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  $\delta$  such that pain requires the same compensation (of \$ 56 per day) as in the benchmark case. This leads to the estimate  $\delta = 7$ . Despite the different evolution of pain (life cycle trajectories are shown in Figure A.3 in the Appendix), the implications for wellbeing and life expectancy are very similar to the benchmark run, as shown in case 16-18 in Table 2 (compared to case 1-3).

**5.4. 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 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.<sup>9</sup> As shown in case 19 and 20 of Table 3 (when compared to case 1-3) 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 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.

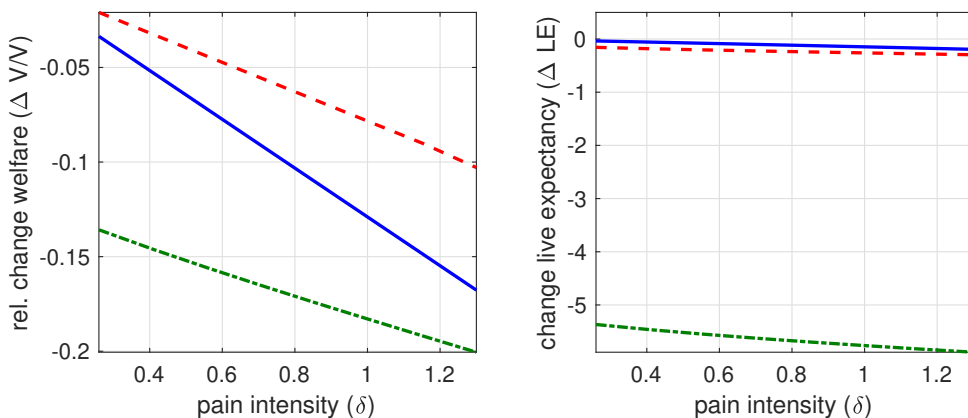
**5.5. Pain Intensity.** We next return to the basic model and discuss in more detail the comparative dynamics with respect to pain intensity  $\delta$ . Results are condensed in Figure 5. Solid (blue) lines show the change in wellbeing and life expectancy (compared to the pain-free benchmark) for alternative 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

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<sup>9</sup>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.

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. (2017), 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 5: PAIN INTENSITY, WELLBEING, AND LIFE EXPECTANCY



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 ( $\eta = 0.6$ ,  $\alpha = 0.03$ ).

Figure 5 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, life time 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 5 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.

**5.6. 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 as  $V$  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 3. 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 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 in case 5-8 of Table 3 the results for a severe pain shock at age 60 ( $\delta = 1.0$ ). 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.

**5.7. Palliative Care.** In this section we consider a pain shock experienced in conjunction with a drastic deterioration of physical health that substantially reduces life expectancy. Such a



Table 3: Pain Onset in Old Age: Effects on Wellbeing and Longevity

case	$\Delta V/V$	$\Delta LE$
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).

scenario is more appropriate to describe chronic malignant pain at the end of life rather than, for example, chronic backpain. In order to check whether the model supports opioid pain treatment in these end-of-life situations we first consider again the severe pain shock ( $\delta = 1$ ) at age 70, which, however, is now accompanied with 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, i.e. by 6.4 years and expected remaining lifetime utility would decline to 5.4 if the last years of life were pain-free. As shown in case 1 of Table 4, severe pain causes an additional loss of welfare of 12 percent. As shown in case 3, OPR treatment is not significantly worse than no treatment although it is still outperformed by light painkillers.

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 row 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 row 5.

Table 4: Pain and Severely Life-Shortening Health Shocks

case	$\Delta V/V$	$\Delta LE$
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

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 status.

