Edited by
Alberto Bucci
Klaus Prettner
Alexia Prskawetz

Human Capital and Economic Growth

The Impact of Health, Education and Demographic Change

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Editors
Alberto Bucci
Department of Economics, Management, and Quantitative Methods (DEMM)
University of Milan
Milan, Italy

Alexia Prskawetz
Institute of Statistics and Mathematical Methods in Economics
Vienna University of Technology
Vienna, Austria

Klaus Prettner
Institute of Economics
University of Hohenheim
Stuttgart, Germany

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Introduction

Alberto Bucci, Klaus Prettner, and Alexia Prskawetz

Beginning with the seminal work by Romer (1986, 1987), theoretical and empirical research on economic growth have experienced a dramatic boom. The *"New"* (i.e., *Endogenous*) *Growth Theory* has focused on productivity advances that stem from embodied and from disembodied technological progress. Thus, in this theory increased human capital accumulation in the form of schooling and education by people (Lucas, 1988) and intensified R&D investments by firms (Romer, 1990) both play a central role in spurring long-term productivity growth. According to recent estimates (Barro, 2013), the variable *"years of schooling at the secondary and higher level for males aged 25 and over"* shows a significantly positive effect on subsequent economic growth: an extra year of male

A. Bucci
Department of Economics, Management, and Quantitative Methods (DEMM), University of Milan, Milan, Italy

K. Prettner
Institute of Economics, University of Hohenheim, Stuttgart, Germany

A. Prskawetz
Institute of Statistics and Mathematical Methods in Economics, Vienna University of Technology, Vienna, Austria

upper-level schooling raises economic growth by 1.2 percentage points per year. In the same study Barro (2013) also shows that the same extra year of male upper-level schooling raises the magnitude of the convergence coefficient from 0.026 to 0.032. This result supports theories that point to a positive effect of human capital in the form of education on an economy's ability to absorb new technologies.

In addition, Romer (1990), Aghion and Howitt (1992), and Jones (1995) have clarified the central role played not only by the quality but also by the quantity of people (i.e., population size) in economic growth through its capacity of influencing an economy's ability to discover new "ideas" (*"More people means more Isaac Newtons and therefore new ideas"*, Jones, 2003, p. 505). However, empirical growth studies for the second half of the twentieth century tend to find a significantly negative coefficient estimate for the log of the total fertility rate (Barro, 2013). Three mechanisms may explain this result: (1) With a growing population, part of the economy's investment is used to provide capital for new workers, rather than to raise capital per worker (*dilution effect*); (2) A higher population growth rate (due to higher fertility) means that increased resources must be devoted to childrearing, rather than to the production of goods and new ideas; (3) Fast population growth constitutes one mechanism by which a poverty trap can emerge and be sustained as shown in *Unified Growth Theory* (Galor and Weil, 2000; Galor, 2005, 2011).

While population growth tends to exhibit a negative correlation with economic growth, a population's overall health status (measured, for example, by the log of life-expectancy at birth, or by the rates of infant– and child– mortality) reveals an unambiguous and significantly positive effect on subsequent economic growth: according to Barro (2013) a rise of initial life expectancy from 50 to 70 years (an increase of 40%) would raise the rate of economic growth by 1.4 percentage points per year.

In conclusion, human capital (either in the form of schooling and education, or in the form of health) and population change are potentially important drivers of per capita income growth in the long-run.

The main objective of this book is to analyze, with a variety of different approaches (theory and empirics) and methodologies (economic, demographic, etc.), the relations among four fundamental variables, (1) Human

capital in the form of health, (2) Human capital in the form of education, (3) Demographic change, and (4) Economic growth. The book consists of 10 contributed chapters that study in detail these four variables and the multiple links existing among them.

In particular, the book deals with the following two broad topics: *"Human capital in the form of education, demographic change, and their economic implications"*, and *"Human capital in the form of health, its economic implications, and the economic policies for a healthier world"*. The two sections of the book are devoted to the analysis of each of these topics.

Human Capital in the form of Education, Demographic Change, and Their Economic Implications

Models of optimal individual education decisions have a long tradition in theoretical economics (see, for example, Uzawa, 1965; Ben-Porath, 1967; Ghez and Becker, 1975; Blinder and Weiss, 1976; Ryder et al., 1976; Haley, 1976). The typical finding of this literature is that an optimal path over the life cycle starts with high education investments in younger ages and involves lower investments later on in life. Lucas (1988), by contrast, investigates the education choice of an infinitely-lived representative agent within a general equilibrium long-run economic growth setting. He finds that the optimal education trajectory is to spend a constant part of available time on education throughout the entire life.

The first part of this book analyzes the (macro-)economic implications of human capital (in the form of education) and of demographic change.

Pelloni et al. start by exploring empirically how institutions and human capital interact with each other, and together influence economic growth. Using a threshold regression model, they find strong nonlinearities in this relationship.

Skritek et al. provide an analytical assessment of the optimal lifetime education decision problem of an atomistic (as opposed to a representative) individual, whose choices have no influence on the labor and capital markets, and demonstrate that the canonical result of Lucas (1988), briefly

summarized above, hinges on the assumption of a representative agent whose choices affect the wage rate and the interest rate.

Human capital and demographic change are able to affect long-run economic growth not only in isolation, but also when considered together. In other words, these two variables are strongly linked to each other. As an example, in a recent application of the Lucas model with the objective of answering the question of whether educational investments can help to mitigate the negative economic effects that are expected from population aging, Edle von Gäßler and Ziesemer (2016) show that, within the Lucas (1988) setting, an increase in education is indeed an optimal response to faster aging.

Demographic change, in general, and population aging, in particular, have become a highly important topic in the economic, social, and political debate of many countries, especially the most developed ones (see, Bloom et al., 2004, 2010). The reason is that demographic changes have important effects not only on the sustainability of social security systems and pension schemes, but also on human capital accumulation, R&D, technological progress, long-run economic growth, and therefore on wage inequality.

In this regard, **Baudin et al.** explain why, beyond average fertility, childlessness matters in itself and what the links between human capital in the form of education and childlessness are.

Afonso et al. show that the decline in the birth rate (and, consequently, population aging) may help explain both the increase in the skill premium and the decline in the R&D intensity observed in many countries starting from the end of the twentieth century.

Based on the concept of generational replacement, the contribution by **Striessnig** presents the view of a demographer on the potential of quantitative forecasts of future populations by age, sex, and level of educational attainment in the analysis of economic development.

Human Capital in the form of Health, Its Economic Implications, and the Economic Policies for a Healthier World

Across countries, per capita GDP is positively correlated with indicators for health, e.g. life expectancy at birth, birth weight, and height (Weil, 2014). The link between health and income is bi-directional. From a theoretical standpoint, endogenous growth theories have stressed the role of human capital for productivity advances (Lucas, 2002). Healthier individuals are more productive, learn more in school, and, because they live longer, face enhanced incentives to accumulate human capital, which ultimately causes long-run economic growth. Moreover, under standard assumptions on individual preferences, as people get richer, the marginal utility of consumption declines and the income effect pushes spending towards more life-saving activities, such as health. From an empirical standpoint, there exists strong evidence for a bi-directional link between health and income. However, the magnitude of these effects is limited, especially when the object of the analysis is to investigate cross-sectional differences. Weil (2014) points out that other (unobserved) factors, which simultaneously raise income and improve health outcomes, such as institutional quality (at the country level) and education (at the individual level), are responsible for the positive observed health-income correlation.

After summarizing the most recent literature on health and economic growth, and using the sample of OECD countries, **Bucci et al.** provide convincing evidence that the level of per capita income is positively affected by a population's health level, proxied by the life expectancy at birth.

In the last decades, health care costs have been rising in most developed and developing countries. For example, health care in the United States is today a 3 trillion US-$ industry, accounting for nearly 18% of GDP. This has helped to increase longevity. Indeed, since 1950 life expectancy at birth has risen by more than ten years in Northern America and Europe and by about twenty-five years in Africa and Latin America. Fertility, at the same time, has declined dramatically over the last few decades,

reaching unprecedented low levels, so that now nearly half of the world lives in countries with below-replacement level fertility (2.1 children per woman). The result is an old-age dependency ratio in developed countries of about 25% that is expected to double by the end of the century. These health– and demographic– trends have already put pressure on pension, health, and elderly care systems almost everywhere. Moreover, according to the World Population Ageing Report of the United Nations (2015), between now and 2030 the number of people aged 60 years and over is projected to grow by 56% and to exceed the number of children aged 0–9. This aging process is especially advanced in Europe and Northern America where people aged 60 and over represent already more than a fifth of the population and will represent more than a fourth by 2030. In a word, these trends will continue to pose a challenge for the sustainability of unfunded pension systems, because they clearly struggle to maintain an adequate level of income support. A similar challenge is posed to the sustainability of current health care expenditures in the face of population aging.

Cipriani and Fioroni analyze the role of the government in such a framework and study the conditions under which governmental policies on health and pensions might lift an economy from a low to a high income equilibrium.

It is now recognized that health and education are largely interlinked in their contribution to economic growth because they both contribute to human capital accumulation. A common approach is thus to treat these two aspects symmetrically when thinking about human capital. However, it is essential to distinguish between health and knowledge (education). Indeed, while the latter can be accumulated without bounds, the former cannot.

According to **Agénor**, this distinction has important analytical and policy implications, especially as far as the allocation of government spending between health and education is concerned. In particular, Agénor shows that growth dynamics depend in important ways on the externalities associated with these two aspects of human capital (knowledge and health, respectively). Depending on the strength of these externalities, increases in government spending on education or health (financed by

a cut in unproductive spending, for example) may have differential and ambiguous effects on economic growth.

The way people value the future is crucially affected by their life expectancy: a higher longevity decreases the effective discount rate, thus pushing individuals to invest more in environmental quality. Of course, the causal link between life expectancy/health and environmental quality may also go the other way around (as confirmed by several studies in medicine and epidemiology). Considering endogenous economic growth makes the picture more complex and interesting: environmental preservation may in fact divert resources from investment (typically in human capital), while on the other hand income growth makes more maintenance possible, but may itself involve more pollution and poorer health.

In their chapter, **Mariani et al.** focus on this intricate interplay between human capital, health (represented by longevity), population, and environmental quality and draw interesting normative conclusions.

Modern demo-economic research has attempted to provide unified explanations of the industrial revolution and of the parallel development of the demographic transition (Galor, 2011; Livi-Bacci, 2017). The industrial revolution and the demographic transition allowed human populations to escape from their long-term stagnation in the *"Malthusian"* regime, characterized by the persistence of a poverty trap at exceptionally high levels of mortality and fertility. Nonetheless, a number of important aspects are still under-researched. For example, in most available explanations, the escape from the Malthusian regime is ultimately triggered by mortality decline. However, in these explanations mortality decline manifests itself as a monotonic, essentially steady process over time. This likely represents a crude approximation of reality. Indeed, exactly as the Malthusian regime was, rather than a static equilibrium, a continued sequence of "stop-and-go" epochs (in which homeostatic mechanisms were able to recover human populations from the crises deriving from recurrent epidemics, wars, and the stochastic appearance of famine), similarly a major part of the *"take-off"*-phase of the industrial revolution (say up to 1850–1900 in industrialized countries) showed a great deal of complexity in relation to the impact of infectious diseases and epidemics on mortality and morbidity (Livi-Bacci, 2017). Today, despite the

increasing rate of diffusion of effective therapies, the battle against such epidemics as HIV/AIDS in Sub-Saharan Africa (SSA) is far from being over.

In their chapter, **Gori et al.** analyze the effects that the diffusion of epidemics might have on the fertility transition, on the amount of resources needed to finance the fight against them, on the resistance to the available medical treatments, and ultimately on economic growth.

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Notes on Contributors

Oscar Afonso is an Associate Professor (with tenure and habilitation) at University of Porto and CEF.UP. Oscar holds a Ph.D in Economics from the University of Porto. His main research interests lie on the fields of Economic Growth and Trade and he has published in journals such as Applied Economics, Journal of Economic Behavior and Organization, Macroeconomic Dynamics, Research Policy, and World Development.

Pierre-Richard Agénor is Hallsworth Professor of International Macroeconomics and Development Economics at the University of Manchester. He has published a large number of articles in leading professional journals on a wide range of issues, related notably to international macroeconomics, development economics, growth theory, labour economics, and gender equality. He is the author of several books, including *Public Capital, Growth and Welfare* (Princeton University Press) and *Monetary Policy and Macroprudential Regulation with Financial Frictions* (MIT Press).

Thomas Baudin is Associate Professor of Economics and Quantitative Methods at IESEG School of Management and Research Fellow at IRES (UCLouvain). His main research interests are economic growth, family economics, and cultural economics.

Alberto Bucci is Professor of Dynamic Macroeconomics and Growth Theory at the University of Milan (Italy). He is senior research fellow at *RCEA* (*Rimini Center for Economic Analysis* – Wilfrid Laurier University, Waterloo, Canada),

research fellow at *CEIS* (*Centre for Economic and International Studies* – University of Rome *"Tor-Vergata"*, Rome, Italy), and research fellow at *CICSE* (*Center for Economic Growth and Development* – University of Pisa, Italy). He holds a Ph.D. in Economics from the *Université catholique de Louvain* (Louvain-la-Neuve, Belgium). His research has been extensively published in leading economics journals (such as the JEDC, the JEBO, the Journal of Human Capital, the Journal of Macroeconomics, and Macroeconomic Dynamics), and falls into the domain of financial economics, dynamic macroeconomics, and the economics of growth (R&D, human capital, innovation, health).

Lorenzo Carbonari is *Assistant Professor* at the University of Rome "Tor Vergata", Department of Economics and Finance, and research fellow at CEIS. His main research areas are macroeconomics (growth, micro-founded models of money) and health economics (pharmaceutical markets, cost-effectiveness analysis).

Giam Pietro Cipriani (PhD, Manchester) is Professor of Economics at the University of Verona. His research interests lie mainly on the edges of macroeconomics, in long-run economic development and demographic change. In particular, he has contributed to the literature on endogenous mortality in the OLG model. Other recent research interests fall in the areas of behavioral economics and economics education. He is an Associate Editor of the Journal of Population Economics and of the International Review of Economics Education.

Jesus Crespo Cuaresma is Director of Economic Analysis at the Wittgenstein Centre for Demography and Global Human Capital (WIC), Professor of Economics at the Vienna University of Economics and Business (WU), and Research Scholar at the International Institute of Applied Systems Analysis (IIASA). He has published numerous articles, inter alia, on education and economic growth in renowned scientific journals and acts as a scientific consultant to the World Bank and the Austrian Institute of Economic Research.

David de la Croix is Full Professor of Economics at UCLouvain and Research Fellow at CEPR. His main research interests are economic growth, family economics, and economic history.

Tamara Fioroni is Assistant Professor at the Department of Economics – University of Verona. She received the PhD degrees in Economics at the University of Pisa. She spent a research period as visiting research student at the Université catholique de Louvain. Her main research interests involve economic

development and demographic transition, distribution of income, human capital accumulation, child labour and non-linear dynamics.

Alexia Prskawetz is Professor of Mathematical Economics at the Vienna University of Technology, executive director at the Vienna Institute of Demography, Austrian Academy of Sciences, one of the Directors at the Wittgenstein Centre for Demography and Global Human Capital (WIC) and research associate at the International Institute of Applied Systems Analysis, Laxenburg, Austria. Her main areas of research are in the economic consequences of population and individual ageing, long run economic growth, and agent based computational demography.

Pedro Mazeda Gil is an Assistant Professor (with tenure) at University of Porto and CEF.UP. Pedro holds a PhD in Economics from the University of Porto. His main research interests are Economic Growth and Industrial Dynamics. He has published in journals such as the Economics Letters, Journal of Economic Behavior and Organization, Journal of Evolutionary Economics, Journal of Money, Credit and Banking, Macroeconomic Dynamics, and Oxford Economic Papers.

Paula E. Gobbi is Associate Professor of Economics at Université Libre de Bruxelles and Research Fellow at CEPR. Her main research interests are economic history, family and household economics.

Luca Gori is Associate Professor of Economics at the Department of Law, University of Pisa.

Arkadii V. Kryazhimskii was a Senior Research Scholar at the International Institute for Applied Systems Analysis (IIASA), Laxenburg, Austria. He was also a Principal Research Scholar at the Steklov Institute of Mathematics of the Russian Academy of Sciences, Moscow. His research interests included optimal control theory, differential games, and ill-posed problems. He passed away in 2014.

Piero Manfredi is Full Professor of Demography at the Department of Economics and Management, University of Pisa.

Fabio Mariani is Professor of Economics at UCLouvain and Research Fellow at IZA. His main research interests are economic growth, family economics, and international migration.

Pedro Cunha Neves is an Assistant Professor at Universidade da Beira Interior and CEFAGE. Pedro holds a PhD from University of Porto. His main research fields are Economic Growth and Inequality. He has published in journals such

as Economic Modelling, Journal of Development Studies, Journal of Policy Modeling, Research Policy, and World Development.

Alessandra Pelloni is currently full Professor of Economics at the Department of Economics and Finance of the University of Rome, Tor Vergata. She previously held positions at the Universities of Warwick and Manchester, UK. She got a M. Sc. In Economics at the London School of Economics and a Ph. D. in Economics at the European University Institute. Her work on topics ranging from the link between long run growth and fluctuations to the theory of optimal capital taxation in models of endogenous growth to the economics of relational goods has appeared in journals including the Economic Journal and the Journal of Public Economics.

Agustín Pérez-Barahona is Professor of Economics at the University of Cergy-Pontoise. His main research interests are economic growth and environmental economics.

Klaus Prettner is Professor of Economics at the University of Hohenheim and speaker of the research network "Inequality and Economic Policy Analysis (INEPA)." His research is primarily concerned with the interrelations between economic growth and inequality, the economic consequences of automation, and the impact of demographic change on long-run economic growth in industrialized economies.

Natacha Raffin is Professor of Economics at the University of Rouen (France). Her main research interests are economic growth, environmental economics and health economics.

Elena Rovenskaya is the Director of the Advanced Systems Analysis Program at the International Institute for Applied Systems Analysis (IIASA), Laxenburg, Austria. She is also a Research Scholar at the Optimal Control Department of the Faculty of Computational Mathematics and Cybernetics, Lomonosov Moscow State University, Russia. Her scientific interests lie in the fields of theory of optimal control and economic-environmental modeling.

Tiago Neves Sequeira is an Associate Professor (with tenure and habilitation) at Universidade da Beira Interior and CEFAGE. Tiago holds a Ph.D in Economics from the NOVA School of Business and Economics (Lisbon, Portugal). His main research interests lie on the field of Economic Growth and he has published in journals such as the Journal of Economic Behavior and Organization, Macroeconomic Dynamics, Oxford Economic Papers, Research Policy, and Scandinavian Journal of Economics.

Bernhard Skritek is currently working at PricewaterhouseCoopers (PwC). He obtained his PhD in Mathematical Methods in Economis at the Vienna University of Technology, Austria and was a Research Assistant at the International Institute for Applied Systems Analysis (IIASA), Laxenburg, Austria. In his thesis, he investigated heterogeneous optimal control problems on the infinite horizon and their economic applications.

Mauro Sodini is Associate Professor of Applied Mathematics for Economics at the Department of Economics and Management, University of Pisa.

Thanasis Stengos joined the Department of Economics at the University of Guelph in 1984, where he now holds a University Research Chair. He currently serves as Associate Editor of the Journal of Applied Econometrics, Empirical Economics, Economics Letters and he is co-editor of the Review of Economic Analysis. His research covers topics in nonparametric econometric methods and nonlinear models, and applications in empirical growth, environmental and energy economics and welfare measurement.

Erich Striessnig is a Research Scholar with the World Population (POP) Program at IIASA and Vienna Institute of Demography (VID) of the Austrian Academy of Sciences. His work focuses on the development of multidimensional population projections, as well as their application to questions related to social transformation processes resulting from the demographic metabolism.

Giovanni Trovato is Professor of Economic Statistics at the University of Rome 'Tor Vergata', Department of Economics and Finance. He is research fellow at CEIS. Following his bachelor's degree in Economics at 'La Sapienza' University (Rome), he gained a PhD in Economics from the University of St. Gallen. His research interests include econometrics, economic growth and finance. His works have been extensively published in leading economics journals such as Journal of Money, Credit and Banking, Journal of Applied Econometrics, Econometrics Journal, Small Business Economics and Macroeconomic Dynamics.

Fabrizio Valenti is an economist and international development specialist with experience working for a wide range of institutions including UNDP, Save the Children, and private consulting companies. He obtained his Ph.D. from the University of Rome Tor Vergata and his research interests focus on the relationship between economic growth and issues such as volatility, human capital, and institutional quality. Recently, Fabrizio has been working on employment and labor in Bangladesh, Sierra Leone and Canada.

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Part I

Human Capital in the Form of Education, Demographic Change, and Their Economic Implications

1

The Non-linearity in the Relationship Between Human Capital and Growth

Alessandra Pelloni, Thanasis Stengos, and Fabrizio Valenti

1.1 Introduction

This chapter offers an empirical investigation of the relationship between human capital and per capita GDP growth across countries. Previous studies have detected strong non-linearities (thresholds) in this relationship, see for example Kalaitzidakis et al. (2001) and Savvides and Stengos (2009). The leading theoretical explanation for these findings is that human capital accumulation by individual agents will confer external economies to other agents, increasing their productivity and producing increasing returns on an aggregate basis. These spillover effects, if strong enough, may result in multiple equilibria including low-growth

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A. Pelloni (✉)
Department of Economics and Finance, University of Rome II, Rome, Italy
e-mail: alessandra.pelloni@uniroma2.it

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or poverty traps. More recently, economists have focused on the quality of institutions in conditioning the interactions between agents. Our empirical analysis factors in this aspect. We also highlight that human capital accumulation by women has a very different impact from human capital accumulation by men.

Our analysis is based on the threshold regression model introduced by Hansen (2000) and, specifically, on its most recent version developed by Kourtellos et al. (2013, 2016). The approach that we take here differs from the semiparametric methodology adopted by Kalaitzidakis et al. (2001) and Savvides and Stengos (2009) to estimate the human capital economic growth nexus. The main difference is that in the previous work any evidence of turning points (thresholds) was made thorough a smooth transition without proper testing. In our current approach we are able to obtain "sharp" turning points and test for their significance. Through all our regressions the dependent variable is the growth of GDP per capita, while human capital and, precisely, primary education—disaggregated by gender—is the threshold variable.

Our empirical procedure is divided in two steps: the first consists in testing for the presence of a threshold in human capital. If the null of no such presence is rejected the next step consists in estimating the threshold regression parameters, dividing the sample in two regimes obtained by separating the observations above and below the threshold. The procedure is repeated using fitted values of human capital, which are estimated using institutional quality as an instrumental variable. This approach is employed to deal with the possible endogeneity of human capital.

T. Stengos
Department of Economics and Finance, University of Guelph, Guelph, ON, Canada
e-mail: tstengos@uoguelph.ca

F. Valenti
International Business Development, KSAR & Associates Inc., Ottawa, ON, Canada
e-mail: fabrizio.valenti@ksar.co

Our results consistently reject the null hypothesis of no thresholds in the effect of human capital on growth, with the exception of fitted female primary education. Otherwise, non-linearities emerge very clearly as slope coefficients change in magnitude from one regime to the other. Comparing results for female primary education and fitted male primary education provides some interesting insights. In fact, the effect of these variables on growth of GDP per capita is in the case of female education much higher in the low-education regime, while the opposite is true for male education.

Overall, regression results confirm the non-linear shape of the relationship between human capital and growth, while showing the importance of institutions and gender. More specifically, our findings suggest that female primary education has an unconditional direct effect on GDP growth, while male primary education has an indirect effect, which depends on institutional quality.

The role of human capital in raising per capita GDP growth has been long debated by economists and policy makers. On the theoretical side, human capital has been widely studied as a driver of growth, with papers stressing its importance both as a stock (Lucas, 1988) and a flow variable (Nelson and Phelps, 1965). While both theories and intuition point toward a positive role, the empirical evidence is not clear-cut. Earlier investigations starting from the seminal paper by Mankiw et al. (1992) found a positive link between human capital and growth of per capita GDP. The measure of human capital adopted by Mankiw et al. (1992)—enrollment rates in secondary education—was however quite restrictive. Klenow and Rodriguez-Clare (1997) showed that the effects of human capital tend to be much smaller using other measures for the variable, while other works such as Benhabib and Spiegel (1994) and Pritchett (1996) found that human capital has an insignificant or even negative effect on economic growth. A way to reconcile this conflicting evidence is to allow for non-linearities in the relationship. This is the approach adopted by Kalaitzidakis et al. (2001) who use non-parametric techniques. Their central finding is that the beneficial effect of human capital on growth is limited to economies with middle levels of human capital (measured by average years of schooling).

The theoretical possibility of non-linearities in the impact of human capital accumulation on economic outcomes is represented in Azariadis and Drazen (1990). In their model the economy can find itself in two different "regimes" depending on whether human capital is above or below a certain threshold level, making multiple equilibria arise. Poverty traps—in the definition of Azariadis and Stachurski (2005): "any self-reinforcing mechanism which causes poverty to persist"—arising from human capital accumulation have been subsequently modeled in many papers, of which we can just mention a few. Berti Cerioni (2001) built a model of "fractal" poverty traps (as defined by Easterly, 2001; Barrett and Swallow, 2006) in which poverty and inequality self-perpetuate themselves since members of the lowest classes need higher returns to invest in education. Santos (2009) builds on this model showing that poverty traps also arise from differences in the quality of education provided to children coming from different social backgrounds.

Acemoglu et al. (2004) make the case that differences in economic institutions are the fundamental cause of differences in economic development. Indeed, a focus on the role of institutions is reshaping the way economists think about development and growth, see e.g. Roland (2014). This revival of institutionalism is based on the increasing awareness in the profession that markets do not emerge spontaneously to create efficient outcomes but that political and legal institutions are needed to provide stabilization, regulation, and legitimation to economic activity. Institutions can be defined as constraints placed by social, formal or informal, rules on human behavior. Social rules are necessary to solve a vast array of problems in economic transactions: from information and commitment problems to cooperation and coordination ones. Different institutional structures will deal with these problems in a more or less desirable and efficient way from the social point of view. For instance, there is a commitment problem when people cannot achieve their goals because of an inability to make credible promises. Formal institutions help to solve the problem by enforcing legal contracts, while informal enforcement may give rise to violence and crimes. To give another example, cooperation problems arise because collective action benefits all but has a private cost only for those who participate in it. Democracy minimizes the private costs of collective action by means of elections. Informal

solutions include riots and conflicts etc. More generally, according to North (1990), the basic institutional framework may make appropriating income produced by others a preferred economic opportunity to a productivity increasing one. When, as in many developing countries such sub-optimal institutional equilibria prevail, education goes into forming skills that are useful for privately remunerative, but socially unproductive or harmful activities as noted by Pritchett (1996).

An important indicator of the quality of institutions in a country is the extent of corruption. Corruption, defined by Bardhan (1997) as "the use of public office for private gains" is indeed endemic in less developed economies (for surveys of the existing literature, see Bardhan, 1997; Jain, 2001; and Rose-Ackerman, 1998).

Human capital accumulation and corruption have been considered together in some recent theoretical papers. According to Costa Vieira and Teixeira (2006) education tends to reduce corruption: in a democratic regime, the higher the literacy rate, the more receptive citizens are to the efforts of the minority parties to unveil corruption in the government. However, Eicher et al. (2009) show that the relationship between human capital and corruption can be more complex: in fact, if it is true that education produces more informed electorates, it is also true that higher human capital means increased average income and, thus, increased corruption rents, an effect first described by Blackburn et al. (2006) in a model without human capital. Economies with intermediate levels of human capital might remain stuck in a poverty-trap-like situation where the average skill level is high enough to create sizable corruption rents, but not high enough to induce enough monitoring. Haque and Hussain (2012) show that human capital accumulation could increase corruption by increasing the efficiency of bureaucrats in being corrupt.

As mentioned above we allow for the possibility of different effects of human capital accumulation by sex. The literature on the gender gap in education is huge and we can only summarize here those of its main findings that are more relevant for our analysis (for some basic facts see Todaro and Smith, 2014, and for an extended overview see World Bank, 2012). A first fact is that females tend to receive less education than males in most low-income developing countries. The rate of return

on women's education is then found to be higher than that on men's (possibly because the marginal girl is likely to be more talented on average than the marginal boy). Increasing women's education induces lower fertility because it raises the opportunity cost of women's time in raising children (Galor and Weil, 1996). There is a large evidence that mother's schooling is positively correlated with children's health and nutrition in developing countries (see Glewwe, 1999). This means that, apart from the positive effects on the aggregate production function and female human capital accumulation, female education has longer term effects on future generations. A third important point to consider is that since women are disproportionately affected by poverty, improvements in their education can have an important impact on breaking the vicious circle of poverty (see World Bank, 2012, and Agarwal, 2016).

These improvements are also less likely to lead to individual positions of power and rent-extraction, which lead to negative social net dividends. Indeed, many behavioral studies have found women to be more trustworthy and socially-minded than men. Dollar et al. (2001) found that, in a large cross section of countries, the greater the representation of women in parliament, the lower the level of corruption.

The rest of the chapter is organized as follows: Sect. 1.2 describes the empirical procedure employed in the analysis, Sect. 1.3 introduces the data utilized in the analysis, Sect. 1.4 presents findings and discusses results, and Sect. 1.5 concludes.

1.2 Empirical Model

In contrast to what was done previously in the literature that relied mainly on semiparametric methods to estimate the effect of human capital on growth, see Kalaitzidakis et al. (2001) and Savvides and Stengos (2009) for example, in the present chapter we use a method that allows for the presence and statistical testing of "sharp" turning points. The empirical procedure adopted in this chapter is based on the threshold regression model introduced by Hansen (1999, 2000) and Caner and Hansen (2004). Specifically, the analysis will employ the extended version of this model, as developed by Kourtellos et al. (2016) that allows for

an endogenous threshold. Institutions will then be introduced as an instrumental variable for human capital, in order to mitigate the risk of endogeneity in human capital. Following a rich literature highlighting the importance that gender plays in the impact of education on economic growth, the regressions presented in this chapter use gender disaggregated data.

The Kourtellos, Stengos and Tan version of the threshold regression model introduces an interesting form of non-linear regression model. It allows for a high degree of flexibility in the functional form and at the same avoids problems like the "curse-of-dimensionality" that affect, for example, non-parametric regressions. The following paragraphs will introduce and explain a general version of said model.

The observed data is divided between the variables of interest, Y, the regressors, X, a set of instruments Z and the threshold variable q. In particular, the data take the form $\left\{y_{i,t}, x_{i,t}, z_{i,t}, q_{i,t}\right\}_{\mathrm{i=1,t=1}}^{\mathrm{n,T}}$, where $y_{i,t}$ and $q_{i,t}$ are real-valued and $x_{i,t}$ and $z_{i,t}$ are m and I vectors of regressors and instruments respectively. Moreover, notice that $q_{i,t}$ may be an element of $x_{i,t}$ and is assumed to have a continuous distribution.

The simple threshold regression model without an endogenous threshold can be written as follows:

$$y_{i,t} = x'_{i,t}\beta_1 + u_{1i,t}, q_{i,t} \leq \gamma \tag{1.1}$$

$$y_{i,t} = x'_{i,t}\beta_2 + u_{2i,t}, q_{i,t} > \gamma \tag{1.2}$$

Equations (1.1) and (1.2) describe the relationship between the variables of interest in each of the two regimes and $\mathrm{q_i}$ is the threshold variable with γ being the sample split threshold. Note that $\mathrm{q_i}$ is observed but the sample split is unknown.

Equations (1.1) and (1.2) can be rewritten by defining y_{1i} and y_{2i} as follows:

$$y_{i,t} = \begin{cases} y_{1i,t}, if\ q_{i,t} \leq \gamma : Regime\ 1 \\ y_{2i,t}, if\ q_{i,t} > \gamma : Regime\ 2 \end{cases} \tag{1.3}$$

Or, in order to condense the model in a single equation, it is possible to use the indicator function ($I(\bullet)$) to define $d_{i,t}(\gamma) = I(q_{i,t} \leq \gamma)$. This leads to:

$$y_{i,t} = d_{i,t}(\gamma)\, y_{i,t} + \left(1 - d_{i,t}(\gamma)\right) y_{2i,t} \tag{1.4}$$

Which is equivalent to:

$$y_{i,t} = d_{i,t}(\gamma)\, x'_{i,t}\beta_1 + \left(1 - d_{i,t}(\gamma)\right) x'_{i,t}\beta_2 + d_{i,t}(\gamma)\, u_{1i,t} + \left(1 - d_{i,t}(\gamma)\right) u_{2i,t} \tag{1.5}$$

This last equation can be rewritten employing the following more compact notation:

$$y_{i,t} = x\prime_{i,t}\beta + x'_{i,t}(\gamma)\,\delta + u_i \tag{1.6}$$

Where:

$$\begin{gathered} u_{i,t} = \mathrm{d_{i,t}}(\gamma)\, u_{1i,t} + \left(1 - \mathrm{d_{i,t}}(\gamma)\right) \mathrm{u}_{2i,t} \\ x_{i,t}(\gamma) = x_{i,t}\mathrm{d_{i,t}}(\gamma) \\ \beta = \beta_2 \\ \delta = \beta_1 - \beta_2 \end{gathered}$$

Allowing for endogeneity of the $x_{i,t}$ introduces an additional equation that links the endogenous variables and the set of instruments. Similarly, if the threshold variable $q_{i,t}$ is endogenous there is an extra equation that links $q_{i,t}$ to the instruments. In this case, the model is augmented by the presence of selectivity correction factors (Inverse Mills Ratios) that ensure that the model yields consistent estimators both of the threshold parameter as well as the slope coefficients. The extended framework of the threshold model with endogeneity has been analyzed by Kourtellos et al. (2016) and has been applied in the context of an empirical growth model in Kourtellos et al. (2013).

The first step of the regression procedure consists in testing for the presence of a threshold: given the simple regression Eq. (1.6) or the extended framework that allows for endogeneity, the null hypothesis of no threshold is:

$$H_0 : \delta = 0 \tag{1.7}$$

A bootstrap procedure, which simulates the asymptotic distribution of the likelihood ratio test, is employed to test for H_0. If H_0 is rejected the next step consists in estimating the threshold regression parameters using concentrated least squares.

1.3 Data

The empirical analysis employs two similar data sets. The first is a balanced five-year period panel of 104 countries starting in 1960 and ending in 2010 and it includes the following variables taken from Penn World Tables: (a) the logarithm of GDP growth per capita averaged over five-year intervals, (b) the logarithm of initial real GDP per capita, (c) the average ratio between investment and GDP, and (d) the logarithm of average trade openness.

Two variables for population are included as well: the logarithm of average population, and the average growth rate of population.

The dataset also includes measures of education, taken from the Barro-Lee educational attainment data set (Barro and Lee, 2013). In particular, these are: the percent enrollment rates in primary, secondary, and tertiary education, the sum of these enrollment rates, and a measure of total average years of schooling. The data is available at both the aggregated and gender-disaggregated level.

The second dataset adds to the above-mentioned variables some measures for institutional quality for which there is, however, data only starting from 1995. These variables will act as instruments to estimate current levels of human capital. Given that institutions are slow moving, causality will be assumed to run from *previous* institutional quality to *current* human capital. The variables for "institutional quality" come from

the 2017 Worldwide Governance Indicators and range from approximately −2.5 (weak governance performance) to 2.5 (strong governance performance). Specifically, they are:

Voice and Accountability	Reflects perceptions of the extent to which a country's citizens are able to participate in selecting their government, as well as freedom of expression, freedom of association, and a free media;
Political stability and absence of violence/terrorism	Measures perceptions of the likelihood of political instability and/or politically-motivated violence, including terrorism;
Government effectiveness	Reflects perceptions of the quality of public services, the quality of the civil service and the degree of its independence from political pressures, the quality of policy formulation and implementation, and the credibility of the government's commitment to such policies;
Regulatory quality	Reflects perceptions of the ability of the government to formulate and implement sound policies and regulations that permit and promote private sector development;
Rule of law	Reflects perceptions of the extent to which agents have confidence in and abide by the rules of society, and in particular the quality of contract enforcement, property rights, the police, and the courts, as well as the likelihood of crime and violence;
Control of corruption	Reflects perceptions of the extent to which public power is exercised for private gain, including both petty and grand forms of corruption, as well as "capture" of the state by elites and private interests

1.4 Empirical Results

In all regressions, the variable of interest, y, is the average growth of GDP per capita, while human capital is the threshold variable. The logarithm of average trade openness, the logarithm of initial real GDP per capita, the average ratio between investment and GDP, the average population growth and a measure of human capital are used as control variables.

For the purposes of this chapter Lagged Primary Education (fitted or not, depending on the analysis being performed) has been selected as a measure of human capital, both as a threshold and as a control variable.

1.4.1 The Threshold Model—Not Accounting for Endogeneity

Given the general econometric procedure previously described, the first step in the analysis consists in testing for the presence of a threshold.

The LM test statistic and the bootstrapped p-value tell whether the null hypothesis is rejected or not, i.e. whether a threshold is present or not. The first stage of the analysis also provides an estimated value of the threshold itself.

While Tables 1.1, 1.2, 1.3, 1.4, 1.5, and 1.6 show descriptive statistics,[1] Table 1.7 presents the results from these tests and estimation using lagged primary education as both a threshold and control variable. The same

Table 1.1 Descriptive statistics, non-fitted data—not disaggregated by gender

	Total	Regime 1	Regime 2
Average GDP per capita growth Mean	0.0212	0.0175	0.0225
Standard deviation	0.0321	0.0370	0.0301
Initial GDP per capita			
Mean	4036.06	3358.96	4275.28
Standard deviation	5699.52	5684.08	5690.14
Average population growth			
Mean	0.0181	0.0214	0.0170
Standard deviation	0.0111	0.0115	0.0106
Average investment/GDP			
Mean	23.40	25.09	22.81
Standard deviation	14.21	23.62	8.79
Average trade openness			
Mean	69.00	68.50	69.18
Standard deviation	53.55	56.14	52.65
Lagged primary education			
Mean	0.3612	0.1357	0.4397
Standard deviation	0.1806	0.0536	0.1386
Observations	1030	266	764

Table 1.2 Descriptive statistics, non-fitted data—gender disaggregated—female

	Total	Regime 1	Regime 2
Average GDP per capita growth Mean	0.0212	0.0156	0.0229
Standard deviation	0.0321	0.0382	0.0298
Initial GDP per capita			
Mean	4036.06	2692.60	4438.78
Standard deviation	5699.52	5216.31	5779.25
Average population growth			
Mean	0.0181	0.0237	0.0165
Standard deviation	0.0110	0.0105	0.0107
Average investment/GDP			
Mean	23.40	24.64	23.02
Standard deviation	14.21	24.89	8.75
Average trade openness			
Mean	69.00	57.85	72.35
Standard deviation	53.55	38.80	56.84
Lagged primary education			
Mean	0.3418	0.0943	0.4162
Standard deviation	0.1932	0.0452	0.1547
Observations	1030	238	792

table also shows the same results using gender disaggregated human capital. In all circumstances, results strongly reject the null hypothesis, i.e. are in favor of the presence of a threshold.[2] Interestingly, the estimated threshold is lower for female education, meaning that lower levels of female educational attainment are required for an economy to move from one regime to the other.

Table 1.8 shows the estimated slope parameters in the absence of a sample split, which are obtained via ordinary least squares. These results are reported as a comparative benchmark to understand the effect that utilizing a threshold regression model has on the findings. Table 1.9, finally presents the estimation results utilizing the threshold regression model. Nonlinearities emerge very clearly from the table, which is evident by observing the differences in the estimated parameters between regimes. Focusing on human capital, it is interesting to notice that not only the coefficients of primary female education are significant in both regimes, but that the value of the parameter is much higher in regime 1 than in regime 2. This suggests that in low-education economies (i.e. those

Table 1.3 Descriptive statistics, non-fitted data—gender disaggregated—male

	Total	Regime 1	Regime 2
Average GDP per capita growth Mean	0.0212	0.0175	0.0223
Standard deviation	0.0321	0.0372	0.0304
Initial GDP per capita			
Mean	4036.06	4720.33	3841.53
Standard deviation	5699.52	7167.13	5196.39
Average population growth			
Mean	0.0181	0.0191	0.0178
Standard deviation	0.0111	0.0117	0.0108
Average investment/GDP			
Mean	23.40	25.80	22.71
Standard deviation	14.21	25.97	8.98
Average trade openness			
Mean	69.00	73.75	67.65
Standard deviation	53.55	61.60	50.95
Lagged primary education			
Mean	0.3801	0.1401	0.4483
Standard deviation	0.1797	0.0514	0.1403
Observations	1030	228	802

that fall under the threshold), female education has a greater effect on per capita GDP growth. These results are even more significant when compared to the estimated coefficients in Table 1.8: not only the slope parameter of female primary education in regime 1 is much higher than the same parameter estimated using OLS, but it is higher than the OLS-estimated total and male primary education parameters as well. Finally, in both Regimes, the coefficients of male human capital are not significant. Apart from that, it is interesting to notice that results are, qualitatively, similar to those obtained with female human capital, i.e. the coefficient for Regime 1 is larger than the coefficient for Regime 2 and the former is larger than the coefficient obtained without a sample split.

Table 1.4 Descriptive statistics, fitted data—not disaggregated by gender

	Total	Regime 1	Regime 2
Average GDP per capita			
growth Mean	0.0260	0.0244	0.0298
Standard deviation	0.0264	0.0254	0.0283
Initial GDP per capita			
Mean	7683.59	10,104.27	2246.68
Standard deviation	18,609.07	21,859.99	33033.43
Average population growth			
Mean	0.0143	0.0135	0.0161
Standard deviation	0.0161	0.0179	0.0110
Average investment/GDP			
Mean	23.10	23.57	22.03
Standard deviation	6.62	5.84	8.03
Average trade openness			
Mean	92.71	98.76	79.12
Standard deviation	57.57	64.55	33.89
Lagged primary education			
Mean	0.2766	0.2535	0.3284
Standard deviation	0.0466	0.0367	0.0117
Observations	409	283	126

1.4.2 The Threshold Model—Accounting for Endogeneity

Considering the literature linking human capital and institution, one could suspect that the results obtained above might be affected by an issue of endogeneity. This problem is addressed by using a Two Stage Least Squares (2SLS) approach, whereas in the first stage we use the following specification:

$$hc = \beta_0 + \beta Institutions \tag{1.8}$$

where *Institutions* is a vector of variables relative to institutional quality, as described in the "Data" section,[3] and β is a vector of parameters.

