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

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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. Some individuals treated with opioid pain relievers develop addiction, which increases their demand for opioids and reduces their welfare and life expectancy through side effects and potential overdose. Nevertheless, individuals prefer opioid treatment if they fail to understand how it causes addiction. Once individuals are unintentionally addicted and access to prescription opioids is discontinued, consumption shifts to illicit opioids (like heroin). 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. I also discuss treatment of 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 people 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.¹ 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 is recommended to be treated by weak opioids like codeine and strong opioid pain relievers (OPRs) such as morphine or oxycodone. Treatment with opioids is very 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,

¹Since 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, instead of switching to heroin. This form of nonmedical OPR use is taken into

account by the proposed theory, which focuses on unintentional addiction of pain patients. However, the theory does not cover the initiation of OPRs for recreational purposes of individuals who never suffered from chronic pain. This form of non-medical use is likely more prevalent among adolescents and young adults whereas chronic pain treatment is more prevalent among the middle-aged and old. The middle-aged are also the group in which OPR-overdose is more prevalent, indicating an initiation of drug use by 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). Analysis of a prescription monitoring program showed that half of the overdose victims in 2010 had an OPR prescription active on the day of death (Dasgupta et al., 2016).

This paper addresses the unintentional transition from pain patient to opioid addict and its implications for health, longevity, and welfare, by proposing an economic theory of pain and painkiller addiction. The theory 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).²

The proposed economic model of painkiller consumption assumes that pain is caused either spontaneously or gradually by the arrival of health deficits and that the sensation of pain is independent from the level of consumption. If pain interacted multiplicatively with the utility experienced from consumption, rich people would experience greater pain than poor people from the same health shock, which seems not to be supported empirically. The feature of

²The 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).

additive separability 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). Although pain and depression share also some symptoms (like, for example, sleeplessness or poor concentration) they are certainly experienced quite differently at a deeper level. These subtleties, however, cannot be represented by economic utility functions (which also do not distinguish between the sensation experienced from buying an ice cream or a t-shirt).

The main difference between the present study and Strulik (2019) is the modeling and analysis of treatment. While in Strulik (2019) treatment is regarded to be always health- and welfare-improving we here explicitly consider its dark side. In particular, individuals may become addicted to the treatment, 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.

The feature of OPR addiction relates the paper to the economic theory of rational addiction (Becker and Murphy, 1988). The standard theory of addiction has been refined in Strulik (2018) by a model that treats the length of life as finite and endogenous and where individuals take the negative repercussions of addictive consumption on health deficit accumulation into account. It has been shown that, in this context, perfectly rational individuals postpone the consumption of addictive goods until old age and then steeply increase consumption with increasing age. The prediction that addictive goods (like cigarettes) are most intensively consumed by the elderly, however, is at odds with observable behavior. In order to generate actually observable age-patterns of addictive good consumption, Strulik (2018) suggested to refine the theory with an element of bounded rationality, namely that individuals fail to understand how the strength of their addiction depends on addictive goods consumption. The present study uses the same idea,

which seems to be particularly relevant in the case of painkiller addiction where pharmaceutical companies and health care providers propagated wrong beliefs about the addictive power of certain treatments. In contrast to Strulik (2018), however, the consumption of addictive goods is driven by the need for pain relief and not by recreational motives and the consumption of addictive painkillers is only one of several options to treat physical pain. Furthermore, Strulik (2018) considered a deterministic model and focussed on smoking as a comparatively mild form of addictive behavior. Here, death is conceptualized as a stochastic event, which allows to include death from drug overdose in the analysis. A particular focus of the present paper is the impact of prices and efficacy of alternative pain treatments on the formation of addiction and the transformation of individuals from pain patients to heroin junkies.

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. A couple of propositions on painkiller use are derived from the general model without explicit solution of the lifetime trajectories. In Section 4, I parameterize and solve the full model and in Section 5 I calibrate it for a benchmark U.S. American and three stylized painkillers (ibuprofen, oxycontin, heroin). In Section 6, I present the main results on painkiller use and its effects on wellbeing and longevity. A sensitivity analysis includes effects on labor supply, analyses for different pain intensities and onsets as well as the use of opioids in palliative care. I investigate the transition to non-medical OPR-use and heroin consumption when prescriptions are discontinued and briefly discuss stylized treatments of addiction (naltrexone, methadone). Section 7 concludes the paper.

2. THE MODEL

Pain is modeled as a health-related spontaneous or gradual downward shift of the utility function. The intensity of pain P depends positively 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 and of different association with health deficits. In an application of the model, 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. Suppose that there exists a variety of painkiller drugs, indexed by j . Let m^j denote the dose and η^j the efficacy of drug j such that taking drug j reduces pain to $g(m^j, \eta^j)$ percent of its untreated value, $\partial g/\partial m^j < 0$, $\partial g/\partial \eta^j < 0$. We assume declining marginal efficacy $\partial^2 g/\partial m^2 > 0$ and $\partial^2 g/(\partial m^j \partial \eta^j) < 0$, i.e. that increasing the dose reduces pain by more for drugs of higher efficacy. For simplicity, we assume that drugs do not interact such that the whole pain reducing power is given by $\sum_{j=1}^n g(m^j, \eta^j)$.

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

$$U = u(c) - P(D, z) \cdot \sum_{j=1}^n g(m^j, \eta^j). \quad (1)$$

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. While such a motive could exist in reality we focus here on experienced pain and/or addiction as the sole motive of painkiller use. The feature that experienced pain and utility from consumption enter aggregate utility additively captures the assumption that rich people (who consume more) do not experience pain differently (i.e. more severely) than poor people. Nevertheless, rich people are willing to pay more to get rid of pain, as it will be shown later. 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, $\partial U / \partial m > 0$.

The modeling of addiction follows in many aspects the theory of rational addiction (built on Becker and Murphy, 1988). However, there are two substantial differences. First, by design, individuals who do not suffer pain from physical health deficits would not start using painkillers. Second, individuals do not correctly anticipate the addictive potential of painkillers. They are in this sense less than perfectly rational, perhaps because they were not fully informed about the addictive power of specific painkillers or because they simply fail to predict how they will get hooked.³

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

³Strulik (2018) shows that the standard theory of rational addiction predicts unobservable life cycle patterns for the consumption of addictive goods when life is finite and addiction is unhealthy: individuals consume little over most of their life and start consuming increasing amounts in old age. Observable life cycle consumption patterns are predicted when individuals do not fully understand how their consumption affects their addiction.