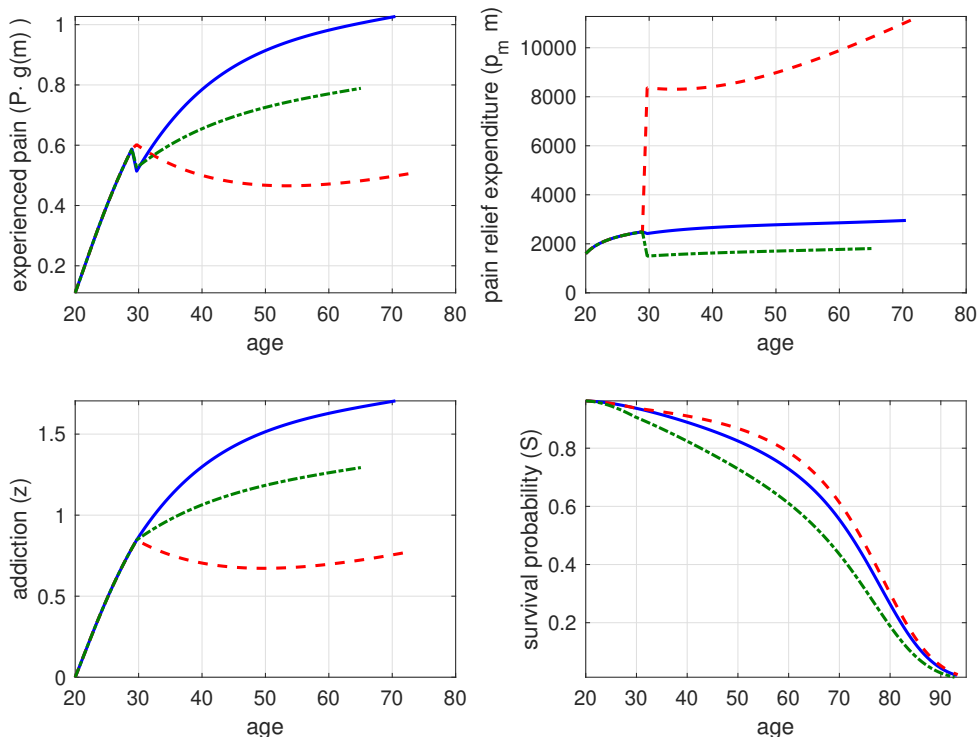
These conclusions do not rest on the onset of severe pain in old age but rather on their appearance in conjunction with drastic, life-shortening shocks in physical health. In order to show this, we next consider a 40-year-old individual who experiences severe pain together with a drastic increase of health deficits from 3.7 to 13.7 percent such that life expectancy at 40 reduces to 4.7 years and expected lifetime utility reduces to 3.9. The effect of severe pain on wellbeing is similar, OPR treatment dominates no treatment, and, if its efficacy raises to 0.8, it dominates treatment by light painkillers. We expect that the preference of OPR raises also at lower efficacy levels if remaining life expectancy is even shorter. Then, however, it becomes increasingly difficult to numerically solve the model.<sup>10</sup>

**5.8. OPR Abuse and Illicit Drug Consumption.** In this section we return to the initial scenario of the onset of light to moderate pain at the age of 20 and consider the transition from pain patient to junkie. For that purpose, we assume that the original source of pain, say backpain, disappears such that any non-addict person 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 manages to receive prescription opioids and support from health insurance such that  $p_m$  and  $\phi_m$  stay at their original values. As shown in the upper left panel of Figure 6, the loss in pain due to absent backpain causes a mere wrinkle in the trajectory of life-cycle pain of

<sup>10</sup>The reason is that model age is measured in years. A re-calibration of the model that measures age in months or days could address this shortcoming.

the addicted. Soon, additional cravings from addiction compensate for the temporary liberation from backpain and the life cycle trajectories for pain, pain relief expenditure, and life expectancy follow the original (benchmark) path. Case 1 in Table 5 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  $\phi_m$  to 100 percent. Keeping everything else from 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. On average, the individual returns slowly 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 5, 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 higher probability of overdose. As a result, life expectancy declines by 10.5 years and lifetime utility declines by 16.8 percent (case 3 in Table 5).

In order to understand why addicted individuals prefer heroin over black market oxycontin although 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.