In other words, this means that when assessing the threshold effect of human capital allowing for endogeneity, the different measures of institutional quality are used as instruments. Since institutions are taken to be "slow moving" variables and as such, institutional quality may be

Table 1.5 Descriptive statistics, fitted data—disaggregated by gender—female

	Total	Regime 1	Regime 2
Average GDP per capita growth Mean	0.2641	–	–
Standard deviation	0.0272	–	–
Initial GDP per capita			
Mean	7342.68	–	–
Standard deviation	18,164.98	–	–
Average population growth			
Mean	0.0149	–	–
Standard deviation	0.0161	–	–
Average investment/GDP			
Mean	22.83	–	–
Standard deviation	6.81	–	–
Average trade openness			
Mean	91.79	–	–
Standard deviation	56.72	–	–
Lagged primary education			
Mean	0.2705	–	–
Standard deviation	0.0372	–	–
Observations	432	–	–

correlated with human capital accumulation, as the quality of education is affected by institutions (inclusion restriction), but the latter as they do not change much over time do not correlate with current growth (exclusion restriction).

A more complete approach allowing for an endogenous treatment of institutions as well would require data on institutional quality going further back in time. Institutional quality, in fact, is only partially available since 1995, while, for example, the data used in the first part of the analysis goes back to 1960. Due to this fact, obviously, the threshold regression will be based on less observations.

The 2SLS results for the linearity tests and the estimated threshold values are presented in Table 1.10. Interestingly, the test results still strongly reject the null hypothesis when using male primary education or total primary education, but show no evidence in support of a sample split for female primary education (Table 1.11).

Table 1.12 presents the slope parameters obtained using the threshold regression model. The results confirm the presence of non-linearities and

Table 1.6 Descriptive statistics, fitted data—disaggregated by gender—male

	Total	Regime 1	Regime 2
Average GDP per capita growth Mean	0.0264	0.0246	0.0296
Standard deviation	0.0272	0.0251	0.0306
Initial GDP per capita Mean	7342.68	10,334.41	1996.18
Standard deviation	18,164.98	22,041.11	2816.90
Average population growth Mean	0.0149	0.0130	0.0182
Standard deviation	0.0161	0.0179	0.0113
Average investment/GDP Mean	22.83	23.46	21.71
Standard deviation	6.81	5.99	7.97
Average trade openness Mean	91.79	100.46	76.30
Standard deviation	56.72	64.70	33.52
Lagged primary education Mean	0.2907	0.2518	0.3604
Standard deviation	0.0673	0.0485	0.0289
Observations	432	277	155

Table 1.7 Linearity test and threshold estimate, non-fitted data

Threshold variable: Lagged primary education			
	Total	Female	Male
LM test	23.7173	25.3344	26.7086
P value	0.0030	0.0020	0.0010
Threshold	0.1849	0.1226	0.2307

the estimated parameters for male fitted primary education are particularly interesting. In fact, not only the estimated values are significant in both regimes, but they show an important change from one regime to the other. In this situation the slope parameter is much higher in regime 2 than in regime 1, which is the opposite of what has been observed in the regressions not accounting for endogeneity regarding female primary education. While the coefficients of male human capital presented in Table 1.9 (non-fitted data) were not significant, it is interesting to notice that the results obtained using fitted data are quite different. In the former

Table 1.8 OLS regression without sample split, non-fitted data

Threshold variable: Lagged primary education			
	Total	Female	Male
Constant	0.0373***	0.0394***	0.0364***
	(0.0127)	(0.0125)	(0.0129)
Initial real GDP per capita	−0.0051***	−0.0054***	−0.0049***
	(0.0012)	(0.0012)	(0.0013)
Average population growth	−0.6064***	−0.5602***	−0.6501***
	(0.1527)	(0.1525)	(0.1515)
Average investment/GDP	0.0003***	0.0003***	0.0003***
	(0.0001)	(0.0001)	(0.0001)
Average trade openness	0.0040***	0.0039***	0.0042***
	(0.0014)	(0.0014)	(0.0014)
Lagged primary education	0.0253***	0.0272***	0.0213***
	(0.0049)	(0.0048)	(0.0050)
R-squared:	0.1711	0.1712	0.1668

***Denotes significance at 1%
**Denotes significance at 5%
*Denotes significance at 10%

case, in fact, the coefficient in Regime 2 was smaller than the coefficient in Regime 1, while the opposite is true when employing fitted data, as already noted in this paragraph.

These results suggest that female primary education has a direct effect on GDP growth per capita, while male primary education has an indirect effect, i.e. it requires good institutions in order to come to fruition. It is not surprising, then, that the effect of the former is higher in the low-education regime, which is, intuitively, associated with lower institutional quality and lower GDP, while the effect of the latter is more pronounced in the high-education regime, which is typical of countries with better institutions and better economies. Thus, a decision-maker wishing to increase GDP growth in a low-education country should prioritize female education, which is coherent with findings coming from a vast literature on development economics and gender.

Table 1.9 Threshold regression, non-fitted data

Threshold variable: Lagged primary education					
	Total		Female		Male
	Regime 1	Regime 2	Regime 1	Regime 2	Regime 1
Constant	0.0543***	0.0058	0.0431***	0.0151	0.0533***
	(0.0187)	(0.0155)	(0.0193)	(0.0153)	(0.0193)
Initial real GDP per capita	−0.0073***	−0.0027*	−0.0061**	−0.0037***	−0.0070***
	(0.0027)	(0.0013)	(0.0030)	(0.0012)	(0.0028)
Average population growth	−0.7712***	−0.3183	−0.6785**	−0.3389*	−0.6740***
	(0.2119)	(0.1822)	(0.2546)	(0.1739)	(0.2356)
Average investment/GDP	0.0001	0.0012***	0.00005*	0.0011***	0.00001
	(0.0001)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Average trade openness	0.0053*	0.0020	0.0051*	0.0020	0.0053*
	(0.0029)	(0.0016)	(0.0035)	(0.0015)	(0.0030)
Lagged primary education	0.0614*	0.0176**	0.0917*	0.0186***	0.0461
	(0.0312)	(0.0074)	(0.0595)	(0.0067)	(0.0456)
Observations	266	764	238	792	228
R-squared:	0.0883	0.1711	0.0666	0.1712	0.0763

Table 1.10 Linearity test and threshold estimate, fitted data

Threshold variable: Lagged primary education			
	Total	Female	Male
LM test	22.4419	12.2213	21.6482
P-value	0.0070	0.4250	0.0050
Threshold	0.2832	–	0.3086

Table 1.11 OLS regression without sample split, fitted data

Threshold variable: Lagged primary education			
	Total	Female	Male
Constant	−0.0062	0.0089	−0.0051
	(0.0189)	(0.0204)	(0.0176)
Initial real GDP per capita	−0.0033***	−0.0046***	−0.0034***
	(0.0011)	(0.0011)	(0.0011)
Average population growth	−0.4193***	−0.3329***	−0.4031***
	(0.1115)	(0.1099)	(0.1140)
Average investment/GDP	0.0008***	0.0007***	0.0008***
	(0.0002)	(0.0002)	(0.0002)
Average trade openness	0.0035	0.0037	0.0048**
	(0.0021)	(0.0022)	(0.0022)
Lagged primary education	0.1056***	0.0954***	0.0889***
	(0.0283)	(0.0343)	(0.0236)
R-squared:	0.2905	0.1036	0.1022

1.5 Conclusions

Different growth rates among countries depend on a multitude of factors. This chapter focused on how the interaction between human capital and institutional quality can influence economic growth.

The first part of the analysis, which did not consider institutional quality, employed a threshold regression model, where the variable of interest was the average growth of GDP per capita and the threshold variable was human capital. Specifically, the employed measure of human capital was lagged primary education, disaggregated by gender. The performed linearity tests strongly rejected the null hypothesis of no threshold in all scenarios. Estimating the parameters of the threshold regression

Table 1.12 Threshold regression, fitted data

Threshold variable: Lagged primary education					
	Total		Female		Male
	Regime 1	Regime 2	Regime 1	Regime 2	Regime 1
Constant	−0.0428***	−0.1962***	–	–	−0.0503
	(0.0203)	(0.0496)	–	–	(0.0199)
Initial real GDP per capita	−0.0027**	−0.0065***	–	–	−0.0019
	(0.0011)	(0.0031)	–	–	(0.0011)
Average population growth	−0.3647***	−1.1004***	–	–	−0.4472***
	(0.1200)	(0.2552)	–	–	(0.1138)
Average investment/GDP	0.0011***	0.0009***	–	–	0.0013***
	(0.0003)	(0.0003)	–	–	(0.0002)
Average trade openness	0.0073***	−0.0074	–	–	0.0071***
	(0.0021)	(0.0056)	–	–	(0.0021)
Lagged primary education	0.1439***	0.9198***	–	–	0.1391***
	(0.0416)	(0.1642)	–	–	(0.0342)
Observations	283	126	–	–	277
R-squared:	0.2899	0.2905	–	–	0.3592

the presence and relevance of non-linearities emerges very clearly, as parameter values change in magnitude when switching from one regime to the other. Interestingly, the effect of female primary education is far larger in the low-education regime.

Linkages between human capital and institutions might create an issue of endogeneity, which is addressed by using a 2SLS approach employing the institutional quality variables as instrumental variables. The results from this analysis support the presence of a sample split when looking at male primary education and total primary education, but show no evidence in favor of a threshold in the case of female primary education. The results for male fitted primary education are particularly interesting, with the estimated effect of male education on GDP growth being much lower in the low-education regime than in the high-education one.

These findings confirm the presence of non-linearities in the relationship between human capital and growth, while showing the importance that institutions and gender have on it. Specifically, regression results suggest that female primary education has a direct effect on GDP growth, independent from institutions, while male primary education works indirectly towards GDP growth with institutions "channeling" this effect. It is then intuitive the powerful effect that female primary education can have in low education economies.

Notes

1. Tables 1.1, 1.2, and 1.3 present statistics for non-fitted data, which is used in this section, while Tables 1.4, 1.5, and 1.6 display statistics for fitted-data, which is employed in the analysis presented in Sect. 1.4.1 of this chapter.
2. If a first threshold is proven to exist, tests for the presence of a second threshold also reject the null-hypothesis in all cases presented in this chapter. These supplementary results provide further evidence to confirm the non-linear relationship between human capital and growth. Similarly, the additional estimated coefficients for human capital reflect the qualitative findings obtained in the first round of threshold estimations. As these results align with and confirm those presented in the rest of the chapter they have been omitted from the presentation for the sake of brevity.

3. These are: Voice and Accountability, Political Stability and Absence of Violence/Terrorism, Government Effectiveness, Regulatory Quality, Rule of law, and Control of Corruption.

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2

Optimal Life-Cycle Education Decisions of Atomistic Individuals

Bernhard Skritek, Jesus Crespo Cuaresma, Arkadii V. Kryazhimskii, Klaus Prettner, Alexia Prskawetz, and Elena Rovenskaya

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2.1 Introduction

One of the main determinants of the aggregate stock of human capital in a society is the individual life-cycle decision of how much time to allocate to education. A thorough understanding of the underlying mechanisms

This chapter is dedicated to the memory of Arkadii V. Kryazhimskii, who passed away while working on it. He is deeply missed.

B. Skritek • A. Prskawetz
Institute of Statistics and Mathematical Methods in Economics,
Vienna University of Technology, Vienna, Austria
e-mail: afp@econ.tuwien.ac.at

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of individual investment patterns in education is therefore of crucial importance for economists and policymakers who are interested in (1) the determinants of human capital accumulation; (2) the consequences of human capital accumulation for macroeconomic outcomes and, particularly, for long-run economic growth; and (3) the economic policies that have the potential to affect the human capital stock of a society.

Models that address the optimal lifetime decision of individuals on investing in education versus participating in the labor market have a long tradition in theoretical economics. Seminal early contributions include work by, among others, Ben-Porath (1967), Ghez and Becker (1975), Blinder and Weiss (1976), Ryder et al. (1976), and Haley (1976). The typical result in these contributions is that the time invested in education changes over the life cycle. Individuals tend to invest more in education in earlier stages of their life and to reduce these investments later. One of the characteristics of this strand of the literature is that the results are obtained in a setting in which the interest rate and the wage rate are exogenously given.

Uzawa (1965) sets up a general equilibrium model with two state variables physical capital and technological knowledge, with the latter being labor augmenting. He postulates that investments in education raise the stock of knowledge over time and that a planner maximizes social welfare by choosing the optimal amount of time to be invested in education. He finds that the social optimum is attained when the capital-labor ratio accumulates at the same rate as technological knowledge. This

J. C. Cuaresma
Department of Economics, Vienna University of Economics and Business, Vienna, Austria
e-mail: Jesus.Crespo.Cuaresma@wu.ac.at

A. V. Kryazhimskii • E. Rovenskaya
International Institute of Applied Systems Analysis (IIASA), Laxenburg, Austria
e-mail: rovenska@iiasa.ac.at

K. Prettner (✉)
Institute of Economics, University of Hohenheim, Stuttgart, Germany
e-mail: klaus.prettner@uni-hohenheim.de

is the case for an interior optimal fraction of time that individuals in the economy spend in education.

Lucas (1988) builds on the contribution of Uzawa (1965) and investigates the choice of human capital accumulation trajectories within a general equilibrium long-run economic growth setting. In contrast to Uzawa (1965) he analyzes the decentralized economy and he allows for human capital externalities, i.e., that there are positive effects of the human capital of one individual on the productivity of other individuals. Altogether, his findings are well in line with Uzawa (1965) in the sense that the optimal education trajectory is to spend a constant part of available time on education throughout the entire infinite life of the decision maker.

In Lucas (1988), a representative individual maximizes her lifetime utility by choosing an optimal consumption path and the optimal allocation of her available time between education (human capital accumulation) and production. The problem that the individual faces is specified as an optimal control problem with two state variables: physical capital and human capital. The existence of a unique solution along a balanced growth path is derived under the assumptions of (1) perfect foresight, (2) perfect competition in labor and capital markets, and (3) that the decision of the agent has an effect on the interest rate and on the wage rate through general equilibrium feedbacks. A recent application of the Lucas model to answer the question whether educational investments can help to mitigate the negative economic effects that are expected in the wake of population aging is provided in the work of Edle von Gaessler and Ziesemer (2016). They show that, within the Lucas (1988) setting, an increase in education is indeed an optimal response to faster aging as represented by an increasing dependency ratio.

In this chapter we provide a rigorous analytical assessment of the optimal life-cycle education decision problem of an atomistic individual. In so doing, we rely on the standard definition of atomistic competition that each single agent is infinitesimally small compared to the rest of the market such that her actions do not affect equilibrium prices[1]. Thus, individual choices have no influence on the labor and capital markets in

our setting. Using this framework, we are able to show that the results of Lucas (1988) hinge on the assumption of an infinitely-lived representative agent whose choices determine the wage rate and the interest rate. The difference between our assumption and the assumption used by Lucas is therefore the following. In our case, the decisions of one individual do not affect the market prices (interest rate and wage rate) because one individual is atomistic. By contrast, in case of the Lucas model, one single representative individual determines aggregate labor supply and aggregate savings such that its decisions on labor supply and saving represent the aggregate household side. Thus, the decisions of an individual do not affect prices in our case, whereas they affect prices in the Lucas model.

While the mechanism proposed by Lucas acts as one possible equilibrium selection rule, it is restrictive and has been criticized on the grounds of being highly difficult to justify and implying misleading results (see, for example, Kirman, 1992; Farmer and Foley, 2009). We therefore relax the restrictive assumption of the representative agent and show that other mechanisms—such as nonlinearities in human capital production with respect to the time invested in education or finite lifetime horizons potentially coupled with plausible effects of aging on education costs—can also lead to the selection of a unique control in the individual optimization problem. The atomistic individual setting that we put forward is arguably a more realistic depiction of the situation in large economies and in economies in which labor market frictions such as minimum wages or binding collective wage bargaining agreements exist that prevent the general-equilibrium responses that are necessary for the balanced growth path solution of the Lucas model to be unique.

Our analysis delivers new and richer results for the time path of optimal education and human capital accumulation. As our central result, we show that, a priori, infinitely many optimal solutions for the time allocation between work and education exist for an atomistic infinitely lived individual. For example, three feasible optimal strategies are (1) allocating the whole available time to education when young and focusing on supplying labor when old (classical schooling); (2) switching back and forth between full-time education and full-time work (educational leave); (3) working part time and educating the rest of the time (continuous

lifetime learning as in the model of Lucas). The intuition for our central finding is that, without an effect of the atomistic individual's labor market decisions on the equilibrium wage rate, individuals only need to determine the optimal life-cycle labor supply, while the *distribution* of the life-cycle labor supply over time can be arbitrary. Income can be transferred over the life cycle to attain the optimal consumption growth path for the given optimal lifetime wage income.

The results derived in this chapter are important because—as this volume shows—human capital accumulation is the main driver of long-run economic development. The positive implications of education as one way to raise human capital have been investigated in the theoretical literature by, for example, Dalgaard and Kreiner (2001), Bucci (2008), and Strulik et al. (2013).[2] These models unambiguously show that higher investments in education raise long-run economic prosperity. This result is also supported by the available empirical evidence. Even in early cross-country growth regressions in the vein of Barro (1991), Sala-i-Martin (1997), and Sala-i-Martin et al. (2004), education is found to be positively associated with subsequent economic growth. More recent studies by de la Fuente and Domenéch (2006), Cohen and Soto (2007), Lutz et al. (2008), Hanushek and Woessmann (2012, 2015), and Cuaresma et al. (2014) confirm this pattern with better data and more modern estimation techniques. Thus, an understanding of the individual incentives for investing in education is crucial for policymakers whose aim is to raise future economic prosperity.

While our analysis focuses on the long-run solution along the balanced growth path, Bella et al. (2017) analyze the stability properties of the long-run equilibrium in the Lucas (1988) model. They find that complex behavior can emerge during the transition. For example, it is possible to have a continuum of solution paths that start at the same initial condition and that are consistent with perfect foresight.

The chapter is organized as follows. Section 2.2 revisits the standard representative agent framework of the life-cycle education decision that leads to the outcome of continuous lifetime education. In Sect. 2.3, we introduce the reformulation of the model for atomistic individuals

whose decisions do not affect labor and capital markets, together with the transformation of the model based on a two-stage formulation. In Sect. 2.4, we solve the model and present our main analytical results. In Sect. 2.5, we present three possible equilibrium selection mechanisms and discuss the effects of finite lifetime horizons and of aging on optimal education patterns. Finally, in Sect. 2.6, we discuss the results and present promising avenues for further research.

2.2 The Representative Agent Formulation

The prototype formulation of the optimal education decision problem of a representative individual is given by the framework put forward in Lucas (1988). In the following, we present this framework and modify it to account for cases in which individual actions do not affect wages and interest rates. This is particularly plausible if individuals are atomistic or if there are economy-wide minimum wages or economy-wide binding collective bargaining agreements on the wage level.

Consider a representative agent who maximizes the discounted flow of utility derived from consumption over her infinite life. At every point in time, t, the agent is endowed with one unit of time that can be split between production of output, $u(t)$, and production of human capital, $1-u(t)$. Effective labor $u(t)h(t)$, where $h(t)$ is the human capital level of the agent, is compensated by the wage rate $w(t)$. Savings are invested on the capital market and earn the real interest rate $r(t)$. Instantaneous utility is iso-elastic and future utility is discounted with the time preference rate $\rho > 0$. The problem of the representative individual is thus given by

$$\max_{c(t),u(t)} \int_0^\infty e^{-\rho t} \frac{c(t)^{1-\theta} - 1}{1-\theta} \, \mathrm{d}t, \tag{2.1}$$

subject to

$$\dot{k}(t) = r(t)k(t) + w(t)h(t)u(t) - c(t), \qquad k(0) = k_0 > 0, \tag{2.2}$$

$$\dot{h}(t) = \chi h(t)[1 - u(t)], \qquad h(0) = h_0 > 0, \tag{2.3}$$

$$u(t) \in [0, 1], \qquad c(t) \geq 0, \qquad \lim_{t \to \infty} e^{-\int_0^t r(s)\,\mathrm{d}s} k(t) \geq 0, \tag{2.4}$$

where $c(t)$ denotes consumption per capita at time t, $k(t)$ refers to physical capital holdings per capita, and $\theta > 0$ and $\chi > 0$ are parameters measuring the relative risk aversion of individuals and the productivity of education, respectively. The *No Ponzi Game Restriction*, $\lim_{t \to \infty} e^{-\int_0^t r(s)\,\mathrm{d}s} k(t) \geq 0$, rules out infinite borrowing to finance consumption.

Assuming $N(t)$ homogeneous agents, aggregate physical capital is given by $K(t) = N(t)k(t)$ and aggregate effective labor in the production process amounts to $L(t) = N(t)h(t)u(t)$. Both aggregate physical capital and aggregate effective labor are used to produce output $Y(t)$ according to a Cobb–Douglas production function with constant technology $A > 0$ and an elasticity of output with respect to physical capital of $\alpha \in (0, 1)$,

$$Y(t) = A\, K(t)^{\alpha} L(t)^{1-\alpha}.$$

Output can be consumed or invested in physical capital. In the standard formulation of the representative agent model by Lucas (1988), the wage rate per unit of effective labor, $w(t)$, and the capital rental rate, $R(t) = r(t) + \delta$, with δ being the rate of depreciation, are determined in competitive markets. Profit maximization at the firm level (assuming that the price of output is chosen as the numéraire) implies

$$w(t) = \frac{\partial Y(t)}{\partial L(t)} = A(1 - \alpha) \left[\frac{K(t)}{L(t)} \right]^{\alpha},$$

$$R(t) = \frac{\partial Y(t)}{\partial K(t)} = A\alpha \left[\frac{L(t)}{K(t)} \right]^{1-\alpha}.$$

Without loss of generality, we assume a stationary population and normalize it to $N(t) = 1$ for all t. Altogether, Lucas (1988) derives a balanced growth path where, the variables of the model grow at constant rates, with the optimal equilibrium allocation for $u(t)$ being determined as the market equilibrium between firms and households and finds a unique solution. In our contribution, we analyze the problem of an atomistic individual whose decisions have no impact on wages and interest rates and show that such a setting has the potential to lead to optimal life-cycle education paths that are better aligned with actual observable patterns.

2.3 Reformulation of the Model for Atomistic Agents

In our reformulation of the model for atomistic agents we assume that the paths of the interest rate and the wage rate per unit of effective labor, $r(t)$ and $w(t)$, are given, known to the agent, and not affected by the agent's decisions on savings and/or labor market participation. The individual maximizes her discounted lifetime utility given by Eq. (2.1), subject to constraints (2.2)–(2.4), conditional on given paths for $r(t)$ and $w(t)$ with $w(t)$ being a positively valued continuously differentiable function and $r(t)$ being a positively valued integrable function.

The optimization problem given by Eqs. (2.1)–(2.4) can be reformulated as a two-stage problem. The individual first chooses the allocation of time between production and education so as to maximize her total discounted income over the life cycle. In the second stage, the individual decides on the optimal distribution of consumption over the life cycle for the given income that results in the first stage of the optimization problem.[3] The result requires the assumption that the function $w(t)$ is such that for any admissible control $u(t)$ and the corresponding trajectory $h(t)$, total discounted lifetime income is finite,

$$\int_0^\infty e^{-r(t,0)} w(t)h(t)u(t)\,\mathrm{d}t < \infty, \tag{2.5}$$

where $r(t,s) = \int_s^t r(\tau)\,\mathrm{d}\tau$ is the accumulated interest rate between time s and time t. If there were some control $u(t)$ for which the integral

above was infinite, this would imply infinite consumption at time $t = 0$. Clearly, this is infeasible from an economic point of view.

We now split the optimization problem (2.1)–(2.4) into two parts. The *education problem* aims to maximize total discounted lifetime income,

$$\max_{u(t)} \int_0^\infty e^{-r(t,0)} w(t)u(t)h(t)\,\mathrm{d}t, \tag{2.6}$$

subject to the dynamics of human capital

$$\dot{h}(t) = \chi h(t)[1 - u(t)], \qquad h(0) = h_0. \tag{2.7}$$

The *consumption problem* aims to maximize the original utility

$$\max_{c(t)} \int_0^\infty e^{-\rho t} \frac{c(t)^{1-\theta} - 1}{1-\theta}\,\mathrm{d}t, \tag{2.8}$$

subject to the dynamics of an auxiliary variable $x(t)$

$$\dot{x}(t) = r(t)x(t) - c(t), \qquad x(0) = x_0 + \int_0^\infty e^{-r(t,0)} w(t)u(t)h(t)\,\mathrm{d}t, \tag{2.9}$$

$$\lim_{t\to\infty} e^{-r(t,0)} x(t) \geq 0. \tag{2.10}$$

The following lemma holds true.

Lemma 2.1 *Problem (2.1)–(2.4) is equivalent to the combination of problems (2.6)–(2.7) and (2.8)–(2.10). Namely, if $(c^*(t), h^*(t))$ is an optimal pair in (2.1)–(2.4), then $c^*(t)$ is a solution to (2.8)–(2.10) and $h^*(t)$ is a solution to (2.6)–(2.7). Also, if $c^*(t)$ is a solution to (2.8)–(2.10) and $h^*(t)$ is a solution to (2.6)–(2.7), then $(c^*(t), h^*(t))$ is a solution to (2.1)–(2.4).*

Proof See Appendix 1. □

Lemma 2.1 establishes the following. If (2.5) is fulfilled, then the original problem given by Eqs. (2.1)–(2.4) is equivalent to the combination of (1) the problem of choosing the allocation of time between production and education in order to maximize total discounted lifetime income and (2) the problem of choosing a consumption path that maximizes utility, given total discounted lifetime income.

The *consumption problem* has been studied in the literature and its optimal solution is given by the following expressions

$$c^*(t) = c_0^* e^{\frac{r(t,0)-\rho t}{\theta}}, \qquad c_0^* = \frac{x(0)}{\int_0^\infty e^{-r(t,0)} e^{\frac{r(t,0)-\rho t}{\theta}} \, \mathrm{d}t}. \tag{2.11}$$

The proof can be found in standard textbooks (see, for example, Acemoglu, 2009, pp. 294–298).

The decomposition of the optimization problem simplifies the mathematical analysis considerably. Intuitively, it shows that we do not need to think of individuals as making their decisions on $u(t)$ and $c(t)$ simultaneously. Instead, at the first stage, individuals maximize their income by optimally choosing the allocation of time between education and work. At the second stage, optimal consumption is determined. This decision does not depend on the actual flow of income but only on its net present value at time zero.

Next, we analyze the *education problem*.

Proposition 2.1 *Let (2.5) be fulfilled, $u^*(t)$ be an optimal control in the education problem, and $h^*(t)$ be the trajectory corresponding to $u^*(t)$. Then there exists an adjoint variable $\xi(t)$ defined as*

$$\xi(t) = \int_t^\infty e^{-r(s,0)} w(s) u^*(s) e^{-\chi \int_t^s [1-u^*(\tau)] \, \mathrm{d}\tau} \, \mathrm{d}s \tag{2.12}$$

that satisfies almost everywhere the adjoint equation

$$-\dot{\xi}(t) = \chi[1 - u^*(t)]\xi(t) + e^{-r(t,0)}w(t)u^*(t). \tag{2.13}$$

The optimal control $u^*(t)$ *maximizes the Hamiltonian*

$$\mathcal{H}(h, u, \xi, t) = e^{-r(t,0)}w(t)hu + \chi\xi h(1 - u)$$

evaluated along the optimal path $h = h^*(t)$, $\xi = \xi(t)$ *over all admissible* $u \in [0, 1]$ *for almost all* $t \in [0, \infty)$. *As a consequence,*

$$u^*(t) = \begin{cases} 1, & \text{if } e^{-r(t,0)}w(t) - \chi\xi(t) > 0, \\ 0, & \text{if } e^{-r(t,0)}w(t) - \chi\xi(t) < 0, \\ \text{singular}, & \text{if } e^{-r(t,0)}w(t) - \chi\xi(t) = 0. \end{cases} \tag{2.14}$$

If $e^{-r(t,0)}w(t) - \chi\xi(t) = 0$ then any $u \in [0, 1]$ maximizes the Hamiltonian. Thus, the maximum principle does not provide any useful information for deriving the optimal control. Solutions for which $e^{-r(t,0)}w(t) - \chi\xi(t) = 0$ are called singular solutions because their value at time t cannot be defined from the gradient being equal to zero. From (2.14), it follows that any solution for which $0 < u^*(t) < 1$ in $[t_0, t_1]$ must be singular in this interval. However, the reverse implication is not true, a singular solution can still take the values $u^*(t) = 0$ and $u^*(t) = 1$ over some intervals.

Proof See Appendix 1. □

2.4 Multiplicity of Optimal Solutions

After reformulating the problem, we now show that, for an individual who faces exogenous wages and interest rates, infinitely many optimal solutions to the life-cycle education problem exist. As a side result, we obtain a condition on the interest rate and the wage rate for a singular solution to exist.

Theorem 2.1 *If an optimal balanced growth path with positive human capital growth exists for the problem given by Eq.* (2.1) *subject to Eqs.* (2.2)–(2.4)*, infinitely many optimal solutions for the education problem exist. They are characterized by*

$$\lim_{t\to\infty} \int_0^t u(s)\,\mathrm{d}s \to \infty. \tag{2.15}$$

We start by formulating one finding that is required for the proof in the form of a corollary.

Corollary 2.1 *If a singular solution exists for the problem given by Eq.* (2.1) *subject to Eqs.* (2.2)–(2.4)*, then*

$$\frac{\dot{w}(t)}{w(t)} = [r(t) - \chi], \quad t \in [0, \infty). \tag{2.16}$$

All admissible controls $u(t)$ that satisfy Eq. (2.15) *lead to the same value of the objective function in the education problem and the discounted future income is proportional to the initial level of human capital,*

$$\int_0^\infty e^{-r(t,0)} w(t)u(t)h(t)\,\mathrm{d}t = h(0)\frac{w(0)}{\chi}. \tag{2.17}$$

This implies that, for a singular solution with a constant wage rate per unit of effective labor and a constant interest rate, the interest rate must satisfy $r(t) \equiv \chi$, while the wage rate per unit of effective labor may be any arbitrary positive constant. This equality represents an equilibrium where both human capital and physical capital grow at the same rate.

Proof of Theorem 2.1 See Appendix 1. □

The following theorem defines the settings in which a balanced growth path exists in our framework.

Theorem 2.2 *Let the wage rate per unit of effective labor be constant,* $w(t) \equiv w > 0$*, let the interest rate be equal to the efficiency of human capital production,* $r(t) \equiv \chi$*, for all* $t \in [0, \infty)$*, and let additionally* $\chi(1-\theta) < \rho$ *and* $0 < (\chi - \rho)/(\chi\theta) < 1$*. Then a balanced growth path exists and the growth rate of both physical and human capital is* $(\chi - \rho)/\theta$*.*

Proof See Appendix 1. □

Note that the optimality of the control and the assertion of the theorem are independent of $k(0)$ and $h(0)$, i.e., the optimality of the control is robust with respect to perturbations in the initial conditions. This implies that, for two agents with different initial human capital $h_1(0) \neq h_2(0)$, and with both agents choosing the balanced growth path solution, the difference $h_1(t) - h_2(t)$ grows exponentially over time, but the fraction $h_1(t)/h_2(t)$ remains constant. The same holds true for physical capital.

The condition $r(t) \equiv \chi$ in Theorem 2.2 may seem restrictive at first glance, but as shown above, the existence of a singular solution implies already that this condition must hold. For completeness, we also mention that there exist reasonable parameter combinations such that the conditions above are met, e.g. $(\theta = 1, \rho = 0.04, \chi = 0.05)$ or $(\theta = 2, \rho = 0.01, \chi = 0.1)$ or $(\theta = 0.5, \rho = 0.06, \chi = 0.1)$.

The intuition behind this result is that individuals only need to determine the optimal life-cycle labor supply, while the *distribution* of the life-cycle labor supply over time does not matter. The reason is that, due to a perfect capital market, income can be transferred over the life cycle to achieve the optimal consumption growth path for the given optimal lifetime wage income. Due to the linearity of the human capital dynamics, all education paths are optimal for which a positive amount of the infinite lifetime is devoted to labor.[4]

Out of the infinitely many solutions that exist, the general equilibrium mechanism in a representative agent setting such as the one proposed by Lucas (1988) identifies one of them. Other possible mechanisms to choose a unique solution are discussed in Sect. 2.5.

Note that if we consider a constant control $u(t) \equiv u_0$, then $h(t) = h(0)e^{\chi(1-u_0)t}$. As shown in the proof of Theorem 2.2, $I(t) = I(0)e^{\frac{\chi-\rho}{\theta}t}$.

Inserting both terms into the equation $I(t) = \chi k(t) + wh(t)$, we obtain information on the evolution of physical capital in the long run:

$$k(t) = \frac{1}{\theta}\left[I(0)e^{\frac{\chi-\rho}{\theta}t} - wh(0)e^{\chi(1-u_0)t}\right].$$

In the long run, $\dot{k}(t)/k(t) = \max\{\chi(1-u_0)t, (\chi-\rho)/\theta\}$. If physical capital $k(t)$ grows at the same rate as human capital $h(t)$ and faster than total income $I(t)$, then the agent is indebting herself increasingly and invests in education. If $k(t)$ grows faster than human capital, the agent is saving in terms of physical capital. The balanced growth path is the solution for which both human and physical capital grow at the same rate.

2.5 Solution Selection Mechanisms and the Effects of Aging on Optimal Education Paths

In this section we discuss some potential approaches to select a unique optimal solution in the optimization problem of the atomistic individual. Our results show that individuals have infinitely many optimal choices for the control $u(t)$. Among the several solution selection mechanisms available to overcome this indeterminacy, we highlight three that introduce a nonlinearity in the Hamiltonian of the education problem: the *general equilibrium* formulation of the model (as in Lucas, 1988), the introduction of a *nonlinearity in the human capital accumulation* function, and the *effects of aging*.

If the interest rate and the wage rate per unit of effective labor are determined endogenously in a general equilibrium setting by the interaction of the choices of the representative agent with those of a representative firm, the problem of this representative agent has a unique solution. This is the case because labor supply is increasing with respect to the wage rate, while labor demand is decreasing. Thus, there exists a unique intersection of labor demand and labor supply at the unique

equilibrium wage. If the representative agent decreases (increases) her labor supply, wages rise (fall) and agents have an incentive to increase (decrease) labor supply accordingly. Consequently, it is optimal to choose a smooth education profile over the life cycle, i.e., to continuously educate oneself. Altogether, the general equilibrium framework acts as a "selector" of a unique optimal choice of the control.

Analytically, for a given control $u(t)$, a production function $Y(t) = F[K(t), L(t)]$, and the assumption that the wage equals the marginal product of labor, it holds that

$$w[t, u(t)] = \left. \frac{\partial F[K(t), L(t)]}{\partial L(t)} \right|_{[K(t),L(t)]=[K(t),u(t)H(t)]}.$$

Inserting this expression into the Hamiltonian of the *education problem*, the optimal control $u^*(t)$ maximizes

$$\mathcal{H}(h, u, \xi, t) = w[t, u(t)]u(t)h(t)e^{-r(t,0)} + \chi\xi(t)h(t)[1 - u(t)]$$

over the set $u(t) \in [0, 1]$. If $w[t, u(t)]u(t) + \chi\xi(t)e^{r(t,0)}$ is a concave function in $u(t)$, then a unique maximum $u^*(t)$ exists. This holds true, for example, for a Cobb–Douglas production function (as used in the model of Lucas, 1988). In the Cobb–Douglas case, the unique optimal control is given by

$$u^*(t) = \begin{cases} \left[\frac{\xi(t)\chi e^{r(t,0)}}{h(t)^{1-\alpha}A(1-\alpha)^2k(t)^\alpha}\right]^{-\frac{1}{\alpha}}, & \text{if } \left[\frac{\xi(t)\chi e^{r(t,0)}}{h(t)^{1-\alpha}A(1-\alpha)^2k(t)^\alpha}\right]^{-\frac{1}{\alpha}} \leq 1, \\ 1, & \text{if } \left[\frac{\xi(t)\chi e^{r(t,0)}}{h(t)^{1-\alpha}A(1-\alpha)^2k(t)^\alpha}\right]^{-\frac{1}{\alpha}} > 1. \end{cases}$$

A second mechanism that acts as a solution selector is a non-linearity in human capital production. In our setting, the linearity of $\dot{h}(t)$ and $\dot{k}(t)$ with respect to $u(t)$ and $h(t)$ together with the equality of marginal returns in equilibrium imply that individuals are indifferent between all controls $u(t)$ that result in the same amount of working time. Thus, switching back and forth between education and work becomes also an optimal behavior for individuals in our setting. Introducing a nonlinearity

with respect to $u(t)$ in the human capital accumulation equation is another possible way to achieve uniqueness in this case (see Uzawa, 1965).

The third mechanism is particularly important in face of an aging population in industrialized countries. Individuals do not live indefinitely as in the Lucas (1988) model and, while human capital might be partially inherited by the children because they observe their parents and learn from them, the assumption of a full inheritance of human capital (which is an alternative interpretation for the infinite lifetime horizon of Lucas, 1988) is very unrealistic. In case of a finite life, an optimal strategy would be to concentrate educational investments on younger ages so as to be able to reap the benefits of education over a longer working life (see, for example, Hazan and Zoaby, 2006; Hazan, 2009; Cervellati and Sunde, 2013; Strulik and Werner, 2016, for discussions of how changes in life expectancy might impact upon optimal education decisions). Changes in the costs of education over the life cycle in the sense that it becomes more difficult to learn as individuals grow older complement this effect. As we all know only too well, assuming such a life-cycle cost pattern of education may not be entirely unrealistic.

2.6 Conclusions and Avenues for Further Research

We show that the optimal life-cycle education problem of atomistic individuals exhibits multiple solutions. In particular, if a balanced growth path exists, all solutions that satisfy the condition $\lim_{t\to\infty} \int_0^t u(s)\,\mathrm{d}s \to \infty$ are optimal. Thus, all of the following stylized categories of individual behavior, which are illustrated graphically in Fig. 2.1, are rational for atomistic agents:

- Educational Leave: Switching back and forth between work and education, e.g., $u(t) \equiv 1$ for $t \in [2n, 2n+1)$ and $u(t) \equiv 0$ for $t \in [2n-1, 2n)$ and $n \in \mathbb{N}$.
- No Learning: Working full time without additional education, $u(t) \equiv 1$ for all $t \in [0, \infty)$.

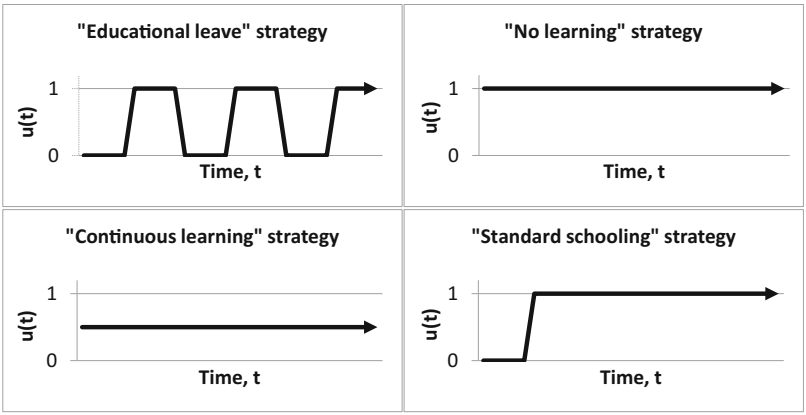

Fig. 2.1 Graphical illustration of potentially optimal solution paths for the fraction of time spent working, $u(t)$

- Continuous Learning: Working part time and educating the rest of the time $0 < u(t) \equiv u < 1$ for all $t \in [0, \infty)$.
- Standard Schooling: Full-time education, $u(t) \equiv 0$, for $t \in [0, s]$ and full-time work, $u(t) \equiv 1$, for $t \in (s, \infty)$.

The assumptions leading to the equivalence of these strategies are the existence of a singular solution, the linearity of human capital dynamics, and the infinite lifetime horizons of individuals, or, in the dynastic interpretation, that human capital is fully inherited. In case of a setting in which agents have finite lives and cannot pass the acquired human capital to children, we would expect a decline in investment in education at older ages. Similarly, if human capital accumulation were less costly for younger individuals than older ones, an optimal solution might imply full-time education in younger ages, perhaps followed by a period of part-time education, and finally full-time work at older ages (until retirement). A non-linear human capital production function would shift the optimal solution either to continuous learning (in case of human capital production being concave in time devoted to learning) or to educational leave (in case of human capital production being convex in time

devoted to learning). Altogether, this implies that our framework is able to deliver potentially realistic age-education profiles. The richness of the results for human capital dynamics under additional assumptions renders the proposed framework particularly promising for further research on optimal individual education paths.

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Appendix 1: Proofs

Proof of Lemma 2.1 Consider an arbitrary admissible control $u(t)$ and its corresponding human capital path $h(t)$ being a solution to Eq. (2.3). Let $x(t)$ and $z(t)$ be solutions to

$$\dot{x}(t) = r(t)x(t) - c(t), \quad x(0) = k_0 + \int_0^\infty e^{-r(t,0)} w(t)h(t)u(t)\,\mathrm{d}t \tag{2.18}$$

and

$$\begin{aligned} \dot{z}(t) &= r(t)z(t) + w(t)h(t)u(t), \\ z(0) &= -\int_0^\infty e^{-r(t,0)} w(t)h(t)u(t)\,\mathrm{d}t. \end{aligned} \tag{2.19}$$

Then, $k(t) = x(t) + z(t)$ for all $t \geq 0$. Note that (2.5) and (2.19) imply that

$$\lim_{t\to\infty} z(t) = \lim_{t\to\infty} -\int_t^\infty e^{-r(s,t)} w(s)h(s)u(s)\,\mathrm{d}s = 0$$

such that the No Ponzi Game condition given by Eq. (2.4) leads to

$$\begin{aligned}\lim_{t\to\infty} e^{-r(t,0)}k(t) &= \lim_{t\to\infty} e^{-r(t,0)}x(t) + \lim_{t\to\infty} e^{-r(t,0)}z(t)\\ &= \lim_{t\to\infty} e^{-r(t,0)}x(t) \geq 0. \end{aligned} \tag{2.20}$$

The state variable $z(t)$ does not play any role for the optimization problem with objective function given by Eq. (2.1) and can be omitted in further considerations.