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. Aside from being drug-specific, α^j is assumed to be individual-specific with $\alpha^j = 0$ for some drugs and some individuals. This captures the feature that not all painkillers are addictive and not all individuals become addicted to a potentially addictive painkiller. The parameter ψ measures the “depreciation rate” of addictive capital, i.e. the rate of disappearance of the physical and mental effects of past consumption of the painkiller.

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 μ is the “natural” force of aging, A and γ reflect the state of medical technology in health maintenance and repair, and a captures environmental effects (as in Dalgaard and Strulik, 2014). Additionally, B^j measures the unhealthiness of painkiller j .

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 ϕ is the out-of-pocket share of health expenditure for maintenance and repair. The price of painkiller drug j is denoted by p_m^j and the associated out-of-pocket share is ϕ_m^j . We allow for the existence of several types of painkillers comprising for example mild analgesics like paracetamol (low efficiency η^j , low harm B^j), more effective and potentially addictive prescription opioids like oxycontin (high η^j , high α^j , low ϕ_m^j), and illicit drugs like heroin (high η^j , high α^j , high ϕ_m^j , and high B^j).

Life satisfaction is conceptualized as individual welfare and defined as discounted utility experienced over the course of life, $\int_0^T S(D, z) U e^{-\rho t} dt$, in which ρ denotes the rate of pure time preference. 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

$$H = S(D, z) \left[u(c) - P(D, z) \cdot \sum_{j=1}^J g(m^j, \eta^j) \right] + \lambda_k \left[w + (r + q)k - c - p\phi h - \sum_{j=1}^J \phi_m^j p_m^j m^j \right] + \lambda_D \mu \left[D - Ah^\gamma + \sum_{j=1}^J B^j m^j - a \right], \quad (5)$$

in which λ_k and λ_D are the costate variables for capital and health deficits. Notice that health deficits reduce expected utility such that $\lambda_D \leq 0$. In contrast to the theory of rational addiction, the evolution of addiction (2) is not taken into account in the Hamiltonian. This feature captures the idea of bounded rationality in addiction, i.e. the phenomenon that individuals do not rationally plan how to become addicted to painkiller consumption.

The first order conditions for a maximum are:

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

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

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

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 λ_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, μB^j , evaluated with the shadow price of health deficits, λ_D . 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

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

$$\frac{\partial S(D, z)}{\partial D} \left[u(c) - P(D, z) \sum_{j=1}^J g(m^j, \eta^j) \right] - S(D, z) \frac{\partial P(D, z)}{\partial D} \sum_{j=1}^J g(m^j, \eta^j) + \lambda_D \mu = \lambda_D \rho - \dot{\lambda}_D. \quad (10)$$

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.

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

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

$$G \equiv -P(D, z) \frac{\partial g(\eta^j, m^j)}{\partial m^j} - \left(p_m^j \phi_m^j + \frac{p \phi B^j}{A \gamma h^{\gamma-1}} \right) \frac{\partial u(c)}{\partial c} \leq 0. \quad (11)$$

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 painkiller use provides $G = 0$. Thus any change of a parameter or variable x that increases G , increases the propensity of painkiller use. Recalling that $\partial^2 g / \partial(m^j)^2 < 0$, we read off (11) that $\partial G / \partial x > 0$ for $x \in \{P, \eta\}$ and for $-x \in \{p_m^j, \phi_m^j, p, A, \phi, B^j\}$. The response to painkiller efficacy follows from $\partial^2 g / (\partial m^j \partial \eta^j) < 0$. 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 in the efficacy of the painkiller (η^j) and declining in the price p_m^j , the out-of-pocket ratio ϕ_m^j , and the severity of side-effects on health (B^j).*

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

Next, we investigate conditions that are conducive to the intake of a painkiller with more severe side-effects.

PROPOSITION 3. *The propensity to substitute a drug i with little side-effects on health for drug j with severe side-effects on health increases with increasing pain if drug j is more effective. It increases if the out-pocket share declines for drug j or increases for drug i , if the price declines for drug j or increases for drug i , if the efficacy increases for drug j or declines for drug i .*

For the proof, we note that $B^j > B^i$ since j is by assumption the less healthy drug. We then see from (11) that drug j is preferred over drug i if

$$F \equiv \left(\phi_m^j p_m^j + \frac{p \phi B^j}{A \gamma h^{\gamma-1}} \right) \frac{\partial u}{\partial c} + P \frac{\partial g}{\partial m^j} - \left(\phi_m^i p_m^i + \frac{p \phi B^i}{A \gamma h^{\gamma-1}} \right) \frac{\partial u}{\partial c} - P \frac{\partial g}{\partial m^i} < 0. \quad (12)$$

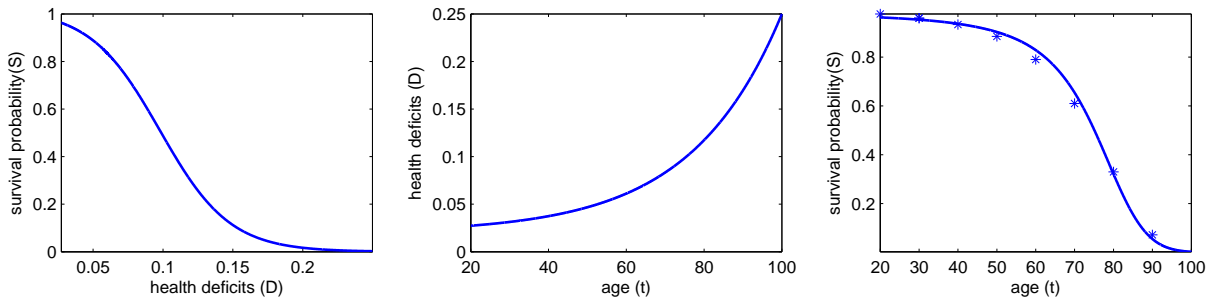
The propensity to take the less healthy drug thus increases with declining F . We have $\partial F / \partial P = \partial g / \partial m^j - \partial g / \partial m^i$, which is negative if drug j is more effective. We obtain $\partial F / \partial \phi_m^j > 0$, $\partial F / \partial p_m^j > 0$, and vice versa for ϕ_m^i and p_m^i . The last part of the proof follows from $\partial^2 g / (\partial m^j \partial \eta^j) < 0$.