Table 5: OPR Abuse, Illicit Drug Consumption, and Treatment

case	$\Delta V/V$	$\Delta LE$
1) continued prescription OPR	-12.6	-5.16
2) black market oxycontin	-9.77	-2.80
3) heroin	-16.8	-10.5
4) ... and information shock (TORA behavior)	-8.81	-1.23
5) ideal therapy	-6.82	-1.50
6) as 4) and $\psi = 0.1$	-1.92	-0.16
7) as 5) and $\psi = 0.1$	-1.89	-0.18

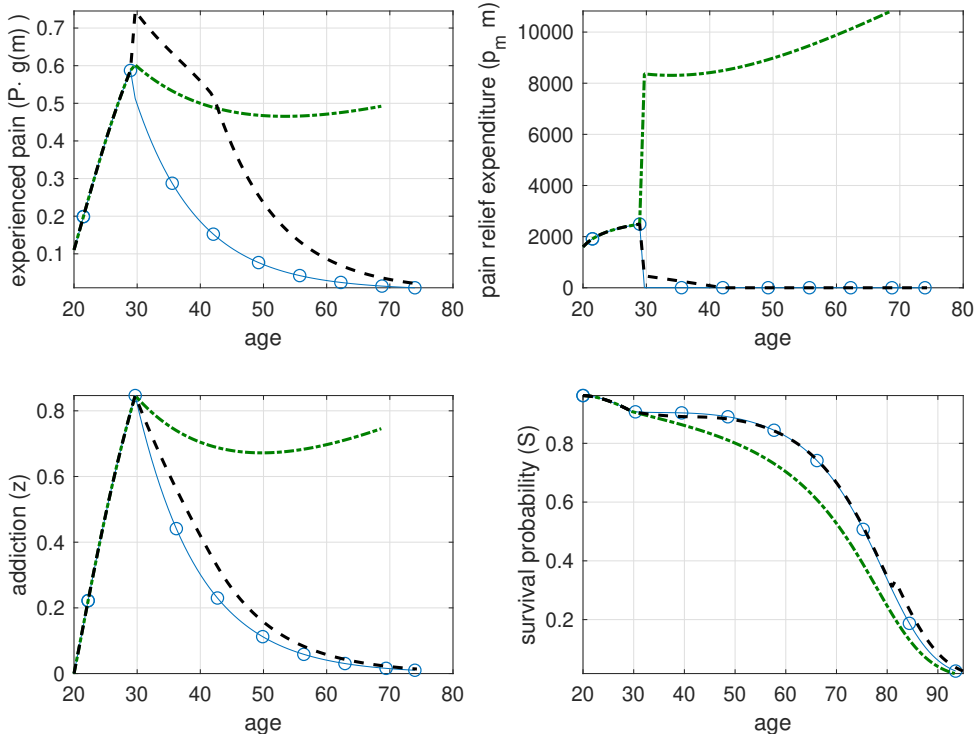
The experiment eliminates chronic pain and (from case 2 on) the prescription of OPRs at age 30. The ideal therapy maintains efficacy of OPRs and eliminates addiction. It reduces the drug price to 1 Dollar (ideal methadon).

These features are next illustrated with a computational experiment that identifies TORA and TICA behavior. As in the experiment of Section 5.2 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 5.2, but now they face others options. They could obtain illicit OPRs from the street at much higher price, or switch to heroin, or quit addictive goods consumption (cold withdrawal). As shown above, TICA individuals prefer to switch to heroin. The implied lifetime trajectories are shown by dashed green lines in Figure 7.

TORA individuals response to the shocks by a small intake of illicit OPRs in order to meliorate the pain from withdrawal and after a short time they completely quit consumption of addictive drugs. The lifetime trajectories are shown by dashed black lines in Figure 7. Notice that quitting occurs much faster than in the scenario of Section 5.2 because now the information about addictive power of OPRs is accompanied by a drastic price increase. The predicted loss of value of life, shown in Table 5, is lower than in case 4) from Table 2 because (by assumption) chronic pain also recedes at age 30. The predicted loss in value of life, however, is of about the same magnitude in both cases. Interestingly, OPR prescription policy has opposing effects on TORA and TICA individuals. A discontinued prescription helps TORA individuals to quit quickly while it induces TICA individuals to take up heroin.