Consider the No Ponzi Game condition in Eq. (2.20). From Eq. (2.18) it follows that

$$x(t) = e^{r(t,0)}\left[k_0 + \int_0^\infty e^{-r(s,0)} w(s)h(s)u(s)\,\mathrm{d}s - \int_0^t e^{-r(s,0)} c(s)\,\mathrm{d}s\right]$$

and so the condition given by Eq. (2.20) requires that

$$\int_0^\infty e^{-r(s,0)} c(s)\,\mathrm{d}s \leq k_0 + \int_0^\infty e^{-r(s,0)} w(s)h(s)u(s)\,\mathrm{d}s. \tag{2.21}$$

This inequality implies that the set of admissible controls $c(t)$ is the largest if the term $\int_0^\infty e^{-r(s,0)} w(s)h(s)u(s)\,\mathrm{d}s$ takes the maximal possible value. Thus, maximizing the right hand side of Eq. (2.21) will allow maximizing utility as given by the objective function in Eq. (2.1). Therefore, the original problem given by Eqs. (2.1)–(2.4) is reducible to the two separate problems put forward above. □

Proof of Proposition 2.1 The statement of the proposition follows from Theorem 4.1 in Aseev and Veliov (2014): Assumption (A1) is fulfilled by the dynamics of the problem, and Assumption (A2) in Theorem 4.1 is

satisfied because the objective function is finite for all admissible controls. For finite objective functions, the notion of weakly overtaking optimality and strong optimality coincide (cf. Definition 2.3 and 2.5 in Aseev and Veliov, 2014). Furthermore, the notion of local weak overtaking optimality is weaker than the property of weak overtaking optimality (cf. footnote 3 in Aseev and Veliov, 2014). Hence, any strongly optimal control is also locally weak overtaking optimal. Thus, the theorem can be applied for this problem and implies that the Maximum Principle holds for the optimal control and the adjoint function can be explicitly stated as in the proposition.

The derivation of functional forms in Proposition 2.1 is purely technical and given in Appendix 2. Note that in the result of Aseev and Veliov (2014) the adjoint multiplier of the objective function in the definition of the Hamiltonian is equal to 1, which simplifies the analysis compared to other results in the literature. □

Proof of Theorem 2.1 We divide the proof into three steps. First, we show that the existence of an optimal balanced growth path with positive human capital growth implies the existence of a singular solution. Then, we derive a necessary condition for an optimal solution to exist. Finally, we show that infinitely many optimal solutions exist.

The assumption of an optimal balanced growth path with positive human capital growth implies that, for some constant $d > 0$, it holds that $d = \dot{h}(t)/h(t) = \chi[1 - u^*(t)]$. Thus, $u^*(t)$ is constant almost everywhere, $u^*(t) \equiv 1 - d/\chi < 1$, for almost all $t \in [0, \infty)$. The boundary value $u^*(t) \equiv 0$ has objective value zero in the education problem, which is clearly not optimal because controls that lead to a positive objective value exist. Therefore, $0 < u^*(t) < 1$ for almost all $t \in [0, \infty)$, which proves that a singular optimal solution exists.

Next, we derive the necessary condition for a singular solution presented in Corollary 2.1 by using the optimality conditions of Proposition 2.1. Consider the switching function $\zeta(t)$, defined as

$$\zeta(t) = e^{-r(t,0)} w(t) - \chi \xi(t).$$

If a control is singular over the interval $[t_1, t_2) \subset [0, \infty)$, then the switching function $\zeta(t)$ is zero almost everywhere, which implies

$$\chi \xi(t) = e^{-r(t,0)} w(t).$$

Furthermore, $\dot{\zeta}(t) = 0$ for $t \in [t_1, t_2)$. Using the adjoint equation (2.13), the derivative of $\zeta(t)$ with respect to time is given by

$$\begin{aligned}
\dot{\zeta}(t) &= -r(t)e^{-r(t,0)}w(t) + e^{-r(t,0)}\dot{w}(t) - \chi\dot{\xi}(t) = \\
&= -r(t)e^{-r(t,0)}w(t) + e^{-r(t,0)}\dot{w}(t) \\
&\quad + \chi[1 - u^*(t)]e^{-r(t,0)}w(t) + \chi e^{-r(t,0)}w(t)u^*(t) = \\
&= e^{-r(t,0)}\left[-r(t)w(t) + \dot{w}(t) + \chi w(t)\right] = \\
&= e^{-r(t,0)}w(t)\left\{\frac{\dot{w}(t)}{w(t)} - [r(t) - \chi]\right\}.
\end{aligned}$$

The condition $\dot{\zeta}(t) = 0$ implies that the first condition in Corollary 2.1 given by Eq. (2.16) is fulfilled.

Note that the conditions $\zeta(t) = 0$ and $\dot{\zeta}(t) = 0$ are independent of the control $u(t)$. If these conditions are fulfilled, every control $u(t)$ satisfies the necessary optimality conditions and therefore infinitely many solutions and infinitely many singular solutions exist. We now show that all of these solutions are optimal. Considering any control $u(t)$, the objective value of the education problem is given by[5]

$$\int_0^\infty e^{-r(t,0)}w(t)u(t)h(t)\,\mathrm{d}t = \frac{w(0)h(0)}{\chi}\left[1 - e^{-\chi \lim_{t\to\infty}\int_0^t u(s)\,\mathrm{d}s}\right]. \tag{2.22}$$

The objective value is thus maximized and equal to $w(0)h(0)/\chi$ for any control $u(t)$ that satisfies Eq. (2.15). This equation is in turn satisfied by infinitely many solutions, which proves the claim of the theorem. By shifting the initial point in time it follows that, along every optimal control, discounted future income is always equal to the current level of human capital multiplied by $w(t)/\chi$. □

Proof of Theorem 2.2 If $r(t)$ and $w(t)$ are constant, it follows that $r(t) \equiv \chi$ for $t \in [0, \infty)$, while the wage rate per unit of effective labor is undetermined, $w(t) \equiv w > 0$.

Consider the maximal achievable income $I(t) := \chi k(t) + wh(t)$, which is equal to the income at time t under a constant interest rate and a constant wage rate if the agent works full time. Taking the derivative of $I(t)$ with respect to time, we obtain

$$\begin{aligned}\dot{I}(t) &= \chi \dot{k}(t) + w\dot{h}(t) = \\ &= \chi[\chi k(t) + wu(t)h(t) - c(t)] + w\{h(t)\chi[1 - u(t)]\} = \\ &= \chi[\chi k(t) + wh(t) - c(t)] = \\ &= \chi[I(t) - c(t)].\end{aligned}$$

Consumption can be rewritten as

$$c^*(t) = I(t)\left[\frac{\rho - \chi(1-\theta)}{\theta\chi}\right]. \tag{2.23}$$

Inserting this expression into the equation for $\dot{I}(t)$, we obtain that the maximal achievable income grows at a constant rate, independently of the control $u(t)$,

$$\frac{\dot{I}(t)}{I(t)} = \frac{\chi - \rho}{\theta}.$$

Due to our assumption $0 < (\chi - \rho)/(\chi\theta) < 1$, there exists $\tilde{u} \in (0, 1)$ such that $\dot{h}(t)/h(t) = \chi(1 - \tilde{u}) = (\chi - \rho)/\theta$. Inserting this $\tilde{u}$ and the expression for $c^*(t)$ into the equation for $\dot{k}(t)$, we obtain

$$\frac{\dot{k}(t)}{k(t)} = \frac{\chi - \rho}{\theta}.$$

Due to Corollary 2.1, the control $u(t) \equiv \tilde{u}$ is also optimal, which concludes our proof of the existence of a balanced growth path. □

Appendix 2: Derivations

Derivation of Eqs. (2.12) and (2.13)

According to Theorem 4.1 in Aseev and Veliov (2014), the adjoint is defined by $\xi(t) = Z(t)I(t)$ with $Z(t)$ being the fundamental matrix solution to the problem

$$\dot{z}(t) = -\chi[1 - u(t)]z(t),$$

and

$$I(t) = \int_t^\infty Z(s)^{-1} e^{-r(s,0)} w(s)u(s)\,\mathrm{d}s.$$

The solution to the differential equation with some initial condition $z(0) = z_0$ is

$$z(t) = e^{-\chi \int_0^t [1-u(\tau)]\,\mathrm{d}\tau} z_0 = Z(t)z_0.$$

Therefore,

$$\xi(t) = Z(t)I(t) = e^{-\chi \int_0^t [1-u(\tau)]\,\mathrm{d}\tau} \int_t^\infty e^{\chi \int_0^s [1-u(\tau)]\,\mathrm{d}\tau} e^{-r(s,0)} w(s)u(s)\,\mathrm{d}s.$$

Simplifying the expression results in Eq. (2.12).

Derivation of Eq. (2.22)

For a given control path $u(t)$, human capital at time t is given by

$$h(t) = h(0)e^{\int_0^t \chi[1-u(s)]\,\mathrm{d}s}.$$

Using this expression, we obtain

$$\int_0^\infty e^{-r(t,0)} w(t) u(t) h(t)\,\mathrm{d}t = \int_0^\infty \left[e^{-r(t,0)} e^{\chi t} w(t)\right] \left[h(0) u(t) e^{-\chi \int_0^t u(s)\,\mathrm{d}s}\right] \mathrm{d}t.$$

Integrating by parts, where the first square bracket is differentiated, while the second is integrated, results in

$$e^{-r(t,0)} e^{\chi t} w(t) \frac{h(0)}{\chi} e^{-\chi \int_0^t u(s)\,\mathrm{d}s} \Big|_{t=0}^{\infty}$$
$$- \int_0^\infty [\dot{w}(t) + \chi w(t) - r(t) w(t)] e^{-r(t,0)} e^{\chi t} h(0) e^{-\chi \int_0^t u(s)\,\mathrm{d}s}\,\mathrm{d}t.$$

Since the wage satisfies Eq. (2.16), the integrand of the second term is zero. Equation (2.16) further implies that $w(t) = w(0) e^{r(t,0)} e^{-\chi t}$, which leads to the first term being equal to

$$\frac{w(0) h(0)}{\chi} \left[1 - e^{-\chi \lim_{t\to\infty} \int_0^t u(s)\,\mathrm{d}s}\right],$$

which is Eq. (2.22).

Derivation of Eq. (2.23)

Equation (2.23) is a representation of optimal consumption—using the notion of "maximal achievable income" $I(t)$—which is not common. Therefore, we sketch how to obtain this formulation from the known equation (2.11).

We start by applying a time shift: exchanging in Eq. (2.11) for c_0^* the integration variable t by s and then the 0 by t, we obtain

$$c^*(t) = c_t^*, \qquad c_t^* = \frac{x(t)}{\int_t^\infty e^{-r(s,t)} e^{\frac{r(s,t)-\rho(s-t)}{\theta}}\,\mathrm{d}s}.$$

Since $r(t) \equiv \chi$ for all $t \in [0, \infty)$, it follows that $r(s,t) = \chi(s-t)$ and the integral in the denominator can be solved:

$$\int_t^\infty e^{-r(s,t)} e^{\frac{r(s,t)-\rho(s-t)}{\theta}} \, \mathrm{d}s = \left[\frac{\theta}{\rho - \chi(1-\theta)}\right].$$

The numerator $x(t)$ is by definition equal to the future discounted income plus today's assets,

$$x(t) = k(t) + \int_t^\infty e^{-r(s,t)} w(s)u(s)h(s) \, \mathrm{d}s,$$

where the integral is, for any optimal control, equal to $w(t)h(t)/\chi$, see Eq. (2.17) and the first paragraph of Appendix 2. Combining the equations above, we obtain Eq. (2.23):

$$\begin{aligned} c^*(t) = c_t^*; \qquad c_t^* &= [\chi k(t) + wh(t)] \left[\frac{\rho - \chi(1-\theta)}{\theta\chi}\right] \\ &= I(t) \left[\frac{\rho - \chi(1-\theta)}{\theta\chi}\right]. \end{aligned}$$

Notes

1. See, for example, the Encyclopædia Britannica (2018); stating that "When the number of sellers is quite large, and each seller's share of the market is so small that in practice he cannot, by changing his selling price or output, perceptibly influence the market share or income of any competing seller, economists speak of atomistic competition[. . .]".
2. In Romer (1990), human capital is the central determinant of economic growth because it is the crucial input in the production of new knowledge. This idea has been formalized and further developed by Funke and Strulik (2000), Dalgaard and Kreiner (2001), Strulik (2005), Bucci (2008), Strulik et al. (2013), Prettner (2014), and Prettner and Strulik (2016). For other beneficial effects of education see Lee and Mason (2010), Venti (2015), and Mason et al. (2016).

3. In the model, the feasibility of such a decomposition rests on the assumptions of the lack of disutility of work beyond the opportunity costs of investing in human capital and that capital markets are perfectly competitive.
4. Note that we assume in Theorem 2.1 that a balanced growth path with positive human capital growth exists and obtain as a result that also the control $u^*(t) \equiv 1$ for all $t \in [0, \infty)$ without human capital growth is optimal. This is, at first glance, a contradiction. But the assumption does not rule out that there exists a balanced growth path without human capital growth. From a mathematical point of view, the theorem could be reformulated to start with "If an optimal singular control exists" but we chose our formulation as economically more intuitive.
5. The derivation of Eq. (2.22) can be found in Appendix 2.

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3

Childlessness and Economic Development: A Survey

Thomas Baudin, David de la Croix, and Paula E. Gobbi

JEL Classification Numbers J11; O11; O40

3.1 Introduction

In her book "No Kids: 40 Good Reasons Not to Have Children", Corinne Maier puts into question the idealized notion of parenthood as a natural behavior. She asks her childless readers whether they are prepared to give up their time, money, and friends for the "*vicious little dwarves*" *that will treat them like their servant and end up resenting them*. In contrast to Corinne Maier, Jody Day, in her book "Living the Life Unexpected: 12 Weeks to Your Plan B for a Meaningful and Fulfilling Future Without

T. Baudin (✉)
IÉSEG School of Management & LEM (UMR 9221), Lille, France
e-mail: t.baudin@ieseg.fr

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Children", states that "*Across the globe, millions of women are reaching their mid-forties without having had a child, (…) most didn't choose this and are silently struggling in a life they didn't foresee. Most people think that women who aren't mothers either couldn't have or didn't want children: the truth is much more complex.*" In the list of arguments and situations discussed in these two books for the general public, many speak to the theory of fertility as it has been developed since Malthus (1807), Sadler (1830), and Becker (1960).

In this paper, we expose and summarize what can be learned by applying the economic theory of fertility to the understanding of childlessness. We claim that analyzing who is more prone to not having babies, and—more importantly—why, is interesting in itself. Moreover, looking at this specific outcome may help to understand the motives behind fertility behavior in general. In other words, analyzing childlessness behavior helps to unravel the factors behind fertility behavior by adding an additional dimension to the reasoning. For example, if we believe that fertility dropped because of factor x, we may wonder whether what we observe in terms of childlessness also squares with this explanation. In technical terms, looking at childlessness in addition to fertility adds restrictions to properly identify the important factors.

Even in historical periods during which demographers would advance that childlessness is identical to sterility, we show that there are patterns which we can learn from. In particular, we will build the case for an economic analysis of childlessness, arguing that childlessness varies both over time and across social classes, independently of the age at first

D. de la Croix
CEPR, London, UK

IRES/LIDAM - Université catholique de Louvain, Louvain-la-Neuve, Belgium
e-mail: david.delacroix@uclouvain.be

P. E. Gobbi
Université Libre de Bruxelles (ECARES), Bruxelles, Belgium

CEPR, London, UK
e-mail: pgobbi@ulb.ac.be

marriage (which is often taken as a proxy for sterility). An unexpected high childlessness rate among the upper classes, which is found both in England and France before the Industrial Revolution, raises many questions about the incentives faced by the upper classes to have children.

Developing countries today can also have high levels of childlessness. They are hidden behind general high fertility rates and the common idea that fertility is the result of "mistakes" and a lack of family planning. We show that childlessness can be caused by extreme poverty and therefore act as a kind of Malthusian preventive check. In practice, childlessness that results from poverty is due to malnutrition, stress, unhealthy environments, and venereal diseases leading to infections. We also evidence that poverty is not the only engine of childlessness in developing countries, since progress toward women's empowerment also drives childlessness.

Childlessness in developed countries is mostly the result of the high opportunity cost of time. As wages become higher, the cost of childcare also increases which reduces the incentives to have large families, and even to become a parent. Nevertheless, in countries like the US, poverty remains an important driver of childlessness, especially among single women.

After having reviewed the evidence on childlessness in Sect. 3.2, we provide a theory to illustrate the main mechanisms described above in Sect. 3.3. Such a theory is useful to understand how major changes in the environment may affect fertility and childlessness. Such changes include an increase in the education of women, in overall non-labor incomes, and in the degree of assortativeness in the marriage market.

3.2 Data

3.2.1 Childlessness in Historical Data

Very little has been said on childlessness for pre-industrial societies. It is often assumed that being married implied a desire for children, and that the observed childlessness rate is a measure of the natural sterility in a population (Leridon, 2008). For England, Wrigley et al. (1997) discuss sterility as a part of their analysis of marital fertility, mainly attributing

sterility to biological factors. They argue that the main factor influencing the sterility of married couples, also named entry sterility, was the mother's age at marriage since only 2.6 and 3.8 percent of women who married at ages 15–19 or 20–24 respectively never bore children, whereas 69.3 percent of women who were first married at the age of 40–44 never bore children. Hollingsworth (1965), who studied the demographics of British nobles based on genealogical records, finds an average childlessness rate at 24 percent, based on all persons ever married, a surprisingly high rate compared with other populations and with the average entry sterility rates for English provincial parishes (Wrigley et al., 1997, pp. 395–397).

In a recent paper, de la Croix et al. (2019) reconsider the English data based on parish records and analyze the social gradient to childlessness using modern econometric techniques. Occupational groups are defined using the professions indicated on the various baptism/marriage/burial registers. In order to compute the childlessness rate (i.e. the share of married couples who never had a child), they use Cox proportional hazard models to estimate the risk of a married couple having a first birth. Couples become at risk of a first birth upon marriage and censoring occurs when the mother dies. They limit their sample to couples whose burial dates are known to ensure that childlessness is not attributed to couples that migrated out of the parish. Figure 3.1 shows the predicted childlessness rate ten years after marriage. These childlessness rates are rather high in general. Marriages in wealthier social groups were more likely to remain childless relative to the poor. This result is surprising and difficult to explain, in particular because the age at marriage is very similar across groups. In their paper, the authors consider various mechanisms: higher rates of venereal diseases, higher consanguinity, and the unique cultural context of marriage among the gentry.

Gobbi and Goñi (2017) look at the childlessness of the British aristocracy in 1650–1882 using the Hollingsworth (2001) database. They show that 30–40% of the peers' daughters who married remained childless around the 1650s (see Fig. 3.2). Compared to the average person in Britain, the childlessness rate of aristocrats was surprisingly high, threatening the continuation of aristocratic family lineages and the maintenance of large estates in the hands of this wealthy elite. Gobbi and Goñi (2017) show that these threats were countered by the introduction of settlements,

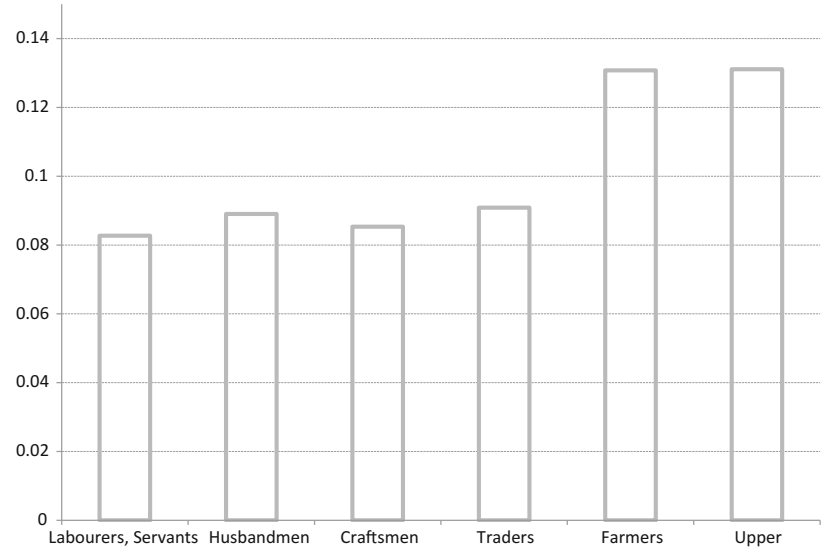

Fig. 3.1 Predicted childlessness rate ten years after marriage by social group—England 1580–1837

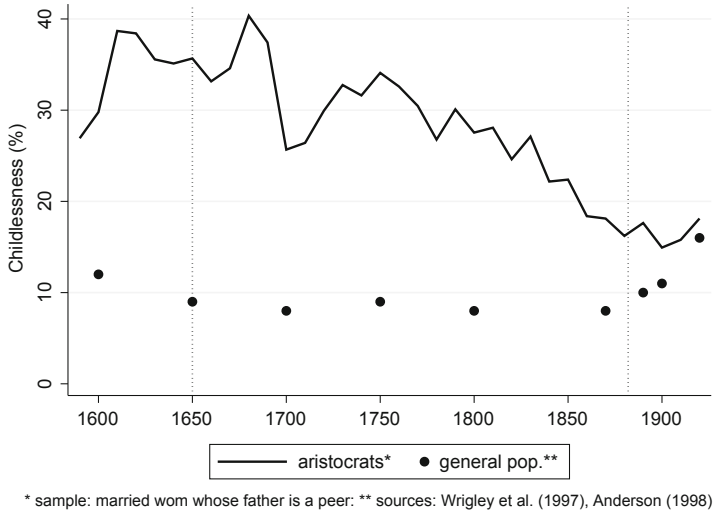

Fig. 3.2 Childlessness rates of British aristocrats by marriage decade

an inheritance scheme combining primogeniture with a one-generation entail of the land. The typical settlement operated as follows: it was signed between a family's head and his heir upon the marriage of the latter. By signing a settlement, the heir committed to passing down the family estate unbroken to the next generation (Habakkuk, 1950). This altered fertility incentives along the extensive margin. In short, those who signed a settlement had to pass down a large inheritance, which they preferred to go to their natural sons rather than to a distant family member. Using genealogical data from Hollingsworth (1964), Gobbi and Goñi (2017) show that families who signed a settlement were more likely to have children than families who failed to sign a settlement because the family's head died before his heir's wedding. Eventually, settlements brought childlessness rates among aristocrats close to the "natural" rate of 2.4 percent (Tietze, 1957), and hence, contributed to the survival of noble family lineages.

In France, scholars have explored childlessness and sterility by social class more directly. Bardet (1983), who reconstituted 5889 complete family histories over the seventeenth and eighteenth centuries, presents a table (p. 300) with the percentage of childless women by year at marriage and social class. The childlessness rate is computed based on women who married before 30 years old, and for whom a complete record of life events exists. Bardet's numbers are presented in Table 3.1. The social gradient was positive: childlessness was more widespread among nobles and shopkeepers than among workers, and there was a positive time trend in the data. On the whole, Bardet's figures are above the natural sterility rate estimated elsewhere (Leridon, 2008).

Taking a fresh look at Bardet's data, Brée and de la Croix (2019) ponder the possible explanations for the rise in childlessness witnessed. Any explanation should also be compatible with the drop in fertility observed over this period, and with the increase in education (as indicated by the increase in the number of people able to sign the marriage register). They find that several explanations appear irrelevant due to the absence of change during the period under consideration: contraception, wet nursing, age at marriage, income, and divorce. Other explanations have been rejected because they are not consistent with the fact that changes

Table 3.1 Childlessness rate by year of marriage and social class

	Social classes				Difference
Marriage	Gentry	Shopkeepers	Craftsmen	Workers	Gentry − Workers
1670–1699	4	4	5	3	1
1700–1729	8	9	7	6	2
1730–1759	11	11	8	6	5
1760–1792	12	13	10	8	4
Δ 1670–1792	+8	+9	+5	+5	

Note: first line should be read as: 4% of the women belonging to the gentry who married before age 30 during the period 1670–1699 remained childless, etc. The last column gives the difference between the childlessness rate of the gentry and that of the workers. The last line computes the difference between the fourth line (1760–1792) and the first line (1670–1699)
Sources: Bardet (1983) p300, author's own calculations

were more pronounced among the higher social groups; they include: drop in mortality and rising secularization. As such, three explanations without any direct evidence for or against them remain: increase in materialism, women's empowerment, and increase in return to education.

A possible increase in the return to education as a driver of the drop in fertility is rejected on the basis of a theoretical argument. If the return to education had increased, a drop in fertility should have been observed, which fits the data well, but also a drop in childlessness, as having children would have become more worthwhile. Indeed, with a higher return to education, children have a brighter future, and hence become more valuable. There is a counterargument to this claim: giving birth to one child that is left uneducated becomes less worthwhile from an individual perspective and less acceptable from a social perspective if the return to education rises. Thus, many very poor women who cannot afford to send their children to school might choose to stay childless instead of giving birth to one child who will then remain uneducated. This, in isolation, would increase childlessness in case of an increasing return to education, in particular in the low social classes, which is an implication rejected by the data as it is mostly the upper classes for which childlessness increases. A last argument in favor of the rise in the return to education storyline is that it could lead to a reduction in the desired number of children and/or to a postponement of fertility as mothers educate themselves longer. Since

there is a stochastic element in having children, reducing the number of desired children or postponing childbirth both increase the risk of ending up childless. This explanation might be relevant for the most recent periods when the mean age at motherhood has increased above 30, but not for the preindustrial era.

Thus, two explanations remain to understand this increased childlessness: the increase in materialism and women's empowerment. The first—increase in materialism—means an increasing availability, variety, affordability, and quality of consumer goods, implying a rising demand for them. According to De Vries (2008), such a change is a key characteristic of Northern Europe for the period from the mid-seventeenth century to about 1830. De Vries (2008) documents that many households began to consume a wider variety and amount of consumer goods, accompanied by an intensification of household labor and engagement with different aspects of the market. Even if an increase in materialism may explain why households desired fewer children, could it also explain why some of them even stopped wanting children altogether? Did certain kinds of new (luxury) goods become accessible or desirable, and was being childfree a requirement to procure them? Van de Walle and Van de Walle (1972) identify two major arguments that were advanced by authors of the eighteenth and nineteenth centuries to explain why women did not want children. The first of these arguments is that women did not want to experience the physical changes of pregnancy, and the second was that women wanted to preserve their freedom and avoid the burden of a pregnant belly. Implicit in these two arguments is the desire to avoid losing one's place in society—not to be kept away from salons and high society. This would then only have concerned the elite.

The second—women's empowerment—can be measured along several dimensions, and each dimension has its own effect on fertility (see de la Croix and Vander Donckt, 2010). Essential dimensions in today's economies are: political empowerment, educational attainment, economic participation and opportunity, as well as health and survival. On the whole, there is converging evidence that the gender gap in Rouen started to shrink along the educational, occupational, and health dimensions during the eighteenth century. This may have reduced fertility

and increased childlessness as long as women inherently desired fewer children than men. For educational attainment, marriage registers can be used to evaluate the basic level of literacy through the quality of someone's signature. Brée and de la Croix (2019) show that the educational gap between (married) men and women shrunk over the period. With regard to the gender gap in economic participation and opportunity, Brée and de la Croix (2019) use the database of famous people built by de la Croix and Licandro (2015) and show that prior to the eighteenth century, only a few women were famous in Rouen. Then, the share of women among famous people increased to 10–12%. Although some of the new occupations held by women do not necessarily correspond to our idea of highly skilled jobs (like playing the role of a soubrette on stage, for example), it is fair to conclude that the gender gap in economic participation and opportunity started to shrink in the eighteenth century.

Four recent papers stress the importance of economic determinants for US data from a historical perspective. Gobbi (2013) shows how childlessness rates and fertility rates co-move over time as a function of shocks to the gender wage gap and to the cost of having children. Aaronson et al. (2014) focus on a quantity-quality trade-off faced by parents and look at how the Rosenwald Rural Schools Initiative in the early twentieth century affected fertility along both the extensive and intensive margins. They show that the expansion of schooling opportunities decreased the price of child quality. This reduction in the price of child quality decreased the proportion of women with the highest fertility rates as expected, but it also led to a decrease in childlessness rates. Baudin et al. (2015) provide a framework to understand the deep causes of childlessness and how their importance has changed over time. As shown in Fig. 3.3 from US census data, childlessness rates were high in the past, decreased and then increased again for more recent cohorts of married women. Baudin et al. (2015) apply structural estimation techniques to historical census data on fertility for the United States, over the period 1871–1964. They evidence that the first drop in childlessness rates for the cohorts of women born between 1910–1935 is due to a decrease in the number of women who remained childless because of poverty, while the increase that followed is due to a rise in the opportunity cost to rear children. The main causes of

Fig. 3.3 Childlessness rates of married women in the United States by birth cohort

childlessness over the past 100 years have changed from poverty-induced necessity to a choice driven by higher levels of income and education among women.

Finally, Bhalotra et al. (2018) study the impact of the introduction of Sulfa drugs in the United States in 1937 on fertility and childlessness. The introduction of antibiotics reduced child and maternal mortality dramatically. By encouraging the postponement of first births and female labor force participation, this revolution has led to a significant increase in childlessness among American women. This explanation is complementary to that of Baudin et al. (2015), as freeing women's time to participate in the labor force contributes to the increase in female education and wages.

3.2.2 Childlessness in Developing Countries

Developing countries remain heterogeneous regarding their demographic situations: some countries like Brazil have almost achieved their demographic transition (Turra and Queiroz, 2005), while others like Nigeria are experiencing the early phases of the latter (Canning et al., 2015).

Thus, contrary to developed countries, it is impossible to provide a unified picture of the dynamics of childlessness along the demographic transition of developing countries. For this reason, Baudin et al. (2019) propose a cross-country analysis of the relationship between childlessness and development. They develop a theoretical framework distinguishing between four types of childlessness. *Natural sterility* reflects situations in which a woman or a couple suffers from an innate biological impossibility of having children; this kind of childlessness is unrelated to the level of education or wealth. *Mortality-driven childlessness* concerns women who have had children, but none of them have survived. *Opportunity-driven childlessness* comes from the economic opportunities offered to a couple and especially to women: the more educated a woman, the higher her potential wage and thus the higher the opportunity cost of the time she does not spend in the labor force.[1] Finally, *poverty-driven childlessness* concerns women who face the heaviest burden of poverty; lack of education and celibacy are important drivers of this kind of childlessness.

The co-existence of poverty- and opportunity-driven childlessness explains why the relationship between the rates of definitive childlessness and the development level of a country is weak *a priori*. Proxying development by the average level of education of the population, we evidence this in the left panel of Fig. 3.4. The weak link between childlessness

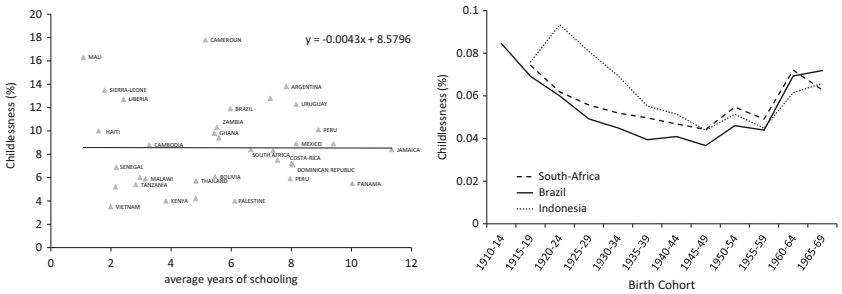

Fig. 3.4 **Left panel:** Relationship between definitive childlessness rates and the average level of education of women aged between 40 and 54 in a set of 36 developing countries. Data from Baudin et al. (2019). **Right panel:** definitive childlessness among married women by cohort of birth. Data from IPUMS-International, authors' own calculations

and development hides a powerful statistical regularity: along the development process, poverty-driven childlessness is progressively replaced by opportunity-driven childlessness. Such a regularity has been conjectured in an alternative framework by Poston and Trent (1982).

As a result, any economic shock or development policy affecting poverty and the economic opportunities offered to women may have an ambivalent effect on the dynamics of childlessness in developing countries. This is what Baudin et al. (2019) evidence in a series of quantitative analyses. For instance, enforcing strict wage equality between men and women would, not surprisingly, reduce the fertility of women with children as higher wages translate into a higher opportunity cost of having children. This increase in the opportunity cost of having children translates into higher childlessness rates among highly educated women. In most cases, this increase is not compensated fully by the decrease in childlessness due to poverty: low-educated women become richer and thus less subject to extreme poverty leading to sterility. The general increase in childlessness rates magnifies the decrease in fertility among mothers. On a set of 36 developing countries, they estimate that closing the gender wage gap would reduce the fertility of mothers by 8.21%, while after taking into account the adjustment of childlessness and marriage rates, the total decrease in fertility would equal 12.97%.

In a study on India, Baudin and Sarkar (2018) confront the fact that the highest childlessness rates are found in the most developed states and among highly educated women to the belief that childlessness in India is synonymous with sterility. They identify a U-shaped relationship between the probability for a woman to end her reproductive life childless and her level of education. This statistical regularity remains valid after controlling for the specific context of caste divisions, religious diversity, as well as state and cohort fixed effects. It reveals that opportunity-driven childlessness is emerging as a new and important form of childlessness in the country. Interestingly enough, the education of husbands reduces the probability of remaining childless, which highlights the persistence of traditional family structures in which the male breadwinner protects women against extreme forms of poverty. In a recent paper, using alternative vocabulary, Iftikhar (2018) also identifies the co-existence of poverty

and opportunity-driven childlessness in Pakistan, a country that shares many specificities with India.

While all developing countries have not finished their demographic transition, some of them are at very advanced stages of the latter. This is the case, for instance, of Brazil, Indonesia, and South Africa, three large countries which are emblematic of their continents. On the right panel of Fig. 3.4, we document the evolution of definitive childlessness rates by birth cohort among married women between 1915 and 1965. The U-shaped pattern of childlessness is remarkable and puts these three countries in a situation close to a large set of developed countries, as shown in the next section.

3.2.3 Childlessness in Developed Countries

The cohorts of women born in developed countries over the course of the twentieth century have experienced rapid changes in the prevalence of definitive childlessness. In Fig. 3.5, we document a pattern of childlessness, which is systematically U-shaped in developed countries.

In their study of childlessness in the US, Baudin et al. (2015) identify three major reasons for being childless: natural sterility, poverty-driven childlessness, and opportunity-driven childlessness. As explained in Sect. 3.2.1, their theoretical framework allows to reproduce the evo-

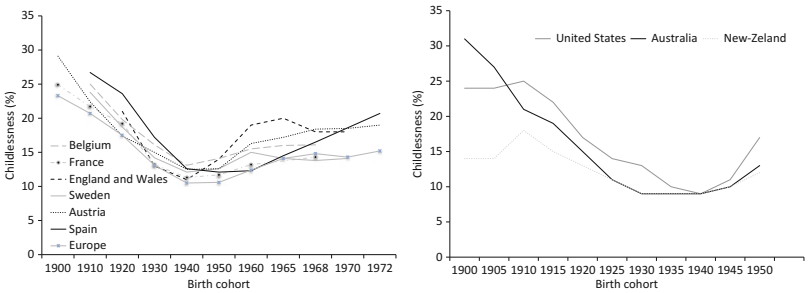

Fig. 3.5 **Left panel:** Childlessness rates at age 40+ among European women by cohort of birth. Data from Sobotka (2017). **Right panel:** childlessness rates among all women at age 45, data from Rowland (2007)

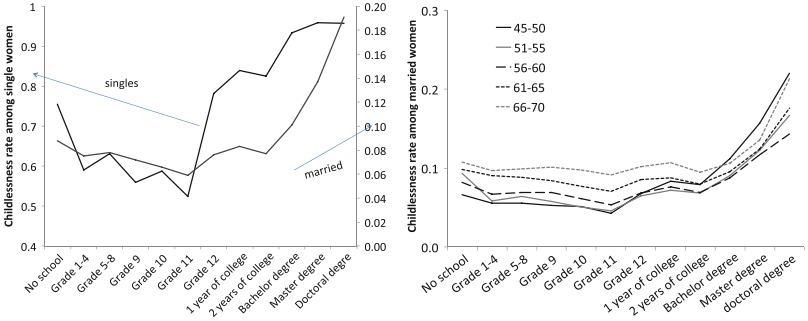

Fig. 3.6 **Left panel:** Childlessness rates at age 45–70 among American women in 1990. **Right panel:** Definitive childlessness rates among American women by cohort of birth, data from Baudin et al. (2015)

lution of childlessness rates in the US in the twentieth century both quantitatively and qualitatively. Beyond this, it also enables to understand what drives the relationship between education and childlessness for a given year. In 1990, American women of age 45–70 were characterized by a U-shaped relationship between their childlessness rate and their education level (see Fig. 3.6, left panel). As shown in Fig. 3.6 (right panel), this fact remains valid even when they disaggregate their data at the cohort level, the same being true for racial differences. It testifies to how, in the richest country in the world, poverty-driven childlessness continues to co-exist with opportunity-driven childlessness. Baudin et al. (2015) estimate that in 1990 in the US, 2.5% of American women were definitively childless because of their poverty, but this percentage increases to 12.1% among women with less than 5 years of schooling. On the other side, 8.9% of American women were childless because of the economic opportunities offered to them, and this percentage reaches 19.4% among women with at least a bachelor's degree.

The persistence of high levels of childlessness among poor women is associated with the persistence of strong economic inequalities in the country. The authors show, in a counterfactual experiment, how an increase in the returns to schooling and its associated rise in the Gini coefficient of labor and non-labor income may increase the rates of poverty-driven childlessness. An increase in the rate of return of one

year of schooling from 9.2% to 12.6% could increase the prevalence of poverty-driven childlessness among American women from 2.5% to 3.3%.

The educational gradient of childlessness in the US is remarkable; this is also the case in countries like England and Germany. Berrington (2017) documents a positive educational gradient of childlessness among English women aged 40–49 born between 1940 and 1969. The absence of a negative educational gradient for low levels of education may be explained, among other factors, by the fact that women are grouped in too large educational groups which does not allow to identify the poorest women. See Baudin et al. (2015) for a discussion of that point. The case of Germany is one of the most interesting. Using micro-census data from 2012, Kreyenfeld and Dirk (2017) evidence a positive educational gradient of definitive childlessness among women born between 1940 and 1964 in former West Germany. Among their counterparts from East Germany, childlessness has a U-shaped relationship with education, whatever the sub-cohort observed and even if education is grouped in very large categories. As shown by Baudin and Stelter (2018a), this is a unique opportunity to appreciate how historical, institutional, and economic factors shaping the level of economic and health poverty in turn shape the structure of childlessness in a population.

3.3 Models

The goal of this section is to illustrate theoretically how an economic shock like an increase in women's education or a change in the degree of assortativeness on the marriage market may change childlessness rates. Here, we build a new version of Baudin et al. (2015)'s framework in which childlessness can only be of two types: poverty driven or opportunity driven. As in Aaronson et al. (2014) and Bhalotra et al. (2018), we introduce a trade-off between the quality and quantity of children. In Sects. 3.3.1–3.3.3, we analyze the properties of a model without marriage. We investigate the relationship between marriage and childlessness in Sect. 3.3.4.

We assume that each woman is characterized by the following utility function:

$$u(c, n, h') = \ln c + \alpha \ln(n + \nu) + \beta \ln h', \tag{3.1}$$

where c denotes the consumption of an aggregated good whose price is normalized to 1. n denotes the number of children this woman gives birth to, while parameter $\nu > 0$ allows for the existence of corner solutions on n. Variable h' denotes the quality of children, it takes the form of human capital in most of the literature. α and β are preference parameters over fertility and child quality, respectively.

Following de la Croix and Doepke (2003), we assume that the quality of children is produced through an investment in education, e; it also depends on the parental human capital, h, such that:

$$h' = (e + \pi)^{\phi} h^{\iota}, \quad \text{with } \{\phi, \iota\} \in \mathbb{R}^{2+} \tag{3.2}$$

where $\pi > 0$ allows for the possibility of not educating children, ϕ captures the weight of education in the production of human capital, and ι captures the intergenerational transmission of human capital.

As in Baudin et al. (2015), we assume that women cannot have children if their consumption is lower than a threshold, $\hat{c}$:

$$c \leq \hat{c} \Rightarrow n = 0. \tag{3.3}$$

This constraint introduces poverty-driven childlessness in a simple way as all women who are too poor to afford a consumption bundle of $\hat{c}$ will be childless. Women maximize their utility with respect to c, n, and e, subject to this minimum consumption constraint and the budget constraint:

$$c + \rho e = (1 - \tau n) w + \Omega \tag{3.4}$$

where $\rho > 0$ denotes the cost of providing one unit of education to all children within the family. We make a somewhat unusual hypothesis as we assume that education is a pure public good inside the family. This

is done for the sake of simplicity, but it will not change the main feature of our model compared to the more usual model in which education is considered as a pure private good inside the family.[2] $\Omega > 0$ is a non-labor income orthogonal to h and w, the wage rate earned by the woman on the labor market.[3] A woman's potential wage is an increasing function of her human capital h. Finally, τ denotes the time that raising and bearing a child requires, time that cannot be spent in the labor force.