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 a 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 is one for non-addicted individuals and declines exponentially in the degree of addiction z . The impact of addiction on overdose death is measured by the drug-specific parameter χ^j .

FIGURE 1: HEALTH-DEPENDENT SURVIVAL AND SURVIVAL BY AGE



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

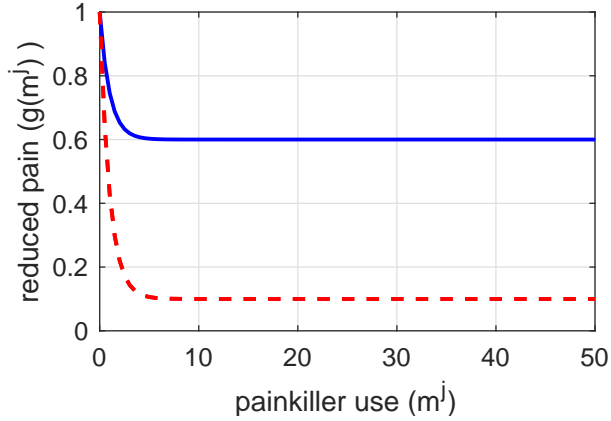
For simplicity we assume that pain intensity is additively separable in health deficits and addictive capital as well as linear in z , $P = \delta D^\omega + \zeta z$, in which δ and ω reflect the influence

of health deficits on pain intensity and ζ reflects the influence of addiction; δ will be the key parameter to evaluate the intensity of pain. For painkiller efficacy we assume a negative exponential function as a continuous approximation of a step function, which would indicate that only a certain (prescribed) dose of m is effective:

$$g(m^j, \eta^j) = 1 - \eta^j + \eta^j e^{-m^j}. \quad (13)$$

The efficacy parameter $\eta^j \in \{0, 1\}$ measures the maximum degree of pain that can be reduced by taking painkiller j . It also provides the marginal efficacy of the first unit of painkiller use, $\partial g(0, \eta^j)/\partial m^j = -\eta^j$. Figure 2 shows the painkiller function for $\eta^j = 0.6$ (solid lines) and $\eta^j = 0.9$ (dashed lines). The exponential function (13) captures well the ceiling effect, i.e. that there exists a maximum level of pain reduction (η^j) beyond which increasing the dosage does not provide additional benefits.

FIGURE 2: PAINKILLER USE AND EFFICACY



Blue (solid)line: $\eta^j = 0.4$; red (dashed) line: $\eta^j = 0.9$.

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

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

$$\frac{\dot{h}}{h} = \frac{r + q - \mu}{1 - \gamma}$$

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

with $g(m^j, \eta^j) = 1 - \eta^j + \eta^j e^{-m^j}$ and $P = \delta D^\omega + \zeta z$. Painkiller demand is obtained as

$$m^j = \max \left\{ 0, \log [(\delta D^\omega + \zeta z) \eta^j] - \log \left[\left(\phi_m^j p_m^j - \frac{p\phi B^j}{A\gamma h^{\gamma-1}} \right) c^{-\sigma} \right] \right\}. \quad (16)$$

5. 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 γ 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, $VOL = \int_0^{\tilde{T}} S(D) e^{-\rho\tau} u(c(\tau)) / u'(c(t))$, in which \tilde{T} fulfils $D(\tilde{T}) = \bar{D}$.

We calibrate the remaining parameters a , A , ρ , and σ such that (i) the reference American expects to die at age 75.5 (male life expectancy at 20 in the year 2000; NVSS, 2012), (ii) the age-weighted health expenditure is 13.3 percent of GDP as estimated for the US in the year 2000 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 σ is in line with recent studies suggesting that the “true” value is probably close to one (Chetty, 2006), or slightly above one (Layard et al., 2008).

The impact of pain on utility is calibrated using a study by Olafsdottir et al. (2017) which estimates the compensation variation for pain, i.e. the additional equivalized household 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 Δw between \$ 20,440 and \$ 52,925. Based on these findings, we estimate

the value of $\delta > 0$ that provides with compensation Δw 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 and the estimate $\delta = 0.51$ for $\Delta w = \$ 52,925$, which we define as the benchmark case of severe pain.

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 we set $\phi_m^j = 0.19$ 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. 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. 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.

Alternatively, pain may be treated by prescription opioid pain relievers (OPRs) and we calibrate a stylized treatment with oxycontin. The main reason to demand opioids is their high efficacy in pain relief. We set $\eta^j = 0.9$ such that the opioid reduces 90 percent of untreated pain. 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 $pm^j = 4,000$.

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 α and ψ are unobserved and potentially individual-specific, we meet the involved parameter uncertainty with a sensitivity analysis. The greatest health risk for the addicted individual 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).

Table 1: Three Types of Painkillers

| | ibuprofen | oxycontin | heroin |
|---------------|-------------------------|--|------------------------------|
| efficacy | low ($\eta = 0.4$) | high ($\eta = 0.9$) | high ($\eta = 0.9$) |
| price | low ($p_m = 30$) | high ($p_m = 500$ or 4000) | medium ($p_m = 400$) |
| out-of-pocket | low ($\phi_m = 0.19$) | low ($\phi_m = 0.19$) or high ($\phi_m = 1$) | high ($\phi_m = 1$) |
| side effects | low ($B = 10^{-5}$) | moderate ($B = 5 \cdot 10^{-5}$) | high ($B = 10^{-4}$) |
| addictive | low ($\alpha = 0$) | high ($\alpha = 0.03$) | high ($\alpha = 0.03$) |
| overdose | low ($\chi = 0$) | high ($\chi = 0.002$) | very high ($\chi = 0.004$) |