Finally, we 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 ( $\alpha^j = 0$ ) and entails similar health effects as controlled prescription OPR use. It is administered at close to zero costs ( $p^j = 1$  dollar). 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 about 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 5. 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

FIGURE 7: SOPHISTICATED RESPONSE TO INCREASING OPR PRICE AND IDEAL THERAPY



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.

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 withdrawal symptoms recede declines from about 7 years to about 0.7 years and the loss in lifetime utility declines further (in absolute values), see case 5 and 7 of Table 5.

## 6. 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 and 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 suffer 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. The small refinement towards imperfectly controlled addiction allows to model the initiation of prescription OPR use by chronic pain patients as well as the transition to illicit OPR use as a mistake. 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 assumption on the efficacy of painkillers, drug prices, out-of-pocket ratios, and the addictive characteristics of OPRs. As an exception, the model endorses the use of opioid treatment 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 gradually withdraw and eventually quit taking OPRs. TICA Individuals do not reduce their consumption, further develop their addiction, and consume even more.

Another computational experiment considered 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, in contrast, are predicted to quickly 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, who suffering from an addiction control problem, 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 10 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 allows to 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 does, for example, neglect intergenerational welfare effects from more restrictive OPR prescription rules.



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APPENDIX

**Derivation of the Euler Equations.** Obtain  $\dot{\lambda}_k$  from differentiation of (6) with respect to age and substitute  $\lambda_k$  and  $\dot{\lambda}_k$  in (9) to obtain (14). Log-differentiate (8) with respect to age and insert (9) to obtain:

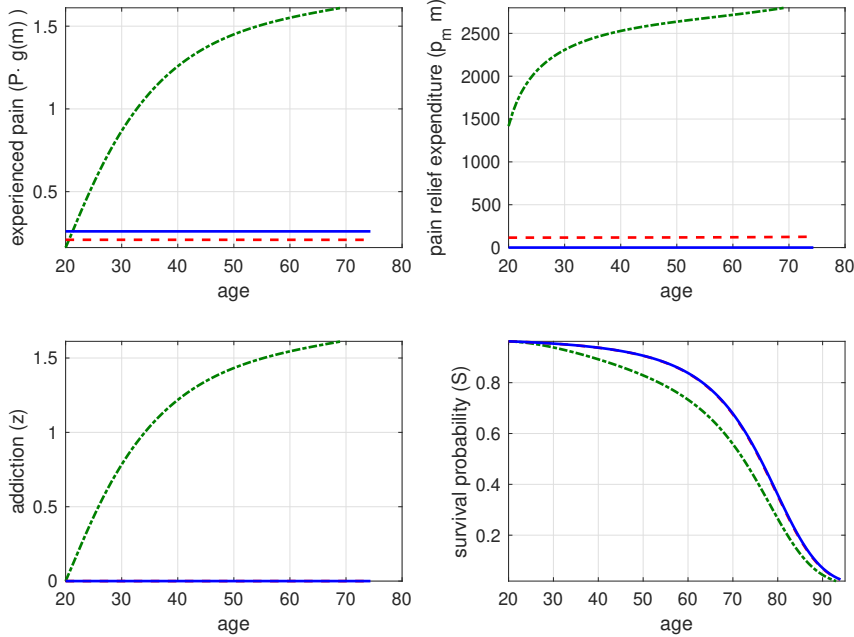
$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma} \left( \frac{\dot{\lambda}_D}{\lambda_D} + r - \rho \right) \quad (\text{A.1})$$

