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**Physiological Aging around the World and Economic
Growth**

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Physiological Aging around the World and Economic Growth*

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Abstract. As the composition of the world population gradually shifts towards older age groups, it becomes increasingly important to understand the influence of aging on macro-economic outcomes of interest. Until now, however, it has been impossible to separate out the role played by demographics from the pure role of aging at the country level. Drawing on research in the fields of biology and medicine, the present study provides data on physiological aging. Our data shows that, over the last quarter of a century, the average person in the global labor force has not grown older in physiological terms. In an application of our panel dataset, we find evidence that accelerated physiological aging causally reduces labor productivity. Taken together, our analysis suggests that if productivity growth has decelerated in recent decades, physiological aging is unlikely to be a contributing force.

Keywords: Physiological Aging; Economic Growth

JEL: O5; I15

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1. INTRODUCTION

As fertility declines and progressively more people reach an advanced age in life, the typical world citizen grows older. As depicted in Figure 1, the median age has risen persistently since the 1970s, and this process is expected to continue throughout the 21st century. According to United Nations “medium fertility forecast” there will be as many people in 2100 above the age of 40 as below; in the richest parts of the planet this is already the case today. Naturally, this trajectory entails changes in the demographic structure of the world population. But it also suggests physiological changes in the average member of the labor force. In the present study we develop a measure of average physiological aging at the country level, investigate its evolution worldwide, and inquire into the likely consequences of aging for productivity growth.

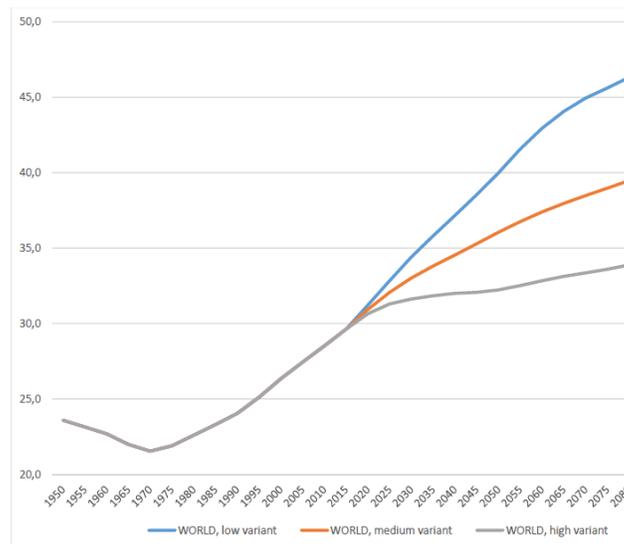


FIGURE 1. Median age of world population: 1950-2100. Note: Data points after 2015 are projections, reflecting low, medium and high fertility projections. Source: UN population division.

Our measure of aging is grounded in the literature on the so-called *health deficit index*, following the seminal work of Mitnitski and Rockwood (2002a,b). For an individual, this index simply records the fraction of a set of health conditions that he or she has.¹ As the index rises towards one the individual is viewed as increasingly frail, and in this sense physiologically older. Conceptually, this notion of aging has its foundations in biological research on why humans age pioneered by Gavrilov and Gavrilova (1991), as discussed below. The index itself contains

¹In practise 30-40 conditions are required. We discuss the principles underlying the construction of the index below.

information on conditions for which disease prevalence generally rises with age; communicable diseases, for example, are thus not admissible. From a macro perspective the *average* health deficit index in a population can be shown to equal the average prevalence rate of relevant conditions. In the end, it can be calculated for people in the labor force; for men and women, respectively, or, for particular age-groups within a population. Below, we construct aggregate health deficits indices for 191 countries at five year intervals between 1990-2015. We validate the resulting index, showing that the macro health deficit index mimics key regularities of the micro counterpart. This is the first contribution of the present study.

The second contribution is to document that, over the last quarter of a century, the average member of the labor force has not grown older in physiological terms, measured by average health deficits. We also find that the variation in the health deficit index across countries has remained relatively stable between 1990 and 2015. Finally, we explain more formally how changes in average health deficits over time in a country can be translated into changes in average “physiological age” and explore the link between changes in the average physiological age of the labor force and in average chronological age in the labor force between 1990 and 2015 across countries. While changes in chronological age is associated with rising physiological age the association is less than one for one. Moreover, our exploratory regressions suggest that the link between physiological age and chronological age is shifting down over time, perhaps reflecting health improving innovations. This result is robust to the control for growth in average income, and holds generally within every continent.

The third contribution is to explore the link between aging and productivity growth. Naturally, the present study is not the first to ponder this link. However, until now “aging” has been captured solely by demographics, and physiological aging is not merely a time dependent process. In practise, therefore, one can reasonably think of different degrees of aging within age-groups as well as between. In addition, of course, the chronological age of an individual has economic ramifications beyond those pertaining to aging per se. For example, we participate in the labor market only in some stages of life, in others we are not (allowed to be) active; in some parts of our lives we have children, in others we do not etc. These effects, and a potential impact of physiological aging, are confounded when only demographic variables are considered. However, by employing the macro health deficit index, we are in principle able to separate the impact of aging from that of the demographic structure on growth. Filtering out the influence

of demographics on labor productivity growth, our main finding is that elevated physiological aging is associated with lower labor productivity. We develop an instrumental variables strategy demonstrating that this link likely can be given a causal interpretation.

The present study is related to several strands of literature. One is an extensive literature that explores the relationship between health and economic outcomes, following the work of Grossman (1972) and Ehrlich and Chuma (1990). A stumbling block for this line of research has been that the core theoretical concept – “health capital” – is an inherently unobservable variable. Nevertheless, inspired by the work of Grossman, a literature has unfolded that makes use of various proxies. In a macroeconomic context, life expectancy at birth is an often used proxy.² Early contributions exploring the impact of health, measured in this way, on growth includes Knowles and Owen (1995), Bhargava et al. (2001) and Bloom et al. (2004).³ Another approach to the study of the bidirectional link between health and socioeconomic circumstances is centered around the concept of health deficits (Dalgaard and Strulik, 2014).⁴ The present study is the first to take this approach in an empirical macroeconomic direction. From an empirical vantage point, the key difference to the standard practise in macroeconomics is that our index focuses more on (age-related) morbidity, rather than mortality.

Another related literature studies the effect of aging on economic growth through demographic changes across countries. An early contribution examining the link between demographic structure and economic growth is Sarel (1995), who uncovers a hump shaped link between age structure and labor productivity, with a peak somewhere in the mid 40s. Broadly similar findings are reported in Lindh and Malmberg (1999), who augment a human capital augmented Solow model with age-group controls. More recent work along similar lines is that of Feyrer (2007), Maestas et al (2016), Kotschy and Sunde (2016), and Acemoglu and Restrepo (2017).⁵ While

²Aside from life expectancy other indicators of health that have been proposed in the macro literature including average height, birth weight and more; see Weil (2014).

³In recent years a debate erupted on the impact of life expectancy on growth, following the work of Acemoglu and Johnson (2007). For installments into this debate see e.g., Cervellati and Sunde (2011); Bloom et al. (2014); Hansen and Lønstrup (2016) on the empirical side, and Hazan (2009), Hansen and Lønstrup (2012); Cervelatti and Sunde (2013) and Strulik and Werner (2016) on the theoretical side.

⁴The basic model has by now been adapted to the study of the link between aging and human capital accumulation (Strulik, 2018); years in retirement (Dalgaard and Strulik, 2017); Fetal origins (Dalgaard, Hansen and Strulik, 2017); the gender-gap in mortality (Schünemann et al., 2017a), and the health gap between married and unmarried individuals (Schünemann et al., 2017b).

⁵Other contributions have focused on the influence of the over-all dependency ratio on growth (e.g. Bloom et al., 2001) and the youth dependency ratio (Kögel, 2005).

demographic change is an important consequence of an aging society, the present paper attempts to filter out these effects so as to focus on physiological aging.

The paper proceeds as follows. In the next section we discuss the underlying reasoning behind the health deficit index, how we adapt it to a macroeconomic context and explore validation tests of the resulting data. Section 3 explores the evolution of average health deficits in the labor force around the world and Section 4 contains our empirical analysis of physiological aging and labor productivity. Section 5 concludes.

2. MEASURING PHYSIOLOGICAL AGING

2.1. Introduction. “Aging” is defined as the intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death (Arking, 2006). But why do we age? A promising strand of literature in biology has sought an answer to the question by drawing on reliability theory from engineering.⁶ The basic idea can be illustrated by the following model (Gavrilov and Gavrilova, 1991). Suppose we view the organism as a whole as consisting of a fixed number of individual parts, which we will refer to as “blocks”. Each block does not age. That is, the failure rate of a block is constant. This assumption captures that the human organism is ultimately constructed from non-aging components.⁷ Next, assume that the blocks are connected in parallel, and that the system as a whole is assumed to survive as long as there is one functioning block remaining. This assumption is motivated by the physiological fact that the human organism is characterized by a great deal of redundancy; as young adults the functional capacity of our organs is estimated to be tenfold higher than needed for mere survival (Fries, 1980). Though each block does not age the passing of time will reduce redundancy in the system as a whole, which leads to an increasing failure rate of the system. Hence, the simple model successfully reproduces a rising death rate with age, in keeping with the Gompertz mortality law. Many extensions of this basic model have been developed, which have lead to new insights into the aging process.⁸ But for present purposes the key point of reliability theory is conveyed by the

⁶Reliability theory is used in engineering to understand the failure rate of mechanical devices. Gavrilov and Gavrilova (1991) were the first to introduce reliability theory into biology.

⁷Superficially, humans age because their organs age, which can be explained by aging tissue and so forth. Eventually, however, a level of disaggregation is reached where the “component” in question is non-aging, like atoms.

⁸For example, if one extends the framework we have just sketched by assuming that the body consists of a number of essential blocks (so that if one fails the organism fails), each of which consists of non-aging elements of which some initially are defect, the model reproduces the Gompertz-Makeham mortality law; see Gavrilov and Gavrilova (1991).

simple model: senescence can be conceptualized as the gradual loss of redundancy, ultimately leading to organism collapse.

Following the reasoning of reliability theory one may thus think of aging as being characterized by increasing frailty. That is, as the redundancy of the human organism shrinks, we become more fragile. An empirical measure of human frailty has been developed by Mitnitski and Rockwood and various coauthors in a series of articles in the form of the *health deficit index* (e.g., Mitnitski et al, 2002a,b; 2005, 2013, 2016; Rockwood and Mitnitski, 2006, 2007). As humans age, they develop an increasing number of disorders, which Mitnitski et al. (2002a) refer to as “deficits”. Some of these deficits may be viewed as relatively mild nuisances (e.g., incontinence) while others are more serious in nature (e.g., strokes). Nevertheless, the notion is that when the number of deficits rises the body becomes more frail. A health deficit index can then be estimated for an individual as the proportion of the total potential deficits (**a**ilment) $a = 1, \dots, A$ that an individual has. That is, the health deficit index of individual i from country c is

$$d_{ic} = \frac{1}{A} \sum_{a=1}^A \mathbf{1}_{ic}(a), \quad (1)$$

where $\mathbf{1}_{ic}(a)$ is an indicator function that takes on the value 1 if individual i suffers from deficit a .

In order to operationalize the health deficit index, one needs to choose a set of deficits to include. Searle et al. (2008) outline five criteria:

- (1) The deficit needs to be associated with health status. Graying hair, for example, would be inadmissible although it is obviously age related.
- (2) A deficit’s prevalence must generally increase with age, although some clearly age-related adverse conditions can decrease in prevalence at very advanced ages due to survivor effects. This rules out communicable diseases that do not feature the required age gradient in prevalence rates, for example.
- (3) The chosen deficits must not saturate too early. For example, as human age it becomes harder to focus on close objects (Presbyopia); by around 55 the disease is nearly universal and thus less than ideal to include.
- (4) The deficits that make up a frailty index must cover a range of systems. If the index becomes too narrowly focused, say on cognitive deficits, it potentially no longer captures overall aging but simply cognitive aging.