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

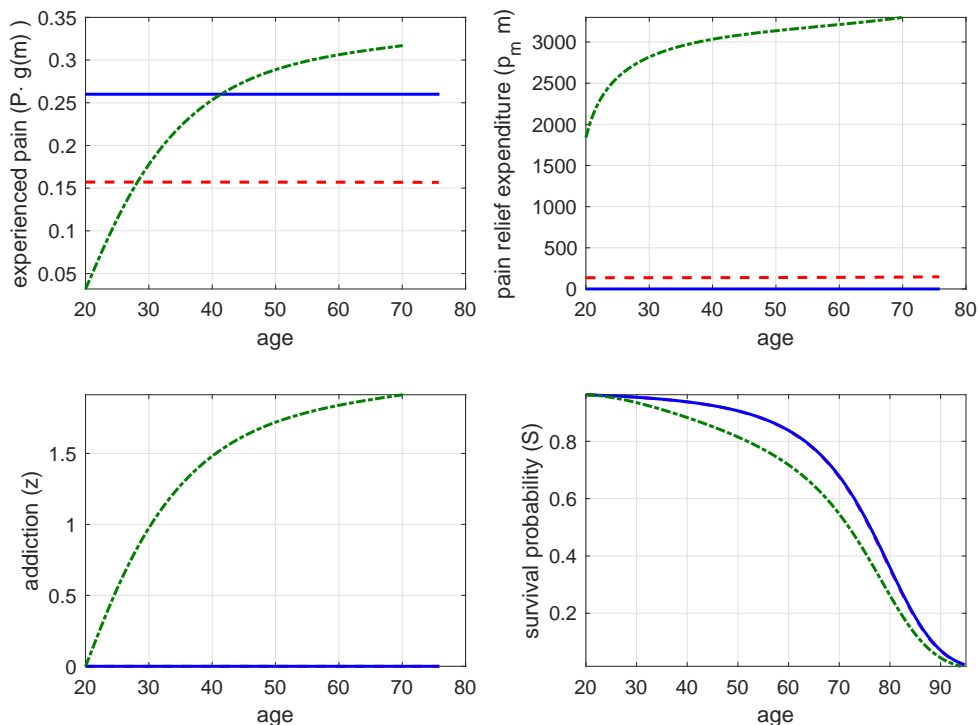
As a form of non-prescribed and illicit pain treatment we consider heroin consumption. We assume that heroin has the same efficacy and the same addictive potential as prescription opioids but a lower market price, a 100% out-of-pocket share, more side-effects, and a higher overdose probability. In terms of morphine equivalents, heroin is available at about one-tenth of the market price of opioid pain medication, 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.

6. PAIN, PAINKILLER USE, WELLBEING AND LONGEVITY: RESULTS

6.1. Benchmark Results. We solve the life cycle decision problem with the relaxation algorithm of Trimborn et al. (2008). The method provides the exact constrained-optimal life cycle trajectories, up to a user-specified approximation error (which is set to 10^{-5}). Figure 3 shows the life cycle trajectories for pain, pain relief expenditure, addiction, and survival probability at any age. Blue (solid) lines show the trajectories for untreated chronic pain at benchmark level of $\delta = 0.26$. For better comparisons, the pain and addiction trajectories end at the age of predicted death, which is at age 75.5 years. The trajectory for survival probability, however, shows the predicted survival 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 ($\eta = 0.9$, $\alpha = 0.03$).

Life cycle dynamics are more interesting in the case of opioid treatment, shown by green (dash-dotted) lines. We see that treatment is initially very effective in removing pain (upper left panel). However, as the individual becomes increasingly addicted (lower left panel), additional pain is created from increasing tolerance. According to the calibration, the pain from addiction surpasses that of the original untreated pain after 20 years of treatment. The individual responds to increasing pain by increasing demand for pain treatment and pain relief expenditure rises. Over the course of life, pain relief expenditure more than 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 5.6 years.

These results are summarized in the first rows of Table 2 using two aggregate indicators, the loss of lifetime utility ΔV and the loss of life expectancy ΔLE , compared to the calibrated pain-free American. The calibrated loss of life satisfaction through untreated pain is $\Delta V = -4.5$ in the benchmark case (compensation equivalent of \$ 56 per day). In relative terms, benchmark pain leads to a reduction of life satisfaction $\Delta V/V = 3\%$. This can be considered a relatively small reduction of life satisfaction when compared to the loss resulting from major depressive disorder (which was estimated as 20% in Strulik, 2019). 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 life satisfaction, which falls short of that of a pain-free individual by only $\Delta V = -2.8$ units. The use of prescription opioids (case 3) reduces the value of life by $\Delta V = 10.8$, i.e. by 8% although it initially improves instantaneous utility due to drastic pain reduction. The loss of life satisfaction comes through three channels: (i) through increasing tolerance and increasing cravings for the opioid such that experienced pain actually increases in the long run; (ii) through shorter life expectancy due to faster deteriorating health and the probability of overdose; (iii) because the expenditure for opioids reduces the funds available for consumptions, savings, and health investments.

6.2. Comparative Dynamics. We next consider the comparative dynamics of OPR treatment using the two aggregate measures of the quality and quantity of life, ΔV and ΔLE . First, we acknowledge that not everybody gets addicted from opioid use. By setting $\alpha^j = 0$, we consider with case 4 in Table 2 an addiction-free individual. Alternatively, this case can be interpreted as the hypothetical scenario that OPR-use bears no risk of addiction. In this case, opioid

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

| case | ΔV | ΔLE |
|---|------------|-------------|
| 1) moderate pain, untreated ($\delta = 0.26$) | -4.5 | -0.03 |
| 2) light painkiller ($\eta = 0.4$) | -2.8 | -0.02 |
| 3) prescription opioid ($\eta = 0.9$) | -10.8 | -5.63 |
| prescription opioid use and... | | |
| 4) no addiction ($\alpha = 0$) | -1.1 | -0.5 |
| 5) strong addiction ($\alpha = 0.06$) | -21.5 | -10.8 |
| 6) higher tolerance ($\zeta = 3$) | -14.8 | -6.3 |
| 7) lower depreciation ($\psi = 0.05$) | -15.8 | -4.9 |
| 8) high out-of-pocket ($\phi = 1$) | -9.8 | -4.9 |
| 9) black market ($p_m = 5000, \phi_m = 1$) | -8.6 | -3.0 |
| aging-related pain ($\omega = 1$) and... | | |
| 10) moderate pain, untreated ($\delta = 0.7$) | -4.5 | -0.08 |
| 11) light painkiller ($\eta = 0.4$) | -2.8 | -0.18 |
| 12) prescription opioid ($\eta = 0.9$) | -10.7 | -5.63 |
| labor market effects for moderate pain ($\delta = 0.26$), $\beta = 20$, and... | | |
| 13) no treatment | -4.6 | -0.03 |
| 14) OPR treatment | -10.9 | -5.63 |
| 12) OPR treatment and $\alpha = 0$ | -1.2 | -0.53 |

