Economic Theories of Health Inequality Across the Life Course

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Economic theories of health inequality across the life course

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Abstract

This chapter will explore what economic theory can offer in terms of improving our understanding of the potential causes of health inequalities over the life course. We use the model by Galama and Van Kippersluis (2019), which is based on the seminal health-capital theory of Grossman (1972), to discuss important insights relevant for health inequality research. We additionally discuss some of the most important sources of critique of the model and sketch directions of how the theory could be extended to do justice to the recent empirical literature refuting some of the theoretical assumptions.

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Introduction

Socially and economically disadvantaged individuals generally experience poorer health outcomes (e.g., Glymour et al. 2014), and the Covid-19 pandemic risks aggravating these inequalities (e.g., Bambra et al. 2020). Health inequality is not only an infringement of equity (Woodward and Kawachi, 2000; Anand, 2002), but the higher prevalence of mortality and morbidity among lower socioeconomic groups also impedes productivity and threatens to undermine economic growth and prosperity (WHO, 2001; Bloom et al. 2019). Reducing health inequality is therefore high on the policy agenda in many countries. For example, the US Centers for Disease Control and Prevention (CDC) launches a Healthy People initiative every decade with the goal of improving the health of all Americans.

However, despite numerous policy efforts, health inequalities – like income inequalities – have not decreased over time, and if anything, are increasing (Meara et al. 2008; Cantu et al. 2021; Case and Deaton, 2021). Apparently, we still lack a full understanding of how health inequalities arise, and the tools policymakers have to reducing disadvantage in socioeconomic and health outcomes appear to be insufficiently effective. Anne Case and Nobel-laureate Angus Deaton famously wrote: “it is extremely difficult to untangle the links between work, earnings, health, and education, without some sort of guiding framework” (Case and Deaton, 2005). In this spirit, this chapter will explore what economic theory can offer in terms of improving our understanding of the potential causes of health inequalities over the life course. We will focus on health-capital theory based on the seminal work of Michael Grossman (1972a;b) and later extensions by Galama (2015), Galama and Van Kippersluis (2015; 2019) and Galama, Lleras-Muney and Van Kippersluis (2018).

The late Adam Wagstaff sketched the basic intuition of the economic approach to understanding health inequalities (Wagstaff, 1986). The basic pillars of the economic approach are: (i) people care about utility or ‘well-being’, which depends on both consumption as well as health, resulting in trade-offs that can help explain why people sometimes engage in unhealthy types of consumption even if this is detrimental to their health; (ii) health is produced on the basis of a production function where medical
care is just one of its inputs; and (iii) there are time and budget constraints such that choices are restricted by the time and money available.

Whereas the basic intuition of the economic approach is still highly relevant, empirical evidence over the last decades has challenged many key assumptions underlying the conventional economic model due to Grossman (1972). We highlight four examples here. First, while Wagstaff (1986) illustrates how income differences lead to health differences in the traditional theory, recent quasi-experimental studies provide evidence that changes in income do not appear to improve health (e.g., O’Donnell et al. 2015; Cesarini et al. 2016).¹ Second, whereas educational attainment is assumed to increase the efficiency of medical care and thereby health (e.g., Grossman, 1972; 2000), and there exists ample empirical evidence for a strong association between education and health (e.g., Freedman and Martin, 1999; Cutler and Lleras-Muney, 2008), recent quasi-experimental studies have established that the causal effect of education on health is much smaller than the raw association suggests and seems to exist only in certain contexts and at certain levels of education (e.g., Galama et al. 2018, Savelyev, 2020; Xue et al. 2020; Barcellos et al. 2021; Fletcher and Noghanibehambari, 2021). A third example is the growing body of evidence on the developmental origins of health and disease (e.g., Barker, 1993; Almond et al. 2018), which the conventional model is silent about. In fact, Dalgaard et al. (2021) show how the traditional health capital model implicitly assumes that initial differences are depreciated away as individuals grow older, whereas empirical evidence suggests that disparities in health start very early in life and continue to widen till around age 60 (e.g., Case, Lubotsky & Paxson 2002). Finally, ample evidence from psychology, and behavioural economics has convincingly demonstrated that human rationality is bounded. For example, people overweight small probabilities and exhibit present bias and loss aversion (e.g., DellaVigna, 2009; Loewenstein et al. 2013). Moreover, preferences are partly shaped by social forces (the so-called ‘habitus’; Bourdieu,

¹ There is however a literature showing beneficial effects of parental income on the child’s health (e.g., Akee et al. 2018), and most quasi-experimental studies on own income rely on one-off windfall shocks that are not necessarily representative for earned income effects on health (see discussion below on generalizability of quasi-experimental studies).
Hence, the rigid assumption of a rational, forward-looking decision-maker who makes choices in a social vacuum is obsolete.