- (5) If a single deficit index is to be used serially on the same people, the items that make up the index need to be the same from one iteration to the next.

No specific deficit is required to enter into the index, since results appear to be unaffected as long as a sufficient number of deficits – 30 to 40 – are included (Mitnitski and Rockwood, 2007; Searle et al., 2008). In light of its simplicity and intuitive nature, it is perhaps unsurprising that the health deficit index has been applied in hundreds of studies until now. The novel element of the present study, from a measurement perspective, lies in computing aggregate health deficit indices for a panel of countries, i.e. we investigate the physiological aging of nations rather than individuals.

2.2. Measuring Physiological Aging at the Country Level. The average deficit index in country c , d_c , is

$$d_c = \frac{1}{P_c} \sum_i^{P_c} d_{ic},$$

where P_c is the size of the population in country c . In light of equation (1) a simple rearrangement of the sum allows us to write the average health deficit index as :

$$d_c = \frac{1}{P_c} \frac{1}{A} \sum_{a=1}^A P_{ac},$$

where P_{ac} is the number of people in country c that suffers from deficit a . Accordingly, in order to work out the aggregate deficit index we simply need to work out the average of A prevalence rates, P_{ac}/P_c , in each country. It should also be clear that if one wishes to calculate average deficits for age group g it is

$$d_{gc} = \frac{1}{A} \sum_{a=1}^A \frac{P_{agc}}{P_{gc}},$$

where P_{agc}/P_{gc} is the prevalence rate of a within age group g in country c . Similarly, one might choose to distinguish between men and women in the population.

Our data on prevalence rates derive from the database created by Vos et al. (2015), which today spans the period 1990 to 2015. The prevalence rates are available for men and women separately, as well as for five year age-groups. When computing the health deficit index, we focus on the population above 20 in keeping with conventions in the literature on health deficits.

In order to provide a relevant validation check of our data, we create average deficit indices for both men and women separately, albeit we resort to the overall average in the growth

analysis. Naturally, in order to construct deficit indices, we only include conditions that abide by the criteria listed in the last section. This leaves us with 45 conditions entering into the deficit indices. These deficits are listed in Appendix A, which also contains the sources for the remaining data used below.

2.3. Validation Checks. An important regularity that has emerged in the literature on health deficits is that the fraction of deficits for a representative individual grows from one birthday to the next at an approximately constant rate from the age of 20 onwards (e.g., Rockwood and Mitnitski, 2016). For example, using data for ten European countries that are represented in the European Survey of Health, Aging and Retirement (SHARE), Abeliansky and Strulik (2018) document that deficits accumulate by two to three percent per year on average by estimating a log-linear specification of the following type:

$$\ln d_{icw} = \theta_c + \theta_\omega + \mu_g \text{Age}_{icw} + \varepsilon_{icw}, \quad (2)$$

where i denotes individuals and g their gender, θ_c denotes country fixed effects and θ_ω is a survey-wave indicator (i.e., time dummies).⁹

Another key regularity that has been confirmed repeatedly in the literature (starting with Mitnitski and Rockwood, 2002a) is that the deficit index follows different trajectories for men and women. Specifically, while μ is greater for men than women, the intercept is smaller for men. This implies an inverse relationship between intercepts and slopes akin to the famous Strehler-Mildvan correlation, also known as the compensation effect of mortality (Mitnitski and Rockwood, 2002a; Strehler-Mildvan, 1960). It is worth observing that the compensating effect of mortality can be accounted for, based on the principles of the reliability theory of aging, if the rate of the primary processes which destroy the organism in aging is a species-specific invariant constant (Gavrilov and Gavrilova, 1991, p. 154); in the model above this means the failure rate of individual blocks is required to be a species specific constant. As a result, a compensating effect of deficits would also be anticipated.

⁹Mitnitski et al (2002a) show that deficits of the representative individual follows $d_t = e + b \exp^{\mu t}$, which is a formula that is structurally identical to the Gompertz-Makeham law of mortality. The regularity observed by Mitnitski and Rockwood (2016) is that μ is about constant after the age of 20. In this setting, then, the log-specification stated above (which imposes $e = 0$) is a useful short-cut if the objective is simply to estimate μ . Abeliansky and Strulik also estimate the non-linear equation on a country-by-country basis. They find μ is fairly constant across countries, albeit higher than that obtained in the log-linear case. In the present case, with a sample of 191 countries we stick with the approximation in order to provide a straightforward comparison with Abeliansky and Strulik's findings.

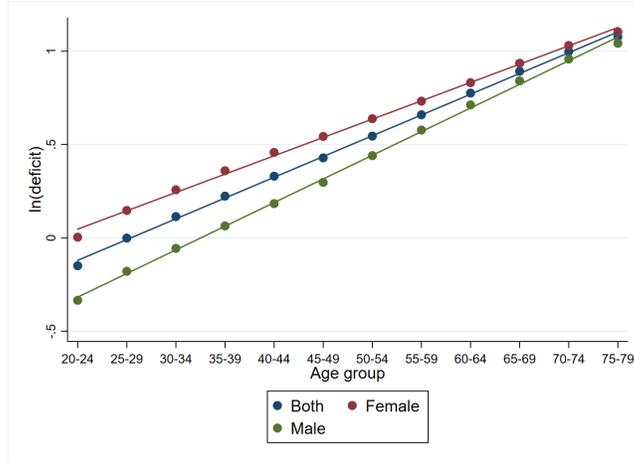


FIGURE 2. Binned scatterplot of log deficits vs five year age-groups: 20-79. Note: The sample includes all countries (191). Country fixed effects and time dummies have been controlled for.

Thirdly, the compensating effect of deficits is also found *between* countries; in countries where the level of deficits is high early in life the speed at which deficits accumulate over the life cycle is lower (see Abeliansky and Strulik, 2018).

As it turns out, our macro health deficit index behaves much like its micro counterpart. Figure 2 shows a binned scatterplot of log (average) deficits across five-year age groups: 20-24, ..., 75-79 for men and women as well as overall.¹⁰ In keeping with the first regularity, the growth rate of the deficit index is very stable. Moreover, the regularity concerning the differentiated evolution of deficits over the life course for the two genders is also borne out: the growth rate of deficits is faster for men than women, but the intercept is larger for women.

Table 1 provides estimates of the growth rate of deficits between the age of 20 and 79; the model that is estimated is equation (2) with time dummies replacing wave controls. Consequently, we estimate the growth rate of deficits considering both sexes together, corresponding to the blue line in Figure 2.

Table 1

In the first column, we consider the full sample. Overall, deficits grow by roughly 11 percent from one five-year age group to the next, implying an annual growth rate of around 2.2 percent, squaring well with the findings of Abeliansky and Strulik (2018). Moving from left to right in the table, the sample changes. Column 2 focuses on the OECD area, whereas the remaining

¹⁰The binned plot groups the x-axis variable into equal-sized bins, computes the mean of the x-axis and y-axis variables within each bin, then creates a plot of these data points. It provides built-in options to control for covariates before plotting the relationship.

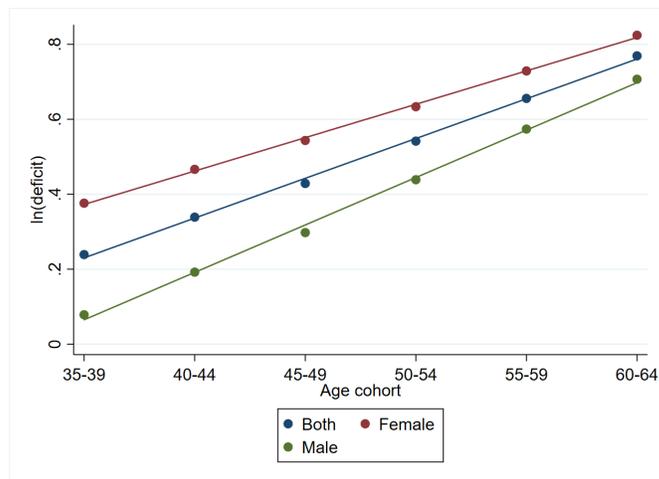


FIGURE 3. Binned scatterplot of log deficits vs age: 35–39 to 60–64. Note: The sample includes all countries (191). Country fixed effects have been controlled for.

columns consider each continent in turn. The growth rate of deficits across the life course is astonishingly stable: from 2.02 percent per year in Europe to 2.28 in Africa.

Table 2

Table 2 allows the growth rate of deficits to differ between the sexes. In keeping with the general message from Figure 2, we find a faster growth rate for men than women. Once again this regularity carries over to all sub-samples and is strikingly stable around the world.

These findings show that the macro health deficit index mimics key regularities in the micro counterpart quite well. At the same time it is worth noting that the evolution of health deficits across the life cycle is assessed – as is standard practise in the literature – using *cross age-group* information. That is, we study the link between deficits and age-groups at given point in time, to gauge its potential life course evolution. Ideally, one would like to track its evolution within a cohort over time, which is usually not possible due to data constraints.

In the present context it is possible to consider cohort variation as a robustness check. Since, for example, individuals that were 35–39 in 1990 are 60–64 in 2015, we can track groups of individuals within the same cohort over part of the life cycle. Figure 3 shows the binned scatterplot which now involves cohort information for individuals 35–39 in 1990 until they are 60–64 in 2015. As can be seen, the fit is as good as the one using cross age-group information to elicit life course information; the standard practise. Moreover, the results reported in Tables 1 and 2 are very

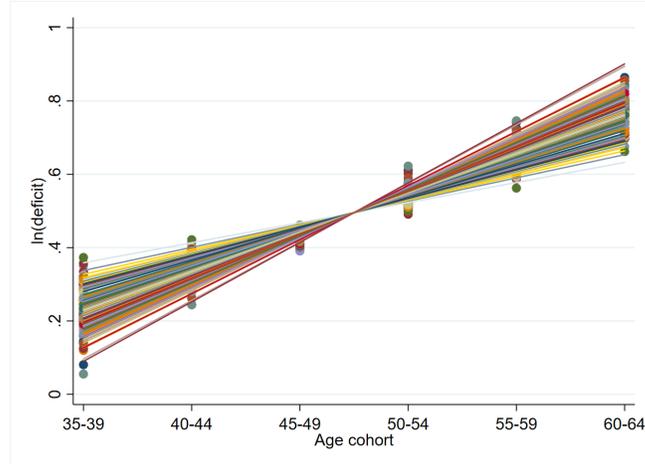


FIGURE 4. Individual deficit trajectories : 35–39 to 60-64. Note: The figure depicts deficit trajectories for 191 countries. Country fixed effects has been controlled for.

similar when relying on cohort data, as documented in tables A1 and A2 in the Supplementary appendix.¹¹

Finally, Figure 4 depict (log) deficit trajectories for all the 191 countries in our sample. As in Figure 3, we track individuals across the life cycle from the age of 35-39 until they are 60-64. Naturally, the individual country profile cannot be clearly identified but a key pattern is obvious: In keeping with the findings of Abeliatsky and Strulik (2018), the compensating effect of deficit accumulation is strongly present across countries. The fact that the level of (log) deficits early in life is a strong predictor of the growth in deficits across the life cycle is something we exploit below, in the context of identifying the impact of physiological aging on productivity.