pain treatment is clearly welfare enhancing compared to no treatment as well as compared to treatment with light painkillers. Life expectancy declines somewhat due to detrimental side effects on health from perpetual opioid use but this does not fully equalize the large positive effect on wellbeing through drastic pain reduction. If addiction is stronger than in the benchmark case, the negative effects of OPR use increase. In case 5 we see that doubling the strength of addiction (to $\alpha = 0.06$) entails an almost proportional reduction of wellbeing (to $\Delta V = -21.5$) and life expectancy ($\Delta LE = -10.8$ years). Case 6 shows similar results for a doubling of pain from addiction (ζ increases from 1.5 to 3.0), capturing an individual with higher negative tolerance and thus faster adaptation to opioid use. Case 7 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 8 we consider an individual who finances OPRs completely out of pocket. Compared to subsidizes 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 9 that abandons access to prescription opioids, which are now bought on the black market at an eightfold higher price. As a result of the drastic increase, individuals reduce demand (average lifetime demand declines by 210 percent compared to case 3) and overdose probability and health repercussions decline such that the loss of life expectancy is “only” 3.0 years compared to 5.6 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 may thus prefer to switch to a less expensive opioid like heroin, a scenario that we discuss in detail below.

We next consider aging-related pain, i.e. pain that increases in conjunction with the development of health deficits. For that purpose, we set $\omega = 1$ and re-calibrate δ such that pain requires the same compensation (of \$ 56 per day) as in the benchmark case. This leads to the estimate $\delta = 7$. Despite the different evolution of pain (life cycle trajectories are shown in Figure A.1 in the Appendix), the implications for wellbeing and life expectancy are very similar to the benchmark run, as shown in case 10-12 in Table 2 (compared to case 1-3). If pain is treated not at all or with light painkillers, its effect on life expectancy is somewhat larger as in the benchmark case. The reason is that individuals experience relatively more pain in old age, which makes health investments for life extension less desirable. In case of OPR treatment, however, these differences are smoothed out by addiction, which develops similarly over the life course as in the benchmark run.

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

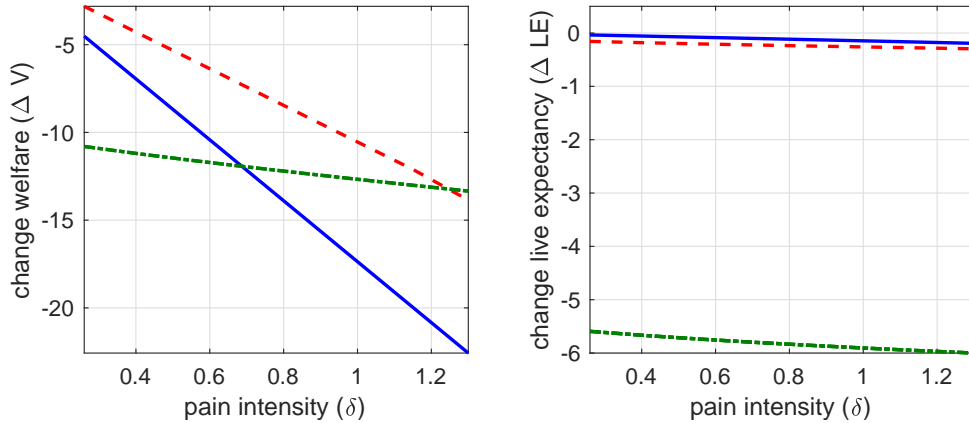
pain leads to 5.2 years earlier retirement and a loss of lifetime income of more than \$ 180,000.⁴ As shown in case 13-15 of Table 3 (when compared to case 1-3) this leads to only marginal changes in the impact of pain and pain treatment on wellbeing and life expectancy. The reason is that the income 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.

6.4. Pain Intensity. We next return to the basic model and discuss in more detail the comparative dynamics with respect to pain intensity δ . Results are shown in Figure 3. Solid (blue) lines show the change in wellbeing and life expectancy (compared to the pain-free benchmark) for alternative untreated pain intensities. Dashed (red) lines show the same outcomes when pain is treated with a light painkiller (ibuprofen) and dash-dotted (green) lines show the outcomes for OPR treatment. All lines originate at benchmark pain ($\delta = 0.26$) and end at fivefold benchmark pain ($5 \cdot 0.26 = 1.3$). At $\delta = 0.51$, pain intensity is at the upper bound of the estimates based on Olafsdottir et al. (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 more than ten times the annual income of the reference American.

Figure 3 shows that lifetime utility declines steeply if pain remains untreated. The loss in wellbeing declines almost linearly from -4.5 to -22.5 , which amounts to 17 percent of lifetime utility. When pain is treated, lifetime utility declines less steeply in pain intensity. The flattest slope is obtained for OPR treatment. An interesting implication of the fact that the slope is flatter for higher painkiller efficacy is that the lines intersect, indicating reversals in wellbeing. For pain intensities greater than about 0.7 (corresponding to a compensation value of \$ 245 per day or \$ 90,000 per year) the green line lies above the blue line and lifetime utility is larger with opioid treatment than without treatment. For pain intensities greater than about

⁴It 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.

FIGURE 3: 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 ($\eta = 0.9$, $\alpha = 0.03$).

1.25 (corresponding to a compensation value of \$ 822 per day or about 300,000 per year), lifetime utility with opioid treatment exceeds that under treatment with a light painkiller. These reversals are obtained although opioid use reduces life expectancy by about 5.5 years, indicating that individuals suffering from great pain benefit from OPR treatment even when the negative effects on increasing tolerance, addiction, and overdose possibility are taken into account. OPR use for individuals with severe pain is thus supported by the model as a rational outcome. Individuals with mild to moderate pain, however, would be better off without OPR use.