Empirical research in economics, and increasingly also in other social sciences and epidemiology, has moved away from traditional ‘controlling for observables’ approaches – e.g., regression adjustment or matching on observables. This has many advantages. Most importantly, it avoids the very bold and unconvincing assumption that the variation in the exposure of interest is fully random after adjusting for a limited set of confounders. In these traditional approaches it is unclear where the variation in the exposure of interest comes from. Consider the example where one is interested in the effect of years of education on health. After controlling for a few confounders, is it really just random that one person has more years of education, or are we simply missing a key unobserved confounder (such as having high quality teachers, parental stimulation, etc.) that made this person go to school one year longer? From such methods, we will never know where the variation across individuals in years of education is coming from, making controlling-for-observables approaches fundamentally unreliable in establishing causal effects.

The empirical revolution in health and economic research essentially launched a shift towards (natural) experiments where the source of variation in the exposure is clear, with recent Nobel prizes in economics awarded to Angrist, Card and Imbens (2021) for the use of natural experiments, and to Banerjee, Duflo and Kremer (2019) for the use of randomized controlled trials. For example, staying with the example of the causal effect of years of education on health, Clark and Royer (2013) exploited an arbitrary birth date cut-off of the Raising of the School Leaving Age (ROSLA) education reform that induced otherwise similar groups, born on either side of the cut-off, to attain different years of schooling (e.g., Clark and Royer, 2013). They found that the reform increased years of education, but did not affect health or health behaviour. The main advantage of this approach is that – given the random-like nature of the difference in education – the likelihood of residual confounding is much lower.

However, these methods are not without critique either (e.g., Deaton, 2010; 2020; Deaton and Cartwright, 2018; Heckman and Urzua, 2010; Imbens, 2010; 2018).
Perhaps most importantly, there are important doubts about external validity of the local treatment effects: "A result that is true in one place, at one time, and under one set of circumstances, will typically not be true in another place, another time, or under different circumstances." (Deaton, 2020).

We believe the focus on natural experiments has brought more transparency to empirical research, and got us closer to establishing causal effects of specific marginal additions to education, or income. However, given the sometimes highly specific nature of natural experiments, in order to make substantial progress in our understanding of the causal effects of socioeconomic status on health, we need to triangulate various empirical research methods, not just limiting ourselves to an opportunistic quest for natural experiments. Moreover, in a world where economics is increasingly dominated by empirical research, it is more important than ever to have theory assist in explaining heterogeneity across the diverse study contexts that the natural experiments span. Theory can help researchers test specific mechanisms to better understand and enhance the external validity of specific empirical studies.

In this review chapter, we will first sketch the model by Galama and Van Kippersluis (2019), which is based on the seminal health-capital theory of Grossman (1972), and discuss its most important insights relevant for health inequality research. Next, we revisit some of the most important sources of critique of the model and sketch directions of how the theory could be extended to do justice to the recent empirical literature refuting some of the theoretical assumptions. We believe advancements in these directions are feasible and anticipate them to occur in the next few years.

**Health Capital Theory**

In Galama and Van Kippersluis (2015; 2019) we developed a theory of disparities in health between SES groups, based on the seminal framework that Grossman (1972, 2000) developed. The theory is developed as a mathematical constrained optimization problem, where a so-called ‘utility’ function is optimized subject to several (dynamic) constraints. By analysing the theory, one can derive ‘optimal’ trajectories for certain choice variables, where optimal means that they provide the highest lifetime utility for a given set of (dynamic) constraints.
In the theory, there are three periods of life, a schooling period up to age $S$, working life up to the retirement age $R$, and a retirement phase that runs until age $T$. In all phases of life, individuals maximize a so-called utility function $U[\cdot]$ that captures the important things they care about

$$\sum_{t=0}^{S} \frac{U[C^h_t, C^u_t, H_t, p_S]}{(1+\beta)^t} + \sum_{t=S}^{R} \frac{U[C^h_t, C^u_t, H_t]}{(1+\beta)^t} + \sum_{t=R}^{T} \frac{U[C^h_t, C^u_t, H_t]}{(1+\beta)^t}$$