More generally, the results depicted in Figure 2 - 4 imply that growth in deficits early in life are very good predictors of growth in deficits in later stages. This relative constancy in growth in deficits holds strong implications for the evolution of the *level* of deficits over the life cycle. It means, in particular, that shocks early in life lead to an accelerated pace of aging; if the initial level increases (due to a shock) and the (exponential) growth rate is roughly constant, the absolute change in the level of deficits needs to accelerate in the remaining years of life, leading to an earlier date of death absent higher investments and/or further shocks. Fundamentally, this captures the notion that aging is an “intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death” (Arking, 2006). Moreover, it also implies

¹¹All tables labeled "Table Ax", that we refer to below, are similarly found in the supplementary appendix.

that shocks early in life are propagated during life, *ceteris paribus*, in keeping with the empirical evidence in the literature on fetal origins (Dalgaard, Hansen and Strulik, 2017).

3. PHYSIOLOGICAL AGING OF NATIONS: 1990-2015

Before we turn to the empirical analysis of how aging impacts productivity, we begin by describing what our data suggests has happened to physiological aging around the world for the last quarter of a century. Specifically, we explore the evolution of the average deficit index for the economically active part of the population, i.e. the population between the age of 20 and 65.¹²

While growth in deficits across the life cycle is positive and fairly constant, changes in *average* deficits in the working age population are not. Figure 5 depicts the evolution of the average health deficits index across 161 countries (unweighted) from 1990 to 2015.¹³ Interestingly, while median age has been on the rise (cf figure 1) the average deficit index has not done the same. In the Supplementary Appendix, we display country trajectories for the individual OECD countries and for individual African nations. The general impression for the OECD is, similar to the world trend, that physiological age has been relatively constant though there are exceptions to the rule. For example, in countries such as Denmark and Switzerland, the average health deficit index has increased between 1990 and 2015. On the poorest continent in the world, average health deficits has as a rule been declining. Once again, however, there are exceptions, such as Mauritius and Seychelles, where deficits have gone up.

Figure 5 also depicts the time path of the extent of variability in average health deficits across the world over time. As can be seen, the cross-country variation does not appear to have changed significantly over the period in focus; in 2015 the standard deviation of log health deficits was 0.09 compared with 0.08 in 1990. A relevant comparison is with average chronological age of the labor force active population (15-65).¹⁴ Much like median age, average age across countries has risen between 1990 and 2015: from about 33 to 35 (in the same sample of countries). The

¹²We choose this focus since the health deficit index, according to the literature in gerontology, is considered a good guide to aging from the age of 20. The age-group 20-65 approximates the working age population (15-65).

¹³These are the countries for which the relationship between aging and productivity can be studied (see also next section).

¹⁴Note that average health deficits at a point in time is the population group weighted average of age-group specific health deficits. Hence, demographic changes directly influences average deficits by changing the weights of individual age-groups.

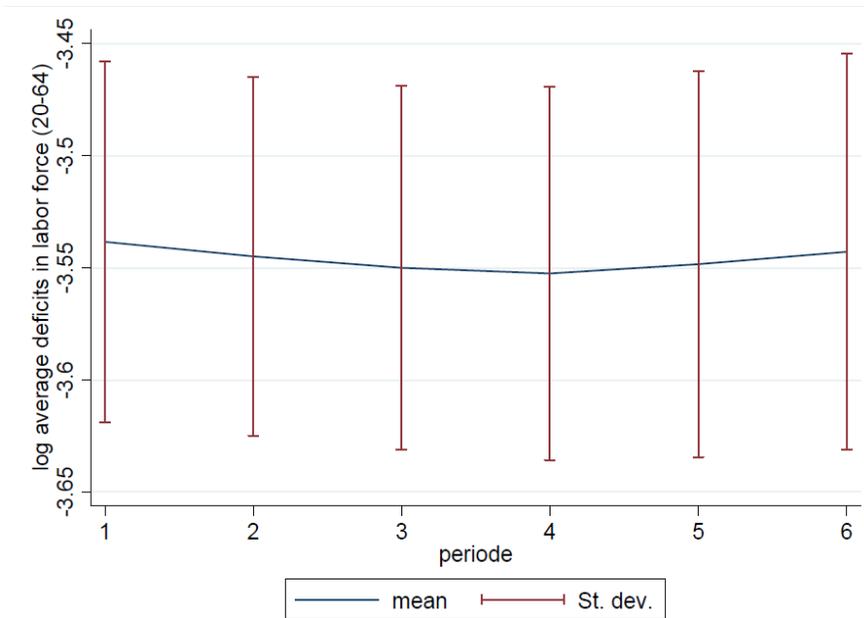


FIGURE 5. Average deficits across the world: 1990-2015. The figure shows the time path of log average deficits across 161 countries at five year intervals.

standard deviation of (log) average chronological age has increased from 0.07 to 0.09.¹⁵ Overall, the regularities depicted in Figure 5 illustrates that demographics and aging are worth separating from one another as they seem to follow distinct trajectories for individual countries as well as on average across countries.

So far the link between health deficits and physiological aging, while quite explicit in theory, has been somewhat indirect. Rising deficits are a consequence of the aging process, and by extension rising average deficits in the labor force can be seen as an indication of rising physiological age. It is, however, possible to make the connection a bit more explicit from a quantitative angle, with the aid of a few simplifying assumptions.

In keeping with the discussion in Section 2.3, assume that the rate at which deficits grow is constant over the life cycle and that it does not change from one cohort to the next.¹⁶ Moreover, suppose this growth rate is constant across countries. In particular, based on the results reported in Table 1 and A1, a reasonable estimate would be a μ in the range two to three percent per year. The constancy of growth in deficits over the life cycle across countries is obviously too strong, as seen from Figure 4. As a result, below we discuss the consequences of relaxing this

¹⁵A figure for the evolution of average chronological age and its standard deviation, corresponding to figure 5 is found in the Supplementary Appendix.

¹⁶The fact that the estimated μ based on cross-section data (Table 1) and cross-cohort data (Table A1) provides evidence that this likely is a reasonable approximation. In our robustness checks we provide further evidence.

assumption. Finally, assume there is an upper limit to deficits, $\bar{d} < 1$.¹⁷ Theoretically, this limit reflects that there is a level of frailty at which the slightest shock is enough for an individual to expire. In theory, this limit should therefore be viewed as biological and thus one that is the same in all countries and does not change over time. If one knew what a reasonable value of \bar{d} is one could calibrate an average “time until death” in the labor force, for any particular country, as the amount of time required to get from an observed level of average deficits to \bar{d} . Yet, as we do not know \bar{d} we cannot do so in a reasonable way. What is possible, however, is to explore the *change* in time until death between two points in time. Say, between 1990 and 2015. In this case \bar{d} is differenced away due to the assumption that μ is constant across time. Hence, in order to explore the link between changes in physiological age (i.e., changes in time until death) and changes in chronological age, we begin by operationalizing this approach in our 161 country sample, assuming $\mu = 0.025$. Appendix B lays out this argument more formally, and also details our robustness checks to which we turn below.

In an effort to explore the link between physiological age and chronological age in a systematic way we run a set of exploratory regressions. Specifically, consider the following model where countries are indexed by c :

$$a_{ct}^p = \theta_c + \theta_t + \beta a_{ct}^c + u_{ct},$$

where a_{ct}^p is average physiological age of the labor force in country c at time t , θ_c and θ_t is a country fixed effect and time fixed effect, respectively, and a_{ct}^c is the average chronological age of the labor force in country c at time t . Finally, u_{ct} is a noise term. As we only have data on the *change* in physiological age between 1990 and 2015 we estimate the first differenced version:

$$\Delta a_{ct}^p = \Delta \theta + \beta \Delta a_{ct}^c + \Delta u_{ct}.$$

Hence, the constant term in the first difference specification reflects the influence from the time fixed effects. Accordingly, a negative constant indicates that the link between physiological age and chronological age has been shifting down over time. To this basic specification we also explore how the results are affected by adding a control for (log) GDP per capita, which in the first difference setting means linking changes in physiological age to growth in GDP per capita.

Table 3

¹⁷For direct evidence of the existence of such an upper boundary for individuals, see Rockwood et al. (2010).

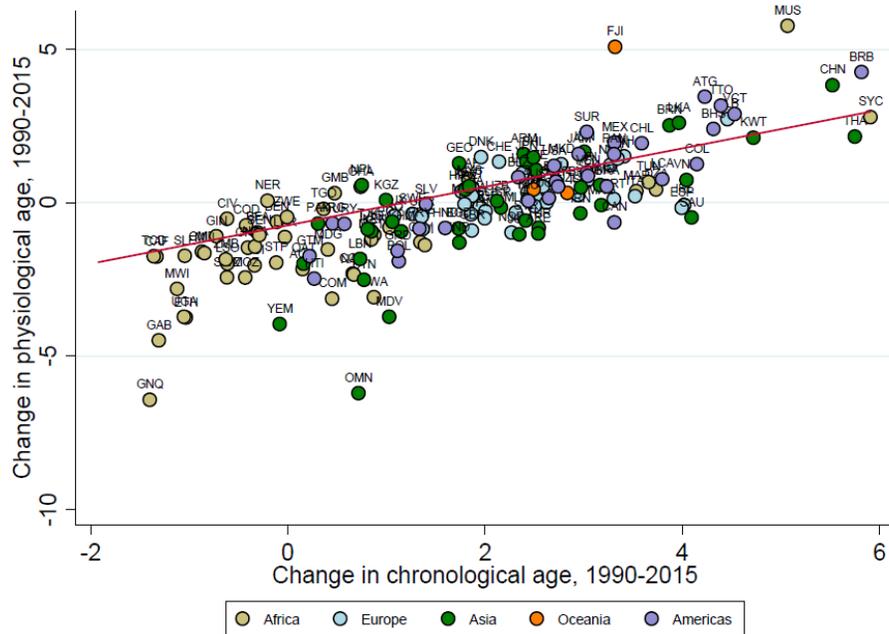


FIGURE 6. Changes in physiological and chronological age across the world, 1990-2015.
 Note: The regression line corresponds to the results reported in Table 3, column 1.

Table 3 reports the results: In column 1 we estimate the model on our full sample of 161 countries. The first thing to note is that when average chronological age in the labor force increases, so does average physiological age. However, the increase does not occur on a one for one basis: each additional year of chronological age is associated with 0.85 years in physiological terms. The second observation is that the link between chronological age and physiological age shifts *down* over time, as evidenced by the constant term (reflecting $\Delta\theta$). The point estimate suggests physiological age in 2015 is about 73% lower than in 1990, *conditional* on chronological age. Since the change occurs over a 25 year period this amounts to an annual reduction of about two percent per year ($=\ln(1.73)/25$). Figure 6 illustrates the bivariate correlation within the world sample.

The next four columns re-estimate the baseline model for each continent. As is clear this does not lead to new insights from a qualitative angle. Quantitatively, however, there are clear differences. First, the link between a^p and a^c appears weaker within Europe than within the remaining continents. Naturally, β is estimated somewhat less precisely, which means that the differences are less glaring if judged by the 95% confidence interval. For example, within Europe we find $\beta \in (.04, .83)$ compared with, say, Asia where the 95% CI is $\beta \in (.59, 1.23)$. Nevertheless, the slope is markedly flatter within Europe than anywhere else. Second, the downward shifts

also vary in magnitude, being greatest in Asia and smallest within Europe, with the remaining continents in between. Insofar as these shifts are (at least in part) of a technological nature, it seems worthwhile to understand their origins. For example, do the larger time fixed effects in Asia than in Europe reflect adoption of medical technology? Exploring such issues is, however, beyond the scope of the present analysis.