We assume that, if τ is positive, then $\tau\nu > \alpha$ in order to ensure that not having children may be an optimal decision.[4] In the following subsections, we impose restrictions on the set of parameters in order to analyze poverty- and opportunity-driven childlessness separately. Relaxing all these restrictions at the same time would impose a large number of other restrictions in order to guarantee the existence of solutions to this problem. In the end, it would be possible to determine state-spaces where each of the properties presented in what follows would prevail.

3.3.1 Opportunity-Driven Childlessness: $\hat{c} = \beta = 0$

This first version of the model abstracts from both investments in education ($\beta = 0$) and poverty-driven childlessness ($\hat{c} = 0$), while it focuses on opportunity-driven childlessness ($\nu > 0$). Each woman maximizes her utility function (3.1) subject to her budget constraint (3.4).

$$c^* = \begin{cases} \Omega & \text{if } w \leq \dfrac{\alpha\Omega}{1+\tau\nu} \equiv \underline{w} \\ \dfrac{(1+\tau\nu)w+\Omega}{1+\alpha} & \text{if } w \in \left]\dfrac{\alpha\Omega}{1+\tau\nu}, \dfrac{\alpha\Omega}{\tau\nu-\alpha}\right[\\ w+\Omega & \text{if } w \geq \dfrac{\alpha\Omega}{\tau\nu-\alpha} \equiv \bar{w} \end{cases}$$

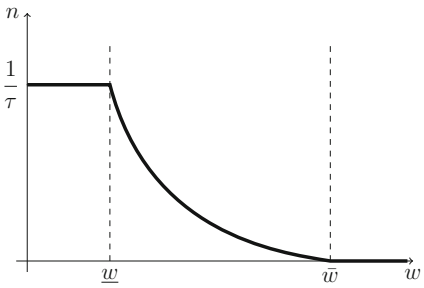

Fig. 3.7 Opportunity-driven childlessness

and

$$n^* = \begin{cases} \dfrac{1}{\tau} & \text{if } w \leq \underline{w} \\ \dfrac{\alpha}{1+\alpha}\dfrac{(1+\tau\nu)w+\Omega}{\tau\nu} - \nu & \text{if } w \in]\underline{w}, \bar{w}[\\ 0 & \text{if } w \geq \bar{w}. \end{cases}$$

In the interior regime, an increase in women's wages raises the opportunity cost of having children, inciting women to substitute private consumption to the quantity of children. As shown in Fig. 3.7, this mechanism prevails only when the wage level is high enough ($w > \underline{w}$) to prevent situations in which mothers' fertility is limited by their reproductive capacity. In the latter situation, already documented by de la Croix and Doepke (2003) for instance, any wage increase has no effect either on fertility or on consumption, as women do not participate in the labor force.

When their wage becomes very large ($w > \bar{w}$), the relative cost of having a child becomes so high that specializing in labor market activities and being childless becomes optimal. This is what we call opportunity-driven childlessness.

Let us consider a situation in which women are heterogeneous regarding their level of education. Any policy making higher education more prevalent would translate into a higher proportion of women enjoying higher levels of education, better economic opportunities, and thus higher

costs of having children. This will translate in the end into an increase in the proportion of women who are childless because of good economic opportunities. As documented by Baudin et al. (2015), more largely, any policy raising women's wages, like closing the gender wage gap, would increase opportunity-driven childlessness.

In this framework, opportunity-driven childlessness appears quite mechanical. In richer models like de la Croix and Pommeret (2018), opportunity-driven childlessness is driven by postponement decisions. In such a set-up, for a given age, highly educated women are those enjoying the best economic opportunities, they thus have the strongest incentives to postpone their first birth. In cases where women are confronted to an especially positive series of economic opportunities, serial postponement may lead either to the inability to have children as fecundity recedes with age, or to the decision to remain childless. Even if richer, the main prediction of this model regarding the effect on childlessness of offering better economic opportunities to women is not different from that obtained with the model developed in this chapter.

3.3.2 Poverty-Driven Childlessness: $\beta = \nu = 0$

In this specification of the model, we abstract again from the quality-quantity trade-off ($\beta = 0$) and rule out opportunity-driven childlessness ($\nu = 0$), but we now let poverty-driven childlessness exist ($\hat{c} > 0$). This specification of the model requires redefining the utility function in the specific case where $n = 0$. We then assume that $u(c, 0, h') = \ln c - P$, where $P > 0$ is an arbitrarily large parameter such that $u(c, 0, h') < u(\hat{c}, n, h')\ \forall n > 0$. It ensures that (*i*) the utility function remains defined even if not differentiable when $n = 0$, and (*ii*) not having children is never a rational decision.

Non-labor income becomes a crucial driver of behaviors as it determines the capacity of women to consume the minimum consumption bundle $\hat{c}$ in extreme situations in which their wages are very small and/or they do not participate in the labor force.

Case 1: $\Omega \in]0, \hat{c}]$
In this situation, the non-labor income is below $\hat{c}$. Hence, participation in the labor force is a necessary but not sufficient condition for a woman to afford to have children. Let us define $\hat{w}$ as the minimum wage such that the minimal consumption constraint (3.3) does not bind: $\hat{w} \equiv (1+\alpha)\hat{c} - \Omega$. Individual behaviors are described as follows:

$$c^* = \begin{cases} w + \Omega & \text{if } \; w \leq \hat{c} - \Omega \\ \hat{c} & \text{if } \; w \in \left]\hat{c} - \Omega, \hat{w}\right] \\ \dfrac{w + \Omega}{1 + \alpha} & \text{if } \; w > \hat{w} \end{cases} \text{ and}$$

$$n^* = \begin{cases} 0 & \text{if } \; w \leq \hat{c} - \Omega \\ \dfrac{w + \Omega - \hat{c}}{\tau w} & \text{if } \; w \in]\hat{c} - \Omega, \hat{w}] \\ \dfrac{\alpha}{1+\alpha}\dfrac{w + \Omega}{\tau w} & \text{if } \; w > \hat{w}. \end{cases} \tag{3.5}$$

Equation (3.5) shows that low-educated women who earn low wages end up childless because of their poverty, which is what we call poverty-driven childlessness. Importantly, the reproductive constraint for having children also gives rise to a Malthusian type of fertility regime. Women with an intermediate wage level and low non-labor income, limit their consumption to $c = \hat{c}$, and any increase in their wage translates into a higher fertility. Once the constraint on $\hat{c}$ does not bind anymore, individual behaviors are aligned with classical Beckerian theory in which higher wages decrease the optimal number of children a woman has.

From Eq. (3.5) and Fig. 3.8, it is clear that, for a given distribution of educational levels among women, any transfer policy increasing Ω would reduce $\hat{w}$ and thus childlessness rates. Nevertheless, the net effect of such a policy on average fertility is ambiguous as the fertility of mothers would increase in the meantime. This simple mechanism highlights how important it is to consider the adjustment of childlessness when one wants to understand how economic and family policies change fertility. This issue is extensively discussed in Baudin et al. (2019).

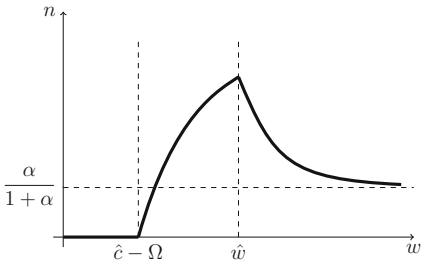

Fig. 3.8 Poverty-driven childlessness when non-labor income is low ($\Omega \in]0, \hat{c}]$)

Case 2: $\Omega > \hat{c}$

When the non-labor income is higher than the minimum consumption that allows a woman to bear a child, poverty-driven childlessness disappears. Furthermore, the Malthusian regime described in the previous sub-case also disappears. From the budget and time constraints, two possible regimes remain, respectively: an interior regime in which no other constraint than the budget constraint binds and a corner solution in which fertility is maximal. We then obtain the following optimal solutions:

$$c^* = \begin{cases} \Omega & \text{if } \ w \leq \alpha\Omega \equiv \bar{w} \\ \dfrac{w+\Omega}{1+\alpha} & \text{if } \ w > \bar{w} \end{cases} \quad \text{and}$$

$$n^* = \begin{cases} \frac{1}{\tau} & \text{if } \ w \leq \bar{w} \\ \dfrac{\alpha}{1+\alpha}\dfrac{w+\Omega}{\tau w} & \text{if } \ w > \bar{w}. \end{cases}$$

In this situation where poverty-driven childlessness does not exist, an increase in the non-labor income increases fertility along its intensive margin in the interior regime only (Fig. 3.9).

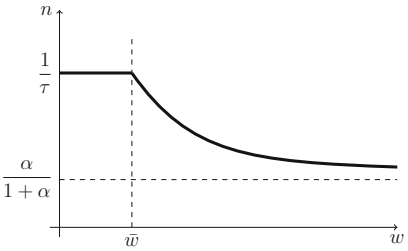

Fig. 3.9 Fertility when non-labor income is high ($\Omega > \hat{c}$)

3.3.3 Quality/Quantity Trade-Off: $\beta > 0$, $\nu > 0$, $\hat{c} = 0$

In this specification of the model, we rule out poverty-driven childlessness and authorize the existence of opportunity-driven childlessness. We extend our framework to investments in the human capital of children.[5] As explained earlier, we assume, without loss of generality, that economies of scale in human capital accumulation are maximum as education is a public good inside the family.

Depending on the values of the parameters, many regimes may prevail in this configuration of the model. In addition to the budget constraint, three constraints may bind: $n \geq 0$, $e \geq 0$, and $n \leq \frac{1}{\tau}$. For a given value of wages w and non-labor income Ω, from none to two of them may bind simultaneously.[6]

Proposition 3.1 *There exists a non-empty state-space* $\mathcal{H} = \{\tau, \nu, \alpha, \beta, \rho, \phi, \iota, \hat{c}\}$ *such that the optimal decisions of the representative individual are described as follows:*

$$c^* = \begin{cases} \Omega & \text{if } w \leq \underline{w} \\ \dfrac{(1+\tau\nu)w + \Omega}{1+\alpha} & \text{if } w \in]\underline{w}, w_e] \\ \dfrac{(1+\tau\nu)w + \rho\pi + \Omega}{1+\alpha+\phi\beta} & \text{if } w \in]w_e, w_n] \\ w + \Omega & \text{if } w > w_n, \end{cases}$$

$$n^* = \begin{cases} \dfrac{1}{\tau} & \text{if } w \leq \underline{w} \\ \dfrac{\alpha}{1+\alpha}\dfrac{(1+\tau\nu)w+\Omega}{\tau w} - \nu & \text{if } w \in]\underline{w}, w_e] \\ \dfrac{\alpha}{1+\alpha+\phi\beta}\dfrac{(1+\tau\nu)w+\rho\pi+\Omega}{\tau w} - \nu & \text{if } w \in]w_e, w_n] \\ 0 & \text{if } w > w_n \end{cases}$$

$$e^* = \begin{cases} 0 & \text{if } w \leq w_e \\ \dfrac{\phi\beta}{1+\alpha+\phi\beta}\dfrac{(1+\tau\nu)w+\rho\pi+\Omega}{\rho} - \pi & \text{if } w \in]w_e, w_n] \\ 0 & \text{if } w > w_n. \end{cases}$$

where $\{w_n, w_e, \underline{w}\} \in \mathbb{R}^{3+}$ *and* $w_n > w_e > \underline{w} > 0$.

Proof See the Appendix. ■

For low wages ($w \leq \underline{w}$), the time constraint binds hand in hand with the non-negativity constraint on educational investments. Women then have a maximal number of children and do not educate them (Fig. 3.10). For higher wages ($w \in]\underline{w}, w_e]$), fertility decreases with wages but the so-called trade-off between the quality and the quantity of children does not yet appear. What appears here is a trade-off between fertility and consumption. The trade-off between the quality and the quantity of

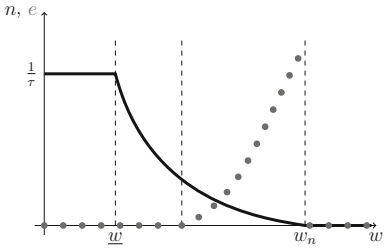

Fig. 3.10 Quality quantity trade-off. Fertility (n) in plain black line and education (e) in gray dots

children appears once $w \in]w_e, w_n]$, and prevails as long as women have children. When wages become very high ($w^f > w_n$), it is optimal to remain childless.

In this specification of the model, opportunity-driven childlessness acts as a barrier to the perpetuation of dynasties lying in the upper tail of the human capital distribution. It can reduce both the average level of human capital within society and inequalities in the distribution of human capital. To the best of our knowledge, this mechanism has never been quantified in the economic literature.

3.3.4 Marriage and Childlessness

Introducing endogenous marriage decisions into a model of fertility and consumption may lead to considerable complexity.[7] For the sake of simplicity, we propose a model in which fertility and consumption decisions inside a marriage are made according to the model presented before. More precisely, we assume that inside a marriage, the woman decides how many children to have, if any, by maximizing her own utility function. This assumption is in line, for instance, with Regalia and Rios-Rull (2001). As most of the literature, we do not assume that men and women meet randomly on the marriage market; we specifically assume assortative mating in terms of education. Any person on the marriage market has more chances to be matched with a partner whose education level is close to his or her own educational level; we will refer to this phenomenon as educational assortativeness or educational homogamy.

From the previous model, we know how a single woman behaves regarding fertility and childlessness. Let us denote n_s her realized fertility, w^f her wage, and Ω^f her non-labor income. We obtained that, except in Malthusian-type regimes, $\frac{dn_s}{dw^f} \leq 0$. Furthermore, whatever $\Omega^f > 0$, $\frac{dn_s}{d\Omega^f} \geq 0$. If we denote $q(w^f, \Omega^f)$ the probability that a woman drawn randomly from the population is childless, then $\frac{dq(w^f,\Omega^f)}{dw^f} < 0$ for low values of w^f (higher wages protect against poverty-driven childlessness), while $\frac{dq(w^f,\Omega^f)}{dw^f} > 0$ for high values of w^f (higher wages increase the time

cost of children and leads to opportunity-driven childlessness). We also learned that $\frac{dq(w^f,\Omega^f)}{d\Omega^f} < 0$.

Let us now introduce married women. We assume that when married, a woman receives part of her husband's labor and non-labor income (denoted respectively w^m and Ω^m), as well as help from her husband to raise children.[8] It implies that, compared to a single woman, a married woman has more non-labor income, while her time cost of raising children is smaller. This assumption is in line with Baudin et al. (2015).

We define the indirect utility of a woman i enjoying a wage rate w_i^f who is married to a man with a wage w^m as:

$$v_i^M = v^S(w_i^f) + \Delta^f(w_i^f, w^m) + \lambda_i^f.$$

$v^S(w_i^f)$ is equal to the indirect utility that she would receive as a single woman, this value may be easily computed from the previous sections. $\Delta^f(w_i^f, w^m)$ denotes the economic surplus coming from marriage (additional income and time coming from the husband). It is straightforward that $\frac{d\Delta^f(w_i^f,w^m)}{dw^m} > 0$, as the richer the potential husband, the higher the economic surplus coming from marriage, while $\frac{d\Delta^f(w_i^f,w^m)}{dw^f} < 0$ as highly educated women rely less on marriage to increase their well-being. λ_i is a love shock representing the quality of the union. We assume that it is drawn from a normal distribution such that:

$$\lambda_i^f \sim \mathcal{N}\left(\frac{\overline{\lambda^f}}{\varepsilon + |w^f - w^m|}, \sigma^f\right).$$

$(\overline{\lambda^f}, \varepsilon) \in \mathbb{R}^{2+}$ are two scalars respectively ensuring the positivity of the average love shock and the existence of the latter for $w^f = w^m$. Let us point out that each pair (w^f, w^m) has its own love shock process: the more homogamous a union in terms of wages (and thus education levels), the higher the expected quality of the match. We assume a constant variance across groups. We also assume that each potential couple may invest a costless infinitesimal amount of time living together in order to know their λ_i.

Computing the expected indirect utility of a woman with a wage w_i^f who is matched with a partner with a wage w^m, we get that all women want to marry *a priori*, such that they all want to invest the small amount of time needed to receive the information about λ_i:

$$Ev_i^M = v^S(w_i^f) + \Delta^f(w_i^f, w^m) + \frac{\overline{\lambda^f}}{\varepsilon + |w^f - w^m|} > v^S(w_i^f)$$

Let us now introduce men. We assume that men can have children only if they are in a relationship. By symmetry with women, we assume that men are characterized by the following indirect utility z_i^M:

$$z_i^M = z^S(w_i^m) + \Delta^m(w_i^f, w^m) + \lambda_i^m.$$

$z^S(w_i^m)$ denotes the indirect utility of a single man earning w^m.[9] The surplus coming from marriage, $\Delta^m(w_i^f, w^m)$, originates from the possibility of having children, as well as from the additional income that the wife brings to the family. This surplus is reduced by the time the husband has to invest in raising children, as well as the potential transfer of income to the wife. For simplicity, we assume, as for women, that this surplus is necessarily positive. Regarding love shocks, men draw them from a distribution which is symmetric to that of women:

$$\lambda_i^m \sim \mathcal{N}\left(\frac{\overline{\lambda^m}}{\varepsilon + |w^f - w^m|}, \sigma^m\right),$$

with $(\overline{\lambda^m}, \varepsilon) \in \mathbb{R}^{2+}$. We let the mean and the variance of love shocks diverge between men and women, as the literature on gender differences in preferences has documented systematic gender differences in preferences regarding for example risk aversion, taste for competition, and family ties.[10]

As for women, we get that all men want to marry *ex ante*. Computing the expected indirect utility of a man with a wage w_i^m who is matched with a partner with a wage w^f, we get that:

$$Ez_i^M = z^S(w_i^m) + \Delta^m(w_i^m, w^f) + \frac{\overline{\lambda^m}}{\varepsilon + |w^f - w^m|} > z^S(w_i^m) \ .$$

Realized Shocks and Marriage Rates

We first assume that a marriage will occur only if, after the realization of the love shock, both partners still want to marry. Then, assuming that the law of large numbers applies, we can compute the proportion of women with a wage w^f matched with a man with w^m who have decided not to marry because they received too bad a love shock. Let us denote the latter, $p^f(w^f, w^m)$:

$$\begin{aligned} p^f(w^f, w^m) &\equiv Prob(\lambda_i^f < -\Delta^f(w^f, w^m)) \\ &= \frac{1}{\sigma^f\sqrt{2\pi}} \int_{-\infty}^{-\Delta^f(w^f, w^m)} e^{-\frac{1}{2}\frac{x - \frac{1}{\varepsilon + |w^f - w^m|}}{(\sigma^f)^2}} dx. \end{aligned}$$

By symmetry, we get that the proportion of men with a wage w^m matched with a woman with w^f who have decided not to marry because they received too bad a love shock is:

$$\begin{aligned} p^m(w^m, w^f) &\equiv Prob(\lambda_i^m < -\Delta^m(w^m, w^f)) \\ &= \frac{1}{\sigma^m\sqrt{2\pi}} \int_{-\infty}^{-\Delta^m(w^m, w^f)} e^{-\frac{1}{2}\frac{x - \frac{1}{\varepsilon + |w^f - w^m|}}{(\sigma^m)^2}} dx \ . \end{aligned}$$

Let us assume that the number of education categories is discrete and let us denote $\tilde{w}$ the number of wage/education categories in the economy. We get that the proportion of married women, m, is the following:

$$m = \sum_{w^f=1}^{\tilde{w}} \sum_{w^m=1}^{\tilde{w}} \xi(w^f, w^m) \left[1 - p^f(w^f, w^m)\right] \left[1 - p^m(w^m, w^f)\right] \quad (3.6)$$

where $\xi(w^f, w^m)$ denotes the proportion of women of type w^f matched with a man of type w^m. Equation (3.6) shows how marriage rates, and therefore childlessness rates, may be influenced by the degree of educational homogamy. A first straightforward effect of increasing homogamy is to reduce the average distance between w^f and w^m, which increases the average quality of unions and marriage rates in turn. In technical terms, $\xi(w^f, w^m)$ increases for w^f close enough to w^m, but decreases for other values of w^f. It implies that, everything else equal, the proportion of couples receiving bad love shocks then recedes, and more couples are willing to marry.[11]

Nevertheless, this composition effect may be counterbalanced by an indirect effect of rising homogamy on the average marriage surplus. Indeed, low-educated women are matched with less educated men, which reduces their marriage surplus, while highly educated women are more often matched with highly educated men, which increases their surplus.[12] The same is true for men. Low-educated men are more often matched with low-educated women for whom the risk of poverty-driven childlessness is higher, while highly educated men are more often matched with highly educated women who are also likelier to remain childless because of economic opportunities. These indirect effects have an undetermined impact on m, but they are second-order effects and thus, even if negative, they cannot dominate the direct composition effect. Since compared to singleness, marriage protects against poverty-driven childlessness and reduces the incentives for not having children among highly educated women, an increase in homogamy therefore decreases childlessness.

We have analyzed the effect of rising educational homogamy under the implicit assumption that the average levels of education among men and women remains constant. As discussed for instance by Iyigun and Lafortune (2016) in the case of the US and by Van Bavel (2012) in the case of Europe, an increase in educational homogamy has emerged hand in hand with an increase in female education, and thus in w^f. When the average level of w^f increases, the marriage surplus for women is again potentially reduced, which contributes to making the indirect effect discussed in the previous paragraph more negative. As a result, the increase in marriage rates due to higher educational homogamy is again

mitigated. In addition, as women are now more educated, fewer of them are childless because of poverty, while more of them are childless because of improved economic opportunities. Knowing this, the final impact of a rise in educational homogamy due to an increase in women's education is ambivalent: it depends on the initial intensity of poverty and the initial level of educational homogamy. From one country to another, the impact of rising educational homogamy may be different.

Interestingly enough, we have given men a rather limited role in fertility decisions inside a marriage; nevertheless, we have shown how their marriage decisions strongly affect childlessness. By refraining (resp. easing) the marriage of low-educated women, they make poverty-driven childlessness increase (resp. recede). The opposite is true regarding highly educated women and opportunity-driven childlessness: by alleviating the time cost of having children, they contribute to limiting the positive link that exists between women's educational attainment and childlessness among highly educated women. As a result, societies strongly promoting gender equity regarding wages and fathers' involvement in children's education are more prone to limit opportunity-driven childlessness.

3.4 Conclusion

Throughout this chapter, we have shown why childlessness matters in itself. It has mainly provided additional, different insights to those coming from the intensive margin of fertility. We have shown that, historically, childlessness rates were large among the wealthiest groups. The reasons are still to be explained and contrast with the outcomes of a Malthusian model of fertility, which is usually used for historical times. Nowadays, childlessness can also be large both in developed and developing countries. Nevertheless, contrary to developed countries, high childlessness rates co-exist with high fertility along the intensive margin (high fertility of mothers) in developing countries. This difference mainly comes from the opposite effect of economic development on the burden of poverty and the economic opportunities offered to women.

We have shown theoretically that accounting for poverty-driven childlessness matters to correctly evaluate the impact that development

policies, reputed to reduce fertility, might have on the latter. Indeed, such policies might reduce childlessness rates and hence lead to an increase in overall fertility rates. Accounting for opportunity-driven childlessness in a quantity-quality model brings the result that childlessness can act as a way to reduce inequalities in the distribution of human capital, a mechanism which still has to be explored quantitatively.

Introducing marriage into an endogenous fertility model that accounts for childlessness also provides new insights: higher educational homogamy per se decreases childlessness as it favors marriage. Nevertheless, as educational homogamy is most of the time accompanied by a rise in female education, it may also be associated with increases in childlessness in the data.

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Appendix: Proof of Proposition 3.1

The aim of this appendix is not to determine how people behave for any couple $\{w, \Omega\}$ but to show that there exists a non-empty state space $\mathcal{H}$ such that agents behave the way described in Proposition 3.1. To do so, we first solve the maximization problem described in the core of the chapter under the following constraint: $n \in]0, \frac{1}{\tau}[$ and $e > 0$. In this pure interior regime, we get that:

$$c^* = \frac{(1+\tau\nu)w + \rho\pi + \Omega}{1+\alpha+\phi\beta} \tag{3.7}$$

$$n^* = \frac{\alpha}{1+\alpha+\phi\beta}\frac{(1+\tau\nu)w + \rho\pi + \Omega}{\tau w} - \nu \tag{3.8}$$

$$e^* = \frac{\phi\beta}{1+\alpha+\phi\beta}\frac{(1+\tau\nu)w + \rho\pi + \Omega}{\rho} - \pi \tag{3.9}$$

From this, we can deduce the level of wages under which such an interior regime may exist and prevail. We get that $n^* > 0 \iff w < \frac{\alpha(\rho\pi+\Omega)}{(1+\phi\beta)\tau\nu-\alpha} \equiv w_n$; $e^* > 0 \iff \frac{(1+\alpha)\rho\pi-\phi\beta\Omega}{\phi\beta(1+\tau\nu)} \equiv w_e$.

Simple computations indicate that $w_n > w_e \iff \Omega < \frac{(\tau\nu-\alpha)\rho\pi}{\tau\phi\beta\nu} \equiv \hat{\Omega}$. Let's now assume that $\Omega < \hat{\Omega}$, we get that $\forall w \in]w_e, w_n[$, the purely interior regime as described in Eqs. (3.7)–(3.9) prevails. For any $w \geq w_n$, $n^* = 0$ implying $e^* = 0$ by definition and so $c^* = w + \Omega$.

We now have to analyze cases where $w \leq w_e$. In this situation, we have to solve the maximization problem under the constraint that $e^* = 0$ and $n^* \in]0, \frac{1}{\tau}[$. In this situation, we obtain that:

$$c^* = \frac{(1+\tau\nu)w + \Omega}{1+\alpha} \tag{3.10}$$

$$n^* = \frac{\alpha}{1+\alpha}\frac{(1+\tau\nu)w + \Omega}{\tau w} - \nu \tag{3.11}$$

$$e^* = 0 \tag{3.12}$$

A simple inspection of Eq. (3.11) indicates that $n^* \leq \frac{1}{\tau} \iff w \geq \frac{\alpha\Omega}{1+\tau\nu} \equiv \underline{w}$. A condition of existence for this regime is that $\underline{w} < w_e$ what is satisfied if $\Omega > \frac{\rho\pi}{\phi\beta} \equiv \tilde{\Omega}$. Let's assume that this condition on Ω is fulfilled too. We then get that $\forall w \in]\underline{w}, w_e]$, the optimal behavior of our representative agent is represented by the set of Eqs. (3.10)–(3.12). The last situation we have to explore is $w \leq \underline{w}$. In this situation, as $n^* = \frac{1}{\tau}$, $e^* = 0$, we get:

$$c^* = \Omega$$

$$n^* = \frac{1}{\tau}$$

$$e^* = 0$$

The advised reader would have noticed that we had to assume $\Omega > \tilde{\Omega}$ and $\Omega < \hat{\Omega}$. For Proposition 3.1 to be valid, there should exist a state-space allowing the condition $\tilde{\Omega} < \hat{\Omega}$ to be fulfilled. We obtain that:

$$\tilde{\Omega} < \hat{\Omega} \iff \frac{\tau\nu - \alpha}{\tau\nu} < 1$$

what is always satisfied. We then get that $\forall\, \Omega \in]\tilde{\Omega}, \hat{\Omega}[$, the representative agent behaves as described in Proposition 3.1 what validates this latter.

Notes

1. The theoretical frameworks of Gobbi (2013) and Aaronson et al. (2014) focus on this type of childlessness.
2. In technical words, the cost of providing one unit of education to n children is equal to ρe; it does not depend on n. Most of the literature assumes that the cost of providing one unit of education to n children equals $\rho n e$. Our assumption does not change our main results qualitatively as it does not prevent the existence of a trade-off between the quality and the quantity of children. See Baudin (2011, 2012) for a generalization.
3. As shown in Jones et al. (2010), because of the log specification of the utility function, a positive non-labor income ensures the negative fertility-income relationship at the aggregate level. As mentioned by them, this could be gifts, lottery income, or bequests.
4. The condition $\tau\nu > \alpha$ should be read as follows: for a given ν, the time cost of having children has to be high enough; while for a given τ, the reservation utility in case of childlessness should be high enough.
5. For the sake of space and simplicity, we do not analyze the specification of the model in which investing in human capital is possible, while opportunity-driven childlessness does not exist but poverty-driven childlessness does. Baudin and Stelter (2018b) study such a framework allowing, furthermore, for the existence of agricultural and industrial goods.
6. Obviously, $n \geq 0$ and $n \leq \frac{1}{\tau}$ cannot bind simultaneously.
7. See Baudin et al. (2015) or Greenwood et al. (2003) for representative examples.

8. Gobbi (2018) provides a semi-cooperative model of household decisions to explain how the time to raise children varies endogenously across households of different education levels.
9. We implicitly assume here that the utility function of men is the same as that of women in the previous sections: $u^m(c^m, n)$ with $u^m(w^m + \Omega, 0) \in \mathbb{R}$ representing the indirect utility of a single man.
10. Hiller and Baudin (2016) and Baudin and Hiller (2019) extensively discuss the interactions between gender differences in preferences and marital behaviors.
11. This effect appears as the movement of the exponential part of the cumulative distribution function of love shocks for men and women.
12. In Eq. (3.6), this effect corresponds to the variations of $\Delta^f(w^f, w^m)$.

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4

Demographic Change, Wage Inequality, and Technology

Oscar Afonso, Pedro Mazeda Gil, Pedro Cunha Neves, and Tiago Neves Sequeira

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O. Afonso (✉) • P. M. Gil
Universidade do Porto, Faculdade de Economia and CEFUP, Porto, Portugal
e-mail: oafonso@fep.up.pt; pgil@fep.up.pt

P. C. Neves
Departamento de Gestão e Economia, Universidade da Beira Interior, Covilhã, Portugal

CEFAGE-UBI, Universidade da Beira Interior, Covilhã, Portugal
e-mail: pcn@ubi.pt

T. N. Sequeira
Universidade de Coimbra, Faculdade de Economia and CeBER, Coimbra, Portugal
e-mail: tms@ubi.pt

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4.1 Introduction

This chapter investigates the consequences of population ageing and demographic change for the long-run performance of economies from the perspective of wage inequality and technology intensity in the process of development.

The literature on intra-country wage inequality reveals that, over recent decades, there has been a rise in the skill premium (i.e., the ratio of high- to low-skilled labor wages or relative wage rate of high-skilled labor), which has been more significant in the Anglo-Saxon countries (e.g., the US and the UK) than in Continental Europe (e.g., France and Germany)—e.g., Violante (2008), and Kurokawa (2014). We seek to explain the observed behaviour of the skill premium with particular attention to the US experience by building on an endogenous growth model with rich demographic structures and dynamics. We do so as demographic variation and, in particular, population ageing, has become an important and comprehensive issue in the economic and social debate, with a special focus on the most developed countries (e.g., Bloom et al., 2004, 2010; The Economist, 2009, 2011; Prettner, 2013).

By considering age specific heterogeneity of households, we introduce demographic structure into a model of R&D-based technological change. Since we are particularly interested in the interplay between technology, economic growth and wage inequality across different types of human capital, a class of model useful to examine our research question is that in which growth is driven by R&D activities that use human capital as an input and R&D effort is determined as the outcome of market forces within a general equilibrium environment.

We start by considering a framework that incorporates heterogeneous human capital (high-skilled and low-skilled workers) and a standard R&D specification, following Romer (1990) and Jones (1995). This will allow us to analyse the impact of demographic change on human capital allocations and the skill premium in an otherwise standard R&D-driven growth model. Later, we extend the framework to consider a general equilibrium Directed Technical Change (DTC) model, following Acemoglu (2002) and in line with, e.g., Neves et al. (2018). We focus on

the DTC theory, as it has often been presented as the most important explanation for the trends observed in the skill premium in the last decades in the US and other developed countries (Chusseau et al., 2008). In addition, it provides an analytical framework that allows examining the interaction between the skill premium, technological-knowledge change and labour endowments, which fits our research objectives. In particular, by contrasting the predictions of the baseline model with the extended model, we are able to identify the quantitative relevance of the DTC channel regarding the observed evolution of the skill premium.

In our framework, the allocation of high-skilled workers between final-good and R&D production is endogenous and dependent on demographic variables. In particular, a lower birth rate or a higher mortality rate implies a lower share of R&D workers in the high-skilled labour population, which, in the baseline model, leads to a lower skill premium. However, when the model is extended to consider DTC, a given change in the birth/mortality rates can have a positive or a negative effect on the skill premium, depending on the elasticity of substitution between high-skilled and low-skilled labour.

We calibrate the two versions of the model to the US economy and run a set of quantitative exercises to confront the predictions of those two versions with observed co-movements in key demographic and macroeconomic variables in the period 1980–2015. In particular, we wish to assess whether the DTC mechanism and its interplay with the demographic structure in the model improves the fit of the replication of the observed skill premium vis-à-vis demographic patterns. As a first step, we consider the extended model with the DTC mechanism and investigate the consequences of the changes (shocks) in the relative endowment of high-skilled labor and in the demographic structure, through shifts in the fertility rate and in the mortality rate—namely on the relative wage of high-skilled labor and the share of R&D labor. As a second step, we shut down the DTC mechanism and re-analyse the effects of those shocks in the macroeconomic variables. By also considering separately the shocks in skills and in the demographic structure, we are able to pin down the quantitative relevance of the DTC channel vis-à-vis the demographic-structure channel, and of their interaction in the model.

This chapter is organized as follows. Section 4.2 presents a growth model in which there is population growth and R&D is performed by human capital. In Sect. 4.3, the dynamic general equilibrium of the model is derived. Section 4.4 extends the model by introducing DTC in which there are two different R&D sectors. Section 4.5 calibrates the models and presents quantitative results. Section 4.6 concludes.

4.2 The Baseline Theoretical Model

This section characterizes a general-equilibrium model of endogenous R&D, which leads to the development of new product varieties (horizontal innovation).[1] The model extends Romer (1990), Jones (1995) and Barro and Sala-i-Martin (2004: ch. 6), by allowing labour heterogeneity, i.e., high- and low-skilled labour, and also overlapping generations. We assume that there are three sectors in the economy: a final-good sector, an intermediate-good sector and an R&D sector. The final-good sector produces a homogenous good, in a perfectly competitive market, by using high- and low-skilled labour and non-durable intermediate goods. Designs are used in the Dixit and Stiglitz (1977) monopolistically competitive intermediate-good sector to produce non-durable intermediate goods. High-skilled labour is also used to produce designs in the perfectly-competitive R&D sector.

In contrast to the representative agent assumption, and in line with Prettner (2013), Prettner and Trimborn (2017), and others, we introduce overlapping generations in the R&D-based growth model in the spirit of Buiter (1988), that is, we consider a continuous-time "perpetual youth" setting (see, e.g., Blanchard, 1985; Barro and Sala-i-Martin, 2004: ch. 3; Acemoglu, 2009: ch. 9). In particular, we assume that total population consists of different generations that are distinguishable by their date of birth denoted as time t_0. Each generation consists of a measure $\mathcal{N}(t_0, t)$ of households at a certain point in time $t > t_0$. In addition, we assume that individuals have to face a constant risk of death at each instant, μ, which does not depend on age. Due to the law of large numbers, μ also denotes the fraction of individuals dying at each instant, i.e., μ is the mortality rate. Population grows at rate $n = \psi - \mu$, where

ψ denotes the instantaneous birth rate. Demographic change can then be analysed by considering shifts in the mortality rate and in the birth rate separately. Decreases in the birth rate lead to both a slowdown of population growth and to population ageing, while decreases in the (age-independent) mortality rate only increase the population growth rate and have no effect on the aggregate age decomposition (Prettner, 2013).

4.2.1 Production Side

The production function of the homogenous final good (which is also the consumption good and the numeraire) at time t is given by:

$$Y(t) = L(t)^{\alpha} H_Y(t)^{\beta} \int_0^{N(t)} X_j(t)^{1-\alpha-\beta} dj, \ 0 < \alpha < 1, \ 0 < \beta < 1, \ 0 < \alpha + \beta < 1. \tag{4.1}$$

Firms in the final goods sector employ low-skilled labour L and high-skilled labour H_Y. Both types of labour are essential to production: the former performs necessary simple tasks, whereas the latter performs complex tasks. Firms also use jth types of nondurable intermediate goods X_j,[2] with N being the number of available types and thus the technological-knowledge frontier. Moreover, α, β and $1 - \alpha - \beta$ are the low-skilled labour, the high-skilled labour and the intermediate goods shares, respectively. Bearing in mind these shares, as we can see later on, the share of high-skilled labour in total labour is assumed to take such a value that the equilibrium level of the relative wage of high- to low-skilled workers (i.e., the skill premium) is greater than unity.

Normalizing the price of the final good to unity, the final goods firm's profit at time t is given by:

$$Y(t) - \int_0^{N(t)} p_j(t) \cdot X_j(t) dj - w_L(t) \cdot L(t) - w_{H_Y}(t) \cdot H_Y(t), \tag{4.2}$$

where p_j is the price of nondurable intermediate j, $w_L(t)$ is the low-skilled labour wage and $w_{H_Y}(t)$ is the high-skilled labour wage. Since the final goods market is competitive, the usual relationships between factor

prices and marginal products are:[3]

$$p_j = L^{\alpha} \cdot H_Y^{\beta} \cdot (1-\alpha-\beta) \cdot X_j^{-\alpha-\beta} \tag{4.3}$$

$$w_L = \alpha \cdot L^{\alpha-1} \cdot H_Y^{\beta} \cdot \int_0^{N(t)} X_j^{1-\alpha-\beta} dj \tag{4.4}$$

$$w_{H_Y} = \beta \cdot L^{\alpha} \cdot H_Y^{\beta-1} \cdot \int_0^{N(t)} X_j^{1-\alpha-\beta} dj \tag{4.5}$$

Firms in the intermediate-good sector need designs (which are invented through R&D activities, as explained below) to produce goods. Once an intermediate-good firm has a new design, it becomes able to produce a new variety of an intermediate good and can retain a perpetual monopoly over its production. The production of an intermediate good requires η units of forgone final output. Thus, the flow of the monopolist's operational profit at a point of time is given by $\pi_j = (p_j - \eta) \cdot X_j$, and the present value of the returns from the operation is then given by:

$$V_j = \int_t^{\infty} \pi_j(v) \cdot e^{-\int_t^v r(\omega)d\omega} dv, \tag{4.6}$$

where r is the real interest rate. Hence, the intermediate-good monopolist faces the demand curve (4.3) and solves the following problem:

$$max \int_t^{\infty} \left(p_j(v) - \eta\right) \cdot X_j(v) \cdot e^{-\int_t^v r(\omega)d\omega} dv. \tag{4.7}$$

The monopolist's optimal production and price (markup) are:

$$X_j \equiv X = \left(\frac{L^{\alpha} \cdot H_Y^{\beta} \cdot (1-\alpha-\beta)^2}{\eta} \right)^{\frac{1}{\alpha+\beta}}, \tag{4.8}$$

$$p_j \equiv p = \frac{\eta}{1-\alpha-\beta}. \tag{4.9}$$

Thus, each monopolist in the intermediate sector produces the same amount, X, charges the same price, p, and has profit $\pi(t) = (p-\eta)\cdot X(t)$ at every period. This also implies that the present value of the monopoly operational profit is the same for each monopolist firm, $V_j = V$.

Each design for a new variety of an intermediate good is invented through R&D activities. We assume that R&D activities require η/N^{ϕ} units of high-skilled labour to invent a new design, with $0 < \phi < 1$. Thus, the existing stock of designs spills over (designs are non-rival goods) and, as the existing stock of designs becomes larger, a lower level of high-skilled labour is required for the invention ("shoulders-of-giants" effect). The invention cost, Z, is then given by:

$$Z(t) = \frac{\eta}{N(t)^{\phi}} w_{H_N}(t), \tag{4.10}$$

where w_{H_N} is the wage for high-skilled labour engaged in R&D.

We assume that there is free entry into R&D. Hence, any firm can pay Z to secure the present value of monopoly profit and thus, in equilibrium, the free-entry condition $V = Z$ is satisfied:

$$V(t) = \int_t^{\infty} \pi(v) \cdot e^{-\int_t^v r(\omega)d\omega} dv = Z(t). \tag{4.11}$$

Differentiating both sides of (4.11) with respect to time yields the no-arbitrage condition:

$$r(t) = \frac{\pi(t)}{Z(t)} + \frac{\dot{Z}(t)}{Z(t)}. \tag{4.12}$$

In addition, because η/N^{ϕ} units of high-skilled labour are required to invent one new design, the aggregate amount of high-skilled labour devoted to R&D is given by $H_N(t) = \dot{N}(t)\left[\eta/N(t)^{\phi}\right]$. We then can obtain:

$$\dot{N}(t) = \frac{1}{\eta} \cdot H_N(t) \cdot N(t)^{\phi} \tag{4.13}$$

In (4.13), $1/\eta$ can be understood as the productivity of high-skilled labour in R&D activities and $0 < \phi < 1$ the size of technological spillovers. Therefore, the technological frontier expands faster if scientists are more productive or technological spillovers are higher. If $\phi = 1$, spillovers are strong enough and developing new designs does not become ever more difficult as the technological frontier expands. If, in contrast, $\phi < 1$, the level of spillovers implies that developing new designs becomes more and more difficult with an expanding technological frontier.

In equilibrium, the high-skilled labour should receive the same wage rate in the final-good sector and in the R&D sector; i.e., $w_H(t) \equiv w_{H_Y}(t) = w_{H_N}(t)$. From (4.4) and (4.5), the skill premium (i.e., the high-skilled wage, w_H, relative to low-skilled wage, w_L) is given by:

$$\frac{w_H}{w_L} = \frac{\beta}{\alpha} \cdot \frac{L(t)}{H_Y(t)}. \tag{4.14}$$

Denoting s as the share of high-skilled labour in the total population, $s \equiv H/\mathcal{L}$, with $\mathcal{L} = L + H$ and $H = H_N + H_Y$, and u the share of R&D workers in the high-skilled labour population, $u \equiv H_N/H$, the skill premium is then:

$$\frac{w_H(t)}{w_L(t)} = \frac{\beta}{\alpha} \cdot \frac{1-s}{s} \cdot \frac{1}{1-u(t)} \tag{4.15}$$

where s is assumed to be constant and exogenously given. We assume that the condition $s < \frac{\beta}{\alpha+\beta}$ is satisfied. Therefore, $\frac{w_H}{w_L} > 1$ holds for any value of $0 < u < 1$ and this condition implies that no high-skilled labour wishes to work as low-skilled labour. From (4.15), the skill premium is higher when: (i) the share of high-skilled labour in production, β , is higher; (ii) the share of low-skilled labour in production, α, is smaller; (iii) the share (i.e., the supply) of high-skilled labour in the total population, s, is smaller; (iv) the share of R&D workers in the high-skilled labour population, u, is higher.