where utility $U[\cdot]$ is provided by healthy consumption $C^h_t$, unhealthy consumption $C^u_t$ and health $H_t$. In simple terms, people care about their consumption and would like to enjoy good health. During schooling years, there is an additional element $p_S$ that captures the (dis)utility of schooling, where some people enjoy the school experience and others do not. $\beta$ is the rate at which individuals discount future utility (capturing that we tend to care more about today than about some far away future). The optimal (in the sense of providing the highest lifetime utility) school-leaving age $S$ and retirement age $R$ are obviously influenced by prevailing policies like minimum school-leaving ages and statutory retirement ages, but are like length-of-life $T$ assumed to be choices made by the individual, subject to a set of constraints.

The first of these is that health depreciates with age at the aging rate $d_t$

$$H_{t+1} - H_t = \mu_t I_t^\alpha - d_t [C^h_t, C^u_t, z_t, H_t]$$

The aging process $d_t$ can be countered through health investments $I_t$ at efficiency $\mu_t$, and the health investment production process is subject to decreasing returns to scale ($0 < \alpha < 1$), which addresses the degeneracy of linear investment models (Ehrlich and Chuma, 1990; Galama, 2015). It also captures an important economic concept, namely that of diminishing returns. In simple terms, people can invest in their health to counteract aging, but higher levels of investment, while still better, are less effective than are smaller levels: exercising 150 minutes a week is good for one’s health, while the additional gains from exercising $>500$ or $>1000$ minutes become smaller and smaller.

Lifestyles and consumption patterns may affect the biological aging rate (Case and Deaton, 2005; see also Forster, 2001). We distinguish healthy consumption $C^h_t$ (such
as the consumption of healthy foods, sports and exercise) from unhealthy consumption $c_t^u$ (such as smoking, excessive alcohol consumption). Healthy consumption provides utility $\partial U / \partial c_t^h > 0$, and is associated with health benefits in that it lowers the biological aging rate $\partial d / \partial c_t^h < 0$. Unhealthy consumption also provides utility $\partial U / \partial c_t^u > 0$ but increases the biological aging rate $\partial d / \partial c_t^u > 0$. The model also allows for the choice of tougher job conditions $z_t$ that increase aging $\partial d / \partial z_t > 0$, but are compensated by a higher wage (allowing for higher levels of utility-enhancing consumption). Finally, the aging rate depends in a flexible way on health, countering the critique by Dalgaard and Strulik (2014) and Dalgaard et al. (2021) that health depreciation in the Grossman model is always higher for the healthy.

In the model, there are three additional constraints. One is the budget constraint, the second is the time constraint, and finally there is the longevity constraint. The budget constraint stipulates that financial assets increase with the interest rate and earned income (or a pension benefit in the retirement phase), which is a positive function of health. Assets decrease with expenditures on healthy and unhealthy consumption, as well as medical care (or other types of health investments, such as healthy behaviour). Over the life cycle one cannot spend more on consumption and health than one earns and inherits. The time constraint simply states that the total time in a day is divided into leisure and time inputs into health investment and consumption, as well as schooling or work depending on the phase of life. Finally, the longevity constraint imposes that health cannot be below a certain minimum threshold, below which life is no longer sustainable, and death is defined as the first moment health reaches this minimum threshold.

**Key insights**

In Galama and Van Kippersluis (2019), we perform comparative dynamic analyses to assess the characteristics of the model and generate empirically testable predictions. Here we summarize the key conceptual insights in an intuitive manner.

1) **Health as one of the inputs into utility, leading to trade-offs**

An important feature of economic theories of health is that the utility function (equation 1), which can loosely be interpreted as life satisfaction (e.g.,
Kahneman and Krueger, 2006), depends not just on health but also on consumption. While health and consumption preferences often are enhancing, in some cases they lead to trade-offs. Many people enjoy a glass of wine, chocolate, and other types of consumption goods and services that are detrimental to health (at least when consumed in larger quantities). Economic theories generally assume that individuals can perfectly navigate these trade-offs, leading to an optimal bundle of healthy and unhealthy consumption and an associated health level.

A consequence of the assumption that any person decides on her optimal bundle of consumption and health is that economists are typically wary of government intervention. In fact, in contrast to the health sciences, economists are not necessarily interested in improving public health at all cost. Only when externalities exist (e.g., second-hand smoke), when individuals are expected to be misinformed or biased (as evidence from psychology and behavioural economics suggests), or when poverty traps exist, such as food deserts and poverty leading to unhealthy lifestyles because of lack of affordability, government intervention is recommended by economists.

