Economic Demography
Felipe Menares

Demographic transition is the main theory explaining the continuing population growth after the 19th century, which began in western society and then happened in parts of Asia and Latin America, until now. In the absence of migration, it started with a pre-transition period in which high fertility and high mortality rates were present - a characteristic of the Malthusian world - which led to very low population growth. In the first stage, we observed an abrupt decrease in mortality followed by a second period where fertility fell while mortality fell at a slower rate, resulting in a huge increase in population. The third phase is characterized by both a low fertility and mortality rate - resulting in population aging - which slows the rates of population growth.

The three stages of demographic transition are explained by the determinants of mortality and fertility rates, where economic and demographic models become crucial. The observed initial decrease in mortality is due to reductions in contagious and infectious diseases, that can be attributed to improvements in public health, such as hygiene and water sanitization. Regarding fertility, I will cover part of the theories explaining the observed trends in this reading list.

Economic theory enters this debate during the mid 20th century, where theories and models to explain economic growth are developed, that is how the well known Solow-Swan model appeared. This model states that the per capita income level is mainly a result of capital per capita. The model assumes that everybody works, and that savings, capital depreciation, and population growth rates are exogenous and constant. Thus, the output per capita is explained through capital deepening or dilution, which is the result of slower or faster population growth, respectively. In this model, the steady state consumption is given by the part that is not saved (invested) of the outcome per capita.

However, the Solow model lacked an age structure component, due to the homogenous population assumption. Here is where demography and economics connect with each other, explaining the role played by population in the economic growth through the demographic transition. Therefore, population can be characterized according to the age profiles, defined by either the younger or the older population divided by the working-age population, known as young or old dependency ratios, respectively. We can also identify the effect of changing population age distribution on overall dependency, measured as the population in the working ages divided by the total population.

Consequently, the output per capita in the Solow model can be decomposed using age profiles in what is known as (i) the support ratio (SR) and (ii) output per worker. The SR is the population-weighted sum across ages of labor income divided by the population-weighted sum of consumption. Output per worker is simply a measure of productivity. As a result, it is possible to identify from these components what is known as a demographic dividend, which is characterized by the shifts in the age structure of the population.

During the first stage of the demographic transition, the decline in mortality happened particularly in infant mortality. This increased life expectancy through the person-years gains occurred in childhood, increasing the total dependency ratio, driven by the young dependency,

decreasing the SR. The second stage is characterized by a decrease in the population growth rate and the dependency ratios. Therefore, according to Solow, the increase in the number of workers as a share of the population, which signified an increase in the SR, can be identified as the first demographic dividend.

The third phase is shaped by low fertility, which unambiguously leads to population aging, decreasing the number of people entering the labor force. In this stage there are two economic changes that increase the SR and productivity, identifying a second demographic dividend. First, following Solow, as a result of the slower population growth, there is an increase in the output per capita through the increase in the capital-labor ratio. Second, the lower fertility due to the lower infant mortality, followed by abortion laws, access to contraception and childcare, allowed women to work and invest in education.

The increase in the female labor participation and fertility decisions can be explained by the value of time model and the quantity-quality model. The first model states that when female wages increase, the higher female labor participation and lower fertility rates are a result of the larger substitution effect that dominates the income effect. That result can explain the decline in the number of desired children while income increases. The second model explains this decision through a quality dimension: with less children, there is more investment in their education increasing human capital. The result of these decisions, on one side, increases the support ratio because of the increase in female labor participation, while productivity increases through the investment in human capital.

Family life cycle and intergenerational transfer are intrinsically related in economics and demography through age composition. As we know, people interact through financial markets and transfer resources from one age group to another, in the form of savings and borrowing decisions that relate to different stages of the life-cycle. Age structure also matters for the international financial market, as capital flows from rich-old populations to poor-young populations, seeking higher rates of returns. In the case of transfers, parents rearing children, provide consumption and investment in their human capital. The same happens with adults caring for their elderly parents. Both of these are examples of private transfers. The public sector transfers are another example in which taxpayers provide public education for children and support for the elderly through pensions, health care and long-term care.

Population aging is the inevitable last stage of the demographic transition bringing challenges to economies. For instance, the public transfer set-up relies heavily on the SR, burdening future generations through taxes. It also affects financial markets through capital deepening: interest rates tend to the zero-lower-bound, which affects the scope of monetary policy. However, there are ways to offset these negative effects: increasing female participation and migration are examples. Capital deepening also increases wages, which promotes increases in labor force participation (mainly women). It also promotes investment in education. Finally, in the U.S, migrants have a higher fertility rate and are net taxpayers: they contribute more than the benefits they receive.

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Showpiece: **Disease and Development: The Effect of Life Expectancy on Economic Growth - Acemoglu & Johnson (2007) - Summary**

The authors exploit the major international health improvements from the 1940s to estimate the relationship between economic growth and life expectancy. They are aware that this relationship can be endogenous, because life expectancy can be explained by the outcomes of interest and vice versa, consequently, OLS estimates will be biased. Thus, life expectancy will covariate with the error term, causing omitted variables bias, since there must be variables other than life expectancy affecting economic growth. To deal with it, they come up with an instrument that allows an exogenous variation that explains the treatment -life expectancy- but not the outcome of interest -income per capita- which is the exclusion restriction.

The instrument should not be related to the component of actual mortality change in each country that results from economic growth or institutional improvements. For that purpose they use data on disease-specific death rates prior to the transition, along with information on the rates at which death rates from different diseases declined, based either on the dates of discovery of disease-specific treatments or worldwide declines in disease-specific mortality, in order to construct a measure of predicted mortality change for every country in their sample.

