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Hungry children age faster

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ABSTRACT

We analyze how childhood hunger affects human aging for a panel of European individuals. For this purpose, we use six waves of the Survey of Health, Aging, and Retirement in Europe (SHARE) dataset and construct a health deficit index. Results from log-linear regressions suggest that, on average, elderly European men and women developed about 20 percent more health deficits when they experienced a hunger episode in their childhood. The effect becomes larger when the hunger episode is experienced earlier in childhood. In non-linear regressions (akin to the Gompertz-Makeham law), we obtain greater effects suggesting that health deficits in old age are up to 40 percent higher for children suffering from hunger. The difference of health deficits between hungry and non-hungry individuals increases absolutely and relatively with age. This implies that individuals who suffered from hunger as children age faster.

1. Introduction

Health shocks that occur early in life influence late-life health outcomes. While most of the earlier studies establishing this link focused on child development in utero (Barker, 1992; Almond and Currie, 2011), more recent studies document a similar pathway for early-life conditions to health in old age. The original "fetal origins" literature matured into a more general "developmental origins" literature (Barker, 2004: Gluckman et al., 2005; Almond et al., 2017). In particular, undernutrition in early stages of life has been found to be detrimental to late-life health. Through the direct biological pathways of epigenetic changes and 're-programming' of bodily functions, undernutrition early in life causes metabolic dysfunction across all ages, resulting in a higher risk of diseases, such as diabetes, hypertension, and cardiovascular diseases later in life (McMillen and Robinson, 2005). Indirect effects are mediated through health behavior, cognitive and non-cognitive development, education, and socio-economic status in adulthood (Case et al., 2005; Kesternich et al., 2015; Havari and Peracchi, 2017).

In this paper, we extend the literature, which so far focused on the link between early-life circumstances and the *state* of adult health, by establishing a link between early-life circumstances and the *changes* in adult health as people get older. Specifically, we show that the experience of a hunger episode in childhood affects biological aging, measured as the accumulation of age-related health deficits with age. Across 14 European countries, elderly women who experienced a hunger episode between age 0 and 4 have developed 30 percent more health deficits by age 50 and 50 percent more health deficits by age 75, compared to women of the same age who did not suffer from hunger in childhood. Effects are somewhat smaller for men and when hunger was experienced from age 4 to 8. The qualitative conclusion, however, remains robust. Early-life nutritional shocks lead to faster biological aging later in life.[1]

The result that undernutrition in childhood affects aging is immediately plausible by noting that the diseases that have been associated with early-life nutritional shocks, like cardiovascular disease, diabetes, and hypertension, are chronic, age-

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[1] Related studies on the impact of childhood hunger on the state of health later in life are Kesternich et al. (2015), van den Berg et al. (2016), and Havari and Peracchi (2017). At a more general level, the influence of childhood exposure to World War II on adult health has been investigated by Kesternich et al. (2014), Halmdienst and Winter-Ebmer (2014), and Akbulut-Yuksel (2017). A large literature documents the impact of nutrition and disease exposure in childhood on adult height as a health indicator, see e.g. Fogel and Costa (1997), Case and Paxson (2008), and Bozzoli et al. (2009). An economic theory establishing the link between childhood nutrition and adult body size is provided by Dalgaard and Strulik (2015, 2016).

related diseases. They can be considered as specific expressions of the general phenomenon of aging, understood as intrinsic, cumulative, progressive, and deleterious loss of bodily function (Arking, 2006). A micro-foundation of the impact of childhood conditions on adult health is provided by the reliability theory of aging. Reliability theory explains why humans and other living organisms age although they are constructed of non-aging elements. A particular challenge is to explain aging such that mortality (the failure rate of the system) follows the Gompertz (1825), Makeham (1860) law (Gavrilov and Gavrilova, 1991). To do so, it has been suggested that one can conceptualize human bodies as systems that achieve reliability not by the initial quality of their elements but through their large redundancy. Indeed, the functional capacity of organs in young adults is estimated to be tenfold higher than needed for mere survival (Fries, 1980). The fact that many elements are initially defective motivates aging according to the Gompertz-Makeham law. The "self-assembly" process of construction of living systems explains why many elements are already defective early in life. The number of initially functioning elements determines the speed of aging and the age at death. Reduced redundancy (of cells, organ tissue, etc.) and increasing initial damage has been attributed to nutritional deprivation in childhood (Gavrilov and Gavrilova, 2004). While there is no direct evidence to support this claim, our study can be interpreted as indirect evidence for a pathway from damages in early life through childhood malnutrition to faster aging.