Use (6) and (8) to replace  $\lambda_k$  and  $\lambda_D$  in (10) and obtain

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

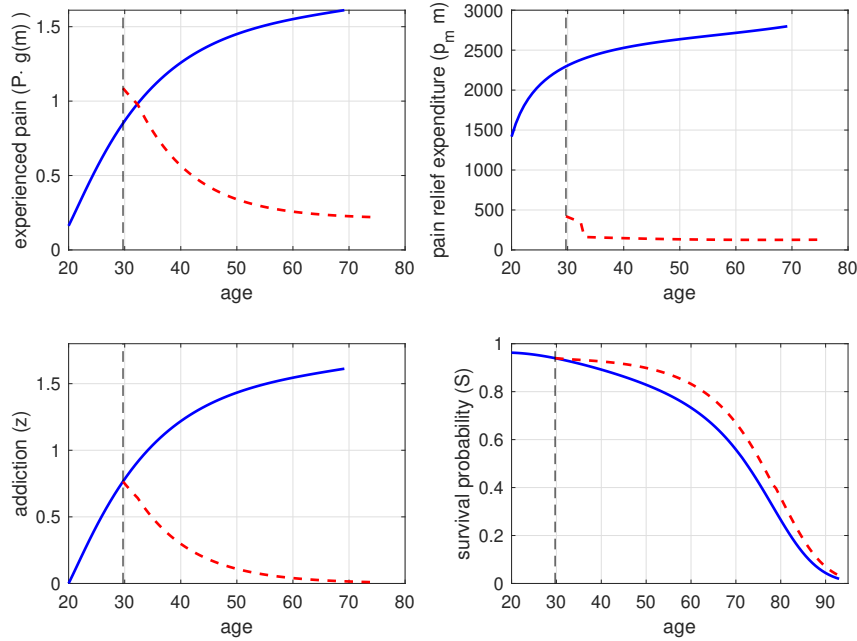
Insert  $\frac{\partial S / \partial D}{S(D, z)}$  obtained from the function parameterized in Section 4.1 and then insert (A.2) in (A.1) to obtain (15). Insert  $\partial g / \partial m^j = -\eta^j \exp(-m^j)$  and the parameterized pain function in (12) and solve  $G(m^j) = 0$  for  $m^j$  to obtain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

FIGURE A.1: PAIN, PAIN TREATMENT, AND HEALTH OUTCOMES



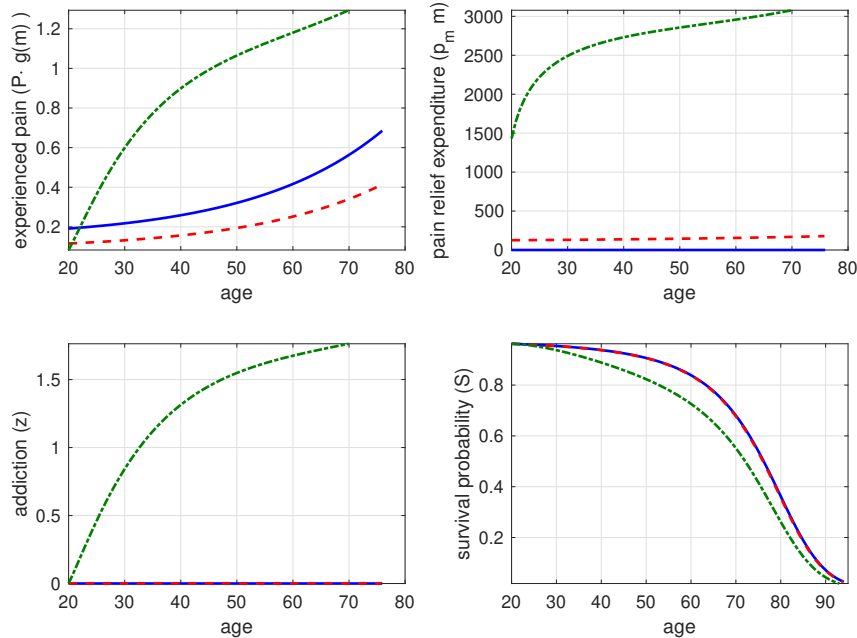
Efficacy of painkillers:  $\eta^j = 0.4$  if  $j$  is oxycontin,  $\eta^j = 0.2$  if  $j$  is ibuprofen. All other parameters as for the benchmark run from Figure 3. Blue (solid) lines: Reference American with untreated moderate pain ( $\delta = 0.26$ ). Red (dashed) lines: common analgesic ( $\eta =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

FIGURE A.2: NEW INFORMATION AT AGE 30: OPRs ARE ADDICTIVE



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 experiment from Figure 4. Blue (solid) lines: TICA addiction; red (dashed) lines: TORA addiction.

FIGURE A.3: 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 ( $\eta = 0.6$ ,  $\alpha = 0.03$ ).