Obviously, the assumption that individuals make rational and optimal decisions is clearly wrong (see also below), but we argue that the assumption of rationality still provides a useful normative benchmark. It is also at least to some extent correct. When faced with new information about, say, the detrimental effects of smoking (e.g., the Surgeon General’s report of 1964), people do respond by quitting or reducing consumption, in particular those of higher SES (something economic theory would predict [see below point 2]).

2) **The health cost of unhealthy consumption**

The theory predicts a central role for a “health cost” of unhealthy behaviours. The choice as to whether to engage in unhealthy consumption or not is not only a function of the direct monetary cost (e.g., the price of a pack of cigarettes) but also of an indirect health cost. This health cost is the marginal
value (in terms of life-time utility) of health lost due to detrimental health behaviours. This cost considers all future consequences of a current health behaviour. In Galama and Van Kippersluis (2019) we find that the health cost increases with wealth (as well as with permanent income\textsuperscript{2} and education) and with the degree of unhealthiness of the good. This leads to the prediction that higher wealth increases demand for healthy and moderately unhealthy consumption goods, but decreases demand for severely unhealthy goods. Our theory may thus provide an economic rationale for the observation that wealthy, high income and educated (permanent income) individuals are more likely to drink moderately, but less likely to drink heavily and smoke (Cutler and Lleras-Muney, 2010; Van Kippersluis and Galama, 2014). Thus, apart from well-established cultural and social determinants of health behaviour, the concept of a health cost has further potential for explaining variation in health behaviours over the life cycle and across SES groups.

3) Wealth and the difference between absolute and marginal utility

The theory predicts that greater wealth, higher earnings and a higher level of education induce individuals to invest more in health, shift consumption toward healthy consumption, and enable individuals to afford healthier working environments. As a result, they are healthier and live longer.

Intuitively, at high levels of wealth individuals enjoy a large basket of consumption. As a result, only limited marginal utility ($\partial U / \partial c_h^t > 0$) or $\partial U / \partial c_u^t > 0$) is gained from yet more consumption. In simple terms, when you own three cars and you have a swimming pool in your backyard, the pleasure from yet another car or another swimming pool is limited. Thus, consuming more when consumption is already high provides relatively small gains in utility ($\partial^2 U / \partial^2 c_h^t < 0$ and $\partial^2 U / \partial^2 c_u^t < 0$). Once again, this is the economic notion (and assumption) of diminishing returns, in this case in the utility function. Still, the absolute level of utility $U[\cdot]$ is very high for high levels of consumption and

\textsuperscript{2} Permanent income is a measure of “life-time income”. If earnings are high over the duration of life, permanent income is high.
a wealthy individual is thus very interested in prolonging the period over which this high level of utility is experienced. The difference between health and consumption is that health extends length of life, providing additional time during which consumption (two cars and a swimming pool) can be enjoyed. This leads wealthy (and high SES more generally) individuals to place a higher value on their health and invest more in it (Becker, 2007; Hall and Jones, 2007). The flipside of this statement, is that lower SES individuals place a lower value on their health. The gains from more consumption are relatively higher for them than are the gains from life extension (i.e., compared to higher SES individuals). Note that the use of the term ‘value’ is not a normative judgement, it is the result of lower SES individuals facing more stringent constraints (lower wealth, lower income, and often poorer health) within the model. The theory thus provides an explanation for the observation that higher SES tend to lead healthier lives (as they can afford it and benefit more from it; see point 4 below).

4) The value of health and health inequalities over the life cycle

As high SES individuals place a higher value on their health, this increases the marginal benefits of healthy consumption, and the marginal costs of unhealthy working environments, and unhealthy consumption. As a result, high SES individuals lead healthier lives, and this gradually leads to a health advantage with age. The more rapidly worsening health of low SES individuals (who engage more in unhealthy behaviour and in more physically / psycho-socially demanding work) may lead to early withdrawal from the labor force and associated lost earnings, further widening the gradient in early- and mid-age. Jointly these behavioural choices gradually lead to growing health advantage for higher SES groups with age, similar to a process of cumulative advantage (e.g., Beckett, 2000; Dannefer, 2003; Lynch, 2003; DiPrete and Eirich, 2006). The model allows for a subsequent narrowing of the SES-health gradient, due to mortality selection but also because the marginal value of health grows when health declines. In simple terms, when health is low, it becomes a primary concern and the value of health increases. With low levels of health people care
more about it. Hence, the theory predicts that low SES individuals after a certain age increase their health investment and improve their health behaviour as a result of their rapidly worsening health. This prediction relies on the assumption that health depreciation can be remedied through curative care and/or improved health behaviours, or at least deteriorate at a slower pace. What is interesting is that the prediction provides an economic behavioural interpretation of the age-as-leveller hypothesis (e.g., Dupre, 2007; see also Hoffmann, 2011 and Fritzell and Rehnberg in this volume). The theory thus allows for a number of stylized facts about the observed life-cycle patterns of the SES-health gradient, although empirical evidence will be needed to discriminate between competing explanations for the same phenomenon.