Finally, in the remaining five columns we introduce growth in GDP per capita as a separate control. Interestingly, whereas GDP per capita does not seem to influence changes in physiological aging within Europe and the Americas, it does work to shift down the link between a^p and a^c on the remaining continents. Notice also, that controlling for income does not change the significance of the time dummies.

These findings illuminate our overall finding from Figure 5. While the demographic structure shifts in favor of groups with higher chronological age, there has been a strong tendency for deficits to decline *within* age-groups, as evidenced by the significance of the constant terms in the regression models above (i.e., the time fixed effects). In the end, these two broad trends have more or less canceled out, producing the result that the average physiological age of the global labor force has remained fairly constant over the last quarter of a century (technically reflected in essentially zero change in log deficits).

As mentioned above, the assumption that the growth rate in deficits across the life cycle is the same in all countries is demonstrably false (cf. Figure 4). Indeed, the growth rate varies systematically with the level of deficits: in places where deficits are high in the 20-24 age-group, μ is lower across the life cycle. This is the compensating effect discussed in the last section. It is thus a natural question to raise if the results above change if the simplifying assumption of a common μ is relaxed.

Appendix B explains how we calibrate a country specific μ . We show, as expected, that the resulting growth rates are strongly and negatively correlated with the level of average deficits. Moreover, we show that the calibrated growth rate of average deficits is in fact approximately constant over time, within countries. After replacing the assumed growth rate of $\mu = 0.025$ with country specific counterparts we redo the analysis above. In the end, the results are very similar to those reported above; see Appendix B for details.

4. THE IMPACT OF PHYSIOLOGICAL AGING ON GROWTH

4.1. Empirical Strategy. In principle, aging may influence growth through a range of channels, both directly (e.g. via human capital) or indirectly (via capital accumulation or technology). For example, a link to human capital could emerge due to a link between aging and experience-based learning, or simply because of a direct “vitality” effect on productivity. In the empirical analysis, we focus on the *reduced-form* impact of physiological aging on economic growth leaving it for future research to explore channels.

To this end, we explore the link between average health deficits, d_{ct} , on GDP per person in the labor force (15-65):

$$\ln y_{ct} = \theta_c + \theta_t + \beta \ln d_{ct} + \mathbf{X}'_{ct} \boldsymbol{\gamma} + \epsilon_{ct}, \quad (3)$$

where θ_c denotes country fixed effect, θ_t denotes time fixed effects, and \mathbf{X}'_{ct} contains, e.g., demographic controls. Specifically, to control for the demographic structure of the labor force, we partition it into ten year age-groups. That is, the demographic controls are (the log of the) fraction of population between 15-24, 25-34, ..., 55-64. Moreover, we also control for log median age in the population. We note that when deploying the baseline demographic controls, the omitted age-groups are the “dependent population” (fraction below 14 and above 65), which therefore constitute the reference group. As an alternative control strategy, we also explore a specification controlling for the youth dependency ratio (fraction below 14), and a variant of the old-age dependency ratio: the fraction of the population above 50 relative to the size of the labor force in the age interval 20-50 following Acemoglu and Restrepo (2017). Aside from demographics, we allow for differences in time fixed effects between the OECD and non-OECD area, continents and regions, respectively, which allows for area specific growth trends. Finally, we demonstrate that our results are robust to controlling for initial (1990) GDP per worker interacted with time-dummies, so as to allow for convergence effects.

The model that we estimate is the first differenced version of equation (3):

$$\Delta \ln y_{ct} = \Delta \theta + \beta \Delta \ln d_{ct} + \Delta \mathbf{X}'_{ct} \boldsymbol{\gamma} + \Delta \epsilon_{ct}, \quad (4)$$

where we focus on the full period available, 1990-2015. As a robustness check, we also explore a shorter observation window, 1990-2000. The motivation is to check whether the time period matters, but also to be able to examine a period prior to the Great Recession (with some

margin). In addition, we also estimate specifications where all time information is exploited (i.e., a five-year panel using the years 1990, 1995, ..., 2015).

It is clear that OLS estimation does not necessarily reveal a causal influence of (growth in) d on (growth of) labor productivity as productivity likely influences the path of average deficits (see Dalgaard and Strulik, 2014). As a result, we also provide IV results. The identification strategy is discussed below along with our results.

4.2. Baseline Results. Table 4 reports the results of estimating equation (4) for the period 1990-2015. In the first five columns, we explore the link between aging and labor productivity with the baseline demographic controls, and in the last four columns, we add median age. p -values are reported in parenthesis. Moving across the columns, we control for differences in trend growth. In the first four columns, trend growth is allowed to differ between, respectively, the OECD/Non-OECD area, between continents and regions. Column 5 allows for convergence effects.

Table 4

In all settings, we find that the average health deficit index is negatively correlated with labor productivity. The point estimate suggests that a change in the average health deficit index of one percent is associated with about six percent lower labor productivity.

Table 5 employs our alternative demographic controls. Otherwise, the testing strategy is the same as in Table 4.

Table 5

Consistent with the findings of Acemoglu and Restrepo (2017), we find that increasing the fraction of older people in the population, relatively to the younger age groups in the labor force is associated with higher productivity.¹⁸ The youth dependency ratio is generally insignificant. In terms of our variable of primary interest – average deficits – the economic and statistical significance are very similar to that reported in Table 4.

As mentioned above, we report additional robustness checks in the appendix. In particular, we explore a shorter observation window, 1990-2000, and we also report the results from estimating equation (3). The results are reported in Table A4 and A5. While the panel estimates are very

¹⁸The authors argue this is caused by automation in production.

similar to those reported in Table 4 and 5, economically and statistically, the point estimates are somewhat larger when focusing on the short first difference.

Overall, the results indicate that average physiological aging is negatively associated with labor productivity. However, these results are solely partial correlations. In the next section we discuss our identification strategy.

4.3. Identification Strategy. A potential concern with the results above is reverse causality. To see the issue more clearly, observe that average deficits in the labor force at a given point in time comprise multiple cohorts. For example, in the 1990 average deficit number for people between 20 and 65, we have cohorts who turned 20 between 1945 and 1990; in the 2015 counterpart they turned 20 between 1970 and 2015. The change in the average deficit index between 1990 and 2015 can therefore be fully accounted for by three types of changes over the period.

First, the change in deficits within cohorts present in the 20-65 age-group throughout the period. That means in practise people who turned 20 between 1970 and 1990. Faster growth in deficits over the life cycle will work to increase growth in average deficits. Second, the change in deficits between age-groups that are caused by a changing demographic structure of the 20-65 age-group between 1990 and 2015. Demographic shifts towards older age-groups will work stimulate growth in average deficits. Third, the entry and exit of age groups. In practise, the exit of age-groups that turned 20 between 1945 and 1970, and the entry of cohorts that turn 20 in 1995 and later. As younger age-groups are healthier than older age-groups this process will work to dampen growth in average deficits.

The entry/exit dynamics is a key reason why simultaneity arises. When labor productivity goes up it implies that lifetime income of “new” cohorts increases relative to previous ones, which in theory implies that these individuals invest more in deficit reduction (Dalgaard and Strulik, 2014). Faster growth in labor productivity will therefore affect the evolution of average deficits, as well as possibly being affected by average deficits. In contrast, the *within* cohort variation should be unaffected by current income since it is determined by cohort specific lifetime income. In practise, of course, it is hard to rule out that also within cohort investments are affected by changes in current income since people do not necessarily pursue an optimal (control based) plan for aging and death.

As discussed in Section 2, however, deficit accumulation *within* cohorts abide by a compensation law: an initially high level of deficits for a cohort is associated with a lower growth rate of deficits across the life cycle. We propose to exploit this regularity in the following way.

The compensation effect of deficit accumulation implies for cohort k that

$$\Delta \ln d_{kt} \propto \eta \ln d_{k0},$$

with $\eta < 0$ and d_{k0} being initial (age 20) deficits for cohort k . Since initial deficits of a cohort in 1990 are unaffected by growth in labor productivity between 1990 and 2015, d_{k0} is a candidate instrument for changes in average deficits between 1990 and 2015. Formally, the reason why d_{k0} is a viable instrument for average deficits is that it explains the growth in deficits *within* cohorts and therefore in average deficits, as mentioned above.

More concretely, consider people who are in the 20-24 and the 35-39 age-group in 1990, respectively. By 2015 they are 45-49 and 60-64, respectively. Taken together they therefore span the full range of age-groups (20-64) within the sample period, 1990-2025. For the group that is 20-24 year old in 1990, d_{k0} is the average deficit index for that age group in 1990. For the 35-39 year old, d_{k0} would be deficits when they were 20-24, which requires data being available outside our observation window (i.e., for 1975). Fortunately, since deficits grow at a constant rate across the life cycle the deficit index for the 35-39 year-old in 1990 is *proportional* to deficits for that age group when they are 20-24 in 1975.

Accordingly, we propose to instrument $\Delta \ln d_t$ with the (log) level of deficits for the 20-24 age-group and the 35-39 age-group in 1990, respectively, so as to attain identification solely through cohort specific deficit accumulation. The initial deficits are in turn determined by conditions at birth and pre 1990 investments (Dalgaard, Hansen and Strulik, 2017). As a result, this strategy should allow us to deal with simultaneity.¹⁹ In a way, this strategy resemblances the more traditional shift-share instrumental-variable approach, which is followed in papers such as Bleakley (2007) and Acemoglu and Johnson (2007). However, while these typically papers exploit human interventions, we leverage a well-documented biological mechanism.

¹⁹With two instruments, deficits for 20-24 year-olds and 35-39 year-olds in 1990, respectively, overidentification tests are viable.

It is worth stressing that since deficit levels in 1990 are likely correlated with the level of productivity in 1990 we follow e.g. Acemoglu and Restrepo (2017) and control for initial productivity interacted with time dummies so as to avoid jeopardizing the exclusion restriction on a prior grounds (i.e., due to the expectation of convergence effects being present).

Finally, observe that while we have focused on the problem of simultaneity in this section, the IV strategy we have described could also deal with potential omitted variables bias. A valid concern is that time-varying factors that impact on growth may be correlated with the evolution of health deficits, which leads to a biased estimate of physiological aging on labor productivity. But shocks to growth between 1990 and 2015 are unlikely to affect initial deficits in 1990. Accordingly, the identification strategy should also address concerns regarding omitted variables.

4.4. IV Results. Table 6 presents our IV estimates, where deficits for 20-24 year-old and 35-39 year-old in 1990, respectively, are used as excluded instruments for growth in average deficits. As is clear the economic significance of aging rises considerably compared with the OLS setting. Based on the Anderson-Rubin test, which is robust to weak instruments, it is clear that average health deficits are statistically significant in explaining economic growth. Also, in every instance, we are unable to reject the exclusion restriction that our instruments are irrelevant for growth, conditional on our controls.

Table 6

The point estimate suggests that an increase in average health deficits by one percent, conditional on the demographic structure of the labor force, reduces labor productivity by about 15 percent.²⁰ In order to assess the economic significance of these results, it is worth recalling that since deficits grow by about 2 percent per year across the life cycle, an increase in the average deficit index by one percent can be thought of as an experiment by which the average physiological age of the labor force is increased by six months. In the interest of completeness, Table A6 reports the results allowing for the alternative set of demographic controls; the results are statistically and economically similar.

²⁰This estimate is about twice the size of the OLS estimate. If the OLS estimates are influenced by simultaneity bias, the interpretation is that the “true” impact of deficits on growth is much stronger than the impact of growth on deficits, since OLS (in theory) produces an estimate that is “in between” the two slopes. Naturally, if omitted variables biases the OLS result the IV results suggests deficits are correlated with (time varying) factors that serve to increase growth.