Thus, we have the following equations:

$$H_Y(t) = (1-u(t)) \cdot s \cdot \mathcal{L}(t);\ H_N(t) = u(t) \cdot s \cdot \mathcal{L}(t);\ L(t) = (1-s) \cdot \mathcal{L}(t) \tag{4.16}$$

where $\mathcal{L}$, the total population, grows at a constant rate $n = \psi - \mu$, $\dot{\mathcal{L}}(t) = n \cdot \mathcal{L}(t)$.

4.2.2 Consumption Side with Demographic Variation

As already stated, total population consists of different generations, distinguishable by their date of birth denoted as time t_0, and each generation consists of a measure $\mathcal{N}(t_0, t)$ of households at a certain point in time $t > t_0$. We normalize the number of members of each household at the date of its birth, t_0, to 1. Households consume and collect income from investments in financial assets (equity) and from labour. Workers have heterogeneous human capital endowments so that, in the aggregate, an economy is endowed with H high-skilled and L low-skilled units of labour that are inelastically supplied to firms. Thus, total labour supply is $L + H = \mathcal{L}$.

4.2.2.1 The Household's Consumption Path

We assume that each household faces a constant risk of death at each instant, which we denote as μ. Due to the law of large numbers, this rate is equal to the fraction of households dying at each instant. Since the population grows at rate $n = \psi - \mu > 0$, the birth rate ψ is higher than μ. Thus, a household, who is born at t_0, wishes to maximize its discounted stream of lifetime utility U as given by:

$$U = \int_{t_0}^{\infty} \log c(t) \cdot e^{-(\mu+\rho)(\tau - t_0)} d\tau, \tag{4.17}$$

where c is the household's consumption of the final good and $\rho > 0$ is the subjective discount rate. Moreover, the household's mortality rate μ augments the discount rate, since the household faces the risk of death and, as a result, does not wish to postpone consumption to the same extent as in case of no lifetime uncertainty. Furthermore, in line with Yaari (1965), we consider that households insure themselves against the risk of dying with positive assets by using their entire savings to buy actuarial notes from a fair life-insurance firm. This firm, in turn, redistributes the

wealth of households who have died to the surviving ones within a certain generation and thus the real rate of return in the household flow budget constraint is augmented by the mortality rate:

$$\dot{k}(t) = (r(t) + \mu) \cdot k(t) + (1 - s) \cdot w_L(t) + s \cdot w_H(t) - c(t), \quad (4.18)$$

where k is the household's total real financial assets. The initial level of wealth $k(0)$ is given and the non-Ponzi games condition $lim_{t\to\infty} e^{-\int_0^t r(s)ds} \cdot k(t) \geq 0$ is also imposed. The Euler equation for consumption and the transversality condition are standard (i.e., they are similar to the ones without lifetime uncertainty):

$$g_c(t) = r(t) - \rho \quad (4.19)$$

$$\lim_{t\to\infty} \lambda(t) \cdot k(t) = 0, \quad (4.20)$$

where $g_c(t) \equiv \frac{\dot{c}(t)}{c(t)}$ and $\lambda(t)$ denotes the shadow price of assets in the present-value Hamiltonian.

4.2.2.2 The Aggregate Consumption Path

As our economy does not feature one single representative household, in this subsection, we use an aggregation rule to come up with the aggregate Euler equation for consumption. As households are heterogeneous with respect to age, they are also heterogenous with respect to accumulated wealth because older households have had more time to build up positive assets. In the appendix, we show that the aggregate Euler equation is:

$$g_C(t) = r(t) - \rho + n - \psi \cdot (\mu + \rho) \cdot \frac{K(t)}{C(t)}, \quad (4.21)$$

where $g_C(t) \equiv \frac{\dot{C}(t)}{C(t)}$ and:

$$K(t) \equiv \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \int_{-\infty}^{t} k(t_0, t) \cdot e^{\psi t_0} dt_0, \quad (4.22)$$

being $K(t)$ the aggregate assets/financial wealth. Since the growth rate of the aggregate consumption per capita, $\bar{c} \equiv C/\mathcal{L}$, is given by $g_{\bar{c}}(t) = r(t) - \rho - \psi\,(\mu + \rho)\,\frac{K(t)}{C(t)}$, it is easy to see that it is always lower than the household's consumption growth, in (4.19). This reflects the fact that, at each instant, a fraction μ of older and, therefore, wealthier households die and are replaced by a fraction ψ of poorer newborn households. As the latter can afford less consumption than the former, the turnover of generations slows down aggregate consumption growth (see, e.g., Barro and Sala-i-Martin, 2004, ch. 3; Acemoglu, 2009, ch. 9).

In the appendix, we also show that the aggregate law of motion for assets (equivalent to the aggregate flow budget constraint for households) is:

$$\dot{K}(t) = r \cdot K(t) - C(t) + W(t), \tag{4.23}$$

where aggregate wages are: $W(t) = \mathcal{L}(0) \cdot e^{(\psi-\mu)t} \cdot [(1-s) \cdot w_L(t) + s \cdot w_H(t)]$. In contrast to individual assets, the evolution of aggregate assets does not depend on mortality. The reason is that the life insurance company redistributes wealth between cohorts such that the mortality effect vanishes in the aggregate. It can then be shown that (4.23), together with the aggregate financial wealth in households' balance sheet, $K(t) = N(t) \cdot Z(t)$, and Eqs. (4.3)–(4.5) and (4.8)–(4.10), is equivalent to the product market equilibrium condition:

$$Y(t) = \eta \cdot N(t) \cdot X(t) + C(t) \tag{4.24}$$

4.3 Dynamic General Equilibrium

The dynamic general equilibrium is defined by the paths of allocations and price distributions $(\{X(\omega,t), p(\omega,t)\}, \omega \in [0,N])_{t\geq 0}$, and the aggregate paths $(C(t), N(t), u(t), r(t), \quad w_L(t), w_H(t))_{t\geq 0}$, such that: (i) households, final-good firms and intermediate-good firms solve their problems; (ii) the innovation free-entry and no-arbitrage conditions are

satisfied; and (iii) markets clear. Total supplies of high- and low-skilled labour are exogenous.

In order to analyse the dynamics of the economy, we need to derive the equations that explain the dynamics of $C(t)$, $u(t)$, and $N(t)$. One differential equation is derived from the aggregate Euler equation (4.21) together with the expression for the rate of return given by (4.12):

$$g_C(t) \equiv \frac{\dot{C}(t)}{C(t)} = \frac{\pi(t)}{Z(t)} + g_Z(t) - \rho + n - \psi \cdot (\mu + \rho) \cdot \Omega(t) \quad (4.25)$$

where $g_Z(t) \equiv \frac{\dot{Z}(t)}{Z(t)}$ and $\Omega(t) \equiv \frac{K(t)}{C(t)}$, with $K(t) = N(t) \cdot Z(t)$. Since $\pi(t) = (p - \eta) \cdot X(t)$, bearing in mind (4.8) and (4.9), the monopolist's operational profits can be rewritten as:

$$\pi(t) = \eta^{\frac{-(1-\alpha-\beta)}{\alpha+\beta}} \cdot (\alpha+\beta) \cdot (1-\alpha-\beta)^{\frac{2-\alpha-\beta}{\alpha+\beta}} \cdot (1-s)^{\frac{\alpha}{\alpha+\beta}} \cdot s^{\frac{\beta}{\alpha+\beta}} \cdot (1-u(t))^{\frac{\beta}{\alpha+\beta}} \cdot \mathcal{L}(t). \quad (4.26)$$

Using (4.4), (4.5) and (4.10) results in equilibrium with $w_{H_Y}(t) = w_{H_N}(t)$:

$$Z(t) = \frac{\eta}{N(t)^{\phi}} \cdot \beta \cdot L^{\alpha} \cdot H_Y^{\beta-1} \cdot \int_0^{N(t)} X_j^{1-\alpha-\beta} dj. \quad (4.27)$$

Taking into account (4.8) and (4.16), this expression can be rewritten as:

$$Z(t) = \beta \cdot \eta^{\frac{2(\alpha+\beta)-1}{\alpha+\beta}} \cdot (1-\alpha-\beta)^{\frac{2(1-\alpha-\beta)}{\alpha+\beta}} \cdot (1-s)^{\frac{\alpha}{\alpha+\beta}} \cdot s^{\frac{-\alpha}{\alpha+\beta}} \cdot (1-u(t))^{\frac{-\alpha}{\alpha+\beta}} \cdot N(t)^{1-\phi}. \quad (4.28)$$

From (4.28), taking logs and differentiating with respect to time yields:

$$g_Z(t) = \frac{\alpha}{\alpha+\beta} \cdot T(t)^{-1} \cdot g_u(t) + (1-\phi) \cdot g_N(t), \quad (4.29)$$

where: $T(t) \equiv \frac{1-u(t)}{u(t)}$, $g_u(t) \equiv \frac{\dot{u(t)}}{u(t)}$ and $g_N(t) \equiv \frac{\dot{N}(t)}{N(t)}$. Substituting (4.26), (4.28) and (4.29) into (4.25), and bearing also in mind (4.13) and (4.16), yields:

$$g_C(t) = \frac{(1-\alpha-\beta)\cdot(\alpha+\beta)}{\beta}\cdot T(t)\cdot g_N(t) + (1-\phi)\cdot g_N(t) + \frac{\alpha}{\alpha+\beta}\cdot T(t)^{-1}\cdot g_u(t) - \rho + n - \psi\cdot(\mu+\rho)\cdot\Omega(t) \tag{4.30}$$

The second differential equation, which shows the dynamics of N, $g_N(t) \equiv \frac{\dot{N}(t)}{N(t)}$, is given by (4.13). However, it will be convenient to rewrite the latter by taking logs and differentiating it with respect to time, so that we get:

$$g_{g_N}(t) \equiv \frac{\dot{g_N(t)}}{g_N(t)} = g_u(t) - (1-\phi)\cdot g_N(t) + \psi - \mu, \tag{4.31}$$

where we also made use of (4.16).

The third differential equation describes the path of u. Considering (4.8) and (4.16), the final-good production function (4.1) can be written as:

$$Y(t) = \eta^{\frac{-(1-\alpha-\beta)}{\alpha+\beta}}\cdot(1-\alpha-\beta)^{\frac{2(1-\alpha-\beta)}{\alpha+\beta}}\cdot(1-s)^{\frac{\alpha}{\alpha+\beta}}\cdot s^{\frac{\beta}{\alpha+\beta}}\cdot(1-u(t))^{\frac{\beta}{\alpha+\beta}}\cdot\mathcal{L}(t)\cdot N(t). \tag{4.32}$$

Bearing in mind (4.32) and then taking logs and differentiating with respect to time:

$$g_u(t) = \frac{\alpha+\beta}{\beta}\cdot T(t)\cdot[g_N(t) - g_Y(t) + \psi - \mu] \tag{4.33}$$

where $g_Y(t) \equiv \frac{\dot{Y}(t)}{Y(t)}$. Bearing once again in mind (4.8), (4.16), and (4.24), we have:

$$C(t) = Y(t) - \eta \cdot N(t) \cdot X(t) = Y(t) \cdot \left[1 - (1 - \alpha - \beta)^2\right], \quad (4.34)$$

and taking logs and differentiating with respect to time, yields $g_C(t) = g_Y(t)$. Hence, also bearing in mind (4.30), Eq. (4.33) can be written as:

$$g_u(t) = T(t) \cdot \left[\frac{(\alpha + \beta - 1) \cdot (\alpha + \beta)}{\beta} \cdot T(t) \cdot g_N(t) + \phi \cdot g_N(t) + \psi \cdot (\mu + \rho) \cdot \Omega(t) + \rho\right]. \quad (4.35)$$

To describe the dynamics of the economy, it will also be convenient to derive a differential equation for $\Omega(t) \equiv \frac{K(t)}{C(t)} = \frac{Z(t) \cdot N(t)}{C(t)}$. Considering the definition and then taking logs and differentiating with respect to time, we get $g_\Omega(t) = g_Z(t) + g_N(t) - g_C(t)$, which, from (4.29) and (4.30), is equivalent to:

$$g_\Omega(t) = \left[\frac{(\alpha + \beta - 1) \cdot (\alpha + \beta)}{\beta} \cdot T(t) \cdot g_N(t) + g_N(t) + \psi \cdot (\mu + \rho) \cdot \Omega(t) + \rho - \psi + \mu\right] \quad (4.36)$$

Together, Eqs. (4.30) (4.31), (4.35), plus (4.36), describe the dynamics of the economy.

Finally, we note that a balanced-growth path (BGP) as a representation of the dynamic long-run equilibrium associated with the dynamical system described above is the aggregate path $(C(t)^*, N(t)^*, u(t)^*)_{t \geq 0}$, along which the growth rates g_C^*, g_N^*, and g_u^* are constant. By considering the dynamic equations above, together with (4.24), (4.8), (4.32), and (4.34), a BGP only exists if: (i) the asymptotic growth rates of consumption, the final-good output and aggregate assets are equal to the growth rate of technological knowledge (the number of varieties) plus the population

growth rate, $g_C = g_Y = g_K = g_N + n$; and (ii) the share of R&D workers, the real interest rate, the real wages, and the technological knowledge growth rate are asymptotically trendless, $g_u{=}g_r = g_{w_L} = g_{w_H} = g_{g_N} = 0$. Notice that the condition $g_Y = g_N + n$ also implies that the per capita growth rate is given by $g_y = g_N$, where $y \equiv Y/\mathcal{L}$.

Under these conditions, we derive the steady-state values of g_N, u (or $T \equiv \frac{1-u}{u}$), and $\Omega \equiv \frac{K}{C}$.[4] Using $g_u = g_{g_N} = 0$ together with (4.31) and (4.35), we find the steady-state values of g_N and Ω as[5]:

$$g_N^* = \frac{\psi - \mu}{1 - \phi}, \tag{4.37}$$

$$\Omega^* = \frac{1}{\psi(\mu + \rho)} \cdot \left\{ \left(\frac{\psi - \mu}{1 - \phi} \right) \cdot \left[\frac{(1 - \alpha - \beta) \cdot (\alpha + \beta)}{\beta} \cdot T^* - \phi \right] - \rho \right\}. \tag{4.38}$$

Moreover, from (4.13) and (4.37), it results that, in steady state, $\frac{N^{*1-\phi}}{H_N^*} = \left(\frac{\psi-\mu}{1-\phi} \right)^{-1} \cdot \frac{1}{\eta}$, which is equivalent to, $\frac{N^{*1-\phi}}{H_Y^*} = \left(\frac{\psi-\mu}{1-\phi} \right)^{-1} \cdot \frac{1}{\eta} \cdot \frac{1}{T^*}$. In turn, from (4.28) and (4.32), it results that $Z = \eta \cdot \beta \cdot N^{-\phi} \cdot H_Y^{-1} \cdot Y$, and thus, from (4.34), $\Omega \equiv \frac{K}{C} \equiv \frac{Z \cdot N}{C} = \frac{\eta \cdot \beta}{1-(1-\alpha-\beta)^2} \frac{N^{1-\phi}}{H_Y}$. Hence, in the steady state, we also have the following relationship between Ω^* and T^*:

$$\Omega^* = \frac{\beta}{1 - (1 - \alpha - \beta)^2} \cdot \left(\frac{\psi - \mu}{1 - \phi} \right)^{-1} \cdot \frac{1}{T^*}. \tag{4.39}$$

The latter together with (4.38) yields:

$$T^* \cdot \left(\frac{\psi - \mu}{1 - \phi} \right) \cdot \frac{1 - (1 - \alpha - \beta)^2}{\beta} \cdot \frac{1}{\psi(\mu + \rho)} \cdot \left\{ \left(\frac{\psi - \mu}{1 - \phi} \right) \cdot \left[\frac{(1 - \alpha - \beta) \cdot (\alpha + \beta)}{\beta} \cdot T^* - \phi \right] - \rho \right\} = 1. \tag{4.40}$$

Then, recalling $T(t) \equiv \frac{1-u(t)}{u(t)}$ and considering the positive root in (4.40), we derive $u^* = \frac{1}{1+T^*}$ as a function of the parameters of the model. It is straightforward to show that (4.40) has two roots of opposite sign (and thus one positive root) if the condition $\frac{(1-\alpha-\beta)\cdot(\alpha+\beta)}{1-(1-\alpha-\beta)^2} \cdot \psi \cdot (\mu + \rho) > 0$ is satisfied, which is always true given the assumptions on the parameters of the model (see Sects. 4.2.1 and 4.2.2, above).

In turn, by using u^* in the skill premium equation (4.15), to get:

$$\bar{w}^* \equiv \left(\frac{w_H}{w_L}\right)^* = \frac{\beta}{\alpha} \cdot \frac{1-s}{s} \cdot \frac{1}{1-u^*}, \tag{4.41}$$

and considering also (4.37), we are able to study the long-run relationship between the mortality and the birth rate, the per capita economic growth rate, $g_y^* = g_N^*$, the skill premium, $\bar{w}^*$, and the aggregate capital-to-consumption ratio, Ω^*.

The following proposition summarizes the comparative-statics results concerning the effect of changes in the structural demographic parameters on u^*, $\bar{w}^*$, Ω^*, and g_Y^*.

Proposition 4.1 *A higher birth rate, ψ, or a lower mortality rate, μ, implies a higher share of R&D workers in the high-skilled labour population, u^*, skill premium, $\bar{w}^*$, and per capita economic growth, g_y^*, and a lower aggregate capital-to-consumption ratio.*

Proof Obtained by checking the partial derivatives of Eqs. (4.37), (4.39)–(4.40), and (4.41) with respect to ψ and to μ, using the Implicit Function Theorem. □

Figure 4.1 illustrates the comparative-statics results.

As in the semi-endogenous growth theory, an increase in the population growth rate (motivated by higher birth rates and/or lower mortality rates) accelerates the steady-state per capita economic growth. In addition, it also has an impact on the allocation of high-skilled workers between final-good and R&D production, as well as in the aggregate capital-to-consumption ratio in steady-state. In particular, the model predicts

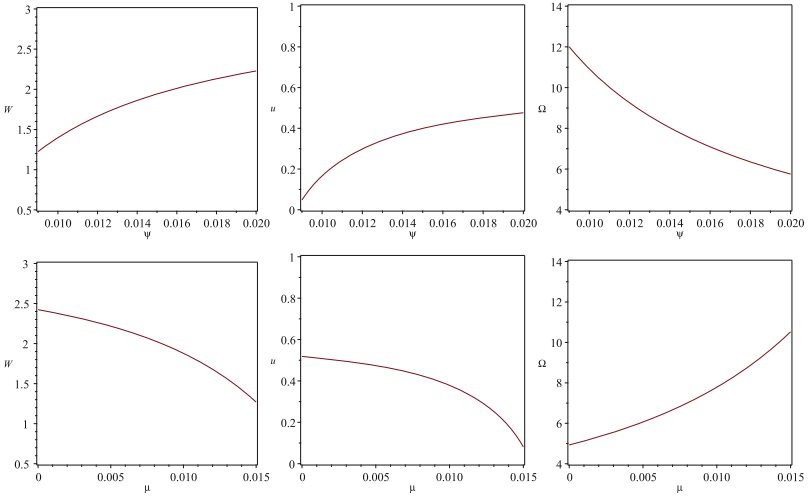


Fig. 4.1 The effect of the birth rate, ψ, and of the mortality rate, μ, on the skill premium, $\tilde{w}^*$, the share of R&D workers in the high-skilled labour population, u^*, and the aggregate capital-to-consumption ratio, Ω^*. $\alpha = 0.4$; $\beta = 0.2$; $\phi = 0.7$; $\rho = 0.02$, $s = 0.3$

that when population grows at higher rates, a higher fraction of the high-skilled population will work in R&D activities and the aggregate capital-to-consumption ratio will be lower. Given that low and high-skilled population are assumed to grow at the same rate, a higher share of R&D workers in the high-skilled population increases the relative supply of low-skilled labour in final-good production, which, through the usual supply effect, leads to an increase in the skill premium (see Eq. (4.14)).

This result also implies that a lower birth rate, and subsequently ageing, implies a lower share of R&D workers leading to a lower effort of the economy in R&D and a lower skill premium. These results imply that we should expect an inversion of the past increasing trend in the skill premium due to the demographic features of current-days societies (e.g. due to lower birth rates that also lead to ageing). Ageing, lower R&D effort, and a decreasing skill premium should be common features of our economies.

4.4 Skill Premium and R&D Labour with Directed Technical Change

In this section, we extend the model by considering the existence of Directed Technical Change (DTC).

DTC is modelled following Neves et al. (2018), with two main differences. First, in order to maintain the comparability with the previous sections, we consider that both skilled and unskilled population grow at the same rate. Second, while in Neves et al. (2018) the allocations of high-skilled labour and R&D workers were assumed exogenous, here we endogenize them, which allows us to investigate how they are affected by demographic changes.

In this section, we are primarily interested in examining whether and how the main results in the previous section are altered (or differ) when a DTC framework is considered.

4.4.1 Production Side

As in the standard DTC literature (e.g., Acemoglu, 2002), we now consider two available technologies: one using low-skilled labour together with low-skilled specific intermediate goods, and another using high-skilled labour together with high-skilled specific intermediate goods. Each final-good firm operates in only one technology, and thus it uses either highskilled or low-skilled labour. The final-good production function in each technology is given by:

$$Y_L(t) = L(t)^{\beta} \int_0^{N_L(t)} X_{j,L}(t)^{1-\beta} dj, \ 0 < \beta < 1 \tag{4.42}$$

$$Y_H(t) = H_Y(t)^{\beta} \int_0^{N_H(t)} X_{j,H}(t)^{1-\beta} dj, \ 0 < \beta < 1$$

where indexes L and H refer to the low-skilled and high-skilled technologies, respectively. In each technology, the share of labour in production is β and the share of intermediate goods is $1 - \beta$.

As in Acemoglu (2002), aggregate output is given by:

$$Y(t) = \left[\gamma Y_L(t)^{\frac{\varepsilon-1}{\varepsilon}} + (1-\gamma)\, Y_H(t)^{\frac{\varepsilon-1}{\varepsilon}}\right]^{\frac{\varepsilon}{\varepsilon-1}} \tag{4.43}$$

with ε standing for the elasticity of substitution between the two technologies (the two technologies are gross substitutes if $\varepsilon > 1$ and gross complements if $\varepsilon < 1$) and $0 < \gamma < 1$ standing for the importance of each technology in aggregate output.

In the presence of DTC, final-good prices in each technology, P_L and P_H, are no longer constant. Thus, the profits of each final-good firm are:

$$P_L(t) \cdot Y_L(t) - \int_0^{N_L(t)} p_{j,L}(t) \cdot X_{j,L}(t)dj - w_L(t) \cdot L(t)$$

$$P_H(t) \cdot Y_H(t) - \int_0^{N_H(t)} p_{j,H}(t) \cdot X_{j,H}(t)dj - w_H(t) \cdot H_Y(t) \tag{4.44}$$

In a context of perfectly competitive final-good markets, the relative final-good prices are equal to:

$$\frac{P_H(t)}{P_L(t)} = \frac{1-\gamma}{\gamma}\left(\frac{Y_H(t)}{Y_L(t)}\right)^{-\frac{1}{\varepsilon}} \tag{4.45}$$

and the factor prices are given by:

$$\begin{aligned} p_{j,L}(t) &= P_L(t) \cdot L(t)^{\beta} \cdot (1-\beta) \cdot X_{j,L}(t)^{-\beta}; \\ p_{j,H}(t) &= P_H(t) \cdot H_Y(t)^{\beta} \cdot (1-\beta) \cdot X_{j,H}(t)^{-\beta} \end{aligned} \tag{4.46}$$

$$\begin{aligned} w_L(t) &= \beta \cdot P_L(t) \cdot L(t)^{\beta-1} \cdot \int_0^{N_L(t)} X_{j,L}(t)^{1-\beta} dj; \\ w_H(t) &= \beta \cdot P_H(t) \cdot H_Y(t)^{\beta-1} \cdot \int_0^{N_H(t)} X_{j,H}(t)^{1-\beta} dj \end{aligned} \tag{4.47}$$

Monopolist's profit maximization in each technology yields the following optimal production and markup:

$$X_{j,L}(t) \equiv X_L(t) = L(t) \cdot \left(\frac{P_L(t) \cdot (1-\beta)^2}{\eta_L} \right)^{\frac{1}{\beta}};$$

$$X_{j,H}(t) \equiv X_H(t) = H_Y(t) \cdot \left(\frac{P_H(t) \cdot (1-\beta)^2}{\eta_H} \right)^{\frac{1}{\beta}} \tag{4.48}$$

$$p_{j,L}(t) \equiv p_L = \frac{\eta_L}{1-\beta}; \qquad p_{j,H}(t) \equiv p_H = \frac{\eta_H}{1-\beta} \tag{4.49}$$

with η_L and η_H standing for the costs of producing one unit of intermediate good of technology L and H, respectively.

We now consider two types of R&D activities: one aimed at discovering new designs to a new variety of an intermediate good of technology L, and the other aimed at discovering new designs to a new variety of an intermediate good of technology H. The amount of high-skilled labour engaged in each type of R&D (scientists) is $H_{N_L}(t)$ and $H_{N_H}(t)$, with $H_{N_H}(t) + H_{N_L}(t) = H_N(t)$. We denote $b(t) \equiv H_{N_H}(t)/H_N(t)$ as the share of total scientists working in R&D for the H technology. Both groups of scientists are paid the same wage, $w_{H_N}(t)$.[6] Thus, the invention costs, the R&D functions and the no-arbitrage conditions for each technology are:

$$Z_L(t) = \frac{\eta_L}{N_L(t)^{\phi}} w_{H_N}(t); \quad Z_H(t) = \frac{\eta_H}{N_H(t)^{\phi}} w_{H_N}(t) \tag{4.50}$$

$$\dot{N}_L(t) = \frac{1}{\eta_L} \cdot H_{N_L}(t) \cdot N_L(t)^{\phi}; \quad \dot{N}_H(t) = \frac{1}{\eta_H} \cdot H_{N_H}(t) \cdot N_H(t)^{\phi} \tag{4.51}$$

$$r(t) = \frac{\pi_L(t)}{Z_L(t)} + \frac{\dot{Z}_L(t)}{Z_L(t)}; \quad r(t) = \frac{\pi_H(t)}{Z_H(t)} + \frac{\dot{Z}_H(t)}{Z_H(t)} \tag{4.52}$$

Using Eq. (4.48) in (4.42), (4.47) and in the expression for monopolist profits, we can write output, wages and profits as functions of the stock

of technological knowledge in each technology, $N_L(t)$ and $N_H(t)$:

$$Y_L(t) = \left(\frac{(1-\beta)^2}{\eta_L}\right)^{\frac{1-\beta}{\beta}} \cdot L(t) \cdot N_L(t) \cdot P_L(t)^{\frac{1-\beta}{\beta}};$$

$$Y_H(t) = \left(\frac{(1-\beta)^2}{\eta_H}\right)^{\frac{1-\beta}{\beta}} \cdot H_Y(t) \cdot N_H(t) \cdot P_H(t)^{\frac{1-\beta}{\beta}} \tag{4.53}$$

$$w_L(t) = \beta \cdot \left(\frac{(1-\beta)^2}{\eta_L}\right)^{\frac{1-\beta}{\beta}} \cdot N_L(t) \cdot P_L(t)^{\frac{1}{\beta}};$$

$$w_H(t) = \beta \cdot \left(\frac{(1-\beta)^2}{\eta_H}\right)^{\frac{1-\beta}{\beta}} \cdot N_H(t) \cdot P_H(t)^{\frac{1}{\beta}} \tag{4.54}$$

$$\pi_L(t) = \frac{\beta(1-\beta)^{\frac{2}{\beta}}}{\eta_L^{\frac{1-\beta}{\beta}}} \cdot L(t) \cdot P_L(t)^{\frac{1}{\beta}};$$

$$\pi_H(t) = \frac{\beta(1-\beta)^{\frac{2}{\beta}}}{\eta_H^{\frac{1-\beta}{\beta}}} \cdot H_Y(t) \cdot P_H(t)^{\frac{1}{\beta}} \tag{4.55}$$

Combining Eqs. (4.53), (4.54) and (4.45), the relative final-good prices and skill premium are given by:

$$\frac{P_H(t)}{P_L(t)} = \left(\frac{1-\gamma}{\gamma}\right)^{\frac{\beta\varepsilon}{\sigma}} \left(\frac{\eta_H}{\eta_L}\right)^{\frac{1-\beta}{\sigma}} \left(\frac{N_H(t)}{N_L(t)}\right)^{\frac{-\beta}{\sigma}} \left(\frac{s}{1-s} \cdot (1-u(t))\right)^{-\frac{\beta}{\sigma}} \tag{4.56}$$

$$\bar{w}(t) = \left(\frac{1-\gamma}{\gamma}\right)^{\frac{\beta\varepsilon}{\sigma}} \left(\frac{\eta_H}{\eta_L}\right)^{1-\frac{\varepsilon}{\sigma}} \left(\frac{N_H(t)}{N_L(t)}\right)^{\frac{\sigma-1}{\sigma}} \left(\frac{1-s}{s} \cdot \frac{1}{1-u(t)}\right)^{\frac{1}{\sigma}} \tag{4.57}$$

where, following Acemoglu (2002), $\sigma = 1 + (\varepsilon - 1)\beta$ is the elasticity of substitution between low-skilled and high-skilled labour in final-good production and N_H/N_L is the technological-knowledge bias.

Equation (4.57) reveals that, as in the baseline model, the skill premium depends negatively on the share of high-skilled labour in total population and positively on the share of R&D workers in the high-skilled labour population.

However, similarly to Neves et al. (2018), in the presence of DTC the influence of the demographic variables on the skill premium is no longer linear, depending on the value of parameter σ. In addition, the skill premium is now also influenced by the technological-knowledge bias, with σ also playing a decisive role in determining how such influence is exerted. As in Acemoglu (2002), if high-skilled and low-skilled labour are gross substitutes ($\sigma > 1$), an increase in the technological-knowledge bias raises the skill premium. On the contrary, if the two types of labour are gross complements ($\sigma < 1$), a more H-biased technology contributes to a lower skill premium. These are important differences to the baseline model, which, as we will show later on, will have a crucial role in explaining the differences in the final results between the two approaches (baseline model vs. DTC model).

4.4.2 Consumption Side

As in the baseline model, the utility maximization problem of each household is defined as:

$$\begin{aligned} max\ U_L &= \int_{t_0}^{\infty} \log c_L(t) \cdot e^{-(\mu+\rho)(\tau-t_0)} d\tau, \\ s.t.\ \dot{k}_L(t) &= (r(t)+\mu) \cdot k_L(t) + w_L(t) - c_L(t) \\ max\ U_H &= \int_{t_0}^{\infty} \log c_H(t) \cdot e^{-(\mu+\rho)(\tau-t_0)} d\tau, \\ s.t.\ \dot{k}_H(t) &= (r(t)+\mu) \cdot k_H(t) + w_H(t) - c_H(t) \end{aligned} \tag{4.58}$$

Using the same aggregation procedures, the aggregate Euler equations and law of motion of aggregate capital are:

$$g_{CL}(t) = r(t) - \rho + n - \psi \cdot (\mu + \rho) \cdot \frac{K_L(t)}{C_L(t)};$$

$$g_{CH}(t) = r(t) - \rho + n - \psi \cdot (\mu + \rho) \cdot \frac{K_H(t)}{C_H(t)} \tag{4.59}$$

$$\dot{K_L}(t) = r(t) \cdot K_L(t) - C_L(t) + W_L(t);$$

$$\dot{K_H}(t) = r(t) \cdot K_H(t) - C_H(t) + W_H(t) \tag{4.60}$$

with $K_L/C_L \equiv \Omega_L$ and $K_H/C_H \equiv \Omega_H$.

From Eqs. (4.48) and (4.42), aggregate output of each technology can be written as $Y_L(t) = \eta_L(t) \cdot N_L(t) \cdot X_L(t) \cdot \frac{1}{P_L(t)\cdot(1-\beta)^2}$ and $Y_H(t) = \eta_H(t) \cdot N_H(t) \cdot X_H(t) \cdot \frac{1}{P_H(t)\cdot(1-\beta)^2}$. Using these results together with Eqs. (4.60) and (4.48)–(4.52), and bearing in mind that $K_H(t) = N_H(t)Z_H(t)$ and $K_L(t) = N_L(t)Z_L(t)$, we obtain, after some algebra, the following expressions for the aggregate consumption by low-skilled and high-skilled households:

$$C_L(t) = \left(2 - \beta - \frac{P_H(t) \cdot Y_H(t)}{P_L(t) \cdot Y_L(t)} \cdot \frac{u(t) \cdot (1 - b(t))}{1 - u(t)}\right) \cdot \beta \cdot P_L(t) \cdot Y_L(t) \tag{4.61}$$

$$C_H(t) = \left(\frac{1 - u(t) \cdot b(t)}{1 - u(t)} + 1 - \beta\right) \cdot \beta \cdot P_H(t) \cdot Y_H(t) \tag{4.62}$$

with $\left(2 - \beta - \frac{P_H(t)Y_H(t)}{P_L(t)Y_L(t)} \cdot \frac{u(t)\cdot(1-b(t))}{1-u(t)}\right) > 0$.

Summing Eqs. (4.61) and (4.62) yields the following linear relationship between total consumption in the economy and total nominal output,[7]

$$C(t) = C_L(t) + C_H(t) = (2 - \beta) \cdot \beta \cdot (P_L(t)Y_L(t) + P_H(t)Y_H(t)) \tag{4.63}$$

which is equivalent to the product market equilibrium condition:

$$P_L(t)Y_L(t) + P_H(t)Y_H(t) = \eta_L(t) \cdot N_L(t) \cdot X_L(t) + \eta_H(t) \cdot N_H(t) \cdot X_H(t) + C(t) \tag{4.64}$$

4.4.3 Dynamic General Equilibrium

In order to describe the dynamics of the economy in the presence of DTC, we start by rewriting the expressions for the invention costs, Z_L and Z_H. Recalling that all scientists (working for either the L and the H-technology) are paid the wage of a high-skilled worker, we replace wages in both the free-entry conditions (both equations in (4.50)) by $w_H(t)$ (defined by the second equation in (4.54)). This yields:

$$\begin{aligned} Z_L(t) &= \beta(1-\beta)^{\frac{2(1-\beta)}{\beta}} \cdot \eta_H^{\frac{\beta-1}{\beta}} \cdot \eta_L \cdot N_L(t)^{1-\phi} \cdot P_L(t)^{\frac{1}{\beta}}; \\ Z_H(t) &= \beta(1-\beta)^{\frac{2(1-\beta)}{\beta}} \cdot \eta_H^{\frac{2\beta-1}{\beta}} \cdot N_H(t)^{1-\phi} \cdot P_H(t)^{\frac{1}{\beta}} \end{aligned} \tag{4.65}$$

Thus, the growth rates of Z_L and Z_H are:

$$g_{Z_L}(t) = (1-\phi) \cdot g_{N_L}(t) + \frac{1}{\beta} \cdot g_{P_L}(t); \quad g_{Z_H}(t) = (1-\phi) \cdot g_{N_H}(t) + \frac{1}{\beta} \cdot g_{P_H}(t) \tag{4.66}$$

Using (4.51), (4.55), (4.65) and (4.66) in (4.52), and plugging the resulting expression for the rate of return in (4.59), we obtain, after some algebra, the following alternative equations for the growth rates of C_L and C_H:

$$\begin{aligned} g_{C_L}(t) = (1-\beta) \cdot \left(\frac{\eta_H}{\eta_L}\right)^{\frac{1-\beta}{\beta}} \cdot \left(\frac{1-s}{s \cdot u(t) \cdot (1-b(t))}\right) \cdot g_{N_L}(t) + \\ + (1-\phi) \cdot g_{N_L}(t) + \frac{1}{\beta} \cdot g_{P_L}(t) - \rho + \psi - \mu - \psi \cdot (\mu + \rho) \cdot \Omega_L(t) \end{aligned} \tag{4.67}$$

$$g_{C_H}(t) = (1-\beta)\cdot\left(\frac{1-u(t)}{u(t)\cdot b(t)}\right)\cdot g_{N_H}(t) + (1-\phi)\cdot g_{N_H}(t) + \frac{1}{\beta}\cdot g_{P_H}(t) - \rho + \psi - \mu - \psi\cdot(\mu+\rho)\cdot\Omega_H(t) \quad (4.68)$$

The growth rates of technological knowledge, g_{N_L} and g_{N_H}, are obtained from (4.51) and are equal to $g_{N_L}(t) = (1/\eta_L)\cdot H_{N_L}(t)\cdot N_L(t)^{\phi-1}$ and $g_{N_H}(t) = (1/\eta_H)\cdot H_{N_H}(t)\cdot N_H(t)^{\phi-1}$. Recalling that $H_{N_H}(t) = u(t)\cdot b(t)\cdot s\cdot\mathcal{L}(t)$ and $H_{N_L}(t) = u(t)\cdot(1-b(t))\cdot s\cdot\mathcal{L}(t)$, we get:

$$g_{g_{N_L}}(t) = g_u(t) - g_b(t)\cdot\frac{b(t)}{1-b(t)} - (1-\phi)\cdot g_{N_L}(t) + \psi - \mu$$
$$g_{N_H}(t) = g_u(t) + g_b(t) - (1-\phi)\cdot g_{N_H}(t) + \psi - \mu \quad (4.69)$$

As for the differential equations for $\Omega_L(t)$ and $\Omega_H(t)$, we use (4.66) and (4.68) and obtain:

$$g_{\Omega_L}(t) = (\beta-1)\cdot\left(\frac{\eta_H}{\eta_L}\right)^{\frac{1-\beta}{\beta}}\cdot\left(\frac{1-s}{s\cdot u(t)\cdot(1-b(t))}\right)\cdot g_{N_L}(t) + g_{N_L}(t) + \psi\cdot(\mu+\rho)\cdot\Omega_L(t) + \rho - \psi + \mu$$
$$g_{\Omega_H}(t) = (\beta-1)\cdot\left(\frac{1-u(t)}{u(t)\cdot b(t)}\right)\cdot g_{N_H}(t) + g_{N_H}(t) + \psi\cdot(\mu+\rho)\cdot\Omega_H(t) + \rho - \psi + \mu \quad (4.70)$$

Moving now to the characterization of steady-state of the economy, we start by noting that in a BGP the stock of technological knowledge has to grow at the same rate in both technologies, that is $g_N \equiv g_{N_L} = g_{N_H}$. This rate is constant and given by (4.38). This implies that: (i) final-good prices also grow at the same rate in both technologies, $g_P \equiv g_{P_L} = g_{P_H}$; (ii) wages, profits and invention costs grow at the rate $\frac{1}{\beta}g_P + n$; (iii) aggregate nominal output, consumption and capital grow at the rate

$\frac{1}{\beta}g_P + g_N + n$; (iv) aggregate real output grows at the rate $\frac{1-\beta}{\beta}g_P + g_N + n$; (v) per capita real output grows at the rate $\frac{1-\beta}{\beta}g_P + g_N$; (vi) the shares defining allocation of high-skilled and R&D labour, the interest rate, the capital-to-consumption ratios, the technological knowledge bias, the relative final-good prices and the skill premium are constant, that is $g_u = g_b = g_r = g_{\Omega_L} = g_{\Omega_H} = g_{N_H/N_L} = g_{P_H/P_L} = g_{\bar{w}} = 0$.

We are mainly interested in examining how demographic variations affect not only the skill premium, $\bar{w}$, in the steady state, but also the allocations of high-skilled labour and R&D workers (defined by u and b) in a DTC framework.[8] With this, we are able to enrich the analysis by Neves et al. (2018), where parameters u and b were taken as exogenous.