5) The importance of being able to influence longevity

Finally, a central prediction of the theory is that the ability of individuals to influence their longevity is crucial in explaining observed associations between SES and health. If life expectancy is fixed and exogenously given, associations between SES and health are small. If, however, life can be extended, SES and health are positively associated and the greater the degree of life extension, the greater is their association. The intuition behind this result is that the horizon (life expectancy) is a crucial determinant of the return to investments in health. Investments in health lead to higher utility and make one more productive, but also importantly can boost life expectancy since mortality is defined as the first instance where health hits a minimum threshold. Now if life expectancy is (perceived to be) fixed, then investments in health will have much lower returns, and high SES individuals will be less tempted to invest in health. This suggests that in settings where it is (perceived as) difficult for wealthier, higher income and higher educated individuals to increase life expectancy (e.g., due to a high disease burden, competing risks, low efficiency of health investment, etc.), health disparities across socioeconomic groups would be smaller. Instead, in contexts where life expectancy is (perceived to be) to a large degree under the control of an individual (e.g., low disease burden, state-
of-the-art medical technology) one would expect stronger disparities in health across SES groups (Galama and Van Kippersluis, 2022).

6) **Interpreting policy changes such as changes in compulsory schooling**

As the utility function in equation (1) illustrates, economic models posit that individuals optimally choose their years of schooling, retirement age, and even length of life. While the latter is obviously a drastic simplification of reality, the idea that individuals consciously choose a certain level of education is less controversial. When accepting the notion that – without any government intervention – individuals would optimally select the years or level of education that is optimal for them (again, in the sense of deriving most lifetime utility from this choice), Galama, Lleras-Muney and Van Kippersluis (2018) derive that imposing a minimum school-leaving age will therefore reduce utility levels for some. After all, if it is optimal to drop out of school at, say, age 16 for a certain individual who really dislikes school, has bad quality teachers and would be better off entering the labour force, yet the government increases the minimum school-leaving age to 17, then this individual will be worse off in terms of lifetime utility. Linking it to the empirics, it is actually this group of people that would have liked to drop out of school but were forced to stay in school under the new rules (sometimes referred to as ‘compliers’) on basis of which the ‘treatment effect’ of raising a minimum school-leaving age is estimated. From a theoretical perspective, it is therefore perhaps not so surprising that most empirical studies of minimum school-leaving ages fail to detect meaningful effects on health. In fact, in line with this reasoning, Avendano, de Coulon and Nafilyan (2020) show limited and if anything, negative effects on mental health between the ages of 16 and 70. This point applies broader than just to minimum school-leaving ages. In fact, any policy reform may interfere with optimal decisions by individuals, and the treatment effects identified on basis of these, so-called, compliers affected by the policy reform may be quite different from the average treatment effects that we typically are interested in (see also Hoffmann and Doblhammer in this volume).
Future extensions

The theory of health disparities we sketched includes health investment, healthy and unhealthy consumption, job choices, and longevity, and is capable of replicating a number of stylized facts regarding socioeconomic inequalities in health over the life cycle. Economic theory is typically stated as a mathematical optimization problem where individuals optimize a certain utility function under constraints, and is therefore essentially an analysis of the benefits and costs of a certain decision. We feel this systematic and formal exposition of a theory is a great tool to generate predictions and hypotheses about real-world behaviour. However, traditionally, too stringent and frankly sometimes implausible – assumptions were historically imposed for mathematical convenience. As touched upon in the introduction, there are a number of empirical findings and regularities that call for further extensions and the loosening of the assumptions of the theory. In our view, these can be grouped into three broad directions: (i) an early childhood phase, (ii) bounded rationality, and (iii) social and contextual influences.