Overall, these results suggest a potentially substantial negative causal impact from physiological aging on labor productivity. In practise, however, it is worth noting that from 1990 to 2015 the health deficit index declined in our 160 country sample by only 0.02 percent, which suggests a productivity stimuli of about 0.3 percent. Hence, while physiological aging does seem to hold a significant influence on labor productivity, its impact over the preceding quarter of a century has been modest according to our estimates.

5. CONCLUDING REMARKS

The present study makes three contributions. First, we provide world-wide data capturing the physiological aging of nations for the period 1990 to 2015. Our measure of aging, the health deficit index, is grounded in the literature on aging within biology and medicine. We show how the health deficits index can be aggregated to the country level in a straightforward way, and that the resulting aggregate index displays key regularities that have been documented for the disaggregate counterparts in the existing literature.

Second, we examine the evolution of physiological aging for the average person in the labor force across the world. An interesting insight is that while the labor force is growing chronologically older, the world it is not on average growing older physiologically as measured by health deficits. This regularity is found within every continent. Some exploratory regression results indicate that this result is due to the fact that the link between physiological age (as defined in the text) and chronological age has been shifting down over time, which possibly reflects health innovations broadly defined. In some parts of the world, rising prosperity possibly has had a similar effect. Accordingly, these processes have worked to counteract the demographic movement towards (chronologically) older age-intervals that generally has been observed across the world. Moreover, we find that global disparities in the health deficit index have remained fairly constant over the preceding quarter of a century.

Third, we use the data to explore the link between physiological aging and labor productivity growth. While the existing literature has investigated the link seen through the lens of demographic shifts, the present study explicitly focuses on the impact of physiological aging, as measured by average deficits in the labor force, conditional on demographics. Our key finding is that physiological aging lowers labor productivity. Our IV estimates suggest that the impact is causal and economically significant. Taken together, our findings suggest that if economic

growth has been declining over the past quarter of a century, physiological aging is unlikely to be part of an explanation in light of its relative constancy over time.

APPENDIX

A. DATA

Health deficit index. The health deficit index is based on prevalence rates for the following diseases (45 in total): Vitamin A deficiency; Esophageal cancer; Stomach cancer; Colon and rectum cancer; Liver cancer; Gallbladder and biliary tract cancer; Pancreatic cancer; Tracheal, bronchus, and lung cancer; Ovarian cancer; Prostate cancer; Kidney cancer; Bladder cancer; Brain and nervous system cancer; Thyroid cancer ; Ischemic heart disease; Cerebrovascular disease; Hypertensive heart disease; Endocarditis; Atrial fibrillation and flutter; Chronic kidney disease; Asthma; Alzheimer disease and other dementias; Parkinson disease; Migraine; Tension-type headache; Other neurological disorders; Schizophrenia; Bipolar disorder; Diabetes mellitus; Osteoarthritis; Low back and neck pain; Sense organ diseases; Rheumatoid arthritis; Larynx cancer; Malignant skin melanoma; Mesothelioma; Leukemia; Cardiomyopathy and myocarditis; Aortic aneurysm; Peripheral artery disease; Other cardiovascular and circulatory diseases; Peptic ulcer disease; Gastritis and duodenitis; Pancreatitis; Other musculoskeletal disorders. The database is being continually updated and expanded and can be accessed via <http://ghdx.healthdata.org/gbd-results-tool>

The aggregate health deficit index is the average of the above listed prevalence rates. To calculate age-group specific deficit indices we rely in the age-specific prevalence rates, but otherwise the procedure is the same, and when we use deficit indices by gender they are calculated using gender-specific prevalence rates.

Other data sources.

- Output side real GDP at chained PPPs and GDP per capita (2011 US\$), from the Penn World Tables, v. 9 (Feenstra et al., 2015). Available at <https://www.rug.nl/ggdc/productivity/pwt/>
- Population shares of various age-groups and median age in the population overall are from United Nations. Available from <https://esa.un.org/unpd/wpp/DataQuery/>

B. PHYSIOLOGICAL AGING OF THE LABOR FORCE

In keeping with the simplified deficit law explored in Section 2.3, assume

$$d_t = (1 + \mu)^t d_{t-1}$$

We will use this approximation to go from (log) deficits to "physiological age" concepts, to begin with. More specifically, we will work with

$$\ln d_t = \mu(t - s) + \ln d_{t-s} \tag{A.1}$$

A relevant metric for "physiological age" at a point in time is *distance from death in years*, τ^d . This we can calculate if we are willing to assume that deficits have an upper boundary, $\bar{d} < 1$. Direct evidence that there exist an upper bound, for individuals, is found in Rockwood et al. (2010). Conceptually, a labor force is then more aged than another if τ^d is relatively lower.

More specifically, distance from death in years in country c at time s is:

$$\ln \bar{d} = \mu \tau_{cs}^d + \ln d_{cs}$$

or equivalently

$$\frac{\ln \bar{d} - \ln d_{cs}}{\mu} = \tau_{cs}^d. \quad (\text{A.2})$$

The **change in physiological age** for a particular country c between time $t+s$ relative to time t is defined as:

$$\tau_{ct}^d - \tau_{ct+s}^d = \frac{\ln \bar{d} - \ln d_{ct}}{\mu} - \frac{\ln \bar{d} - \ln d_{ct+s}}{\mu} = \frac{\ln d_{ct+1} - \ln d_{ct}}{\mu}$$

Hence, the change in physiological age captures the change in “years until death”. If $\tau_{ct}^d - \tau_{ct+s}^d > 0$ it implies that distance to death is shorter at $t+s$ than t , which means a physiologically older population. In the text above we calculate $\frac{\ln d_{2015} - \ln d_{1990}}{\mu}$ using $\mu = 0.025$; Figure 6 compares changes in physiological age to the change in chronological age (average age in labor force) between 1990 and 2015.

The calculation above assumes that the growth in deficits *is the same* in all countries. In practise, this is not true, as seen in Figure 4. In fact, growth rates tend to vary in a systematic way, reflecting a compensation effect: initially high deficits are associated with slower growth in deficits. As a result, one may legitimately be concerned that the results discussed in the paper are sensitive to the “constant across-countries μ ” simplification. As a robustness check we therefore allow μ to differ *between* countries while assuming it is constant *within* countries over time. In order to calculate the growth rate, we proceed in the following way.

As documented in Figure 4 deficits converge around the age of 50 between countries. Denote by \tilde{d} the level of deficits at this “compression point” (in practise, $\ln \tilde{d} \approx .5$, as can be seen). Denote by d_{acs} the level of deficits in age-group $a < 50$, in country c at time s . Then the country specific annual growth rate of deficits can be calculated as

$$\frac{\ln \tilde{d} - \ln d_{acs}}{50 - a} = \mu_c.$$

In particular, choosing the age-group 35-39 we calibrate the annual growth rate by applying average deficits for 35-39 year-olds $\ln d_{35-39cs}$ and dividing by 50-37 in the year $s=2015$. In order to calculate changes in physiological aging we simply replace μ by μ_c in the formula above, yielding:

$$\tau_{ct}^d - \tau_{ct+s}^d = \frac{\ln d_{ct+1} - \ln d_{ct}}{\mu_c}.$$

In the Supplementary Appendix we provide the results that correspond to those reported in the text above, which pertain to the simple case of invariant μ across countries. We also provide evidence that albeit μ in practice does vary across countries, in the expected systematic way, the assumption that it is constant *across time* within countries (at least over the time horizon considered here) is a reasonable approximation. Δ

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Supplementary Appendix to: Physiological Aging around the World and Economic Growth

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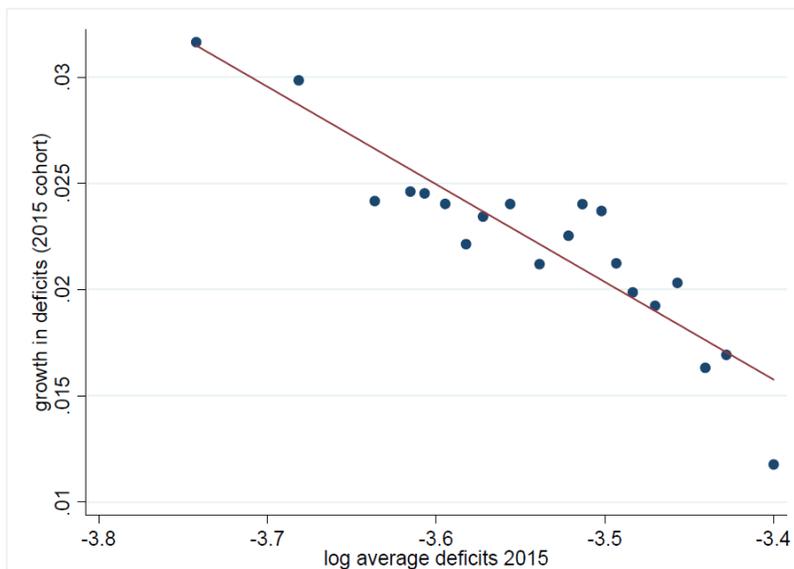


FIGURE 1. The figure shows a binned scatterplot of average growth rate in deficits, calculated using data on average deficits for 35-39 year-olds in 2015 vs log average deficits in the labor force in 2015. Notes: (i) The underlying data involves 161 country observations. (ii) by the label "cohort" we refer to the fact that the growth rate pertains to the five-year cohort that is 34-39 in 2015

1. CHANGES IN PHYSIOLOGICAL AGING: ROBUSTNESS

As discussed in Appendix B of the paper it is possible to calibrate growth in deficits across the life cycle country-by-country. Figure 1 provides a visualization of the link between the resulting growth rate and the level of average deficits across 161 countries. As can be seen, there is a strong negative link in keeping with the compensating effect in deficits, discussed in Section 2 of the paper. As we also discuss in Appendix B of the paper, in order to calculate the change in physiological age over time within countries we need to assume that the growth rate of deficits across the life cycle is fairly constant over time, within countries. Figure 2 shows the correlation between the calibrated growth rate in deficits for two different cohorts: The cohort that is in the age-interval 35-39 in 2015 and 1990, respectively, meaning they enter into the labor force between 1950 and 1975. Recall, the reason why we focus on this age-group is two fold: (i) our cohort analysis in Section 2 of the paper – which involves following people from 34–39 to 60-64 – demonstrates that average growth in deficits varies across countries in a systematic way consistent with compression (see Figure 6 in the paper); (ii) the compression point occurs around the age of 50. Hence, the calibrated growth rates are made in the vicinity of the compression point. As can be seen from the figure there is a very strong correlation

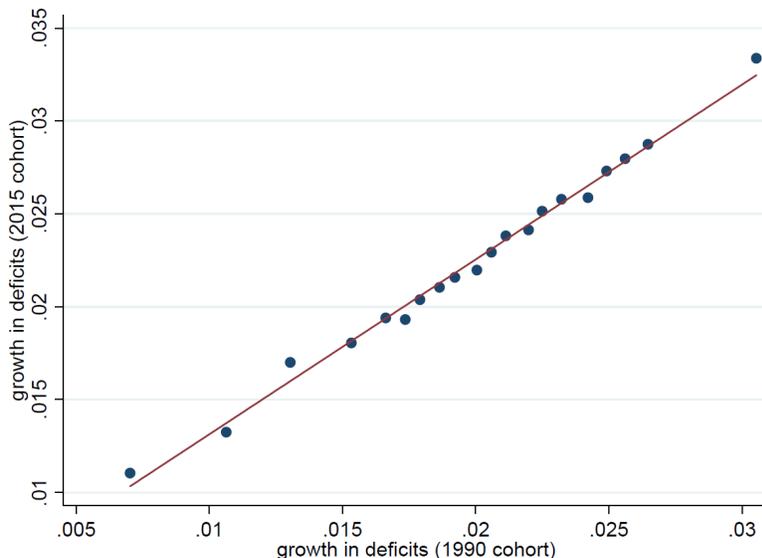


FIGURE 2. The figure shows a binned scatterplot of average growth rate in deficits, calculated using data on average deficits for 35-39 year-olds in 1990 and in 2015, respectively. Notes: (i) The underlying data involves 161 country observations. (ii) by the label "cohort" we refer to the fact that the growth rate pertains to the cohort that is 35-39 in 2015 and 1990, respectively.

between the two series. The simple correlation, using all cross-country observations, is 0.95. A simple bivariate regression returns a slope estimate of .94. This suggests that the simplifying assumption that μ is constant across time (yet varies across countries) is a fairly reasonable one.