In this paper, we follow Mitnitski et al. (2001) and measure health and aging by a health deficit index (a frailty index). This index measures the number of health deficits that a person has at a given age relative to the number of potential health deficits that he or she may have. Health deficits include mild ones as well as serious disabilities. The exact choice of deficits is not crucial provided that sufficiently many indicators are present in the index (see Rockwood and Mitnitski, 2006, 2007 for a methodological background). The seminal papers by Mitnitski et al. (2002a,b) catalyzed a very large research program with hundreds of studies applying the methodology.[2]

The quality of the deficit index is mostly (but not exclusively) demonstrated by its predictive power for death at the individual level, and for mortality at the group level. The prediction of mortality can be so accurate that chronological age adds insignificant explanatory power when added to the regression (Rockwood and Mitnitski, 2007). The elimination of chronological age in the determination of aging and death is the ultimate goal of any successful theory of aging (see Arking, 2006). Mitnitski et al. (2002b) show that the R^2 in a simple log–log regression of the health deficit index and the mortality rate is around 0.99. Of course, predicting individual death is much harder since death is stochastic at the individual level but the health deficit index frequently outperforms age and other conceivable indicators of individual health.[3]

Economically speaking, the health deficit index is a stock variable that accumulates as individuals age. We exploit this feature to measure the speed of aging by the increase of health deficits from one birthday to the next. We use six waves of the Survey of Health, Aging and Retirement in Europe (SHARE dataset) and construct a health deficit index consisting of 38 symptoms, signs, and disease classifications for individuals aged 50 and above. We use information on hunger episodes in childhood from retrospective life data (wave 3 of SHARE). We then regress the health deficits of individuals at a given age on age and the hunger indicator. Employing a log-linear regression analysis, we find that childhood exposure to hunger leads to about 20 percent more health deficits at any given age in old age. Since health deficits increase exponentially with age, this means that the difference of health deficits between exposed and non-exposed individuals gets larger as individuals get older. In non-linear regressions, akin to the Gompertz-Makeham law, we find even larger effects of hunger on aging and that the difference in late-life health between exposed and non-exposed individuals also increases in relative terms. In short, the factor by which the health deficit index of formerly hungry individuals exceeds the health deficit index of non-hungry individuals increases with age. This means that individuals exposed to hunger do not only age faster but that the speed of aging accelerates faster than for non-exposed individuals.

One reason for the success of the health deficit index is that it is easily understood and implemented. Since there are no degrees of freedom in weighing the health deficits, the index is comparable across different studies and across different populations (Searle et al., 2008). The convenience of the health deficit index becomes particularly salient when we compare it with health capital, i.e. the latent variable in the focus of many studies in health economics (following Grossman, 1972). More importantly, the notion of health capital is not conducive to the understanding of early-life origins of late-life health. The reason is that according to health capital theory, healthy people (i.e. those with a large health capital stock), by assumption, lose more health capital through depreciation (see Almond and Currie, 2011). This creates an equilibrating force such that initial health differences (e.g. from exposure to nutritional shocks) depreciate as individuals age such that they play asymptotically no role in old age. The health deficit model (Dalgaard and Strulik, 2014), in contrast, predicts just the opposite. Small initial health differences are amplified with age such that the difference in health deficits between initially well-nourished and malnourished individuals becomes larger over time (Dalgaard et al., 2017). Subsequently, the consequences of initial health differences become salient and noticed for the first time when individuals reach old age. In the context of our study, this means that evidence for a widening health gap between individuals exposed to hunger and well-nourished children refutes the health capital model and supports the health deficit approach.

This paper builds on our earlier work (Abeliansky and Strulik, 2018) where we investigated the similarity (and possible invariants) of human aging in a sample of European countries but did not study the impact of early-life health shocks. Here, we use the same data (extended by wave 6) and contribute to the literature of "developmental origins" by investigating the impact of childhood hunger on the speed of aging in old age.

The paper is organized as follows. In Section 2 we describe the data. In Section 3 we estimate the relationship between the health deficit index, age, and hunger in childhood using log-linear panel regressions. In Section 4 we continue with non-linear (Gompertz-Makeham style) regressions and in Section 5, we estimate the impact of the length of the hunger episode on health and aging. Section 6 concludes.

[2] Originally, the methodology was established by Mitnitski, Rockwood, and coauthors as the frailty index. Newer studies also use the term health deficit index (e.g. Mitnitski and Rockwood, 2016), which seems to be a more appropriate term when the investigated population consists, to a significant degree, of non-frail persons. A handful of studies have investigated the health deficit index (frailty index) using the SHARE dataset (Romero-Ortuno and Kenny, 2012; Harttgen et al., 2013; Theou et al., 2013; Romero-Ortuno, 2014; Abeliansky and Strulik, 2018).

[3] The simple unweighted health deficit index performs well most likely because health deficits are connected, see Mitnitski et al. (2017). For studies on the association between health deficits and mortality using the SHARE dataset, see Romero-Ortuno and Kenny (2012) and Theou et al. (2013).