Starting with the effects on u and b, we recall that in a BGP $g_{\Omega_H} = 0$. Thus, from the second expression in (4.70), Ω_H^* is given by:

$$\Omega_H^* = \frac{1}{\psi(\mu+\rho)} \cdot \left\{ \left(\frac{\psi-\mu}{1-\phi} \right) \cdot \left[(1-\beta) \cdot \left(\frac{1-u^*}{u^* \cdot b^*} \right) - \phi \right] - \rho \right\} \tag{4.71}$$

Using the same procedure as in the derivation of equation (4.39), Ω_H^* can also be expressed as:

$$\Omega_H^* = \left(\frac{\psi-\mu}{1-\phi} \right)^{-1} \cdot \frac{u^* \cdot b^*}{(1-u^* \cdot b^*) + (1-u^*) \cdot (1-\beta)} \tag{4.72}$$

Joining (4.71) and (4.72) yields:

$$\left(\frac{(1-u^* \cdot b^*) + (1-u^*) \cdot (1-\beta)}{u^* \cdot b^*} \right) \cdot \left(\frac{\psi-\mu}{1-\phi} \right) \cdot \frac{1}{\psi(\mu+\rho)} \cdot \left\{ \left(\frac{\psi-\mu}{1-\phi} \right) \cdot \left[(1-\beta) \cdot \left(\frac{1-u^*}{u^* \cdot b^*} \right) - \phi \right] - \rho \right\} = 1 \tag{4.73}$$

Equation (4.73) defines implicitly u^* as a function of b^* and of the parameters of the model. When $b = 1$, all scientists are working for

the $H-$technology and there is no DTC. In this situation, Eq. (4.73) becomes similar to (4.40) (for the particular case $\alpha = 0$). However, when there is DTC, one part of the scientists works for H technology and the other for the L-technology, and thus $b < 1$. In this case, u and b have to be jointly determined. To do so, we must derive the expressions for Ω_L^* as well:[9]

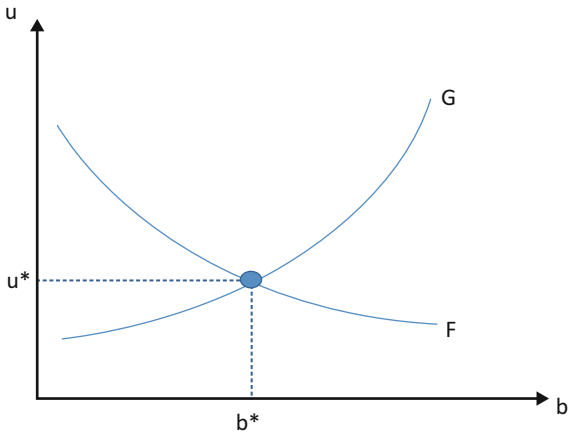
$$\Omega_L^* = \frac{1}{\psi(\mu+\rho)} \cdot \left\{ \left(\frac{\psi-\mu}{1-\phi} \right) \cdot \left[(1-\beta) \cdot \left(\frac{\eta_H}{\eta_L} \right)^{\frac{1-\beta}{\beta}} \cdot \left(\frac{1-s}{s \cdot u^* \cdot (1-b^*)} \right) - \phi \right] - \rho \right\} \tag{4.74}$$

$$\Omega_L^* = \left(2 - \beta - \frac{P_H Y_H}{P_L Y_L} \cdot \frac{u^* \cdot (1-b^*)}{1-u^*} \right)^{-1} \cdot \eta_H \cdot \left(\frac{\eta_L}{\eta_H} \right)^{1/\beta} \cdot \left(\frac{\psi-\mu}{1-\phi} \right)^{-1} \cdot \left(\frac{s \cdot u^* \cdot (1-b^*)}{1-s} \right) \tag{4.75}$$

Joining (4.74) and (4.75) yields:

$$\left(2 - \beta - \frac{P_H Y_H}{P_L Y_L} \cdot \frac{u^* \cdot (1-b^*)}{1-u^*} \right) \cdot \left(\frac{1-s}{s \cdot u^* \cdot (1-b^*)} \right) \cdot \eta_H^{-1} \cdot \left(\frac{\eta_H}{\eta_L} \right)^{1/\beta} \cdot \left(\frac{\psi-\mu}{1-\phi} \right) \cdot \frac{1}{\psi(\mu+\rho)} \cdot \left\{ \left(\frac{\psi-\mu}{1-\phi} \right) \cdot \left[(1-\beta) \cdot \left(\frac{\eta_H}{\eta_L} \right)^{\frac{1-\beta}{\beta}} \cdot \left(\frac{1-s}{s \cdot u^* \cdot (1-b^*)} \right) - \phi \right] - \rho \right\} = 1 \tag{4.76}$$

Equations (4.73) and (4.76) jointly determine the steady-state values of the share of R&D workers in total high-skilled population, u^*, and the share of scientists working for the H-technology, b^*. Applying the Implicit Function Theorem to (4.73), it is straightforward to see that



F represents the combinations of μ and b that satisfy eq. (4.73);
G represents the combinations of μ and b that satisfy eq. (4.76).

Fig. 4.2 Steady-state values of u and b

$\frac{du^*}{db^*} < 0$, which means that the locus that represents the combinations of u^* and b^* satisfying (4.73) is negatively sloped. On the other hand, and recalling that $\left(2 - \beta - \frac{P_H Y_H}{P_L Y_L} \cdot \frac{u \cdot (1-b)}{1-u}\right) > 0$, it is also straightforward to see that $\frac{du^*}{db^*} > 0$ for Eq. (4.76), implying that the locus that represents the combinations of u and b satisfying (4.76) is positively sloped. The intersection of both locuses defines u^* and b^*—see Fig. 4.2.

The effect of demographic changes on u^* and b^* can be summarized in the following Proposition:

Proposition 4.2 *A higher birth rate, ψ, or a lower mortality rate, μ, implies a higher share of R&D workers in the high-skilled labour population, u^*, and has an indeterminate effect on the share of R&D labour working for the H-technology, b^*.*

Proof Obtained by checking the partial derivatives of Eqs. (4.73) and (4.76) with respect to ψ, μ, u^* and b^*, using the Implicit Function Theorem. □

An increase in the birth rate (or a decrease in the mortality rate) raises the value of the left side of Eq. (4.73). Thus, for a given u^*, (4.73) is satisfied only if b^* assumes a higher value (or alternatively, for a given b^*, if u^* assumes a higher value).[10] This translates into a shift of the F locus to the right/upward.

An increase in the birth rate (or a decrease in the mortality rate) will also raise the value of the left side of Eq. (4.76). In this case, for a given u^*, (4.76) is satisfied only if b^* assumes a lower value (or alternatively, for a given b^*, if u^* assumes a higher value).[11] This translates into a shift of the G locus to the left / upward.

As Fig. 4.3 shows, the final effect on the steady-state share of R&D workers, u^*, will be unequivocally positive, as in the baseline model without DTC. The impact on the steady-state share of R&D labour working for the H-technology, b^*, can be positive or negative, depending on the relative strength of the shift of each curve. In the next section, a quantitative exercise will be performed where we analyse how b^* reacts to demographic changes for specific values of the model's parameters.

Moving now to the analysis of the impact of demographic changes on the steady-state skill premium, recall that the skill premium depends on u and on the technological-knowledge bias, N_H/N_L (see Eq. (4.57)). Therefore, we must first determine the expression for the steady-state technological knowledge bias.

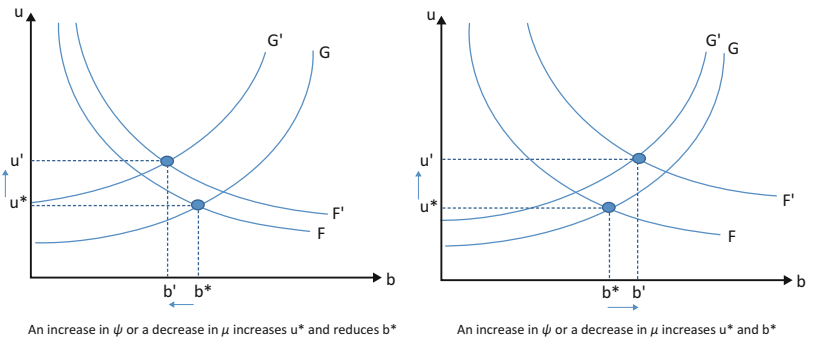


Fig. 4.3 Impact of an increase in the birth rate or a decrease in mortality rate on u^* and b^*

Substituting (4.55) in (4.52), and bearing in mind that in a balanced growth path $\frac{\dot{Z}_H}{Z_H} = \frac{\dot{Z}_L}{Z_L} = \frac{1}{\beta} g_P + n$, the steady expressions for Z are given by:

$$Z_L(t) = \frac{\beta(1-\beta)^{\frac{2}{\beta}} \cdot \eta_L^{\frac{\beta-1}{\beta}} \cdot L(t) \cdot P_L(t)^{\frac{1}{\beta}}}{r - n - \frac{1}{\beta} g_P};$$

$$Z_H(t) = \frac{\beta(1-\beta)^{\frac{2}{\beta}} \cdot \eta_H^{\frac{\beta-1}{\beta}} \cdot H_Y(t) \cdot P_H(t)^{\frac{1}{\beta}}}{r - n - \frac{1}{\beta} g_P} \tag{4.77}$$

In addition, from both equations in (4.50), we have that $Z_L(t) \cdot N_L(t)^{\phi} \cdot \eta_L^{-1} = Z_H(t) \cdot N_H(t)^{\phi} \cdot \eta_H^{-1}$. Replacing $Z_L(t)$ and $Z_H(t)$ by (4.77) and using (4.56), the expression for the technological-knowledge bias in a BGP is given by:

$$\frac{N_H(t)}{N_L(t)} = \left(\frac{1-\gamma}{\gamma}\right)^{\frac{\varepsilon}{1-\phi\sigma}} \left(\frac{\eta_H}{\eta_L}\right)^{\frac{1-\sigma-\beta}{\beta(1-\phi\sigma)}} \left(\frac{s}{1-s} \cdot (1-u^*)\right)^{\frac{\sigma-1}{1-\phi\sigma}} \tag{4.78}$$

where the condition $1 - \phi\sigma > 0$ must verify in order to guarantee the stability of the system.

Using this expression in (4.57), the steady-state skill premium is equal to:

$$\bar{w} = \left(\frac{1-\gamma}{\gamma}\right)^{\frac{\beta\varepsilon(1-\phi\sigma)+\varepsilon(\sigma-1)}{\sigma(1-\phi\sigma)}} \left(\frac{\eta_H}{\eta_L}\right)^{\frac{\beta(1-\phi\sigma)(\sigma-\varepsilon)+(1-\beta-\sigma)(\sigma-1)}{\beta\sigma(1-\phi\sigma)}} \left(\frac{s}{1-s} \cdot (1-u^*)\right)^{\frac{\sigma-2+\phi}{1-\phi\sigma}} \tag{4.79}$$

Proposition 4.3 *A higher birth rate, ψ, or a lower mortality rate, μ, implies a lower skill premium if $\sigma > 2-\phi$, and a higher skill premium if $\sigma < 2-\phi$.*

Proof Obtained by joining the results of Proposition 4.2 with the partial derivatives of Eq. (4.79) with respect to u^* □

Equation (4.79) shows that, as in the baseline model without DTC, variations in population growth are transmitted to the steady-state skill premium via u^*. However, differently from the baseline model, u^* can influence the skill premium positively or negatively, depending on the degree of substitutability between high-skilled and low-skilled labour in the production of final goods, σ.

This results from the fact that, in the presence of DTC, variations in u influence the skill premium not only through the usual supply effect, but also through the market size and the price channels, which operate via the technological knowledge bias. In fact, an increase in u^*—caused by a higher birth rate or a lower mortality rate—turns high-skilled labour relatively less abundant in the production of final goods. Through the supply effect, this will contribute to increase its relative reward and, thus, the skill premium; through the market-size channel, it will contribute to reduce the technological knowledge bias, since the L-technology, which now uses a relatively more abundant factor, becomes more profitable; and through the price channel, a higher u^* will contribute to increase the technological knowledge bias, as it raises the relative final-good prices in the H-technology, making it more attractive to invest in it. Thus, through the market-size and the price channels, demographic changes impact the technological knowledge bias and the skill premium in different directions.

The direction of the overall effect on the skill premium will depend on the degree of substitutability between high-skilled and low-skilled labour in the production of final goods. In particular, as in Acemoglu (2002), when the degree of substitutability is sufficiently high such that $\sigma > 2 - \phi$, the market-size effect tends to be stronger, and a higher value of u^* will reduce the steady-state skill premium. In this case, a higher education effort, captured by a higher s, will raise the skill premium. On the contrary, when both types of labour are gross complements or their degree of substitutability is weak such that $\sigma < 2 - \phi$, a higher u^* will raise the steady-state skill premium. In this case, a higher education effort will decrease the skill premium.

Thus, while in the model without DTC the lower birth rate concomitant with the ageing process leads to a lower skill premium, in the model with DTC this only happens when both types of labour are

gross complements or their degree of substitutability is weak such that $\sigma < 2 - \phi$. On the contrary, when the degree of substitutability is sufficiently high such that $\sigma > 2 - \phi$ and the market effect is dominant, the ageing process (due to a lower birth rate) may be coincident with a higher skill premium.

These results relate to those presented in Neves et al. (2018) in the sense that here the elasticity of substitution between high and low skilled labour also plays a key role in determining the direction of the impact of demographic changes on the skill premium. However, they differ from Neves et al. (2018) in two aspects. First, in Neves et al. (2018) the direction of the impact of demographic changes on the skill premium was determined not only by the degree of the elasticity of substitution, but also by the difference between the growth rates of high and low-skilled population and by the effect of population growth on the real interest rate, thus rendering the relationship between the skill premium and demographic variables more complex. Second, while in Neves et al. (2018) changes in birth and death rates influence the skill premium because of the existence of heterogeneous population growth, here such changes influence the skill premium because they modify the share of R&D workers in the high-skilled population. Thus, our model presents a mechanism through which the skill premium is influenced by demographic changes in a context in which high and low-skilled population grow at the same rate.

4.5 Quantitative Application

In this section, we calibrate the models in order to assess their steady-state properties and compare them to data for the US for the present, which we roughly consider to be the period between 1980 and 2015 (Sect. 4.5.1). This is important because previous research has focused on the skill premium behaviour for this period, highlighting the level of that variable vis-à-vis previous decades.

Next, we introduce historical data in the crucial parameters (share of human capital, s, death rate, μ and birth rate, ψ), concerning the period 1950–1980, to calibrate and evaluate the behaviour of two of the endogenous variables, the share of human capital allocated to R&D

(u) and the skill premium ($\bar{w}$), after shocks on those parameters in two distinct moments of time (Sect. 4.5.2). By considering the two models presented in the previous sections, the calibration exercise allows us to identify the quantitative importance of the demographic channel vis-à-vis the DTC channel in both the contemporaneous and the future evolution of u and $\bar{w}$.

4.5.1 Calibration and Steady-State Values

Our aim in this subsection is to show that the models proposed in this chapter, which consider the demographic transition within endogenous-growth frameworks that account for wage inequality, are able to replicate stylized facts of the US economy.

Most parameters values are chosen according to the existing literature. The share of physical capital is set to 0.35, consistent with a widely accepted stylized fact (see e.g. Grossman et al., 2017). The share of low-skilled and high-skilled labour in final-good production in the baseline model, α and β, are both 0.325, which is consistent with Mauro and Carmeci (2003). Accordingly, the share of low-skilled and of high-skilled labour in the respective production function in the DTC model is 0.65. The intertemporal discount rate (ρ) is set to 0.02, as in Barro and Sala-i-Martin (2004) and many others. In accordance with Neves and Sequeira (2018), the R&D spillover ϕ is set to 0.852 for the base model. For the DTC model, ϕ is set to a somewhat smaller value, 0.7. This smaller value is necessary to satisfy the stability condition $1 - \phi\sigma > 0$ in the latter and is also within the range of the values revised in Neves and Sequeira (2018). The percentage of human capital in total population (s) is the average between 1980 and 2010 of the share of population with tertiary education in the US, taken from Barro and Lee (2010). Death and birth rates (μ and ψ) are taken from United Nations' (2017) data for the US. We use the average between 1980 and 2015 to correspond to the present period.

The quantification of the DTC model implies the calibration of a set of additional parameters. We consider an elasticity of substitution, ε, equal to 1.5, in line with established consensus (e.g. Goldin and Katz, 2007;

Katz and Murphy, 1992). For example, Card and Limieux (2001) support this choice as, when pooling a combined sample of men and women, the elasticity of substitution between college and high school graduates is in the range of 1.1–1.6. The share of the high-skilled intensive good in final production (γ) is set to 0.3. This value is in the range of values for the white collar labor share in the US presented in Voigtlä nder (2014). Finally, we set u and b in the DTC model to match the empirical ratio between high-tech and low-tech production ($\frac{P_H Y_H}{P_L Y_L}$)—see Eq. (4.75)—which is set to 0.4 according to Wolf and Terrel (2016).[12] Table 4.1 summarizes the calibrated parameters and the main steady-state values. The value for η_H (as a level parameter) is set to allow for a good replication of the skill premium in the present, and is maintained throughout the different exercises. The value for η_L is normalized to 1. Note that, in this calibration, $\sigma = 1 + (\varepsilon - 1)\beta > 2 - \phi$. In this case, not only does the increased historical effort explain the rising skill premium that occurred in the US in the last decades of the XXth century—which is a feature that previous literature has been eager to replicate—but also a lower birth rate and ageing imply a higher skill premium, meaning that ageing (occurring due to demographic transition and lower birth rates) may be a process consistent with the increasing skill premium. Our calibration values are grounded in the literature and data and encompass a sufficiently strong market effect such that the increase in skills which occurred in the XXth century is consistent with the rising trend in the skill premium observed in the last decades of that century in the US.

The first important thing to note concerning the steady state results (in Table 4.1) is that both models in this chapter are able to replicate reasonably well the level of the skill premium when compared to the data reported in Goldin and Katz (2007: Figure 1, Table A1.8; the average value for 1980, 1990 and 2000 is 1.682). As for the empirical R&D share, there are several issues in its calculation, especially relating to the stock of

Table 4.1 Parameter and steady-state values for the present period

Model	α	β	ρ	ϕ	γ	ε	s	μ	ψ	$\frac{P_H Y_H}{P_L Y_L}$	u	$\bar{w}$
Baseline	0.325	0.325	0.02	0.852	–	–	0.46	0.0086	0.0143	–	0.33	1.74
DTC	–	0.65	0.02	0.7	0.3	1.5	0.46	0.0086	0.0143	0.4	0.78	1.67

human capital to be considered as a reference, i.e., as the denominator in the share. In 2014, the US had nearly 900,000 scientists engaged in R&D in private businesses according to the OECD statistics,[13] which should account for the human capital allocated to R&D, H_N. If we consider the total human capital, H, as the total population with tertiary education (according to Barro and Lee, 2010—data capitalized to 2014), then the corresponding denominator H would be nearly 128 million people, and the share $u = \frac{H_N}{H}$ should be nearly 0.7%, quite lower than the values obtained by our models. However, if we consider that the human capital considered to be the potential supply of R&D, H, would be measured by the number of PhDs in Science and Engineering, then the denominator would be around 1,060,000 in 2014 (according to the National Science Foundation, 2017), yielding a share $u = \frac{H_N}{H}$ much closer to what is obtained by our DTC model.

Thus, the quantitative exercise in this subsection allows us to conclude that both models proposed in this chapter can account for the current empirical values of the skill premium. However, they differ somewhat in the steady-state value of the R&D allocation share, with the DTC model featuring a better fit to the empirical values.

4.5.2 Shocks

In this section, we perform a number of quantitative exercises to analyse the contribution of two broad types of shocks for the evolution of the skill premium, $\bar{w}$, and of the R&D effort, u: the education shock (measured by the shift in the human capital share, s) and the demographic shock (measured separately by the shift in the birth rate ψ—birth shock—and in the death rate μ—death shock).[14] We do so by considering that the economy has an initial ('past') steady state that corresponds to the period 1950–1980 in the data and that a set of shocks follow in two distinct moments of time ('present' and 'future') and move the economy to two new steady states. Thus we start by calibrating s, ψ and μ with historical values for 1950–1980 for the US. The values of the parameters used for the calibration of the initial period are presented in Table 4.2.

Table 4.2 Parameter values for the past and shocks for the present and future periods

Model	α	β	ρ	ϕ	γ	ε	$\frac{P_H Y_H}{P_L Y_L}$	μ	ψ	s
Baseline (past)	0.325	0.325	0.02	0.852	–	–	–	0.0095	0.020	0.2
DTC (past)	–	0.65	0.02	0.7	0.3	1.5	0.4	0.0095	0.020	0.2
Shock for the present	–	–	–	–	–	–	–	0.0086	0.0143	0.46
Shock for the future	–	–	–	–	–	–	–	0.0092	0.0121	0.62

As can be seen from the analysis of Table 4.2, there are differences with respect to Table 4.1 due to the consideration of the past period 1950–1980 to calibrate the (past) steady state in the model, and also the consideration of two more sets of values for the key parameters, to account for the present and the future shocks. For s, we consider Barro and Lee's (2010) data averaged between 1950 and 1980 (past value), averaged between 1980 and 2010 (to account for the present shock), and capitalized to 2020 assuming the initial value of 2010 and the same growth rate as the one occurred between 2005 and 2010 (to account for the future shock). For ψ and μ, we use birth and death rates (respectively) from the United Nations, assuming averages between 1950 and 1975 (past values), averages for the period 1980–2015 (to account for the present shock), and projections for 2015–2050 (shock for the future).

As a first exercise, we analyse the behaviour of $\bar{w}$ and u by considering joint shifts in the human capital share (education shock) and in the birth and the death rate (demographic shocks). The second exercise shuts down the birth shock, while the death and the education shocks remain. The third exercise shuts down both demographic shocks and thus only the education shock remains.

We present the results for both the baseline and the DTC models in Fig. 4.4.

Results in Fig. 4.4 highlight two main important features: the baseline model predicts an opposite direction for the skill premium when compared to the DTC model, following all the shocks (compare Fig. 4.4a with c). This is due to the expected fact that the DTC model includes the market effect which, for the considered high degree of factor substitutability, implies a rising skill premium following an education shock. Notwithstanding, demographic shocks imply the same qualitative path as

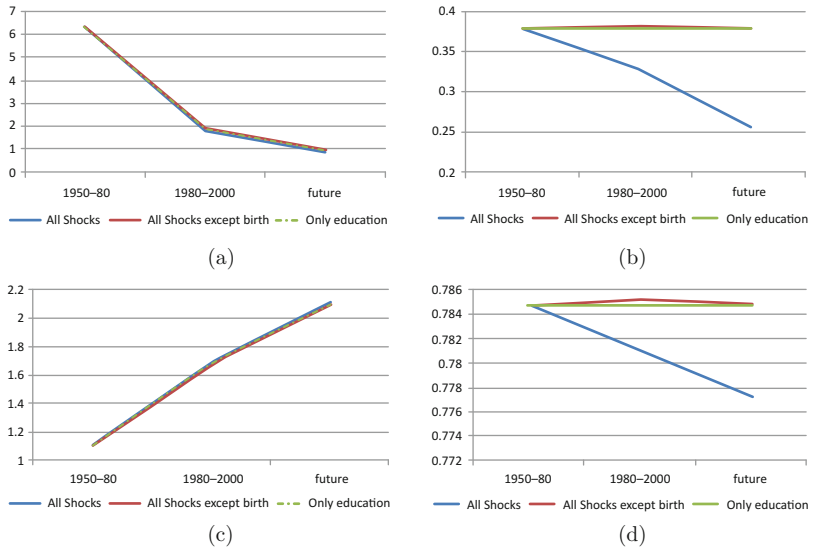


Fig. 4.4 Decomposition of Shocks: all shocks (blue line), all shocks except birth (red line), only education shock (green line). **(a)** Skill Premium—Baseline model. **(b)** R&D share—Baseline model. **(c)** Skill Premium—DTC model. **(d)** R&D share—DTC model

the education shock. In fact, in both models, both demographic shocks reinforce quantitatively the education shock. One should note however that, even in the DTC model, the birth shock (the strongest of the demographic shocks) accounts for about 10% of the evolution of the skill premium, while the death shock has a very small impact (see Fig. 4.4c and note that the green and the red lines almost overlap, just like in Fig. 4.4a). As far as the R&D effort is concerned, the analysis of Fig. 4.4b and d shows that the baseline model and the DTC model imply very similar paths following the education shock, on one hand, and the demographic shocks, on the other hand. The death shock has a very small influence on the R&D share trajectory (note, again, that the green and the red lines almost overlap). When considering all the shocks (which is the same as adding the birth shock and consequently ageing—to the red line—that already considers the education and the death shocks), there is evidence of declining R&D effort,[15] which is, however, quantitatively

more important in the baseline model than in the DTC model. While this decline accounts in the future for almost 40% of the initial value in the baseline model, in the DTC model it barely accounts for 1.5%.

Second, to further highlight the results from the demographic shocks, we isolated birth and death shocks and show the results in Fig. 4.5. This highlights that the birth shock helps the declining trend of the skill premium in the baseline model (see Fig. 4.5a) while helping the rising trend of the skill premium in the DTC model (see Fig. 4.5c). Moreover, it helps the declining R&D share both in the baseline and in the DTC model. The quantitative effects are quite a bit larger within the baseline framework (accounting for 20% of the shift in the skill premium and 25% of the shift in the R&D share in the baseline model and nearly 7% and 1% respectively within the DTC model), which is consistent with the analysis of the results in Fig. 4.4. Moreover, the death shock has a negligible influence on the skill premium implied by the baseline model. It implies a hump-shaped evolution of the R&D share (see Fig. 4.5b),

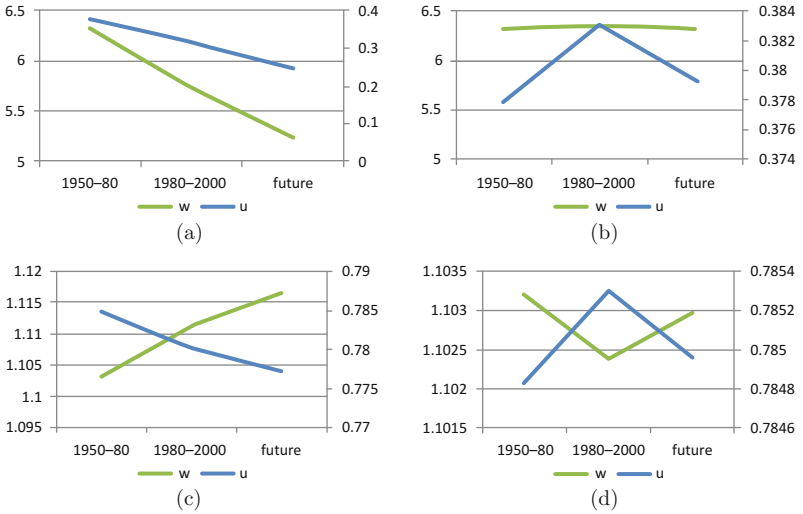


Fig. 4.5 Skill Premium ($\bar{w}$)—left scale, green line, and R&D share (u)—right scale, blue line, in the baseline and DTC model—birth versus death shocks. (**a**) Birth Shock—Baseline model. (**b**) Death Shock—Baseline model. (**c**) Birth Shock—DTC model. (**d**) Death Shock—DTC model

due to the fact that the demographic projections imply a slight rise of the mortality rate for the future (2015–2050). The implied variations in the R&D share following a death shock are less than 20% of the total variation. If the economy followed a DTC model, the death shock would imply a U-shaped trajectory for the skill premium, which is at odds with the trajectory predicted by the baseline model. However, it predicts a hump-shaped trajectory for the R&D share, which is consistent with the baseline model (see Fig. 4.5d). Quantitatively the effects of the death shock in the DTC model are rather small, nearly 0.8% for the skill premium and even less for the R&D share.

The quantitative exercises that illustrate the models with demographic changes presented in this chapter highlight that the important difference introduced by the DTC framework regards the evolution of the skill premium. As far as the skill premium is concerned, the demographic shocks follow and reinforce the education shock. The strongest of the demographic shocks—the birth shock, also associated with ageing—accounts for no more than 7% of the total variation of the skill premium in the DTC model. However, both theoretical approaches predict the productivity slowdown associated with R&D. Again the birth shock is quantitatively more important than the death shock, implying a small variation of nearly 1% of the R&D share.

4.6 Concluding Remarks

We introduce demographic structure by considering age specific heterogeneity of households into models of R&D-based technological change. Indeed, since we are particularly interested in the interplay between technology, economic growth and wage inequality across different types of human capital, a natural model class to examine our research question is the growth framework in which the R&D activities use human capital as an input and R&D effort is market driven. We start by considering a framework that incorporates heterogeneous human capital that encompasses the R&D specification in line with Jones (1995) as a special case. Later, we extend the framework to consider a general equilibrium DTC model, following Acemoglu (2002).

We discover that in the baseline model, a lower birth rate or a higher mortality rate implies a lower share of R&D workers in high-skilled labour population and a lower skill premium. However, the alternative DTC model may imply a rising skill premium following declines in the birth and the death rates for different elasticities of substitution. These results indicate that the demographic changes of the second half of the XXth century may have accounted for at least part of the stylized facts related to the (declining) productivity and the (increasing) skill premium. In particular, the declining birth rate (and the consequent process of ageing) observed in this historical period contributed to the decline in R&D productivity, and for a sufficiently high degree of input substitution, to the increasing skill premium. Additionally, also the decrease in the death rate may also have contributed to the rising skill premium.

Quantitatively, it is shown that the decline in the birth rate and, consequently, ageing may help explain the increase in the skill premium and the decline in the R&D intensity observed at the end of the XXth century, with non-negligible effects that can account for around 7% (DTC model) of the total skill premium variation.

As an extension on the topic, it would be insightful to exploit other channels through which one would be able to analyse the interaction between demographic change and wage inequality, namely the households' human capital choice (based on, e.g., Grossman and Helpman, 1991, Ch. 3 and 5.2; Sequeira, 2007), structural unemployment and labor market institutions (following, e.g., Afonso et al., 2018; Neto et al., 2019), and healthcare activities (based on, e.g., Kuhn and Prettner, 2016; Jones, 2016). We believe these are promising avenues for future work.

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Appendix

In this appendix we derive the aggregate Euler equation for consumption and the aggregate law of motion for assets (Eqs. (4.21) and (4.23)). We consider that the population grows at rate $n = \psi - \mu > 0$ and we normalize the initial population size to $\mathcal{L}(0)$ such that the size of a generation born at $t_0 < t$ at a certain point in time t is:

$$N(t_0, t) = \psi \cdot \mathcal{L}(t_0) \cdot e^{\mu(t_0 - t)} = \psi \cdot \mathcal{L}(0) \cdot e^{nt_0} \cdot e^{\mu(t_0 - t)} = \psi \cdot \mathcal{L}(0) \cdot e^{\psi t_0} \cdot e^{-\mu t}. \tag{4.80}$$

Integrating over all generations yields the population size as:

$$\mathcal{L}(t) = \int_{-\infty}^{t} \psi \cdot \mathcal{L}(0) \cdot e^{\psi t_0} \cdot e^{-\mu t} \cdot dt_0 = \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \int_{-\infty}^{t} e^{\psi t_0} dt_0. \tag{4.81}$$

Hence, we can define the aggregate consumption as:

$$C(t) = \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \int_{-\infty}^{t} c(t_0, t) \cdot e^{\psi t_0} dt_0. \tag{4.82}$$

Differentiating this equation with respect to time yields:

$$\dot{C}(t) = \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \int_{-\infty}^{t} \dot{c}(t_0, t) \cdot e^{\psi t_0} dt_0 - \psi C(t) + \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} c(t, t) e^{\psi t}. \tag{4.83}$$

In order to find $g_C(t)$, we can reformulate the household's optimization problem in Sect. 4.2.2.1, by considering:

$$\underset{c(t_0, \tau)}{\text{Max}} U = \int_{t}^{\infty} \log c(t_0, \tau) \cdot e^{(\mu + \rho)(t - \tau)} d\tau, \tag{4.84}$$

subject to its lifetime budget restriction, stating that the present value of lifetime consumption expenditures has to be equal to the present value of

lifetime wage income plus initial assets:

$$\int_t^{\infty} c(t_0,\tau)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}d\tau = k(t_0,t) + \int_t^{\infty}(1-s)\cdot w_L(\tau)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}d\tau + \int_t^{\infty} s\cdot w_H(\tau)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}d\tau \quad (4.85)$$

From the first order condition to this problem results:

$$c(t_0,\tau)^{-1}\cdot e^{(\mu+\rho)(t-\tau)} = \lambda(t)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds} \quad (4.86)$$

Hence, in time $\tau = t$, we have $c(t_0,\tau) = \frac{1}{\lambda(t)}$ and we can write $c(t_0,\tau)^{-1}\cdot e^{(\mu+\rho)(t-\tau)} = c(t_0,\tau)^{-1}\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}$, which, in turn, implies that:

$$\int_t^{\infty} c(t_0,\tau)\cdot e^{(\mu+\rho)(t-\tau)}d\tau = \int_t^{\infty} c(t_0,\tau)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}d\tau. \quad (4.87)$$

So, from (4.85):

$$c(t_0,t) = (\mu+\rho)\cdot\left[k(t_0,t) + \int_t^{\infty}(1-s)\cdot w_L(\tau)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}d\tau + \int_t^{\infty} s\cdot w_H(\tau)\cdot e^{-\int_t^{\tau}(r(s)+\mu)ds}d\tau\right], \quad (4.88)$$

where the first term in brackets, $k(t_0,t)$, is the financial wealth, which depends on the date of birth, and the second and third terms represent the wealth resulting from work, which is independent of the date of birth because the productivity is age independent. Thus, optimal consumption in the planning period is proportional to total wealth with a marginal propensity to consume of $(\mu+\rho)$.

By using (4.88), we can write the aggregate consumption in (4.82) as:

$$C(t) = (\mu + \rho)\,\psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \int_{-\infty}^{t} k(t_0, t) \cdot e^{\psi t_0} dt_0 + (\mu + \rho) \cdot \mathcal{L}(0) \cdot e^{(\psi - \mu)t} \cdot \left[\int_t^{\infty} (1 - s) \cdot w_L(t) \cdot e^{-\int_t^{\tau} (r(s) + \mu) ds} dt + \int_t^{\infty} s \cdot w_H(t) \cdot e^{-\int_t^{\tau} (r(s) + \mu) ds} dt \right]. \tag{4.89}$$

Newborn households do not have financial wealth because there are no inheritances; thus, $c(t,t) = (\mu + \rho) \cdot \left[\int_t^{\infty} (1 - s) \cdot w_L(t) \cdot e^{-\int_t^{\tau} (r(s) + \mu) ds} dt + \int_t^{\infty} s \cdot w_H(t) \cdot e^{-\int_t^{\tau} (r(s) + \mu) ds} dt \right]$ and $C(t,t) = \mathcal{L}(0) \cdot c(t,t) \cdot e^{(\psi - \mu)t}$ holds for, respectively, each newborn household and each newborn generation. Bearing in mind these last expressions for $c(t,t)$ and $C(t,t)$ and putting (4.83), (4.89) and (4.19), together yields Eqs. (4.21) and (4.22) in the text.

Now, in order to get an expression for the dynamic behaviour of aggregate assets, K, we need to apply the following to integrate over all generations alive at time t. Differentiating (4.22) with respect to time yields:

$$\dot{K}(t) = \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \cdot \int_{-\infty}^{t} \dot{k}\,(t_0, t) \cdot e^{\psi t_0} dt_0 - \psi \cdot K(t) + \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \cdot k\,(t, t) \cdot e^{\psi t}. \tag{4.90}$$

Since $k\,(t,t) = 0$ and bearing in mind (4.18), it results that:

$$\dot{K}\,(t) = -\mu \cdot K(t) + (r(t) + \mu) \cdot \psi \cdot \mathcal{L}(0) \cdot e^{-\mu t} \cdot \int_{-\infty}^{t} k(t_0, t) \cdot e^{\psi t_0} dt_0$$

$$-\mu \cdot \mathcal{L}(0) \cdot e^{-\mu t} \int_{-\infty}^{t} c(t_0, t) \cdot e^{\psi t_0} dt_0 + \mathcal{L}(0) \cdot e^{-\mu t} \cdot e^{\psi t} \cdot [(1 - s) \cdot w_L(t) + s \cdot w_H(t)]_{-\infty}^{t},$$

which implies that the aggregate law of motion for assets (equivalent to the aggregate flow budget constraint for households) is given by Eq. (4.23) in the text.

Notes

1. Using a model with vertical innovations would not change the results.
2. We consider X_j as non-durable goods rather than durable goods because we focus on the effects of technological progress rather than on the effects of capital accumulation.
3. Profit maximization and the assumption of perfect competition imply that factors are paid their marginal products. Note that, by simplification, usually from now on, the time argument is dropped.
4. Notice that Ω is constant in the long-run equilibrium as a result of $g_C = g_K$ along the BGP.
5. Bearing in mind Eqs. (4.28) and (4.37), we also find that $g_Z^* = (1 - \phi)g_N^* = n = \psi - \mu$.
6. As in the baseline model, $w_H(t) \equiv w_{H_Y}(t) = w_{H_N}(t)$.
7. Given that final-good prices are no longer normalized to 1, real output, Y_L and Y_H, differs from nominal output, $P_H \cdot Y_H$ and $P_L \cdot Y_L$.
8. Note that the effect on the growth rate of output per capita is the same as in the baseline model.
9. We use the same procedures as in the derivation of Eqs. (4.71) and (4.72).
10. Note that both u^* and b^* influence the left side of Eq. (4.73) negatively.
11. Note that while u^* influences the left side of Eq. (4.76) negatively, b^* influences it positively.
12. Gil et al. (2016) presented evidence of a roughly similar ratio for the present days in the European countries.
13. Available at https://stats.oecd.org/Index.aspx?DataSetCode=PERS_OCCUP.
14. As usual in the literature, we assume that these shocks are unantecipated and are perceived as permanent by the economic agents.
15. This is consistent with the productivity slowdown phenomenon of the last quartile of the XXth century. For recent evidence and explanation for this productivity slowdown, see, e.g. Sequeira et al. (2018).

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5

The Demographic Metabolism Model of Human Capital Formation

Erich Striessnig

5.1 Introduction

In studying the drivers of social change, researchers tend to overlook the most genuinely demographic form of transformation resulting from the simple fact that no two cohorts ever go through the same historic experiences. Events like the two world wars of the twentieth century, hyperinflation, mass unemployment or the Vietnam War and the movement for civil rights have left their marks on the generations that went through these collective experiences during their formative years. In consequence, the principles they followed later on in life, their normative views, attitudes, and behaviors were shaped in ways that were very different from their parents' and grandparents' generations. As one cohort replaces another in the population pyramid (or in the workforce) and as one generation follows another, social transformation happens in parallel.

E. Striessnig (✉)
Wittgenstein Centre for Demography and Global Human Capital (IIASA, VID/ÖAW, WU), Vienna, Austria
e-mail: erich.striessnig@oeaw.ac.at

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This is in essence the idea behind the "Demographic Metabolism" (DM), first proposed by sociologist Norman Ryder (1965) and recently adopted and further elaborated by Wolfgang Lutz (2013). The DM describes how societies change as a consequence of cohort replacement, i.e. intergenerational changes in which young people with different characteristics gradually move up the age pyramid and change the composition of the society they live in with respect to critical dimensions. The speed at which social transformations take place depends on two parameters. One is the rate of "demographic turnover" (Goldstein, 2009), which can be very rapid in societies in the early stages of the demographic transition due to their high rates of fertility and mortality and very slow in societies that are characterized by high life expectancy and low rates of fertility. The other parameter that determines the speed of transformation will depend on how different subsequent cohorts are from each other with respect to those critical dimensions of social heterogeneity.

The reason why the DM is easily overlooked as an engine for social change is its tacitness: While societies can experience change in a number of ways, often very abruptly, e.g. in the form of sudden-impact natural disasters, man-made catastrophes like Chernobyl, or disruptive technological innovations (that can all be summarized as period effects impacting on all cohorts at the same time), the slow and steady change which societies undergo as a consequence of cohort replacement happens gradually and is therefore more difficult to detect. Nevertheless, it can be very impactful and often more lasting in its consequences than period effects. Once established, new norms and attitudes that follow from the specific early-life experiences of entire generations can be very difficult to change back.

One crucial consequence of this property is the DM's use for predicting future societal developments, from which derives its value in demographic forecasting. Lutz (2013) referred to the DM as "A Predictive Theory of Socioeconomic Change". If for any given social phenomenon of relevance we can identify robust cohort effects, the DM can be used to predict their future impact, as the people shaping tomorrow's societies are already alive today. By studying their individual characteristics (in particular place of residence, health status, level of education, cognitive abilities, religion or

identity), which tend to be "sticky" along cohort lines or change following predictable patterns, we can make projections about those societies in the future using demographic scenarios (Lutz and KC, 2011; Striessnig and Lutz, 2016; Lutz and Muttarak, 2017). Likewise, the DM can be used for demographic "backcasting". As will be shown in this chapter, if certain characteristics do not change over the life course, the fact that people carry those characteristics along with them over most of their adult lifespan allows us to make inferences on the past, when data may not yet have been collected or simply lacks sufficient quality for conducting empirical analysis. As we shall see, reconstructing the past through the DM can open formerly untapped research venues.

The DM can be useful in a host of applications, such as cultural change (Gullickson, 2001), attitudinal shifts in gender roles and tolerance of homosexuality (Brooks and Bolzendahl, 2004; Andersen and Fetner, 2008; Pampel, 2011), future religious composition (Lutz and Skirbekk, 2012), trust within a society (Arolas, 2016), or increasingly important questions of identity (Lutz et al., 2006; Striessnig and Lutz, 2016). The dimension of social heterogeneity that this chapter is focusing on, though, is human capital formation. As theorized by Becker (1962) and formalized by Ben-Porath (1967), due to the finite number of periods over which returns to past investments in human capital can be realized, most people invest in their human capital early in life. Therefore, disregarding for the moment sources of human capital that continue to change later in life (such as on-the-job training and commitments to healthy lifestyles), at least the formal level of schooling for most people fulfills the aforementioned criterion of being sticky over the life course and thus can be modeled using the DM approach.

Unlike unified growth theory and overlapping generations (OLG) models with endogenous fertility (Galor, 2011; Galor and Weil, 2000), as well as vintage models of human capital (Boucekkine et al., 2002; Cervellati and Sunde, 2005; De La Croix et al., 2008) that include realistic demography as well, the DM does not entail a model of human behavior that would explain how the decisions leading to social change come about. Rather it describes the demographic mechanism by which the replacement of mostly still less educated cohorts by cohorts that already benefited from the collective experience of educational expansion

early in their lives leads to drastic social repercussions which can in turn be modeled e.g. using empirical growth regressions.

First of all, though, we want to look more closely at the origins of the DM in the writings of twentieth century sociologists Karl Mannheim and Norman Ryder. We will then look at the methodological advancements in the field of demography that were decisive in promoting the DM into a predictive theory of socioeconomic change, before highlighting the importance of the DM in understanding past and future human capital formation. Finally, the chapter discusses the implications of DM theory, as well as population projections deriving from it with respect to human development.

5.2 The Origins of the Demographic Metabolism

Even when Karl Mannheim first started writing about the process of social change under the title of "The Problem of Generations" (Mannheim, 1928), the idea that societies can change through cohort replacement was not new. We find evidence for similar ways of thinking among the pre-Socratics, just as much as in Confucius and already those ancient thinkers were deeply concerned that with their own generation's disappearance, society will be doomed as the young people of their days could not be entrusted with the responsibilities of public office and maintaining the order that had been established by them and their predecessors. The thoughts that inspired Mannheim more directly, though, dated back only to the late nineteenth and early twentieth centuries when (art) historians tried to explain the succession of epochs by the succession of different generations carrying different tastes, styles, and ways of interacting with their social environment. The "problem" of course consisted in finding the right criteria for drawing the line between two subsequent generations. This is what Mannheim picks up in his essay.