In Tables 3A we provide exploratory regression results that correspond to those reported in Table 3 in the paper.

Table 3A

As can be seen the results are qualitatively similar, with one major exception: within the Asian and Americas samples the coefficient for changes in average chronological age *exceeds* 1. At the same time, of course, the time fixed effects suggests the link between a^p and a^c has been shifting down over time to a considerable extent. Hence, if one were to ignore the constant term (i.e., ignoring the time fixed effects), the coefficient shrinks well below one in both samples (not shown, but available upon request). Hence, it remains true that as average chronological age has been rising across the world, physiological age has changed to a much lesser extent. But the finding also suggests that the processes responsible for the time fixed effects – possibly innovations – is crucial in ensuring slower deficit accumulation and thus longer lives. This insight mirrors, albeit is distinct from, Samuel Prestons (1975) finding that the link between income

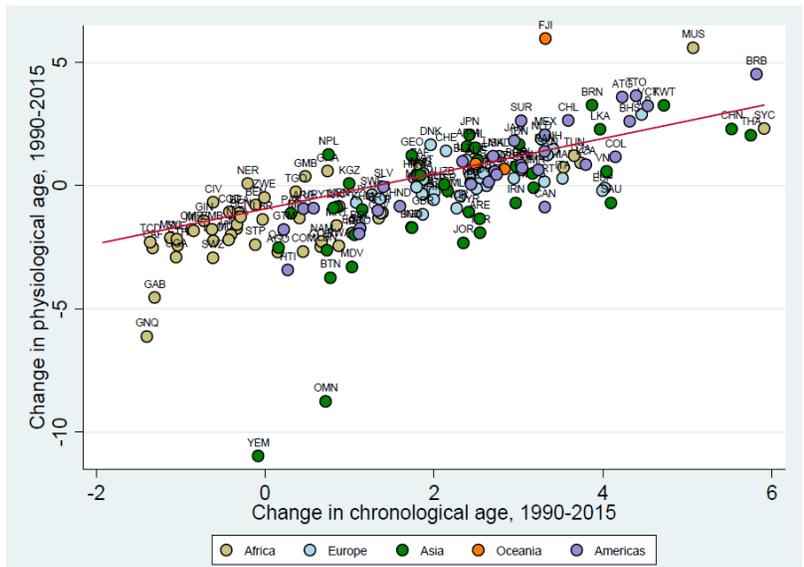


FIGURE 3. Changes in physiological and chronological age across the world, 1990-2015.
 Note: The regression line corresponds to the results reported in Table 3A, column 1.

and life expectancy has been shifting up over time, possibly due to innovative activity, and that this process is the main driver of rising longevity.¹

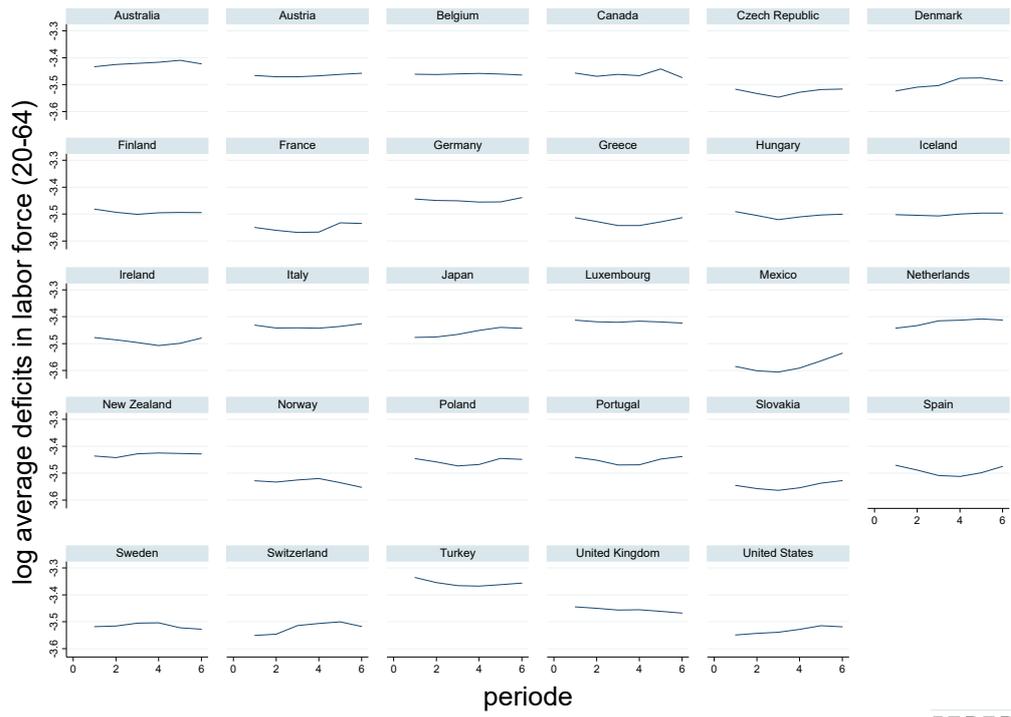
In the interest of completeness Figure 3 depicts the partial correlation, corresponding to Figure 6 in the paper. As can be seen the results are fairly similar.

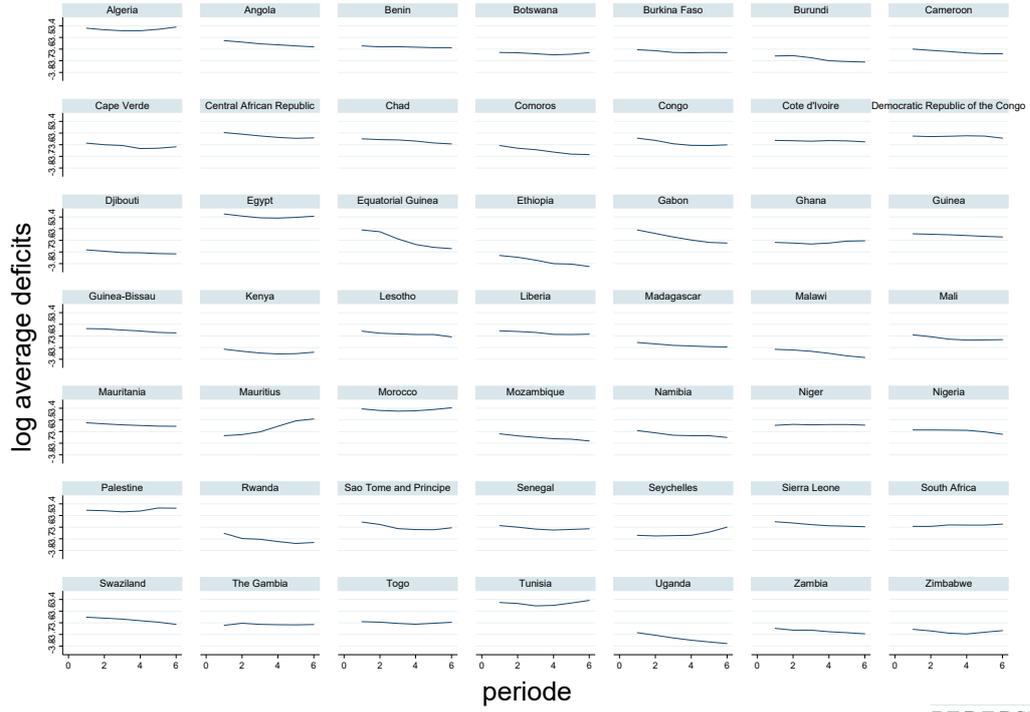
2. SUPPLEMENTARY EMPIRICAL RESULTS

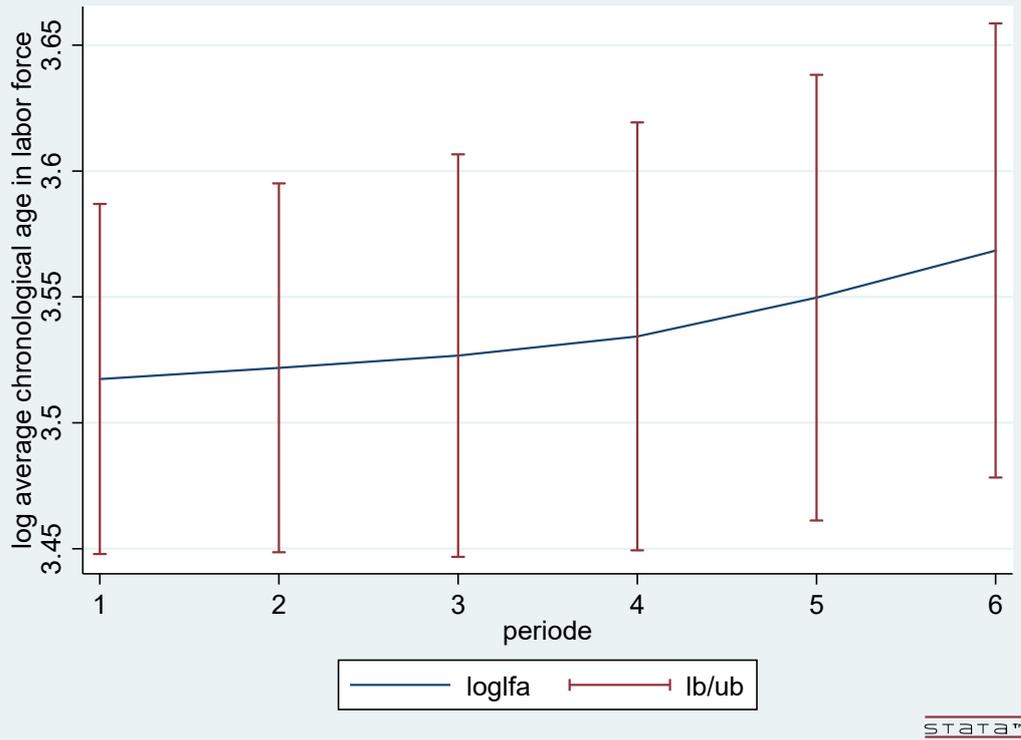
Content:

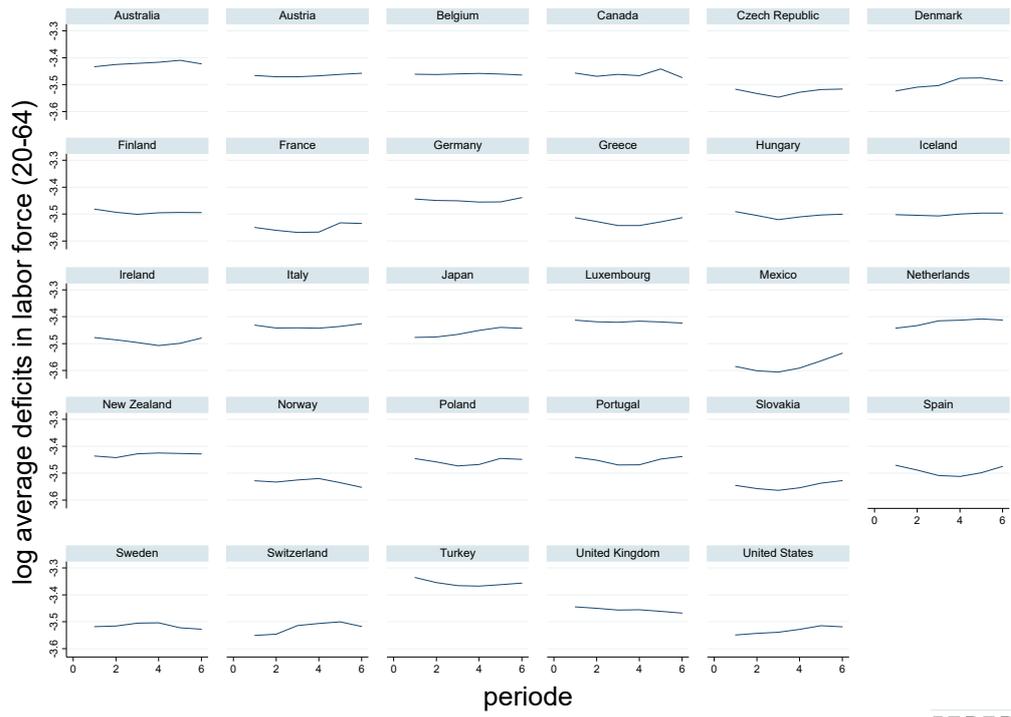
- Country trajectories for OECD countries and African countries in log average health deficits
- Time path of log average chronological age and its standard deviation 1990-2015.
- Tables A1-A6.