6.5. 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. We begin with a case of moderate pain ($\delta = 0.26$) occurring at age 60. At this age, the value of remaining lifetime utility without pain is 70.2 and the remaining life expectancy without pain is 19.9 years. Results are summarized in case 1-4 of Table 3. If pain remains untreated, the individual experiences a loss in lifetime utility of 2.2 years, which is in relative terms about the same loss as it was obtained for chronic pain from age 20 onwards (3.1 percent). The impact on lifetime utility of light painkiller use (-1.4) and OPR treatment (-6.7) is also scaled in a similar way to remaining lifetime utility. An interesting observation is that the effect of OPR treatment on life expectancy is much smaller than in the benchmark case. Life expectancy declines by “only” 0.55 years, i.e. by 2.7% (compared to 7.4% in the benchmark

case with chronic pain from age 20). This indicates that for the onset of pain 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 lifetime utility with OPR use than without pain treatment.

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

| case | ΔV | ΔLE |
|--|------------|-------------|
| Pain onset at age 60, moderate pain ($\delta = 0.26$). | | |
| 1) no treatment | -2.2 | -0.00 |
| 2) light painkiller | -1.4 | -0.01 |
| 3) OPR treatment | -6.7 | -0.55 |
| 4) OPR, slow addiction ($\alpha = 0.015$) | -3.4 | -0.30 |
| Pain onset at age 60, severe pain ($\delta = 1.0$). | | |
| 5) no treatment | -8.6 | -0.01 |
| 6) light painkiller | -5.2 | -0.03 |
| 7) OPR treatment | -7.9 | -0.63 |
| 8) OPR, slow addiction ($\alpha = 0.015$) | -4.5 | -0.35 |
| Pain onset at age 70, severe pain ($\delta = 1.0$). | | |
| 9) no treatment | -4.9 | -0.00 |
| 10) light painkiller | -2.9 | -0.01 |
| 11) OPR treatment | -3.9 | -0.25 |
| 12) OPR, slow addiction ($\alpha = 0.015$) | -2.1 | -0.13 |

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, OPR treatment improves lifetime utility compared to no treatment although treatment with light painkillers still outperforms OPR treatment. When addiction proceeds slower as in the benchmark case, OPR treatment improves lifetime utility even compared to treatment with light painkillers, as shown in case 8 (compared to case 6). Qualitatively, these results are preserved when the severe pain shock hits at age 70 (as shown in case 9 to 12). These results rationalize the use of OPR in old age when pain is intensive and addiction develops slowly. Otherwise even in old age, opioid treatment reduces wellbeing compared to the use of light painkillers.

6.6. Palliative Care. The above conclusions change when pain is experienced in conjunction with a drastic deterioration of physical health that substantially reduces life expectancy. Such a scenario is more appropriate to describe chronic malignant pain at the end of life rather than,

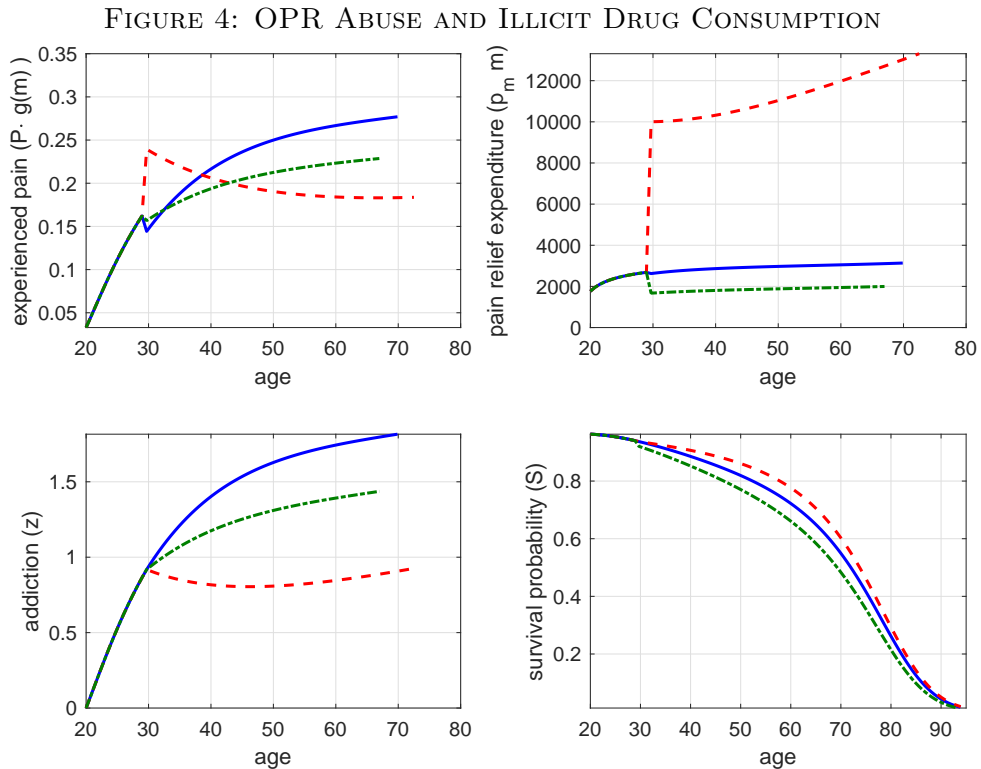
for example, chronic backpain. In order to demonstrate that the model supports opioid pain treatment in these end-of-life situations we first consider again the severe pain shock ($\delta = 1$) at age 70, which, however, is now accompanied with a spontaneous increase in the health deficits index D from 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 lifetime utility would decline to 5.4 if the last years of life were pain-free. As shown in case 1 of Table 1, the presence of severe pain adds another loss in wellbeing of -0.64, i.e. wellbeing declines by 11.8 percent, which is relatively speaking much greater than the loss through benchmark chronic pain (which was 3 percent). In this case, OPR treatment dominates no pain treatment and treatment with light painkillers because the expected remaining lifetime is simply too short for a fully developed opioid addiction.