Mannheim starts out distinguishing two opposing and in his view reductionist approaches to the problem of generations that were prevalent among his antecessors. The one that is closer to what is about to be

introduced in this chapter is the "positivist" approach that tries to tackle the problem quantitatively. Following this view, which according to Mannheim is reflective of Cartesian rationalism predominant since the French enlightenment, generations can be distinguished by identifying measurable characteristics embodied by their members. Once these characteristics have become sufficiently different between two subsequent groups of people, we can speak of a new generation. The remaining problem then boils down to the simple task of calculating the right number of time periods after which one generation replaces another. The antagonist of this view in Mannheim's depiction is the "romantic-historical" approach predominating in the German-speaking world. "Firmly eschewing the clear daylight of mathematics", the proponents of this view follow a more qualitative approach trying to identify different generations' "entelechy", i.e. their inner aim defining how they experience life and the world.

While clearly not convinced of either approach, Mannheim seems more amenable to the view attributed to the French school of thought and tries to find a synthesis with the second view attributed to his German compatriots by defining a generation through its "social location" (*soziale Lagerung*). Similar to how members of a specific social class share certain properties, members of a generation are thought of as sharing one social location, which they carry along as the generation evolves temporally. Just like the existence of social classes creates restrictions in people's movement spaces and development opportunities, their social location predetermines them to ways of thinking and world views specific to their generation. The supposed rigidity of social locations, as well as its homogeneity within each generation then invariably leads to social change.

This is where Mannheim has to be put in contrast to the view proposed in this chapter. First of all, his focus was geared entirely toward the past, trying to contribute to the debate around the driving forces of history, rather than toward the future. The DM as it is laid out here clearly aims at making quantifiable predictions about the future. Secondly, generations the way Mannheim sees them are internally homogenous and their inner contradictions can be safely ignored. In contrast, the DM as a predictive

theory of social change considers cohorts as being composed of people with clearly distinguishable "social locations", which in some cases can even change over time as people age, for example when people attain higher levels of education and this way change their social location. Thus, while firmly holding on to the idea of measurability of important characteristics, the DM fully accounts for the heterogeneity observed within contemporary societies and predicts social change in future societies in response to the changing proportions of people possessing these characteristics within individual cohorts.

Following Mannheim's contribution, which became translated to English only after the war, the demographer Norman Ryder picked up the topic in his article on "The Cohort as a Concept in the Study of Social Change" (Ryder, 1965). Ryder coined the term "Demographic Metabolism", but—like Mannheim before him—remained entirely conceptual and failed to evaluate his theory quantitatively. Moreover, just like Mannheim, Ryder only refers to the potential of the DM to study the past but does not see the potential for forecasting future social transformations along cohort lines. Ryder defines the DM as the "massive process of personnel replacement" (1965, 843) which is shaped by the demographic forces of fertility and mortality, as well as the lives that people live in between those endpoints. Provided that fertility offsets mortality, societies—in contrary to individuals—can live forever. But in the absence of individual mortality, these societies "would resemble a stagnant pond" (ibid.), as individuals according to Ryder lack flexibility to change over their lifetime and so would societies composed of those stagnant individuals if older cohorts were not constantly replaced by new ones.

The part of the legacy of Ryder that clearly goes beyond Mannheim is his definition of cohort, which has since become canonical among demographers. Ryder sees it as "the aggregate of individuals (within some population definition) who experienced the same event within the same interval" (p. 845). Conventionally, the event defining a cohort is seen to be birth. But Ryder's definition does not restrict us to this narrow view and we can define cohorts in any number of ways. In addition, unlike in Mannheim's view, Ryder's notion of cohort is not monolithic,

entailing the possibility of heterogeneity, which makes him an important predecessor of the approach introduced in this chapter of dividing cohorts into groups based on certain characteristics, such as their endowment with human capital.

But the new approach proposed here transcends Ryder in his "complete cohort determinism" (Lutz, 2013, 286) and no longer sees individuals as determined by the cohort that they were born into. Rather, it allows for changes in people's characteristics over the individual life course, either in consequence of aging or due to the transformational period effects described in this chapter's introduction or possible interactions between the two. This acknowledges the possibility of "learning" beyond school age (or the formative years during which a young person's socialization takes place more generally) and such individual level learning experiences to then contribute to social change at the aggregate level. As a consequence, under this new conception of the DM, Ryder's predicament of the "stagnant pond" would not materialize even if individuals were immortal because changes within birth cohorts can in itself represent a force of socioeconomic change. The relative strength of cohort effects as compared to period and age effects will, however, depend on the specific phenomenon studied. At least in the case of human capital formation, which we are focusing on here, people do not cease to evolve as they age. They continue to make further experiences, they get trained on the job, and parts of their human capital depletes and becomes useless or at least less productive. For practical purposes, though, we are usually interested in measurable aspects of human capital, which in many cases leaves us with nothing but the individual characteristic of highest educational attainment that is stagnant for most people after school leaving age.

5.3 Accounting for Population Heterogeneity

The methodological advancements that opened up the possibility for further developing the DM into a theory of social change with predictive power came from multi-dimensional cohort component analysis (Rogers, 1975; Keyfitz, 1985). Pioneered by researchers at the International Institute for Applied Systems Analysis (IIASA) in the 1970ies, this new

approach toward population heterogeneity provided the tools to model changes over the life course, which have since revolutionized the way demographers do forecasting.

Demography has always been the study of the size and composition of populations and from its very beginnings, when it originated from the discipline of "political arithmetic", population forecasts were an essential component of the demographic toolkit, be it for military planning, budgetary considerations or the analysis of ongoing population trends and their future implications. For the longest part of human history, though, population scientists were looking at populations as if they were homogeneous and forecasts were made simply by applying some assumed future growth rate to a given initial population, ignoring the underlying complexity of population structures. Because even under the conditions of a pre-Malthusian world, where populations were more stable and growth rates did not vary as dramatically as in the recent centuries since the beginning of the demographic transition, this traditional approach ignored important fluctuations in population structures that were the consequence of past wars, pestilence, or famine.

The cohort-component projection model that has increasingly come into fashion during the second half of the twentieth century marks a major first step toward recognizing these important irregularities by distinguishing members of a society by age and sex. Of the numerous transitions that demography deals with, the most fundamental transition is of course the unidirectional transition from life to death, which can be described for any population by a (single decrement) life table. A life table is constructed based on age-specific mortality rates, allowing demographers to determine the probability of survival, as well as the corresponding remaining years of life at any age within a population. Such tables are typically calculated separately for men and women, because observed patters of age-specific survival vary substantially by sex, and are then used in cohort-component projection models to describe future population dynamics. In contrary to a model considering only one joint growth rate for the entire population, the cohort-component model accounts for irregularities in a population's age distribution, including age- and sex-specific differences in fertility, mortality and migration. But

while clearly an improvement over the simple exponential growth rate model, the assumption of all members of one cohort sharing the same characteristics with respect to the drivers of population change remains highly questionable.

This limitation is overcome in multi-dimensional demography where the drivers of population change can differ for different population sub-groups as defined by additional characteristics besides just age and sex. This more nuanced view of population change paved the way for simultaneously projecting sub-populations with different fertility, mortality, and migration schedules, while accounting for important membership transitions between different sub-populations. Not surprisingly, many of the topics that demographers deal with nowadays have to do with people's transitions between different group affiliations over specific time intervals, including the transition to adulthood, marriage, parenthood, transitions in and out of the labor force, or the transitions between different levels of education. These group affiliations are important study subjects in their own right, but they become relevant in the context of the DM as in parallel with group affiliation also people's attitudes and behaviors tend to change with often widespread societal implications.

As the multi-dimensional model was first conceived in the context of regional population studies, the distinguishing characteristic that researchers first started to consider was people's geographical sub-region. Rather than assigning the same probability of conceiving, dying, or changing place of residence to every individual member of a population with the same age and sex, the multi-state cohort component projection model projected sub-regional populations forward allowing the corresponding rates to vary also by smaller spatial units. But considering different provinces is by far not the only way of accounting for sub-national heterogeneity in populations. Researchers have also looked at different marital states (Schoen and Nelson, 1974), labor force participation (Willekens, 1978), or highest level of educational attainment (Lutz, 1994) as important criteria for distinguishing relevant sub-populations, which at the same time required the generalization of the simple, single-decrement life table. The multi-state cohort component model adds the possibility that in some cases transitions can go either way, as in the

case of marriage (increment-decrement life tables), or through multiple, competing processes, as when considering different causes of death (multiple-decrement life tables).

To set up a multi-state population projection we first need to define our state space that can consist of different provinces between which migrations are possible, different education levels that people can attain (typically only in ascending order), or labor force status which can change from employed to unemployed and back at various stages in a person's life. If we run our projection for only one region and distinguish the population by three different levels of educational attainment (low, medium, high) and by whether they are employed or unemployed, our state space contains six possible states, to which we need to add a seventh state in which the children not yet participating in the labor market will find themselves. Women of birth-giving ages are exposed to state-specific risks of fertility and give birth to children that grow older by one year in each calendar year while being exposed to state-specific mortality and migration rates throughout their life. As soon as these children are old enough to start working, they switch to one of the six adult states depending on which education level they attained, which at the same time affects their probability of being employed.

This simple example shows how the multi-state population projection model can be used to assess the consequences of the DM in quantitative terms. By projecting populations by relevant characteristics in addition to just age and sex, we not only achieve a more accurate forecast of future human numbers, as those characteristics help us to better account for the underlying population heterogeneity with respect to the drivers of population change, but we can at the same time quantify how future societies will be different from today as a consequence of the changing composition of their populations with respect to these characteristics.

5.4 Population Dynamics by Level of Education

Education has been shown to be one such characteristic that is at once highly relevant in explaining differentials in demographic and economic behaviors and at the same time a major determinant of social heterogeneity. While for theoretical reasons it would be preferable to look at human capital, which can be defined in a variety of ways, usually including some measure of human health (e.g. medical care or vitamin consumption), as well as on-the-job training that adds to individuals' knowledge, skills and abilities (Becker, 1962), for practical purposes we often have to conform ourselves with the level of formal educational attainment (Lutz and KC, 2011). At the aggregate level, this measure can then serve as proxy for a population's skill base or the stock of knowledge that is available to either generate economic output, human well-being, or to prevent the destruction thereof, as in the case of natural disasters and wars. Comparable data on health, skills and informal training is available to a far lesser degree. Flow measures of human capital, such as school enrollment rates, do not inform us about the stock of human capital that is available within a society at a given point in time and aggregate measures, such as mean years of schooling, ignore the underlying distribution of capabilities with respect to important confounders within a society. While in measuring human capital it would certainly be desirable also to account for differences in the quality of schooling (Hanushek, 2013), such information is available only for a small number of (mostly OECD) countries. For older cohorts that have left school some time ago information is even more limited.

Numerous studies have highlighted the strong relationship between education and drivers of population change. Higher levels of education are strongly linked to fertility decline, particularly in low-income countries (Cochrane, 1979; Cochrane et al., 1990; Bongaarts, 2010; Skirbekk and KC, 2012), and have also been shown to matter greatly in explaining mortality differentials (Elo and Preston, 1996; KC and Lentzner, 2010; Baker et al., 2011; Lutz and Kebede, 2018). In some countries, notably the US, differentials by level of education have even been on the rise lately (Olshansky et al., 2012; Sasson, 2016). Not surprisingly, researchers

have long called for education to be routinely included in the study of population dynamics (Lutz et al., 1998). While time series on school enrollment are readily available for large numbers of countries, times series on education stocks among adult populations are typically hard to come by. Reconstruction efforts using interpolation or the perpetual inventory method (Barro and Lee, 1996, 2013) often suffer from the lack of quality of the underlying base data leading to unexplainable jumps in the derived time series and large scale inconsistencies in regression analyses conducted using such datasets (Cohen and Soto, 2007; Goujon et al., 2016).

Making use of the multi-state cohort component methodology described earlier, Lutz et al. (2007) therefore suggested an alternative approach to reconstructing education stock data. Education carries the methodological advantage of being—for the most part—persistent over the life course. With the exception of unidirectional transition probabilities between different education levels at younger ages, which can be modeled from school enrollment data, no assumptions have to be taken in order to model future attainment levels within a cohort, as those can be derived from the DM: If we know the proportion of people with a specific level of education aged 50–54 today, the proportion of people with the same level of education aged 45–49 5 years earlier can be calculated applying age, sex, and education-specific mortality and migration rates according to

$$N_{a-5,t-5}^{edu,sex} = \frac{N_{a,t}^{edu,sex}}{sr_{a,t}^{edu,sex}}$$

where

N refers to the number of people
a refers to the five year age group
t refers to calendar year
edu refers to the highest educational level attained
sex refers to the gender of individuals
sr refers to the proportion of people surviving in the country (i.e. not deceasing from the population through death or out-migration) between time $t - 5$ and t in each age, sex, and education category.

This approach has since led to the reconstruction of past population by age, sex, and level of educational attainment back to 1950 in 170 countries, as well as the forward-projection under different future scenarios to 2100 for 201 countries of the world (KC et al., 2010; Lutz et al., 2014; Lutz et al., 2018). The reasons why these forecasts can be conducted over such long time horizons, as well as for a large number of countries, where data availability often impedes the production of other types of forecasts, are related to the inertia in the population system. First of all, the forecasts rely on slowly changing stocks of people that are mostly already born in the base-year of the forecast. Second, these people acquire many of their stable characteristics, e.g. their education, during their formative years early in life, such that no assumptions about future changes in these characteristics have to be taken. Third, in the majority of countries today life expectancies range well beyond 70 years, adding to the long term stability of demographic forecasts.

As this model of human capital formation relies only on the most recent and therefore less error-prone data, the reconstruction of past educational attainment offers the possibility to revisit earlier findings about the role of human capital in different dimensions of human well-being. For example, the relationship between education and economic growth has long been disputed and the lack of high quality data on education has been blamed in part for the ambiguous findings (Benhabib and Spiegel, 1994; Pritchett, 2001; Cohen and Soto, 2007). Moreover, the measure of human capital used in that research (mean years of schooling) brushed over important details regarding the vintage structure of human capital. Considering the full age distribution of human capital available from the reconstructed human capital history, Lutz et al. (2008) found consistently positive and significant effects of education on economic growth. In line with the DM approach, the research showed that growth expansions in emerging economies took place once better-educated young cohorts replaced their antecessors in the labor force, thus increasing the speed of technological innovation and adoption.

In related research on the demographic dividend, Crespo Cuaresma et al. (2014) used the reconstructed dataset to revisit the conventional theory of the dividend being spurred by changes in populations' age structures (Bloom et al., 2003; Bloom and Canning, 2008). In addition to the age structure effect, they were able to show important effects of human capital accumulation that preceded the changes in age structure. Their model suggests that—rather than exogenous declines in fertility by themselves—increasing levels of education lead to reductions in fertility in the first place, freed female labor to participate in the workforce, whilst raising the opportunity cost of better-skilled female labor and boosting productivity. Besides the role of human capital in the creation of more democratic institutions (Lutz et al., 2010), researchers have recently also used the data for "Redrawing the Preston curve" (Preston, 1975). While Preston interpreted his original finding of the relationship between income and mortality as the effect of medical progress and health care over and above the effect of income, Lutz and Kebede (2018) find a mediating role for education.

5.5 Applying the Demographic Metabolism to Human Capital Formation

In the next section, we want to look at how the changing educational composition, described by scenario-based population projections using the multi-state cohort-component model, affects our assessments of future societies' human capital formation. The social change resulting from repeated cohort replacement can best be illustrated by looking at the evolution of three-dimensional population pyramids over time. Figure 5.1. shows the case of Singapore, with colors representing the proportions of men (left) and women (right) with different levels of education across all age groups on the vertical axis. The gray bars at the bottom of the pyramids correspond to the population under the age of 15 where a breakdown by level of education is not available.

The pyramid on the left shows that back in 1950 Singapore was still a developing country. Only among the young people had the recently

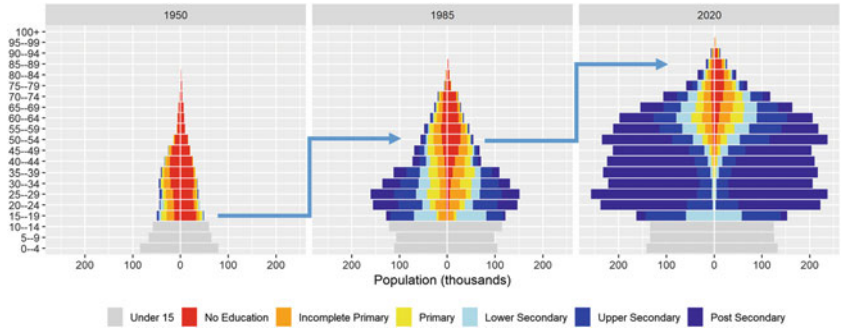

Fig. 5.1 Age and education pyramids for Singapore (1950, 1985, 2020). Colors indicate highest level of educational attainment. Children under the age of 15 are depicted in gray

initiated education expansion already had some visible effects. Among the elderly, particularly women, the vast majority of the population still had no education at all. Not surprisingly, poverty was widespread and the country was heavily in the grip of malaria. But the efforts started in the 1950 continued and over the next 35 years Singapore went through a phase of rapid economic growth that was propelled to a large extent by rapid demographic turnover and replacement of the uneducated older cohorts by younger, far better educated ones (Lutz et al., 2008). By 1985, the DM has moved the younger cohorts born since 1950 up the population pyramid following the blue arrow. But they did not only get older, they also became increasingly more educated as they went on to replace their parents' and grandparents' generations and became the new majority among the population of working ages. The greater availability of skilled labor and entrepreneurial qualification attracted new industries and created higher productivity employment opportunities. Also, malaria had been successfully eradicated.

While increased human capital by itself may not be enough to explain all of Singapore's development success story, there is also no doubt that governance and the right macro-economic policy strategies alone would not have been enough in Singapore and many other Asian tiger countries to spur such rapid economic development. As Fig. 5.1. illustrates, this

change took place along cohort lines and the DM was one of its major engines.

Following the transformation that Singapore has been undergoing as a consequence of the DM for another 35 years, in the picture on the right we see that by 2020 only very few of those uneducated young people visible in the 1950 pyramid will still be alive, whereas down at the bottom of the pyramid we witness the consequences of the education expansion, especially among women, on fertility. In parallel with Singapore's unprecedented school expansion, fertility levels started declining and became another important precondition for the economic growth miracle that followed. While back in 1950, the total fertility rate in Singapore was still at 6.6 children on average per woman, by 1985 it had declined to below 1.7 and today it is at 1.2. Many commentators across Eastern Asian countries see this development as part of a growing aging crisis, about to intensify once the largest baby-boom cohorts start to retire. But they ignore the growing human capital of the smaller young cohorts in countries such as Singapore. They will be more productive than their less educated parents and grandparents in the past and there is strong evidence that better-educated people also age more healthily (Weber et al., 2014), thus slowing down human capital depletion in old age.

Whether these profound changes in the population structure of Singapore will eventually lead to ever higher pension ages in the future can of course not be predicted based on the DM, as that would also strongly depend on political feasibility. Nevertheless, studying today's demographic circumstances in Singapore through the lens of the DM and predicting this society's future development forward along cohort lines there is also reason for optimism. If in addition to the economic arguments ecological reasoning is applied, low fertility (in response to strong investments in human capital) that leads to less emissions (if the population growth effect that reduces aggregate emissions outweighs the emission-enhancing economic growth effect from increased human capital) have to be endorsed rather than opposed (Striessnig and Lutz, 2014).

But human capital formation viewed through the DM not only affects how we see shrinking and rapidly aging future societies under environmental constraints. A DM-based perspective also affects our view

of future societies' adaptive capacities with respect to climate change (Lutz and Muttarak, 2017). In studying future environmental impacts on human societies, researchers tend to pose the challenges of tomorrow to the societies of today or to put it in the words of the IPCC's Fifth Assessment Report: "superimpose biophysical 'futures' onto present-day socioeconomic conditions" (IPCC, 2014). An example is the attempt to model the potential future effect of climate change on malaria transmission under observed present day social conditions (Tanser et al., 2003). This is of course only realistic under Ryder's assumption of the "stagnant pond" which ironically would offer perfect breeding conditions for the mosquitos spreading the disease. Under real world conditions, though, that can be projected forward using multi-state cohort component methodology, future societies will be very different and hopefully much more resilient in every respect than today's and therefore able to face the challenges posed by man-made climate change with greater vigor. The replacement of less-environmentally aware older cohorts by young people that have been exposed to more ecological ways of thinking and post-materialist values from almost the very beginning of their lives is going to affect environmental attitudes, behaviors, as well as political decision making—once they start to form the majority among the electorate—in the future.

This view has led to the inclusion of education as part of "the human core" of the so called Shared Socioeconomic Pathways (SSPs, O'Neill et al., 2014; KC and Lutz, 2017). These qualitative global development narratives, which are now widely used by the integrated assessment modeling community to describe alternative futures with regard to both mitigation and adaptation challenges arising from climate change, have been translated into consistent quantitative assessments of future trends, not only in population, but also economic development (Leimbach et al., 2015), global urbanization (Jiang and O'Neill, 2017), as well as energy, land-use and greenhouse gas emissions (Riahi et al., 2017) and can help gain a better and joint understanding of how future societies will be different from today's. Figure 5.2. looks at the Democratic Republic of Congo (DRC), one of the places most severely hit by the malaria epidemic today, as well as under future climate scenarios (Moukam Kakmeni et al., 2018) under three out of the five SSPs. In the 2010–2015 period,

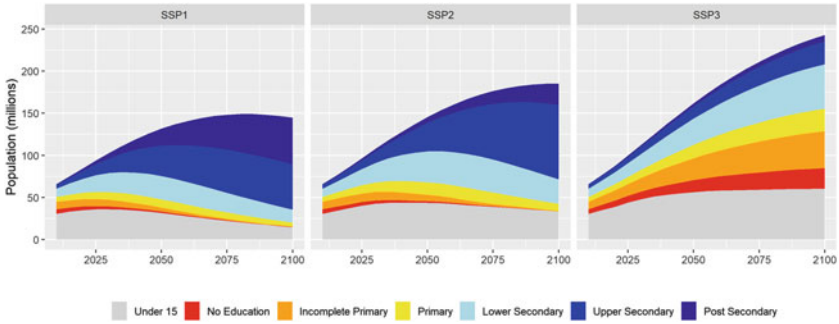

Fig. 5.2 Future population scenarios by level of educational attainment to 2100 on the basis of Shared Socioeconomic Pathways (SSP1, SSP2, SSP3) for the Democratic Republic of Congo

fertility in the DRC was still at a staggering 6.4 children on average per woman, life expectancy was at 56.7 years for men and 59.5 for women, while net migration played only a negligible role in DRC's population dynamics. The most optimistic SSP1 in the leftmost panel describes a world following the paradigm of sustainable development, corresponding in demographic terms to the assumption of low fertility (TFR of 1.27 by the end of the century), low mortality (life expectancy of 79.9 years for men and 84.5 for women) and medium levels of migration (128.000 more people leaving the country compared to those coming in over the last five years of the century), as well as a rapid educational expansion, similar to the Singaporean experience in the past. The effect of education on fertility leads to a much lower population by the end of the century, despite low mortality arising from improved sanitation, nutrition, medical care and reductions in disease prevalence e.g. malaria.

There is, however, also a distinct possibility that the future of global development will not be guided by sustainability, but rather by increasing social and economic inequality, both between developing and the developed parts of the world, as well as within developing countries. This pessimistic counter-narrative (SSP3) is reflected in stagnating school enrollment rates and a delay in the already belated demographic transition of the DRC. In consequence, both fertility (TFR of 2.5) and mortality (life expectancy of 57.8 years for men and 63 years for women) are

assumed to remain relatively high until the end of the century, while the deep segregation between the developed and the developing world hardly allows for population exchange between these two parts of the world. Population size and structure with respect to important dimensions of population heterogeneity will entail far more reasons for pessimism in the DRC than under the optimistic SSP1 scenario, or the most likely "middle of the road" SSP2 scenario, that assumes medium levels of all three drivers of population change until the end of the century (TFR of 1.78, life expectancy of 70.3 for men and 75 years for women, as well as 300.000 more people leaving the country over the five year period from 2095–2100 than coming in). Considering the vastly different educational compositions, these three scenarios also imply vast differences in the prospects for future human well-being. By 2100, SSP1 describes the DRC as a nation of 145 million (still less than double of today's population) mostly well-educated and therefore healthy and wealthy people that will be capable of dealing with the consequences of climate change. Under SSP3, on the other hand, the DRC's population is predicted to more than triple and due to the failures in past developmental policies and the lack of international cooperation, those 243 million, mostly poorly-educated Congolese, will be far more vulnerable to environmental threats.

Besides differences between future scenarios, members of future societies will not be equally vulnerable to risks deriving from climate change, but rather suffer from differential vulnerability at the individual, community and national levels (Butz et al., 2014). Vulnerability can again be linked to important individual level factors, such as age, sex, education, income or place of residence and inform future assessments of societies' vulnerability to climate change following the DM approach. Moreover, a better understanding of the future composition of populations with respect to their demographic characteristics under different future narratives can help identify specific risk groups, as well as policy priorities to reduce foreseeable loss of human lives and material damages.

5.6 Conclusion

This chapter has presented a theory of social change based on the notion of Demographic Metabolism and applied it to the case of human capital formation. While the idea of social change through cohort replacement is not new, only through recent methodological advancements in formal demography that have led to the multi-dimensional demographic model by age, sex and level of education, has it become quantifiable. As shown by a growing body of literature, potential applications of the concept are far reaching, but the focus on human capital formation has special appeal. Human capital is one of the main determinants of social heterogeneity and reveals marked differences within and between societies. As a consequence, investments in human capital not only have a large number of social, economic and health benefits among human populations but also represent an efficient adaptation strategy with respect to the future challenges arising from anthropogenic climate change.

For reasons of measurability and data availability, the indicator that is typically used to follow international trends in human capital—forward and backward in time—is the individual characteristic of highest level of educational attainment. While the workings of the DM can be exemplified by many other relevant characteristics, as long as data from different observation periods is available from sufficiently large, age-structured sample populations, looking at education greatly simplifies matters, as for most people educational attainment does no longer change after they have left school, which typically happens early in life. Therefore, changes in cohort composition only have to be considered during the formative ages and less assumptions are required in projecting observed trends in human capital formation forward or backward along cohort lines.

The DM approach builds upon the basic assumption that any human population can be subdivided by certain measurable characteristics. If membership in the resulting sub-groups of society remains stable over the life course (as in the case of ethnicity or native language), or changes only marginally (as in the case of education or some very strong normative convictions), then the described tools provided by multi-dimensional demography allow us to model the societal evolution with respect to the

social phenomenon of interest as the relative size of the different subgroups changes through cohort replacement. A less stable phenomenon that is characterized by heavily changing group membership along the life course does not necessarily render the DM inapplicable, provided that the transitions or membership changes follow certain identifiable and predictable patterns. While the uncertainty ranges of the resulting predictions will be larger in such cases, due to the length of the human lifespan the DM still remains the theory in the social sciences with perhaps the largest predictive power even over decades into the future.

In light of this noteworthy property, it is all the more remarkable that the DM has so far not been appropriated by the wider community of social researchers. Even within the field of demography, up until its recent rediscovery through Lutz (2013), only a few articles were following the paradigm. Neighboring fields such as economics have made use of the DM to an even lesser degree. This can in part be explained by a lack of communication between the different strains of literature, e.g. population economics, demographic forecasting and growth accounting. Considering the wide range of possible applications and its usefulness in forecasting and reconstructing sociodemographic developments, it can only be hoped that the DM reaches a wider audience.

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Part II

Human Capital in the Form of Health, Its Economic Implications, and the Economic Policies for a Healthier World

6

Health and Income: Theory and Evidence for OECD Countries

Alberto Bucci, Lorenzo Carbonari, and Giovanni Trovato

JEL Classification I15, I25, J24, O41, C14

6.1 Introduction: Motivation and Background

This chapter takes Mankiw–Romer–Weil seriously. In their famous 1992's paper, they demonstrated that a standard Solow (1956) model *augmented* with the inclusion of the accumulation of human as well as physical capital can provide a better understanding of the international differences

A. Bucci
Department of Economics, Management, and Quantitative Methods (DEMM), University of Milan, Milan, Italy
e-mail: alberto.bucci@unimi.it

L. Carbonari (✉) · G. Trovato
Department of Economics and Finance, University of Rome "Tor Vergata", Rome, Italy
e-mail: lorenzo.carbonari@uniroma2.it; giovanni.trovato@uniroma2.it

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in income per capita and that the existing disparities in saving rates, education and population change can account for most of the cross-country variations in living standards. Though, the Mankiw et al. (1992)'s story neglects the role of health, so in their framework the following two questions still remain open. Across the world, what is the role of health in determining a country's degree of economic development (i.e., its level of real per capita income)? What is health's role in explaining cross-country differences in economic development? In this chapter we try to fill this gap in their analysis.

Our starting point is therefore the recognition that human capital can appear either in the form of education/schooling, or in the form of health. In order to test this new *"augmented Solow model"*, we include in our regressions proxies for human capital accumulation and population's health as additional explanatory variables. Moreover, we deal with the potential endogeneity between income and health by developing a parsimonious statistical approach that consents to take into account different possible sources of heterogeneity across countries. In this regard, we assume that cross-country heterogeneity may be modeled through the inclusion of a latent effect which allows for a posterior classification of each country of our sample based on its own value of the latent variable. In this way, we are able to study the role that health-differences can play in explaining income-differences across countries, with a special focus on OECD ones, where results seem to be more ambiguous. In brief, the aim of our analysis is to assess quantitatively the relative contribution of the health-variable (in addition to, and separately from, the education-variable) in accounting for the international differences in per capita income within a sample of OECD countries.

Yet, before doing this we first provide a broad overview of the main theoretical, as well as empirical, literature that has analyzed the multifaceted relation between health, income, and economic growth and in which our contribution can be placed.[1]

According to the report that followed the second consultation of the World Health Organization's Commission on Macroeconomics and Health held in Geneva in 2003 (WHO, 2003):

> *. . . Socio-economic development can be achieved only by rigorously promoting the implementation of pro-poor policies within a viable development strategy, financed through a significant increase in health investments. We have witnessed important achievements, including heightened attention among policy-makers to the health of the poor and recent increases in assistance for health. But more determination and resources are needed to meet the real health needs of the poor. Failure to act promptly and decisively will result in countless additional deaths and illness from preventable causes, trapping individuals and families in poverty and hindering economic growth and development . . . .*

At the heart of such a declaration is the belief that sizeable gains, in terms of economic growth and development, can still be reaped from health improvements. Bloom et al. (2014a), for example, estimate that, over an 18-year period, five categories of non-communicable diseases can together reduce labor supply and capital accumulation in China and India in such a way to cause an overall 34 trillion dollars' worth damage for lost output.

Apparently, the answer to the fundamental question of whether a better health is able to support higher productivity and standard of living seems trivial: of course, yes! After all, healthy people lose less time from work due to ill-health and, when working, are more productive (both physically and mentally). In a word, better health would seem to contribute without any doubt to increase *directly* labor market participation and workers' productivity (Strauss and Thomas, 1998; Bloom and Canning, 2000; Schultz, 2002). At the same time, better health appear to have also a number of other *indirect* positive effects on the level (and growth rate) of per capita income through, for example, a change in the individuals' incentives to invest in human and physical capital, to save, and eventually through a change in the population growth rate, as well.

However, four major difficulties do immediately arise when one tries to assess the overall (direct and indirect) effects of health on income, growth and development and to summarize the ultimate results that the

existing literature has already reached in the field. The first is represented by the *measurement issue*. Health is measured differently across the various studies (the measures employed in microeconomic analyses are generally diverse from those used in macroeconomic models). Moreover, while some works focus on the influence of specific diseases on income per capita,[2] others instead look at the economic effects of more "aggregative" measures of health (such as life expectancy or survival).

The second issue has to do with *causality*: the correlation between health and income can result either from income causing health, or from health causing income, or from some other factor(s) causing both, or else from some combination of the previous three channels all together.

The third issue concerns the existence of health's *'partial-equilibrium' effects* (i.e., those arising from holding all other factors fixed), as opposed to its *'general equilibrium' effects* (i.e., those stemming from a framework where other factors that respond to improved health are explicitly and jointly considered in the analysis). In other words, while some studies focus only on the proximate (or direct) impact of health, others attempt to capture also its indirect economic effects.

Last but not least, the final issue is related to the *composition of the sample* (developing vs. developed countries, or pre-demographic-transition vs. post-demographic-transition countries), with regard to which the consequences of health improvements are evaluated and that in empirical studies play a crucial role in determining the sign of the possible correlation between health and income.

In the last few years there has been an upsurge in the empirical and theoretical work on the nexus between health and income. In what follows we briefly review such advancements, with a special eye on the four major difficulties briefly outlined above.

The measurement of health: Research examining the link between health and economic outcomes generally employs two types of health measures: health inputs and health outcomes (Weil, 2007, pp. 1268–1269). Health inputs are all those physical factors that may influence an individual's health. These include (but are not limited to) nutrition at various points in life (in utero, in childhood, in adulthood); exposure to various forms of pathogens; availability of medical care, etc. Health outcomes are all those personal characteristics that are determined either

by an individual's health inputs or her/his genetic endowments (life expectancy, survival rate, height, the ability to work harder and produce more than others, etc.). In turn, health outcomes can be either non-observable—a notable example in this respect is what Weil (2007, 2014) defines as *'human capital in the form of health'*, that measures how health affects one's ability to produce output[3]—, or observable (in this case health outcomes are called 'health indicators').

An extensive literature examines the effects of health, defined and measured in several ways, on *individual* (as opposed to national or aggregate) economic outcomes. Examples of this approach include Fogel (1997), Behrman and Rosenzweig (2004), Black et al. (2007), Almond (2006), Bleakley (2007), and Miguel and Kremer (2004), among many others. Fogel (1997) studies the effects of better nutrition on output and labor-supply per worker over an interval of 200 years.[4] Behrman and Rosenzweig (2004) use variations in birth weight among identical twins to identify the effect of fetal nutrition on education and wages among adults. Their main estimate regresses the gap in the dependent variable (height, log wages, or schooling) between a pair of twins on the gap in fetal growth. They find that a one-unit difference in fetal growth (measured in ounces per week of gestation) leads to a difference of 0.657 years of schooling, 3.76 centimeters of adult height, and 0.190 gap in log wages. Black et al. (2007) perform a similar analysis using data on Norwegian men. For the full sample, their within-twin-pair estimate of the effect of log birthweight on log earnings is 0.24. Almond (2006) shows that individuals exposed to Spanish influenza in utero had lower education attainment and higher rates of disability than surrounding cohorts. Bleakley (2007) and Miguel and Kremer (2004) find that treatment with deworming drugs increases school attendance. Overall, microeconomic studies are mainly aimed at assessing the economic consequences of health inputs (or morbidity-measures) at an individual level. On the contrary, in macroeconomic models population health is usually measured by life expectancy, or some other mortality-measures (as opposed to the morbidity ones).[5] It is therefore quite difficult to compare studies that use such different notions of health.

Causality: While it is now accepted that high levels of population health go hand in hand with high levels of national income, it still remains disputable whether better health is the consequence or the cause of higher income (Adams et al., 2003). The most important piece of evidence of a positive correlation between income and health is represented by the so-called *'Preston Curve'* (after the path-breaking work by Preston, 1975), that shows the link between GDP per capita (on the horizontal axis) and life expectancy at birth (on the vertical axis). One intuitive reason for this positive correlation to exist is that higher income generally allows for better food, shelter, cleaner water and sanitation, and enhanced medical treatments. Moreover, countries that are richer can afford higher expenditures on public health. According to Preston (1975), the increase in life expectancy experienced in a country over time can be decomposed into two different parts: the part due to higher income (this is the movement along the Preston curve), and the shift in the whole curve. Preston's calculations showed that less than one quarter of the average mortality-improvement observed between 1930 and 1960 was due to movement along the curve, with the remainder due to shifts in the curve. In other words, income gains were not the primary source of health improvements (health interventions can improve individuals' health without the need for previous improvements in their incomes). As far as the causal link between income and health is concerned, Pritchett and Summers (1996), Bloom and Canning (2000), Bloom, Canning, and Sevilla (2003b), and Bloom and Fink (2014) all focus on the channel that runs from health to income, while Cutler et al. (2006) and Hall and Jones (2007) are more recent examples of how the reverse causal channel (going from income to health) can work. The existence of complementarity between health and education make the causality problem even worse as education exhibits the same two-way causality in its association with income (Becker, 2007).

Two alternative methods are employed by the literature to assess the overall effects of health on income and economic growth.[6] The first aggregates the results of Mincerian wage-regressions of the return on individual health to derive the macroeconomic effects of population health. The second, instead, relies on the estimation of a generalized aggregate production function that decomposes human capital into its

components (including health). While most of the studies based on both methods point to a positive effect of health on economic growth, the size of such effect remains, however, subject to debate (it is generally found to be small in the first and large in the second type of studies). Weil (2007) has been among the first to use the available microeconomic estimates to quantitatively evaluate the importance of health at the macroeconomic level. More specifically, he constructs a framework in which estimates of the effect of variation in health-inputs on individual wages can be used to generate estimates of how differences in health, as measured by observable outcomes, contribute in turn to differences in national income. In so doing, Weil (2007) extends the development accounting methodology of Klenow and Rodriguez-Clare (1997) and Hall and Jones (1999) to include also a measure of health. He finds that eliminating health gaps among countries would reduce the variance of log GDP per worker by about 9.9 percent. The conclusion is therefore that while the estimated effect of health on income is positive and economically significant, it is also much smaller than existing estimates derived from cross-country regressions would suggest. Bloom et al. (2004, pp. 2–4), for example, report the results of thirteen such studies, which mostly reach similar quantitative results. Their own estimate, which comes from regressing residual productivity (after accounting for physical capital and education) on health measures in a panel of countries observed every 10 years over 1960–1990 is that a one-year increase in life expectancy raises output by 4 percent. In a paper companion to Weil (2007), Shastry and Weil (2003) calibrate a production function model of aggregate output using microeconomic estimates of the return to health. They assume a stable relationship between average height and adult survival rates so that when adult survival rates improve they can infer a rise in population heights. Using estimates of the effect of height on worker productivity and wages from microeconomic studies they calibrate what health improvements in the form of changes in adult survival rates should mean for aggregate output. They find (Table 3, p. 395) that cross-country gaps in income levels can be explained in part (20.1%) by differential levels of physical capital, in part (21.6%) by differential levels of education, and in part (19%) by differential levels of health. Hence, over half of cross-country differences in income levels can be explained by these three factors, the

remainder of the gap (39.3%) being ascribed, instead, to differences in total factor productivity.

In an influential macro-level paper addressing the issue of whether health may or not have an effect on income, Acemoglu and Johnson (2007) use panel data for 47 countries and exploit the drop in mortality from specific infectious diseases, due to the international epidemiological transition, as an instrument for the change in life expectancy. This identification strategy makes use of the fact that the mortality rate from these diseases was exogenous in 1940, because no treatments, medication, or vaccines were available before that time. Starting from 1980, instead, all these diseases can be treated or prevented in all countries, due to medical advances. After regressing per capita income growth on the increase in life expectancy between 1940 and 1980, Acemoglu and Johnson (2007) report a positive but insignificant effect of increased life expectancy on *aggregate GDP*, and a positive and significant effect on population growth. The overall effect on *GDP* per capita is found to be negative (which means that countries that experienced larger exogenous health improvements saw lower gains in income per capita). Acemoglu and Johnson (2007) attribute their findings to the fact that increases in health result mainly in large increases in population. In turn, as it is well known from Solow (1956), the capital dilution effect associated to a faster population growth reduces income per capita at the steady state. Therefore, in the end improved health lowers per capita income. However, the Acemoglu and Johnson (2007)'s methodology has been challenged as it regresses economic growth against health improvements without including initial health in the model. As such, the negative correlation between health improvements and economic growth shown in their data may simply be the consequence of the fact that countries starting with better health economically grow faster (while experiencing smaller improvements in health) than those other countries starting instead with lower initial health conditions and that at the same time experiment larger health enhancements.[7] To study this possibility, Aghion et al. (2011) and Bloom et al. (2014b) include initial health in the Acemoglu and Johnson (2007) regressions and find that, indeed, the negative causal effect vanishes.[8] More specifically, Aghion et al. (2011) combine the so-called Mankiw-Romer-Weil (1992)'s approach (whereby

output growth is correlated with the rate of improvement in human capital) with the so-called Nelson-Phelps (1966)'s approach (whereby a higher level of health should spur growth by facilitating technological innovation), and look at the joint effect of health and health accumulation on economic growth. After running cross-country growth regressions over the period 1960–2000, they show that the level and the accumulation of health have significant positive effects on per capita income growth.[9] Finally, they find a weaker relationship between health and growth over the contemporary period in OECD countries. According to them, this result can explained by the fact that only gains in life expectancy below 40 years are significantly correlated with per capita income growth.

Direct vs. indirect health's economic effects: As already mentioned above, better health can have not only *direct* but also *indirect* economic effects. Indeed, if on the one hand healthier people are able to work longer and harder (which leads, respectively, to higher labor market participation and higher productivity levels), on the other hand better health has also a number of different indirect economic effects that operate through, for example, a change in the individuals' incentives to invest in human and physical capital, a change in their incentives to save, and eventually a change in the population growth rate. In particular, concerning the effect of better health on population growth, Weil (2007, p. 1266) observes that such effect may be ambiguous, as in their words:

> *. . . In the short run, higher child survival leads to more rapid population growth. Over longer horizons, however, lower infant and child mortality may lead to a more-than-offsetting decline in fertility, so that the net rate of reproduction falls . . . .*[10]

Further than the (direct and/or indirect) effects of life expectancy on income, reductions in mortality may also increase individual wellbeing by not only extending the lifetime horizon but also by improving people's quality of life (Becker et al., 2005; Murphy and Topel, 2006). By looking only at the direct, or proximate, effects of health on income, and thus holding constant the level of physical capital, education, the quality of institutions and so forth, Weil (2007) concludes that the size of the impact is relatively small, and definitely smaller than the estimated effect of health

on economic growth that is obtained from cross-country regressions. To translate Weil (2007)'s conclusions in numbers, he finds that a health improvement that raises life expectancy by five years would increase labor productivity by 3.6 percent and output per capita in the steady state by the same amount. In order to have a raw idea of what these figures might imply notice that along the 2010's Preston curve an increase in life expectancy of five years would have been associated with a *doubling* of output per capita.