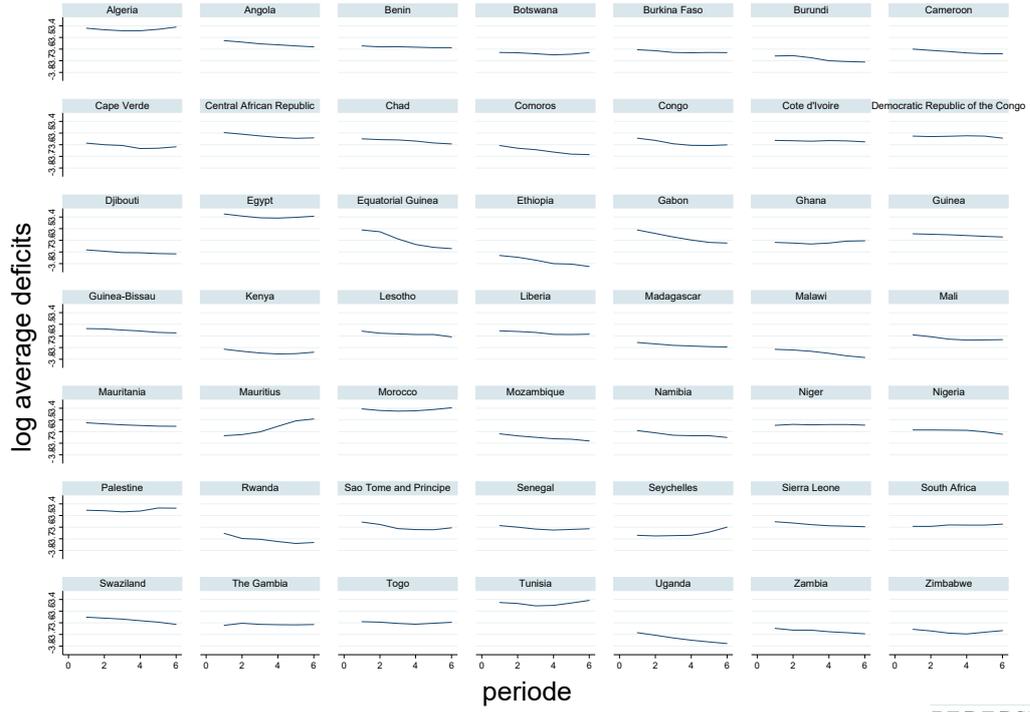
¹Preston, S. H. (1975). The changing relation between mortality and level of economic development. *Population studies*, 29(2), 231-248.











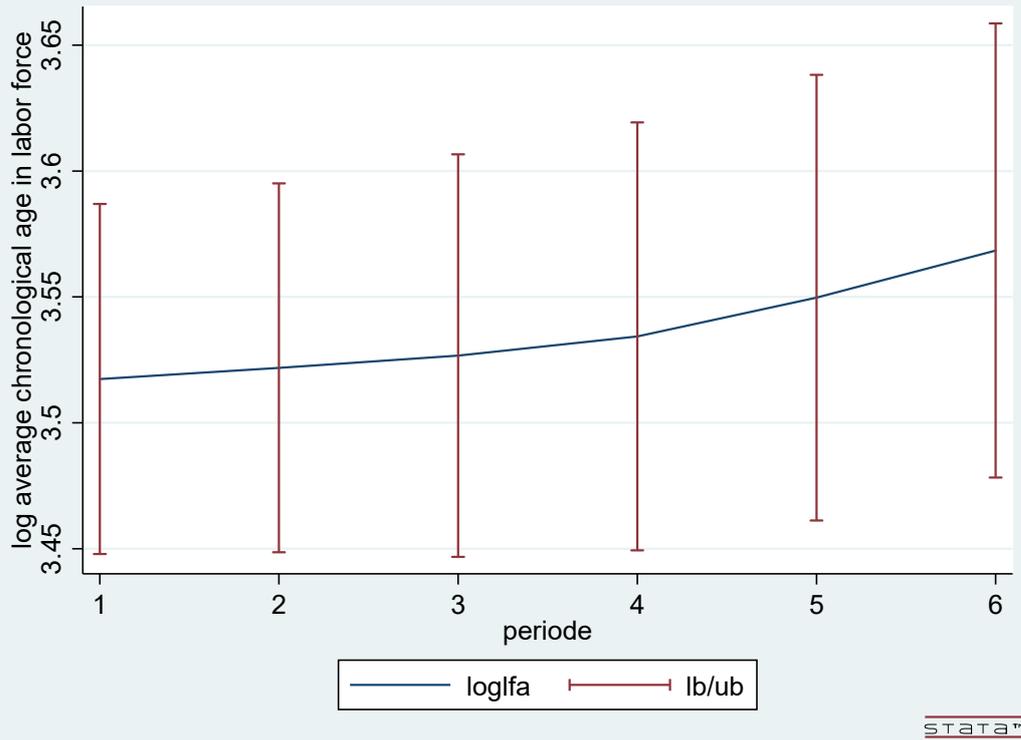


Table 1: Age and Health Deficits, using age-period data (both sexes)

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Log Deficits					
Age	0.111 (0.000)	0.102 (0.000)	0.106 (0.000)	0.110 (0.000)	0.110 (0.000)	0.114 (0.000)
Country FE	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	OECD	Europe	America	Asia	Africa
R-squared	0.987	0.974	0.983	0.988	0.989	0.994
Observations	13,824	2,160	2,808	2,736	3,096	3,816

P-values in parentheses

Table 2: Age and Health Deficits, using age-period data (by sexes)

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Log Deficits					
Age x Female Indicator	0.0981 (0.000)	0.0836 (0.000)	0.0903 (0.000)	0.0930 (0.000)	0.101 (0.000)	0.102 (0.000)
Age x Male Indicator	0.127 (0.000)	0.127 (0.000)	0.126 (0.000)	0.133 (0.000)	0.120 (0.000)	0.128 (0.000)
Country FE	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes
Sex FE	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	OECD	Europe	America	Asia	Africa
R-squared	0.983	0.975	0.983	0.987	0.988	0.989
Observations	27,648	4,320	5,616	5,472	6,192	7,632

P-values in parentheses

Table3: Physiological age and chronological age: 1990-2015

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	Change in physiological age									
Change in chronological age	0.854 (0)	0.435 (0.0311)	0.989 (4.25e-10)	0.843 (7.28e-07)	0.908 (1.13e-06)	0.882 (0)	0.378 (0.0625)	1.013 (2.07e-09)	0.853 (2.09e-07)	0.961 (6.61e-07)
Change in log GDP per capita						-0.672 (1.53e-05)	0.371 (0.128)	0.273 (0.357)	-0.785 (1.68e-05)	-0.804 (0.0220)
Constant	-1.737 (0)	-0.775 (0.0949)	-1.900 (5.11e-06)	-1.552 (0)	-2.118 (7.42e-05)	-1.339 (0)	-0.870 (0.0546)	-2.155 (0.000201)	-1.153 (2.87e-08)	-1.525 (0.00161)
Sample	Full	Europe	Americas	Africa	Asia	Full	Europe	Americas	Africa	Asia
Observations	161	38	32	49	42	161	38	32	49	42
R-squared	0.611	0.218	0.750	0.549	0.472	0.657	0.242	0.754	0.648	0.541

p-values in paranthesis

Table 4: Aging and labor productivity: 1990-2015

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	Change in log GDP per worker									
Change in log deficits	-4.733 (0.0572)	-4.849 (0.0598)	-4.098 (0.115)	-5.209 (0.0761)	-5.998 (0.0106)	-4.546 (0.0768)	-4.672 (0.0762)	-4.009 (0.132)	-5.052 (0.0969)	-5.884 (0.0152)
Change in log population share 15-24	-0.575 (0.185)	-0.606 (0.231)	-0.293 (0.597)	-0.458 (0.379)	-1.488 (0.000102)	-0.373 (0.484)	-0.405 (0.482)	-0.156 (0.803)	-0.316 (0.613)	-1.371 (0.00257)
Change in log population share 25-34	-0.0937 (0.856)	-0.114 (0.826)	-0.175 (0.727)	-0.307 (0.545)	0.0158 (0.969)	-0.250 (0.675)	-0.277 (0.649)	-0.312 (0.592)	-0.434 (0.476)	-0.0670 (0.894)
Change in log population share 35-44	0.236 (0.572)	0.215 (0.629)	0.424 (0.362)	0.328 (0.463)	-0.379 (0.333)	0.0992 (0.814)	0.0728 (0.872)	0.288 (0.532)	0.218 (0.628)	-0.442 (0.256)
Change in log population share 45-54	-0.155 (0.732)	-0.147 (0.750)	-0.357 (0.441)	-0.301 (0.520)	0.356 (0.387)	-0.252 (0.592)	-0.245 (0.608)	-0.428 (0.367)	-0.365 (0.454)	0.299 (0.485)
Change in log population share 55-64	0.769 (0.0485)	0.766 (0.0506)	0.703 (0.0786)	0.601 (0.183)	0.858 (0.00913)	0.538 (0.269)	0.530 (0.281)	0.526 (0.287)	0.440 (0.427)	0.737 (0.0795)
log GDP per worker 1990					-0.280 (6.78e-05)					-0.276 (5.32e-05)
Change in log median age						0.956 (0.378)	0.971 (0.375)	0.790 (0.463)	0.709 (0.549)	0.497 (0.585)
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time x										
OECD	No	Yes	No	No	No	No	Yes	No	No	No
Continents	No	No	Yes	No	No	No	No	Yes	No	No
Regions	No	No	No	Yes	No	No	No	No	Yes	No
R-squared	0.511	0.511	0.523	0.535	0.596	0.516	0.516	0.526	0.537	0.597
Observations	161	160	160	160	161	161	160	160	160	161