In the first stage, they show that the predicted mortality has a large impact on changes in life expectancy starting in 1940 but no effect before 1940, supporting the absence of pretends previous 1940s, it also has no effect on the log GDP. In the last half of the 20th century there were rapid gains in life expectancy associated with the international epidemiological transition in which modern health technologies were quickly diffused to the developing world, which is the framing for the instrument.

In the second stage, they find the striking results that predicted life expectancy has no effect on GDP growth and it has a negative impact on log GDP per capita. They also find a 1 percent increase in life expectancy leads to a 1.7-2 percent increase in population growth rate and a large effect on birth rates. Thus, the mechanisms involved in the negative impact on log GDP per capita are explained through Solow, where large population growth rate, decreases the capital labor ratio and decreases income per capita. The coefficient of life expectancy on log GDP per capita is −1.32 and is statically significant, implying that a country in which life expectancy rose from 40 to 60 would experience a 41% decline in log GDP per capita, holding other factors constant.

It is clear that life expectancy rises as a result of the decrease in infant mortality, shown in the positive impact on births, with the resulting increase in population. A worry here is that this is just an age structure effect due to rising youth dependency when mortality falls. Therefore, they showed that the working age dependency ratio has also a negative impact on log GDP per capita. Consequently, they claim that the negative economic effects of rapid population growth

more than compensated for direct economic benefits from better health, and so income per capita fell.

Bloom et al Comments - AJ Reply

Bloom et al revisited the relationship, arguing that the striking results found by AJ can be attributed to the instrument used. They say that it is very likely that mortality was not randomly distributed among these sets of countries. Rather, as a consequence of the narrow sample, the largest gains in life expectancy were in the countries where life expectancy was lowest. They show that correlation between initial life expectancy and the subsequent change in life expectancy is −0.97. Initial life expectancy is also correlated with subsequent growth of income per capita (correlation of 0.50).

The latter correlation is to be expected: in a model of conditional convergence, any factor that affects steady-state income per capita will also affect growth, conditioning on initial income. This means that countries have different trajectories depending on their own productivity and technology. They argue that life expectancy falls into this category, since there is abundant evidence that health raises individual productivity. This is why the initial level of health cannot be excluded from a regression in which income growth is the dependent variable.

When they re-run the AJ analysis, including both initial life expectancy and initial income on the right-hand side (the latter to control for convergence dynamics), the AJ result goes away. In their reply to this critique, AJ focuses on the effect of controlling for initial life expectancy, they say that it is very highly correlated with the change in life expectancy, and so in a mechanical sense there is no surprise that putting both on the right-hand side of a regression kills the statistical significance of the change in life expectancy. However, controlling for life expectancy in 1900, their results survived.

Finally, they are also challenged by the fact that most of this effect should appear in the long run, not just after a few decades. AJ replied this by saying that they already covered this long run impact by using the differences in differences approach to measure the impact of this increase of life expectancy between 1940 and 1980, and 1940 and 2000.

Felipe Menares - comments

There are issues regarding the identification of causal effects, since we are not able to have a proper counterfactual in this kind of set up. Therefore, the instrument must be really clean to claim causal effect. In that regard, we can say that it is at least controversial to choose diseases that behave and have different outcomes in rich countries compared to poor countries. As a matter of fact, it is likely that mortality is associated with other variables which are also related to economic growth, which is determined by the differences in productivity among these countries and have different paths in rich and poor countries in the second half of the past century.

Therefore, it is possible that unobserved factor(s) that predicted rapid population growth in countries with low life expectancy in 1940 also predicted slow income growth (for reasons unrelated to population or health) in such countries. Countries that had low life expectancy in 1940 differed in a systematic way from those that had high life expectancy: they had different environments, colonial history, levels of institutional development, and demographic histories. It would not be surprising if some element in that set of characteristics had a direct effect on subsequent population or income growth. Thus, it is likely that the instrument is only affecting countries that have these characteristics, low income countries with lower initial life expectancy.

Even though the authors run regressions to address the heterogeneity between high and low-middle income countries, there are 37 out 47 countries that are considered low-middle income countries, with a barely significant coefficient on the negative effect of life expectancy on GDP per capita, one of the most controversial results. Therefore, it is plausible to think that these gains in GDP per capita growth can be explained by the difference at the moment where the countries were with respect to GDP level (convergence) and life expectancy (infant vs elderly).

They claim that the negative impact on log GDP per capita is explained by the faster population growth, offsetting any gains due to the increase in life expectancy. It would be interesting to understand how this population growth relates to other variables that are not necessary related to economic growth or health. The larger population growth rate found is explained mostly through increase in births, which is possible due to the usual delay that the decline in fertility has after mortality falls. However, this can be also reflecting the increased number of reproductive age females resulting from lower mortality, which is independent of the lower infant mortality.

Finally, the paper tries to answer one of the main questions in the literature, the relationship between health and economic growth. Even though scholars have not yet agreed on its direction, the authors believed that life expectancy should impact economic growth. They use instrumental variables in order to solve usual problems in this setup. While the ideal design would be randomized life expectancy to every country and see the causal effect, this is unrealistic. Therefore, an alternative approach would be to implement their empirical strategy to similar countries, not only grouping them by income, but rather by institutional development and disease affected. This can be combined with the use of demographic variables such as life expectancies at different ages, among others.