Table 4: Pain and Severely Life-Shortening Health Shocks

| case | ΔV | ΔLE |
|---|------------|-------------|
| Pain onset at age 70, severe pain ($\delta = 1.0$) and $\Delta D = 0.05$. | | |
| 1) no treatment | -0.64 | -0.00 |
| 2) light painkiller | -0.39 | -0.00 |
| 3) OPR treatment | -0.27 | -0.04 |
| Pain onset at age 40, severe pain ($\delta = 1.0$) and $\Delta D = 0.1$. | | |
| 4) no treatment | -0.48 | -0.00 |
| 5) light painkiller | -0.29 | -0.00 |
| 6) OPR treatment | -0.17 | -0.03 |
| Pain onset at age 40, less severe pain ($\delta = 0.51$) and $\Delta D = 0.1$. | | |
| 7) no treatment | -0.24 | -0.00 |
| 8) light painkiller | -0.15 | -0.00 |
| 9) OPR treatment | -0.14 | -0.03 |

These conclusions do not rest on the onset of severe pain in old age but rather on their appearance in conjunction with drastic 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. Also in this case, the effect of severe pain on wellbeing (-0.48) is large in a relative sense (12.3 percent) and, again, OPR treatment dominates no treatment and light painkiller use (case 4-6 in Table 4). In case of less severe pain, however, OPR treatment is again dominated by light painkiller use, as shown in cases 7-9 of Table 4.

6.7. 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 4, we assume that the individual manages to receive prescription opioids and support from health insurance such that p_m and ϕ_m stay at their original values. As shown in the upper left panel of Figure 4, the loss in pain due to absent backpain causes a mere wrinkle in the trajectory of life-cycle pain of the addicted. Soon, additional cravings from addiction compensate for the temporary liberation from 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 backpain (case 3 in Table 2).



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.

A more likely scenario, however, is that prescription OPR treatment is terminated and opioid abuse is satisfied by demand from the black market. Referring to the calibration from Section 5 such a change is captured by an eightfold price increase as well as by an increase of the out-of-pocket share ϕ_m to 100 percent. Keeping everything else from the benchmark calibration, red lines in Figure 4 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 4) and a substantial increase in pain, in particular initially when the individual fails to maintain the level of prescription OPR use (upper left panel).

Alternatively, the individual 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 3 to the calibrated values, we find that indeed heroin consumption is preferred over black market OPR use. The implied life cycle trajectories are shown by dash-dotted (green) lines in Figure 4. 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 8.3 years and wellbeing declines by 14.6 (case 3 in Table 5).

In order to understand why addicted individuals prefer heroin over black market oxycotin although the implied lifetime utility is lower, recall that 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 fully rational addict in the Becker-Murphy (1988) sense 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. Notice that the form of bounded rationally assumed

in order to explain OPR addiction and unintended heroin use does not require that individuals ignore their addiction or are in any way myopic. Individuals are modeled as forward looking maximizers of lifetime utility with otherwise rational expectations. In their optimization calculus they take their addiction level z into account. They fail, however, to predict how their addiction will develop. Formally, they do not take into account the equation of motion (2) but consider z as a given state variable. This can be regarded as a relatively mild assumption of bounded rationality since even doctors, psychologists, and neuroscientist do not fully understand the individual-specific determinants of addiction.

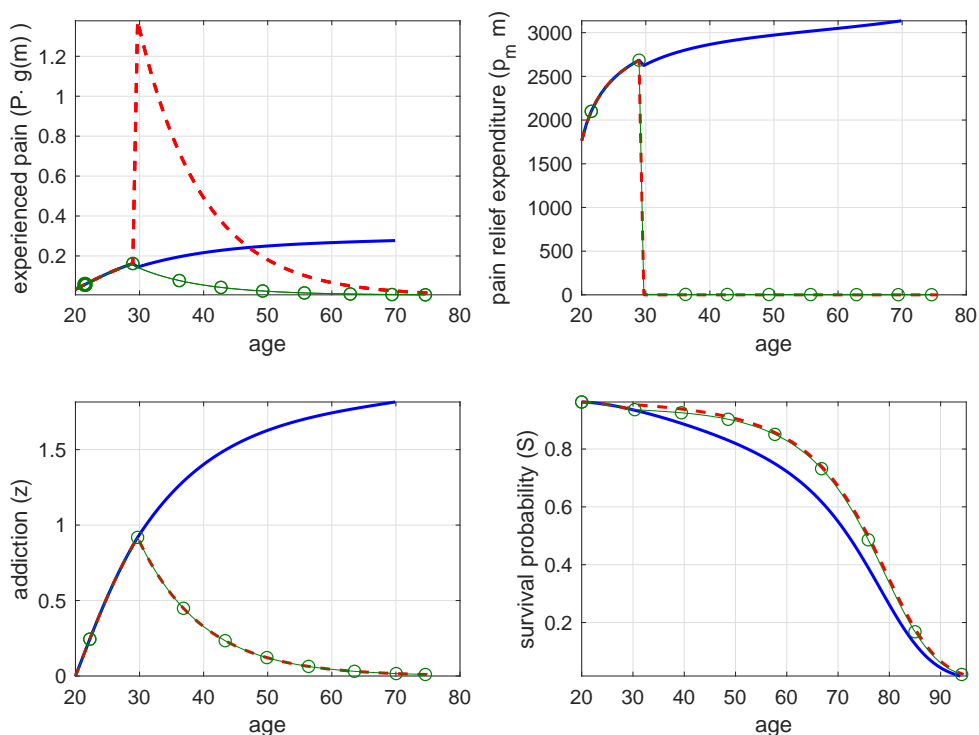
Table 5: OPR Abuse, Illicit Drug Consumption, and Treatment

| case | ΔV | ΔLE |
|-----------------------------------|------------|-------------|
| 1) continued prescription OPR | -10.6 | -5.48 |
| 2) black market oxycontin | -8.9 | -3.31 |
| 3) heroin | -10.8 | -8.41 |
| 4) withdrawal (ideal naltrexone) | -6.8 | -0.33 |
| 5) ... and $\psi = 1$ | -2.3 | -0.33 |
| 6) no addiction (ideal methadone) | -3.4 | -1.01 |
| 7) ... and $\psi = 1$ | -1.7 | -0.39 |

6.8. Addiction Treatment. Finally, we consider alternative forms of addiction treatment. For better comparison, solid (blue) lines in Figure 5, reiterate the lifetime trajectories for continued prescription OPR use from Figure 4. Dashed (red) lines show the trajectories when access to addictive drugs is shut off. As shown in the upper left panel, this leads to initially very high pain from withdrawal as long as addiction recedes. It also leads to a substantial gain in health and survival probability (lower right panel) and an almost complete recovery of life expectancy. As shown in case 4 of Table 5, life expectancy declines by only 0.3 years. The still substantial loss of wellbeing of 6.8 and is thus almost exclusively due to pain from withdrawal. The same trajectories and conclusions are obtained when efficacy η drops to zero for heroin and other opioids. Such an outcome would be achieved by an ideal opioid antagonist. An opioid antagonist, like naltrexone, disables the effects of opioids. A great practical problem is of course the maintenance of the treatment by suffering addicts. An ideal opioid antagonist would solve this problem (perhaps by the operative implementation of a permanent slow-release reservoir). We could re-use the method of Olafsdottir et al. (2017) and compute the compensation value of such an ideal antagonist. This would be the payment that lifelong heroin users would need to

receive in order to achieve the same lifetime utility as under withdrawal at age 30. It provides the willingness to pay for an ideal naltrexone and the cure from heroin addiction. This leads to the estimate of \$ 205,000 per year or \$ 560 per day, a compensation value much higher than the estimates for mild to moderate pain.