First, the theory predicts that traditional socioeconomic advantages like higher income, educational attainment, and wealth, will lead to more health investment and eventually better health. However, despite a very strong association between socioeconomic status and health, recent studies that seek to estimate causal effects of income or education on health outcomes show only limited or no evidence for these theoretical hypotheses (e.g., Clark and Royer, 2013; Cesarini et al. 2016; Avendano et al. 2020). While sometimes methodological arguments are brought up, it seems fair to say that at least some, and arguably a substantial, portion of the socioeconomic health gradient is due to third factors influencing both SES and health. Examples of such third factors include time preferences (e.g., Fuchs, 1982) and cognitive and non-cognitive skills (e.g., Conti et al. 2010; Bijwaard et al. 2015; Strulik, 2018), characteristics which themselves are shaped by prenatal and early-life factors (e.g.,

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3 For example, some argue this could be due to a difference between the Local Average Treatment Effect (LATE) that natural experiments typically uncover versus the Average Treatment Effects on the Treated (ATT) that OLS seeks to estimate.
Barker, 1995), family socioeconomic status (e.g., Hart and Risley, 1995; Currie, 2009; Almond et al. 2018), and also genetic variants (e.g., Boardman et al. 2015; Diewald in this volume).

The traditional Grossman model treats education as exogenously given. Galama and Van Kippersluis (2022) allow individuals to invest in both education as well as health, but their model starts around age 16, where wealth and health endowments at that age are given. An important extension of the theory is therefore the inclusion of a distinct childhood phase of life. One might treat the child as passive, in the sense that parents make decisions regarding time and financial investments in their children, but the children themselves do not. The child’s own adult phase, where he/she makes conscious decisions, then is shaped by preferences and constraints, both of which are shaped by conditions at conception (e.g., genes, family SES), in the pre-natal period (e.g., maternal behaviour) and the early childhood phase (e.g., parental attention). Such a model would do justice to the overwhelming evidence for an influence of genetic and early-life factors implicated in both health and SES, and potentially account for inequalities in health across SES groups, which arise at very young ages (e.g., Case, Lubotsky & Paxson, 2002).

A second promising direction is to move away from the assumption of rationality and allow for systematic and predictable irrationality in decision-making. There is ample evidence from psychology and behavioural economics that individuals are at most boundedly rational, and deviate from it in predictable ways. Examples include individuals disproportionately overweighting the present (a so-called present bias, e.g., Laibson, 1997; O’Donoghue and Rabin, 1999), overweighting small probabilities, disproportionately valuing certain over uncertain outcomes (e.g., Tversky and Kahneman, 1992; Prelec, 1998), and being subject to temptations, especially in stressful situations (e.g., Loewenstein and O’Donoghue, 2004). Unhealthy behaviours are a case in point. The benefits are immediate and certain: lighting up a cigarette or late-night snacking provides immediate gratification. But the costs are mostly in the future and highly uncertain: a higher risk of developing disease and premature mortality sometime in older age. Moreover, recent evidence suggests that “scarcity” in terms of money (or time) may impede the capacity to rationally calculate and weigh
these future costs (e.g., Mani et al. 2013) and education and cognition influence the accuracy of longevity expectations (Bago d'Uva et al. 2020). Hence, SES clearly influences the extent to which individuals make rational decisions and there may be substantial pay-offs to incorporating deviations from rational risk and time preferences into the theory.

A final promising extension is incorporating the role of social contexts, peer groups and social and cultural capital into the theory. The traditional model is one of individual decision making, where an individual optimally makes decisions in a social vacuum. However, empirically, it has been well-established that decisions are shaped by social contexts, in particular peer effects and social expectations (e.g., Nakajima, 2007; Heckman, Flyer and Loughlin, 2008; Cawley and Ruhm, 2011). There is therefore a need to incorporate social and cultural capital, along the lines of Bourdieu (1986), into health capital theory.

**Discussion**

While the scientific evidence on the drivers of the socioeconomic gradient in health has rapidly expanded, the mechanisms through which health differences across socioeconomic groups emerge and persist are still relatively poorly understood. Economic theory can help guide empirical studies in identifying mechanisms through which specific socioeconomic indicators and health interact. We believe that the frontier needs to be pushed both theoretically as well as empirically. Theoretically, the model could incorporate a childhood phase in which parents invest in the cognitive skills, non-cognitive skills and health of children, and where later-life preferences and constraints are shaped by this childhood phase and genetic and environmental differences. Empirically, we have to find a middle ground between internal and external validity, observational and (quasi-)experimental studies, where theory could help in bridging this gap.
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