Table 5: Aging and labor productivity: 1990-2015 (alt. demographic controls)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	Change in log GDP per worker									
Change in log deficits	-4.271 (0.0578)	-4.371 (0.0611)	-4.058 (0.0831)	-4.597 (0.0691)	-4.654 (0.0304)	-4.807 (0.0363)	-4.902 (0.0392)	-4.387 (0.0617)	-4.932 (0.0568)	-5.026 (0.0221)
Change in log ratio of old to young workers	0.620 (0.00966)	0.658 (0.0191)	0.523 (0.0554)	0.532 (0.0733)	1.071 (0.000147)	0.626 (0.00954)	0.663 (0.0190)	0.478 (0.0812)	0.539 (0.0726)	1.053 (0.000201)
Change in log youth dependency ratio	-0.416 (0.155)	-0.383 (0.219)	-0.138 (0.671)	-0.0924 (0.788)	-0.582 (0.0280)	0.320 (0.581)	0.357 (0.552)	0.587 (0.312)	0.405 (0.471)	-0.0358 (0.947)
Log GDP per worker 1990					-0.214 (0.000312)					-0.204 (0.000377)
Change in log median age						1.414 (0.0759)	1.420 (0.0764)	1.480 (0.0786)	1.078 (0.204)	1.033 (0.170)
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time x										
OECD	No	Yes	No	No	No	No	Yes	No	No	No
Continents	No	No	Yes	No	No	No	No	Yes	No	No
Regions	No	No	No	Yes	No	No	No	No	Yes	No
R-squared	0.504	0.504	0.517	0.530	0.566	0.519	0.519	0.531	0.537	0.574
Observations	161	160	160	160	161	161	160	160	160	161

Table 6: Aging and labor productivity - IV estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
VARIABLES	Change in log GDP per worker							
Change in log deficits	-18.50 (0.000197)	-16.62 (0.00137)	-12.00 (0.00319)	-14.52 (0.00642)	-18.48 (0.000254)	-16.60 (0.00173)	-12.15 (0.00334)	-14.77 (0.00628)
Change in log median age					-0.0217 (0.983)	-0.0407 (0.966)	-0.387 (0.667)	-0.524 (0.605)
Age group controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time x								
log GDP per worker 1990	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
OECD	No	Yes	No	No	No	Yes	No	No
Continents	No	No	Yes	No	No	No	Yes	No
Regions	No	No	No	Yes	No	No	No	Yes
Anderson-Rubin test (p-value)	0.0000	0.0000	0.0070	0.0114	0.0000	0.0001	0.0086	0.0129
OID test (p-value)	0.2349	0.1746	0.3546	0.5242	0.2333	0.1733	0.3479	0.5357
R-squared	0.460	0.502	0.593	0.562	0.460	0.502	0.592	0.559
Observations	161	160	160	160	161	160	160	160

Table A1: Age and Health Deficits, using cohort data (both sexes)

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Log Deficits					
Age	0.108 (0.000)	0.102 (0.000)	0.106 (0.000)	0.105 (0.000)	0.108 (0.000)	0.109 (0.000)
Country FE	Yes	Yes	Yes	Yes	Yes	Yes
Cohort FE	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	OECD	Europe	America	Asia	Africa
R-squared	0.984	0.971	0.981	0.984	0.985	0.994
Observations	4,608	720	936	912	1,032	1,272

Cohorts followed over time are individuals in the age groups: 35-39, 40-44, 45-49, and 50-54 in 1990.

P-values in parentheses

Table A2: Age and Health Deficits, using cohort data (by sex)

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Log Deficits					
Age x Female Indicator	0.0921 (0.000)	0.0797 (0.000)	0.0876 (0.000)	0.0838 (0.000)	0.0955 (0.000)	0.0937 (0.000)
Age x Male Indicator	0.129 (0.000)	0.127 (0.000)	0.130 (0.000)	0.120 (0.000)	0.127 (0.000)	0.128 (0.000)
Country FE	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes
Sex FE	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	OECD	Europe	America	Asia	Africa
R-squared	0.976	0.969	0.979	0.980	0.984	0.983
Observations	9,216	1,440	1,872	1,824	2,064	2,544

Cohorts followed over time are individuals in the age groups: 35-39, 40-44, 45-49, and 50-54 in 1990.

P-values in parentheses

Table A3: Physiological age and chronological age: 1990-2015 (country specific growth in deficits)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	Change in Physiological Age									
Change in chronological age	0.921 (0)	0.413 (0.107)	1.070 (8.29e-08)	0.862 (1.05e-07)	1.157 (0.000576)	0.960 (0)	0.349 (0.188)	1.105 (7.68e-07)	0.872 (3.13e-08)	1.258 (0.000183)
Change in log GDP per capita						-0.918 (7.79e-05)	0.410 (0.126)	0.387 (0.361)	-0.758 (6.72e-07)	-1.524 (0.0185)
Constant	-1.954 (0)	-0.688 (0.275)	-2.064 (0.000259)	-1.563 (0)	-3.050 (0.00252)	-1.410 (0)	-0.792 (0.189)	-2.426 (0.00526)	-1.178 (1.37e-09)	-1.926 (0.00895)
Sample	Full	Europe	Americas	Africa	Asia	Full	Europe	Americas	Africa	Asia
Observations	161	38	32	49	42	161	38	32	49	42
R-squared	0.512	0.151	0.681	0.587	0.352	0.575	0.174	0.687	0.681	0.465

Table A4: Aging and labor productivity: 1990-2000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	change in log GDP per worker									
Change in log deficits	-7.574 (0.00542)	-8.106 (0.0206)	-7.946 (0.00735)	-10.01 (0.00819)	-9.629 (0.00159)	-7.123 (0.00964)	-7.620 (0.0249)	-7.471 (0.0134)	-9.261 (0.0159)	-9.629 (0.00159)
Change in log population share 15-24	-1.167 (0.000294)	-1.301 (0.0163)	-1.179 (0.00177)	-1.612 (3.57e-05)	-1.708 (9.14e-05)	-1.157 (0.000210)	-1.291 (0.0137)	-1.186 (0.00168)	-1.618 (3.07e-05)	-1.708 (9.14e-05)
Change in log population share 25-34	0.155 (0.689)	0.0905 (0.848)	0.153 (0.691)	-0.516 (0.255)	-0.227 (0.580)	0.0813 (0.867)	0.00862 (0.988)	0.0362 (0.942)	-0.684 (0.221)	-0.227 (0.580)
Change in log population share 35-44	-0.338 (0.108)	-0.384 (0.122)	-0.276 (0.222)	-0.482 (0.0241)	-0.299 (0.154)	-0.507 (0.205)	-0.568 (0.209)	-0.502 (0.206)	-0.795 (0.0591)	-0.299 (0.154)
Change in log population share 45-54	0.726 (0.00167)	0.763 (0.00160)	0.796 (0.00186)	0.896 (0.00201)	1.097 (0.000193)	0.571 (0.157)	0.597 (0.124)	0.607 (0.142)	0.620 (0.142)	1.097 (0.000193)
Change in log population share 55-64	1.377 (0.000300)	1.394 (0.000592)	1.400 (0.000482)	1.287 (0.00351)	1.621 (4.51e-05)	1.254 (0.00198)	1.259 (0.00269)	1.248 (0.00335)	1.056 (0.0264)	1.621 (4.51e-05)
log GDP per worker 1990					-0.144 (0.00508)					-0.144 (0.00508)
Change in log median age						0.761 (0.641)	0.829 (0.613)	0.961 (0.557)	1.382 (0.410)	
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time x										
OECD	No	Yes	No	No	No	No	Yes	No	No	No
Continents	No	No	Yes	No	No	No	No	Yes	No	No
Regions	No	No	No	Yes	No	No	No	No	Yes	No
R-squared	0.271	0.270	0.281	0.321	0.348	0.273	0.273	0.284	0.327	0.348
Observations	161	160	160	160	161	161	160	160	160	161

Table A5: Average deficits and labor productivity: 1990-2000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
VARIABLES	log GDP per worker									
log deficits	-5.673 (0.0335)	-5.948 (0.0351)	-4.995 (0.0879)	-5.589 (0.0794)	-6.881 (0.00973)	-5.730 (0.0366)	-5.950 (0.0400)	-5.077 (0.0890)	-5.776 (0.0743)	-6.829 (0.0116)
log population share 15-24	-0.562 (0.00730)	-0.594 (0.0199)	-0.445 (0.0774)	-0.524 (0.0249)	-0.950 (8.30e-06)	-0.570 (0.00904)	-0.595 (0.0229)	-0.449 (0.0793)	-0.543 (0.0238)	-0.944 (9.82e-06)
log population share 25-34	-0.0841 (0.649)	-0.213 (0.332)	-0.0647 (0.730)	-0.113 (0.574)	-0.399 (0.0663)	-0.0756 (0.698)	-0.212 (0.363)	-0.0346 (0.860)	-0.0683 (0.749)	-0.409 (0.0758)
log population share 35-44	-0.0922 (0.571)	-0.0844 (0.623)	-0.111 (0.517)	-0.130 (0.504)	-0.221 (0.182)	-0.0687 (0.736)	-0.0832 (0.704)	-0.0525 (0.813)	-0.0324 (0.895)	-0.245 (0.217)
log population share 45-54	0.424 (0.00595)	0.364 (0.0277)	0.249 (0.103)	0.379 (0.0254)	0.605 (0.000304)	0.452 (0.0175)	0.366 (0.0746)	0.307 (0.120)	0.476 (0.0258)	0.577 (0.00335)
log population share 55-64	0.468 (0.0302)	0.507 (0.0251)	0.408 (0.106)	0.376 (0.190)	0.674 (0.00324)	0.493 (0.0521)	0.508 (0.0609)	0.456 (0.119)	0.458 (0.149)	0.650 (0.00971)
log GDP per worker 1990					-0.0793 (0.0521)					-0.0792 (0.0515)
log median age						-0.148 (0.815)	-0.00706 (0.991)	-0.317 (0.639)	-0.570 (0.428)	0.149 (0.807)
Observations	966	960	960	960	966	966	960	960	960	966
R-squared	0.429	0.435	0.452	0.455	0.480	0.429	0.435	0.452	0.457	0.480
Countries	161	160	160	160	161	161	160	160	160	161

Table A6: Aging and labor productivity - IV estimates (alt. demographic controls)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
VARIABLES	Change in log GDP per worker							
Change in log deficits	-17.32 (0.000988)	-15.36 (0.00869)	-11.01 (0.0106)	-13.26 (0.0125)	-14.94 (0.000562)	-13.22 (0.00513)	-9.169 (0.0158)	-12.21 (0.0120)
Change in log median age					1.615 (0.0368)	1.471 (0.0508)	1.465 (0.0673)	1.321 (0.130)
Change in log ratio of old to young workers	2.837 (4.36e-05)	2.538 (0.00149)	1.780 (0.00210)	1.990 (0.00526)	2.394 (1.49e-05)	2.132 (0.000633)	1.417 (0.00449)	1.793 (0.00412)
Change in log youth dependency ratio	-0.475 (0.153)	-0.611 (0.0998)	-0.240 (0.424)	-0.235 (0.497)	0.353 (0.508)	0.110 (0.845)	0.460 (0.376)	0.380 (0.456)
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time x								
log GDP per worker 1990	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
OECD	No	Yes	No	No	No	Yes	No	No
Continents	No	No	Yes	No	No	No	Yes	No
Regions	No	No	No	Yes	No	No	No	Yes
Anderson-Rubin test (p-value)	0.0000	0.0001	0.0126	0.0092	0.0001	0.0002	0.0392	0.0113
OID test (p-value)	0.1487	0.1144	0.3122	0.4639	0.1396	0.1228	0.4151	0.4424
R-squared	0.373	0.430	0.546	0.512	0.458	0.498	0.587	0.543
Observations	161	160	160	160	161	160	160	160