FIGURE 5: ADDICTION TREATMENTS: WITHDRAWAL AND METHADONE



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

As an alternative treatment we consider opioid replacement therapy with a drug that relieves the narcotic cravings without contributing to addiction, like methadone. An ideal methadone would provide the same efficacy as heroin ($\eta = 0.9$) with zero impact on addiction $\alpha = 0$ and it would provide the same side effects and risk of overdose as controlled prescription opioid use. It would also be administered at zero cost. The implied life cycle trajectories under these assumptions are shown by green (dash-dotted) lines in Figure 5. In contrast to naltrexone, the ideal methadone treatment eliminates the utility cost from craving and pain recedes right from the start of the treatment. As a result, the loss of lifetime utility declines to 3.4, half of that from withdrawal (case 4 in Table 5). The gain in life expectancy, however, is smaller due to the negative effects from continued drug use. Again, we can compute the (additional) compensation

value for methadone, i.e. from transiting from case 4 to case 6. This provides an amount of \$ 36,000 per year, or \$ 98 per day, a value in the ballpark of compensation value of moderate pain.

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 6.9 years to 0.69 years and the loss in lifetime utility declines further (in absolute values), see case 5 and 7 of Table 5.

7. 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 who, however, do not fully understand how the consumption of opioid-based painkillers contributes to the development of an opioid addiction. This small refinement of rational addiction towards bounded rationality 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 are susceptible to addiction experience drastic reductions in wellbeing and life expectancy as unintended consequences of their addiction. In most cases, individuals would be better off in the long-run if they would go for a less effective non-addictive pain treatment. 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. Remaining lifetime is simply too short in this case for addiction to unfold.

With computational experiments the model highlighted the policy dilemma of implementing more restrictive OPR prescription rules. Individuals whose OPR prescription is discontinued and who developed an opioid addiction have a strong incentive to switch to illicit opioid consumption and to prefer cheaper but more dangerous drugs like heroin. Computational experiments with respect to addiction treatment suggest that individuals display a high willingness to pay for treatments that alleviate the termination of drug use. Alternative, non-pharmacologic treatments of pain and addiction such as yoga and cognitive behavioral therapy (CBT) are perhaps more difficult to capture by the current framework. Other forms of opioid use, however, could be easily integrated in the model framework. For example, misuse of prescription opioids (e.g. crushing tablets and injecting the substance) could be introduced as an intermediate step as well

as the transition to an even more powerful and deadly opioid such as fentanyl. Mild extensions of the model are conceivable, which capture the joint use of several prescription painkillers or the supplement of prescription OPRs with street purchases. A mild reformulation of the utility function could capture also recreational OPR use. These features fell prey to Occam's razor in order to constrain the length of the paper.

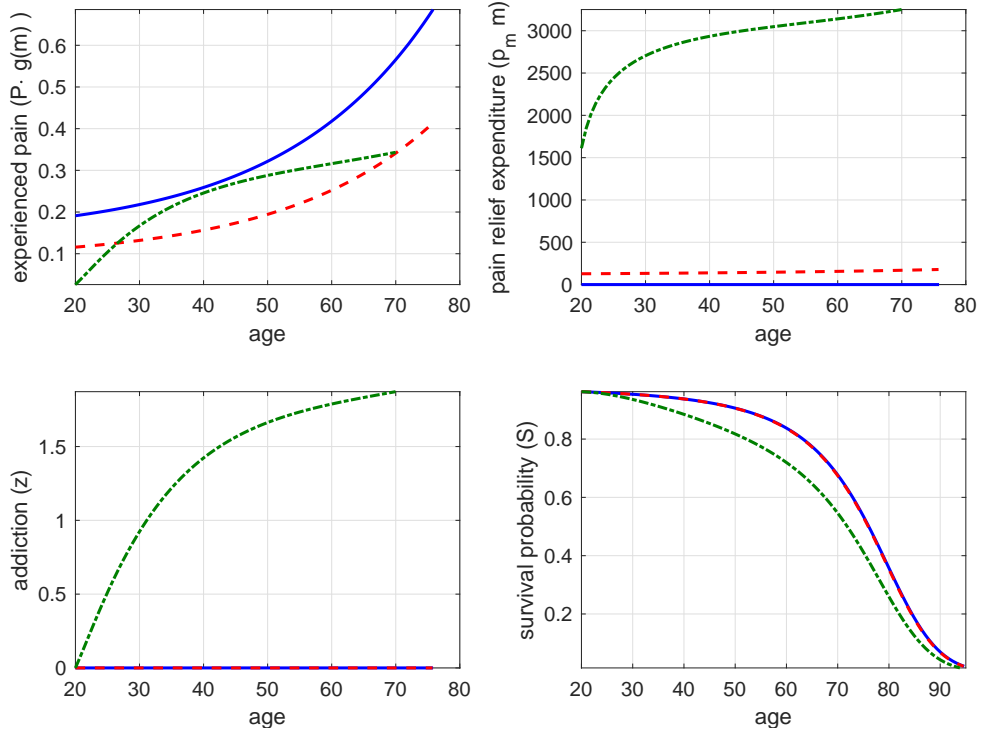
A limitation of the model is its focus on the pain patient. By highlighting individual decision processes and their consequences on wellbeing and life expectancy the analysis neglects to consider the behavior of health providers and the pharmaceutical industry as well as macroeconomic context and social 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.

APPENDIX

Derivation of the Euler Equations.

Aging-Related Pain.

FIGURE A.1: 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 ($\eta = 0.9$, $\alpha = 0.03$).

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