All this said, the positive effects ultimately accruing to economic growth from a better health (no matter how it is measured) through, say, people's education are now unquestionable. Ben-Porath (1967) was among the first to show that if individuals live longer then investments in human capital are more likely to pay off because the working life is lengthier. This implies an increase in the return to human capital accumulation, hence higher incentives to invest in skills. De la Croix and Licandro (1999), Kalemli-Ozcan et al. (2000), and Boucekkine et al. (2002, 2003) build models in which a decline in mortality produces greater investments in individual human capital and, therefore, a rise of economic growth. Chakraborty (2004) reaches similar conclusions in a model where longevity is made endogenous by public health investments. Cervellati and Sunde (2005) and Soares (2005) consider settings in which the mortality decline pushes parents to have (fewer but) better educated children. Bleakley and Lange (2009), and Jayachandran and Lleras-Muney (2009) provide robust and convincing evidence that higher life expectancy increases educational attainments at the individual level.

Concerning the possible impact of health improvements on economic growth by way of individuals' saving decisions, Blanchard (1985)[11] analyzes the growth effects of an increase in life expectancy in developed economies by replacing the representative agent assumption of the standard neoclassical growth theory with an overlapping-generations structure in which individuals face a constant risk of death. In his framework, an increase in life expectancy raises aggregate savings and therefore, according to the canonical mechanism underlying the neoclassic growth model, the growth rate of the economy during the transition to the steady state. Hurd et al. (1998) find that increased expectation of longevity leads

to greater household's wealth in the United States. Bloom et al. (2003a) find an effect of life expectancy on national savings, using cross-country data. Lee et al. (2000) argue that rising life expectancy accounts for the boom in savings in Taiwan since the 1960s. Finally, Zhang and Zhang (2005) construct a three-period overlapping-generations model showing that rising longevity reduces fertility and enhances savings and schooling investment, even though these effects are empirically quite small.

The recent regression results of Madsen (2016) clearly show that health has been highly influential for economic growth since 1870 for the 21 OECD countries considered not only through human capital investment, but also through ideas-production, the two core drivers of modern technological change and economic growth. A direct (and intuitive) effect of health on human capital is that sick children are more often absent from school (Mayer-Foulkes, 2005; Bundy et al., 2006; Currie et al., 2010). More importantly, illness can severely diminish the learning capacity of students because of reduced concentration in the classroom, cognitive impairment, and stigma (Holding and Snow, 2001; Alderman et al., 2005; Mayer-Foulkes, 2005; Bloom and Canning, 2009). Furthermore, chronic poor health can adversely affect ideas production because it impairs creativity and entrepreneurship (Howitt, 2005). Last but not least, recent research shows that societies with high pathogen stress are less innovative, less open to new ideas, and display introversion (Schaller and Murray, 2008; Fincher et al., 2008). According to Madsen (2016)'s findings, while working-age mortality rates are highly significant determinants of ideas production, school-age mortality rates (as proxies for morbidity rates) are especially influential on secondary and tertiary school enrollment, suggesting that health affects not only learning but also enrollment rates, and ultimately human capital accumulation.

Another channel through which health improvements may (indirectly) affect economic growth and development is represented by the so-called *'demographic dividend'*.[12] A common feature of every episode of demographic transition (Lee, 2003; Bloom and Canning, 2009) is that it generally starts with a reduction in mortality rates, while birth rates still remain high. With some delay, fertility also drops. The delay between the initial reduction in mortality and the subsequent decline in fertility

induces the typical hump-shaped pattern of population growth, which initially increases (due to lower mortality rates), but eventually slows down, if reduced fertility more than compensates the initial increase in population. As fertility begins to fall, the overall dependency ratio (the sum of the youth and the old-age dependency ratios) may decrease, as well. If this happens, the resulting *'demographic dividend'* releases resources that can be invested in further health, education and infrastructure improvements, so speeding up the transition toward a phase of sustained long-run growth. All this ultimately suggests that the demographic transition plays a central role in determining the sign of the effect of life expectancy on income per capita growth (see Cervellati and Sunde, 2011, and our discussion below).

Ashraf et al. (2009) undertake a detailed analysis of the different channels though which health affects output. Hence, they go beyond the static analysis of Weil (2007) in order to uncover also the dynamic effects of health shocks. Their simulation model allows for several channels through which health improvements may have an economic impact, including the effect of better health on human capital investment, the change in population growth triggered by increased survival rates, and the possible response of fertility to increased child survival. However, as in Weil (2007), the effect that they find is still relatively modest: an increase in life expectancy from forty to sixty years would raise GDP per capita in the long run by only 15 percent, and for the first thirty years after such an increase, output per capita would be lower than if health had not improved at all. Overall, their results imply that causation from health to income does not drive much of the observed cross-country correlation between the two variables.

Health's effects across different sample-compositions: Weil (2007, p. 1295, 2005, pp. 153–161) suggests that health's positive effect on GDP is stronger across poor countries. For rich countries, instead, the existing empirical evidence is mixed. For instance, while Rivera and Currais (1999a, b, 2003, 2004) find a positive effect of health expenditure growth on productivity growth for OECD countries (as well as the Spanish regions), Knowles and Owen (1995, 1997), and McDonald and Roberts (2002), reject the hypothesis that life expectancy is a statistically

significant explanatory variable for productivity growth in high-income countries. Bhargava et al. (2001) estimate a negative effect of the adult survival rate on economic growth for the US, France, and Switzerland. Hartwig (2010) looks at health-data gathered for 21 OECD countries, and finds no evidence that either health care expenditure or the rise in life expectancy positively Granger-cause per capita GDP growth. On the contrary, when per capita GDP growth is regressed on its own lags and on lags of per capita health care expenditure growth in a panel Granger-causality testing framework, the coefficients for lagged health care expenditure growth are robustly negative (even though the statistical significance of the negative coefficients is not robust to the choice of the GMM estimator).

Recently, Cervellati and Sunde (2011) have tested the hypothesis that the causal effect of life expectancy on income per capita growth is non-monotonic, and convincingly show that the inception of the demographic transition represents an important turning-point in the analysis of the sign of such causal relation. In more detail, they document the presence of a strong and robust positive causal effect of life expectancy on income per capita in *post-transitional countries* (in which it is also possible to observe a significantly negative effect of life expectancy on population growth). On the other hand, they notice a negative (although sometimes insignificant) causal effect of life expectancy on income per capita in *pre-transitional countries* (where the effect of life expectancy on population growth is generally positive). In sum, Cervellati and Sunde (2011)[13] show that longevity improvements can stimulate economic growth only if a country has already undergone the demographic transition from high to low rates of fertility and mortality. Otherwise, such improvements merely translate into greater population growth. Overall, their results are important because they provide a novel explanation for the generally mixed empirical evidence about the effects of life expectancy on income growth. As a matter of fact, their explanation, rather than focusing on the use of possibly different instrumentation strategies, is based instead on the key role played by the composition of the sample in terms of pre– vs. post–transitional countries as it is proved that the impact of life expectancy on income growth is more positive the larger the presence in the sample of

countries that, from the point of view of the demographic transition, can be defined as *"post-transitional"*.

After reviewing the main literature that has studied the relation between health and income (level and/or growth rate), in the next section of this chapter we derive our *"augmented Solow model"*, while we develop the econometric analysis in Sect. 6.3. In this section we present the estimates and show how unobserved heterogeneity can help explaining differences across countries. A discussion of the results along with a comparison with the contributions closest to ours is provided in Sect. 6.4. Section 6.5 concludes.

6.2 The Environment: A New *"Augmented Solow Model"*

Consider an economy where the rates of saving (s), population growth (n) and technological progress (g) are all exogenous. In this economy there are four rival inputs: physical capital (K), raw labor (L), human capital in the form of education (E), and human capital in the form of health (H). Following Barro (2013, Eq. 2, p. 352), we assume that production at time t takes the following Cobb-Douglas form:

$$Y_t = K_t^{\alpha} E_t^{\beta} H_t^{\gamma} (A_t L_t)^{1-\alpha-\beta-\gamma}, \quad 0 < \alpha, \beta, \gamma < 1, \quad 0 < \alpha + \beta + \gamma < 1 \tag{6.1}$$

Notice that in Eq. (6.1) we assume that there are decreasing returns to all capital ($0 < \alpha + \beta + \gamma < 1$), and that raw labor, the stock of physical capital, the stock of human capital in the form of education and the stock of human capital in the form of health are considered as four different inputs in the same aggregate production function. This means that output depends not only on *'conventional'* inputs (such as physical capital, raw labor, and human capital in the form of schooling),[14] but also on workers' health. Moreover, Eq. (6.1) assumes that technological progress (i.e., the growth over time of the level of technology, A_t) is labor-augmenting, and that the contribution to total GDP of raw labor, human capital in the

form of education and human capital in the form of health (as reflected, respectively, by the elasticities $(1-\alpha-\beta-\gamma)$, β, and γ) is potentially dissimilar across each other and different from that of physical capital, as well. For the sake of simplicity, the total of labor input (L) is also assumed to correspond to total population. The dynamics of the size of population and the level of technology are exogenous and obey, respectively to:

$$L_t = L_0 e^{nt} \tag{6.2}$$

$$A_t = A_0 e^{gt}. \tag{6.3}$$

The number of effective units of labor is $A_t L_t$, and grows at rate $(n+g)$. Physical capital, human capital in the form of education and human capital in the form of health are three reproducible factor inputs. The economy-wide budget constraint is:

$$Y_t = K_t^{\alpha} E_t^{\beta} H_t^{\gamma} (A_t L_t)^{1-\alpha-\beta-\gamma} = C_t + I_{E,t} + I_{H,t} + I_{K,t}. \tag{6.4}$$

Thus, the same production function applies to physical capital, education, health, and consumption: once produced, one unit of forgone consumption can be transformed costlessly into either one unit of physical capital or one unit of human capital in the form of schooling or one unit of human capital in the form of health.

After defining by:

$$k_t \equiv K_t / A_t L_t, \quad e_t \equiv E_t / A_t L_t, \quad h_t \equiv H_t / A_t L_t, \tag{6.5}$$

the variables K_t, E_t and H_t per unit of effective labor, it is possible to express the production function in intensive form as:

$$y_t \equiv Y_t / A_t L_t = k_t^{\alpha} e_t^{\beta} h_t^{\gamma}. \tag{6.6}$$

Let s_k, s_e and s_h be, respectively, the exogenous fractions of total income invested in physical capital, education and health, with $s \equiv s_k + s_e + s_h$

being the total saving rate of the economy. The evolution of the three capital stocks is determined as follows:

$$\dot{k}_t = s_k y_t - (n + g + \delta) k_t. \tag{6.7}$$

$$\dot{e}_t = s_e y_t - (n + g + d) e_t. \tag{6.8}$$

$$\dot{h}_t = s_h y_t - (n + g + d) h_t. \tag{6.9}$$

We continue to follow Barro (2013, p. 353) in assuming that the exogenous depreciation rate for physical capital ($\delta > 0$) differs from the exogenous depreciation rate for education and health ($d > 0$).

Eqs. (6.7), (6.8) and (6.9) imply that the economy converges to a steady state equilibrium (defined by $\dot{k}_t = \dot{e}_t = \dot{h}_t = 0$) in which:

$$h^* = \left[\frac{s_k^{\alpha} s_e^{\beta} s_h^{1-\alpha-\beta}}{(n + g + d)^{1-\alpha} (n + g + \delta)^{\alpha}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}}. \tag{6.10}$$

$$e^* = \left[\frac{s_k^{\alpha} s_h^{\gamma} s_e^{1-\alpha-\gamma}}{(n + g + d)^{1-\alpha} (n + g + \delta)^{\alpha}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}}. \tag{6.11}$$

$$k^* = \left[\frac{s_e^{\beta} s_h^{\gamma} s_k^{1-\beta-\gamma}}{(n + g + d)^{\beta+\gamma} (n + g + \delta)^{1-\beta-\gamma}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}}. \tag{6.12}$$

Substituting Eqs. (6.10), (6.11) and (6.12) into the production function in intensive form (Eq. 6.6) gives the steady state level of per capita income, $(Y_t/L_t)^*$:

$$\left(\frac{Y_t}{L_t}\right)^* = (k^*)^{\alpha} (e^*)^{\beta} (h^*)^{\gamma} A_t = \left[\frac{s_e^{\beta} s_h^{\gamma} s_k^{\alpha}}{(n+g+d)^{\beta+\gamma} (n+g+\delta)^{\alpha}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}} A_0 e^{gt} \tag{6.13}$$

This equation shows how in the steady state per capita income depends on the rates of investment in education, physical capital and health, the rate of population growth, the rate of technical change, and the depreciation rates for schooling/health and physical capital. After taking logs of both sides of Eq. (6.13), this equation can be transformed into:

$$\ln\left(\frac{Y_t}{L_t}\right)^* = \ln A_0 + gt - \left(\frac{\beta+\gamma}{1-\alpha-\beta-\gamma}\right)\ln(n+g+d) - \left(\frac{\alpha}{1-\alpha-\beta-\gamma}\right)\ln(n+g+\delta) + \left(\frac{\beta}{1-\alpha-\beta-\gamma}\right)\ln(s_e) + \left(\frac{\gamma}{1-\alpha-\beta-\gamma}\right)\ln(s_h) + \left(\frac{\alpha}{1-\alpha-\beta-\gamma}\right)\ln(s_k) \tag{6.14}$$

In order to make more explicit the role of health in determining the steady state level of per capita income, from Eq. (6.10) we first obtain:

$$\left(\frac{\gamma}{1-\alpha-\beta-\gamma}\right)\ln(s_h) = \left(\frac{\gamma}{1-\alpha-\beta}\right)\ln(h^*) - \frac{\alpha\gamma}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(s_k) - \frac{\beta\gamma}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(s_e) + \frac{\gamma(1-\alpha)}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(n+g+d) + \frac{\gamma\alpha}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(n+g+\delta)$$

Then, after plugging the last expression into Eq. (6.14), we finally obtain:

$$\ln\left(\frac{Y_t}{L_t}\right)^* = \ln A_0 + gt + \left(\frac{\beta}{1-\alpha-\beta}\right)\ln(s_e) + \left(\frac{\alpha}{1-\alpha-\beta}\right)\ln(s_k) - \left(\frac{\beta}{1-\alpha-\beta}\right)\ln(n+g+d) - \left(\frac{\alpha}{1-\alpha-\beta}\right)\ln(n+g+\delta) + \left(\frac{\gamma}{1-\alpha-\beta}\right)\ln(h^*) \tag{6.15}$$

Equation (6.15) yields an expression for the steady state level of per capita income as a function of (some of) the same variables already mentioned earlier and, more importantly, of the level of health in the population (h^*). In the next Section we implement empirically the theoretical model presented here.

6.3 Econometric Analysis

As pointed out in the previous section, the effect of health on real GDP is far from univocal. A large body of both theoretical and empirical literature shows a positive impact of health on economic growth, but for

rich countries, the existing empirical evidence is mixed (Hartwig, 2010). In this section, we revisit the question whether health capital formation stimulates the growth of real income in advanced countries.

Data. Our sample consists of 31 high income OECD countries along the period 1995–2010.[15] The data are from the Penn World Table 8.1 (PWT hereafter) and the World Bank (WB, hereafter).[16] The variables taken into account are real income, physical capital, population, education, public expenditure on health and life expectancy at birth. We measure the population growth rate as the average rate of growth of the working-age population, where working age is defined as 15 to 65. We measure s_k as the average share of real investment (including government investment) in real GDP, and we use the human capital index provided by PWT and the life expectancy at birth provided by the World Bank as proxies of s_e and s_h, respectively.[17] For simplicity, we assume $d = \delta$, i.e. human and physical capital have the same depreciation rate. Summary statistics are provided in Table 6.1, in which all variables are measured in logarithms.

Econometric strategy. In order to deal with the reverse causation between the level of real per capita GDP and country health status (see Weil, 2014; Tamakoshi and Hamori, 2015; Linden and Ray, 2017), we apply a Bivariate Finite Mixture Model (BFMM, hereafter; see Alfò and Trovato, 2004; Alfò et al., 2008; Lu et al., 2016; Ng and Mclachlan, 2014; Owen et al., 2009; Yu et al., 2014), which allows for parameter heterogeneity among countries with similar fundamentals. With regard to our research question, the advantage of this approach is that it allows to consider, as source of unobserved heterogeneity, the endogeneity between per capita income and health status. Moreover, through this estimation procedure we are able to perform a cluster analysis: we sort countries into groups based on the homogeneity of the conditional joint distribution of their income levels and life expectancies with respect to the estimated unobservable factors.

Following Linden and Ray (2017), therefore, we assume that real GDP levels and life expectancy are jointly correlated in some time points.

The BFMM allows to deal with non-trivial correlation structure. For instance, omitted covariates may affect *both* real GDP and aggregate health. It is well known that when responses are correlated (in our

Table 6.1 Summary statistics

Variable	Source	Obs.	Mean	Std. Dev.	Min	Max
Life expectancy	WB	620	78.53	2.80	67.54	83.59
HC index	PWT	620	3.23	0.32	2.07	3.73
Capital share	PWT	620	0.26	0.05	0.14	0.56
$(n + g + d)$	PWT	620	0.57	0.01	0.17	0.32
Public expenditure on health (% GDP)	WB	589	6.28	1.44	1.38	10.05
Real per capita GDP	PWT	620	34,497	12,412	9221	95,176

case, real GDP level and life expectancy), the univariate approach is less efficient than the multivariate one.[18]

Recalling Eq. (6.15), to check if the empirical model is affected by endogeneity between $ln(y)_{it}$ and $ln(h)_{it}$, we estimate the following two equations:

$$E\left(\ln(y)_{it} \mid \ln(h)_{it}, \epsilon_{it}\right) = \alpha + \beta_1 \ln(h)_{it} \tag{6.16}$$

$$E\left(\ln(h)_{it} \mid \ln(y)_{it}, \vartheta_{it}\right) = \delta + \beta_2 \ln(y)_{it} \tag{6.17}$$

In Eq. (6.16) we have a reverse regression in which life expectancy is the response and the level of per capita GDP is the covariate.

Both the estimated parameters β_1 and β_2 are positive and significant ($\hat{\beta}_1 = 1.236$ with s.e. $= 0.019$ and $\hat{\beta}_2 = 0.646$ with s.e. $= 0.009$, see Table 6.2).

In order to consider the process under investigation based on a multivariate joint density, we model per capita GDP level (y_{1it}) and life expectancy (y_{2it}) at time $t = 1, \ldots, T$ as a bivariate process. Vectors of outcome-specific p_{jt} covariates have been recorded for each country and will be denoted by $\mathbf{x}_{it}$ and $\mathbf{z}_{it}$. We consider the case where covariates differ across outcomes and are, respectively, $x_{it} = [ln(s_k)_{i,t}, ln(s_e)_{i,t}, ln(n + g + d)_{i,t}, ln(s_h)_{i,(t-1)}]$ and $z_{it} = [ln(y)_{i,(t-1)}]$.[19] Following the usual notation, for multivariate data, let $y_i = (y_{i1}, y_{i2})$ denote respectively the vector of observed per capita level of GDP and measure of health (i.e. life expectancy) for the *i-th* country, *i=1, ... ,n* in the analyzed time-window.

Table 6.2 Real per capita GDP and health

	β	Std. Err.	z	$P > \lvert z \rvert$	[95% Conf. Interval]	
Real per capita *GDP*						
Health	1.236	0.019	64.330	0.000	1.199	1.274
Constant	6.468	0.078	82.800	0.000	6.315	6.621
Health						
Real GDP	0.646	0.010	64.960	0.000	0.627	0.666
Constant	−3.648	0.117	−31.120	0.000	−3.878	−3.419

We can now write the empirical counterpart of Eq. (6.5) as:

$$E\left(\boldsymbol{y_i}|\boldsymbol{X}_i, \boldsymbol{Z}_i, \vartheta_{it}\right) = \begin{cases} E\left(\boldsymbol{y}_1|\boldsymbol{x}_i, \epsilon_{it}\right) = \alpha_i + \beta_1 \ln\left(se\right)_{i,t} + \beta_2 \ln\left(sk\right)_{i,t} + \beta_3 \ln\left(h\right)_{it} + \beta_4 \ln\left(ngd\right)_{it} \\ E\left(\boldsymbol{y}_2|\boldsymbol{z}_i, \vartheta_{it}\right) = \delta_i + \ln\left(y\right)_{i,t-1} \end{cases} \tag{6.18}$$

According to the previous assumptions, we estimate a linear model for the response variables $y_i = (y_{i1}, y_{i2})$, in which some covariates are missing, collinear or describe a non-linear relationship with GDP levels.

Notice that the intercept terms vary across countries in order to capture country-specific features, i.e. $\alpha_i = \ln\left(A_0 + gt\right) + u_{1i}$ and $\delta_i = \delta + u_{2i}$ are random intercepts in which u_{1i} and u_{2i} are specific random terms that follow any a priori distribution. The set of subjects and the outcome-specific random coefficients appear additively in the linear predictor. This assumption, however, can be relaxed by associating random parameters to some elements of the covariates' set, generalizing our specification to a random coefficient model. In other terms, the values $u_{ij} \in U$, with $i=1, \ldots, n$, for response $J = 1,2$, represent individual-specific features varying over the data set in an unknown way. According to Kiefer and Wolfowitz (1956), they can be treated as drawn from N i.i.d. random variables u_i with a common unknown density function $g(\cdot)$.

Given the assumption of conditionally independence and treating the u_i's as nuisance parameters and integrating them out, the corresponding likelihood function can be rewritten as follows:

$$L\left(\bullet\right) = \prod_{i=1}^{n} \int_{U} f_i\left(y_i|x_i, z_i, u_i\right) = \prod_{i=1}^{n} \int_{U} \left[\prod_{J} \prod_{t=1}^{T} y_{ijt}, x_{it}, z_{it}, u_{ij}\right] d\boldsymbol{G}\left(\boldsymbol{u}_i\right) \tag{6.19}$$

where u represents the support for $G(\boldsymbol{u})$, the distribution function of u_i. Model parameters can be estimated through the above marginal likelihood. In this context, the random component represents mean zero deviations from the fixed part, i.e. the country-specific latent effects u_i capture the country variability in the dynamic process of the *technological*

factor. Various alternative parametric specifications may be proposed for modeling random effect distribution. However, parametric specifications of the mixing distribution can be restrictive and are generally unverifiable.

As proved by Lindsay (1983a, b), the maximum likelihood estimator is concentrated on a support of cardinality at most equal to the number of distinct points in the analyzed sample.

Therefore, the integral of the likelihood may be approximated by a sum on a finite number K of locations:

$$L(\bullet) = \prod_{i=1}^{n} \left[\sum_{k=1}^{K} f_i\left(y_i | x_i, z_i, u_k\right) \pi_k \right] = \prod_{i=1}^{n} \sum_{k=1}^{K} f_{ik} \pi_k \qquad (6.20)$$

where f_{ik} is the response density distribution for the *k-th* component of the BFMM, where the intercept terms vary across countries in order to capture country-specific features.

Finally we assume that unobserved heterogeneity affects outcomes in correlated ways, i.e. the latent effects in the two regression equations are correlated.

Results. Table 6.3 reports the estimates for the OLS fixed effects model, for the *restricted* Solow model and for three alternative specifications of the BFMM. The results strongly support our *augmented* version of the Solow model, with education and health. Equation 6.15 shows that the *augmented* model predicts that the coefficients of s_k, s_e, s_h and $(n + g + d)$ sum to zero. The (implicit) estimated values for α, β and γ show that this restriction is not rejected by the data.

All three bivariate finite mixture models show a better global fit in comparison with the OLS model. This appears immediately from Fig. 6.1, in which we overlay the empirical density function for real per capita GDP (left panel) and for life expectancy (right panel), obtained via BFMM(1) (dash-dotted line) and OLS model (dashed line), to those corresponding to observed data (solid line).[20]

The estimates are all significant: the coefficients on s_k, i.e. the capital share, and on the sum of the rates of change in population and in technological progress plus depreciation $(n + g + d)$ are in line with the literature while the elasticity of the per capita GDP to life expectancy is

Table 6.3 Estimates

	OLS FE	Restricted model	BFMM(1)	BFMM(2)	BFMM(3)
Real per capita *GDP*					
s_e	0.675***	0.399***	0.177***	0.358***	0.120***
s_k	0.409***	0.318***	0.145***	0.197***	0.128***
$n+g+d$	−0.556***		−0.076***	−0.252***	0.205***
s_h	0.802***	0.943***	0.399***	0.458***	0.447***
Constant	8.162***	4.955***	3.625***	4.648***	6.499***
$\hat{\alpha}$		0.120***			
$\hat{\beta}$		0.150***			
$\hat{\gamma}$		0.354***			
Life expectancy					
Lagged Real per capita GDP			0.607***	0.447***	0.407***
Public exp. on health					0.023**
Population growth				3.400***	
Lagged life expectancy				0.362***	0.543***
Constant			−5.542***	−4.142***	−3.897***
σ^2			0.014	0.011	0.013
σ^2_{GDP}			0.015	0.011	0.016
$\sigma^2_{GDP,health}$			0.049	0.027	0.051
$\sigma_{GDP,\,health}$			−0.028	−0.029	−0.029
k			5	6	6
Log-likelihood			2187.14	2534.37	1076.05
Observations	620	620	3059	3059	1240

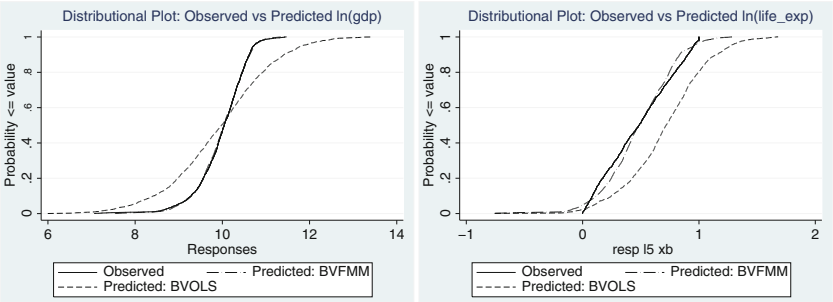

Fig. 6.1 Empirical density functions

0.399. The bottom half of Table 6.3 reports the estimates for the health equation: life expectancy is positively and significantly associated to an increase in the real per capita GDP, with an elasticity of 0.607. A positive impact of the population growth rate (0.407) and the lagged value of life expectancy (0.543) are found in the BFMM(2), in which also the public expenditure on health is slightly positively associated (0.023) to the aggregate level of health of a country. The BFMM(3), which has a richer specification of the equation for life expectancy, presents similar estimates but also an unpleasant positive parameter of the depreciation term $(n + g + d)$.

As previously mentioned, a useful by-product of the BFMM approach is that it allows clustering of countries on the basis of the posterior probabilities estimates $\hat{u_{ij}}$. Notice that each component is characterized by homogeneous values of estimated latent effects, i.e. conditionally on the observed covariates, countries belonging to the same cluster show a similar *structure*, at least in the long-run. The latent variables, therefore, may capture the effect of missing covariates, i.e. those factors not included in the *augmented* Solow model. Using AIC, CAIC and BIC criteria, BFMM(1) identifies 5 clusters of countries while 6 clusters are obtained via BFMM(2) and BFMM(3). Clusters' composition does not change significantly moving across different specifications. In the following, for the sake of brevity, we focus on the BFMM(1).[21]

Estimated locations are shown in Table 6.4, while corresponding clusters are reported in Table 6.5. Finally, Fig. 6.2 provides the rootogram

Table 6.4 BFMM(1), locations and probabilities

Cluster	Real per capita GDP		Life expectancy		
k	*loc.*	*s.e.*	*loc.*	*s.e.*	Prob.
1	−0.276	0.024	0.498	0.041	0.097
2	0.055	0.022	−0.107	0.040	0.322
3	−0.104	0.023	0.187	0.040	0.194
4	−0.008	0.023	0.019	0.040	0.194
5	0.158	0.024	−0.277	0.030	0.195

Note: *k*, number of mixture components selected by penalized criteria; *loc.*, locations; *s.e.*, locations' standard errors. Prob., prior probability of belonging to that local area. The probabilities are for both equations in the bivariate model

of the posterior probability, which shows that the mixture components are well separated one from each other, i.e. no significant mass can be found in the middle of the unit interval (no overlapping components).

Looking at the first column of Table 6.4, we see that the random terms positively affect the level of the real per capita GDP of the countries belonging to clusters 2 (0.055) and 5 (0.158), which are also those with the higher average level of per capita GDP (10.11 and 10.43, respectively). Symmetrically, the same partition emerges when we move to the third column of the Table. In fact, the model suggests that in clusters 2 and 4 can be found some unobserved factors, not directly captured by the model, that are harmful for health, reducing life expectancy. The opposite happens for countries grouped in the remaining clusters.

6.4 Discussion

Our estimates show that, at least for the sample of OECD countries, population's health positively and significantly affects the level of per capita income. This finding is consistent with our theoretical model, in which the typical capital *"dilution effect"*, due to the increase in population induced by a better aggregate health, is offset by the increase in productivity arising from healthier workers.

Apparently, this result is in contrast with that of Acemoglu and Johnson (2007). We reckon that this discrepancy is due to the following reasons.

Table 6.5 Clusters

	Country	Real per capita GDP	Life expectancy
Cluster 1			
	Poland	9.25	72.61
	Portugal	9.47	72.72
	Republic of Korea	9.03	69.49
	Mean	9.25	71.61
Cluster 2			
	Australia	10.18	76.16
	Austria	10.04	74.85
	Belgium	10.09	75.18
	Canada	10.22	76.55
	France	10.06	76.03
	Germany	10.06	74.79
	Iceland	10.23	77.75
	New Zealand	9.97	75.40
	Sweden	10.18	77.42
	United Kingdom	10.05	75.49
	Mean	10.11	75.96
Cluster 3			
	Estonia	9.64	71.78
	Greece	9.67	75.35
	Hungary	9.48	70.89
	Japan	9.94	77.52
	Slovakia	9.79	73.77
	Spain	9.74	76.35
	Mean	9.71	74.28
Cluster 4			
	Czech Republic	10.06	75.40
	Finland	10.01	74.69
	Ireland	9.81	74.69
	Israel	9.98	76.34
	Italy	9.95	76.18
	Slovenia	10.07	76.68
	Mean	9.98	75.66
Cluster 5			
	Denmark	10.17	75.34
	Luxembourg	10.63	74.57
	Netherlands	10.17	76.70
	Norway	10.24	76.91
	Switzerland	10.45	77.14
	United States	*10.43*	*74.54*
	Mean	10.35	75.87

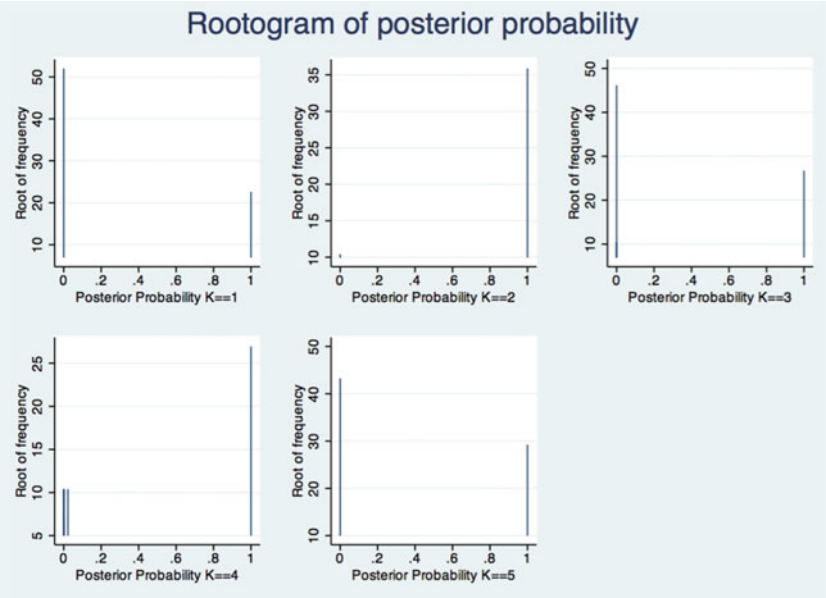

Fig. 6.2 Rootogram

Differently from them, we have restricted our attention to a sample of high income countries (for which they find non-significant estimates) and we have followed an alternative econometric route to deal with the potential endogeneity between life expectancy and per capita income.

Our evidence, instead, seems to be more in accord with Cervellati and Sunde (2011): by moving from a radically different perspective, they also find that the effect of life expectancy on income per capita is positive for the high-income countries (see Table 10 in their Online Appendix). Their argument, however, relies on demographic issues. Although we do not directly tackle such issues, BFMM(2) and BFMM(3) include the population growth rate and/or the lagged value of life expectancy (as a proxy for aggregate health) to account for demographic forces.

Our classification shows that for the group of countries with the highest average income, namely Denmark, Luxembourg, the Netherlands, Norway, Switzerland and the United States, the random component strongly affects the level of per capita GDP, while the effect is strongly

negative for the group with the lowest average income. This difference can be explained as the consequence of the gap in terms of aggregate efficiency, which is actually due to differences in institutional factors. To this extent, along with Mankiw et al. (1992) we may argue that fiscal, education and innovation policies together with political stability will end up among the ultimate determinants of cross-country differences in income. Richest countries are also characterized by having unobservable factors that are harmful for the level of population's health. The strength of these factors, however, declines when we move from BFMM(1) towards the less parsimonious specifications of the equation for aggregate health, provided by BFMM(2) and BFMM(3).

6.5 Concluding Remarks

Following Mankiw et al. (1992), in this chapter we have argued that international differences in income per capita are best understood using a new *augmented* Solow growth model in which output is produced from physical capital, raw labor, human capital in the form of education, and human capital in the form of health. One of the predictions of this model is that the long-run level of real per capita GDP of a country is positively affected by the level of health of its population. We test this prediction by using data from the sample of OECD countries, along the period 1995–2010. As it is standard in this literature, we use life expectancy at birth as a proxy for population health. To deal with the endogeneity problem between health and income we estimate a Bivariate Finite Mixture Model. The empirical analysis corroborates our theoretical finding.

Interestingly, our semi-parametric approach allows countries classification. Despite public expenditure on health positively affects per capita income, through its effect on life expectancy, cluster membership does not change significantly when this kind of public intervention is included as a covariate in the equation for life expectancy. Our estimates also indicate that the richer the country, the stronger the role of unobservable factors in explaining the level of per capita income. Remarkably, the richest countries in the sample are also characterized by having unobservable factors that are harmful for the level of population's health. Our Bivariate

Finite Mixture approach is able to measure local variation in the observed data. Consequently, it makes our *augmented Solow model* with human capital accumulation (via education) and health—conditionally on heterogeneous groups—a useful tool to understand the differences among countries in long-run per capita income.

Notes

1. Recent surveys of the literature that analyzes some aspects of the relationship among health, income, and economic growth/development can be found in Weil (2014) and in Bloom et al. (2018).
2. As an example, using GMM-based panel data methods, Suhrcke and Urban (2010) find a causal negative effect of cardiovascular mortality on subsequent economic growth in high-income countries for the time span 1960–2000. Hyclak et al. (2016) show that across the OECD countries, most of the correlation between cardiovascular mortality and income per capita arises from variations within the Eastern European countries.
3. *"... We do not observe human capital in the form of health directly, but presumably it is some combination of ability to work hard, cognitive function, and possibly other aspects of health ..."* (Weil, 2007, p. 1268).
4. Examining the historical evolution of body size and calorie consumption in the United Kingdom, Fogel (1997) concludes, among other things, that over the period 1780–1980 better nutrition raised labor input per work-aged adult by a factor of 1.96.
5. Hartwig (2010, Table 1, p. 315) reports the results of fourteen studies that analyze the relation between health and macroeconomic growth, using different measures of health.
6. In this chapter we are interested to the channel that goes from health to income. Leading proponents of the view that there is a large structural effect of health on income are also Sachs (2001) and Fogel (1997).
7. Aghion et al. (2011) document that growth of life expectancy is strongly negatively correlated with initial life expectancy across countries over both the 1940–1980 and 1960–2000 periods. In other words, there has been a massive process of world-wide convergence in life expectancy in the last few decades (see also Becker et al., 2005). Indeed, it is intuitive that in countries where life expectancy is initially high (due to already well-developed, highly efficient, and well-equipped health-care systems),

further improvements in population's health can only be achieved at extremely large health-investment costs. Moreover, if such costs are so big to overwhelm the potential economic benefits related to further health improvements, then the ultimate consequence of the amelioration of a population's health conditions can definitely be a worsening of the general economic performance (i.e., the GDP growth rate of a country). This is consistent with Bhargava et al. (2001)'s finding that the effect of health on the GDP growth rate is larger in developing countries than in developed countries. For example, they estimate that for the poorest countries a 1% change in the adult survival rate is associated with an approximate 0.05% increase in the economic growth rate. The parameter estimates imply large positive effects of the adult survival rate on economic growth for countries such as Burkina Faso, Burundi, the Central African Republic, India, Ivory Coast, and Nigeria. For highly developed countries, such as USA, France and Switzerland, the estimated effect of adult survival rate on economic growth is, instead, found to be negative.

8. Consistently with Lorentzen et al. (2008)—who use exogenous variation across countries, such as climatic factors, geographical features, or disease indices, as instruments for differences in life expectancy—, Aghion et al. (2011) find that the initial level of life expectancy has a positive impact on the average rates of investment in physical and human capital, while both the growth and the initial level of life expectancy help reducing fertility. Hence, health variables are an important determinant of economic growth. Doppelhofer et al. (2004), using Bayesian averaging models techniques, show that the initial level of life expectancy is certainly one of the most robust causes of economic growth.
9. Other empirical results, however, contradict such finding. Controlling for initial health, Hansen (2014) finds no significant effect of the change (or level) of longevity on GDP per capita among U.S. states. Hansen and Lönstrup (2015) show that, when implementing a three-point panel (with international data from 1900, 1940, and 1980) and controlling for initial health and country fixed effects, increased longevity appears to play a negative role on GDP per capita.
10. Concerning the multifaceted relation among survival, life expectancy, mortality, fertility, population growth, and ultimately income, papers that find a positive impact of life expectancy on income (abstracting, however, from any possibly-related change in fertility) include de la Croix and Licandro (1999), Kalemli-Ozcan et al. (2000), Blackburn and

Cipriani (2002), Boucekkine et al. (2002, 2003), Lagerlöf (2003), and Bar and Leukhina (2010b), among many others. Galor and Moav (2002) study the role of survival and natural selection in the so-called fertility transition. Papers in which greater life expectancy causally implies a fall in fertility include Kalemli-Ozcan (2002, 2003), Boldrin and Jones (2002), Soares (2005), Strulik (2008), and Bar and Leukhina (2010a). Whether reductions in mortality cause fertility reductions or not is still a highly debated issue in the empirical literature.

11. See also Buiter (1988).
12. See, among others, Bloom and Williamson (1998), Bloom et al. (2003b), Mason et al. (2016) and Bloom et al. (2017).
13. See also Cervellati and Sunde (2015).
14. See, as a notable example, Mankiw et al. (1992, Eq. 8, p. 416).
15. The countries in the sample are: Australia, Austria, Belgium, Canada, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Iceland, Ireland, Israel, Italy, Japan, Luxembourg, the Netherlands, New Zealand, Norway, Poland, Portugal, Republic of Korea, Slovakia, Slovenia, Spain, Sweden, Switzerland, United Kingdom, the United States.
16. For more information on the PWT see: http://www.rug.nl/ggdc/productivity/pwt/.
17. A lot of empirical literature uses, as a proxy for health-status, the health expenditure (for example Hartwig, 2010 and Tamakoshi and Hamorri, 2015); others measure it using life expectancy (Linden and Ray, 2017). In our estimates, we use the public expenditure on health and the population growth rate as regressors in the equation for our measure of health, i.e. life expectancy.
18. See Zellner (1962) and Davidson and Mackinnon (1963) for a detailed discussion of this topic in the SUR context.
19. We also run two alternative specifications of the Bivariate Finite Mixture model: BFMM(2), in which $z_{it} = [ln(y)_{i,\,(t-1)}, life\ expectancy_{i,\,t-1}, n_t]$, and BFMM(3) in which $z_{it} = [ln(y)_{i,\,(t-1)}, life\ expectancy_{i,\,t-1}, ln(public\ exp\ .\ on\ health)_{i,\,t}]$.
20. Similar pictures can be obtained by overlaying the empirical density functions that originate from BFMM(2) and BFMM(3). Notice that between the alternative models estimated, BFMM(1) is the one that provides the most accurate separation of the estimated locations.

21. Details on BFMM(2) and BFMM(3) not presented in the chapter are available upon request. Notice that, while all the clusters originated by BFMM(1) pass the Shapiro–Wilk test for residuals' normality, this is not true for BFMM(2) and BFMM(3). BFMM(1) and BFMM(2) show a similar global fit and produce a very similar classification.

    In particular, by moving from the former to the latter, (1) we observe a merge between BFMM(1)' groups 1 and 2 into BFMM(2)'s group 1; (2) BFMM(1) group 5 splits into two groups, one of which contains only Luxembourg and (3) BFMM(1)'s group 4 moves into BFMM(2)'s group 5.

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7

Health Spending, Education and Endogenous Demographics in an OLG Model

Giam Pietro Cipriani and Tamara Fioroni

7.1 Introduction

Health care costs have been rising almost everywhere in the last few decades. In the United States the health sector is, today, a $3 trillion industry accounting for nearly 18% of GDP, up from 14 per cent in 2000 and 5 per cent in 1960. This is well above the average of the OECD countries, which is still about 9 per cent of GDP, with the group of high income countries (including Germany, France, Japan and Canada) spending a higher fraction, around 11 per cent, than the low income countries (OECD, 2017). In particular, public health expenditure as a fraction of total health expenditure has increased over the years and

G. P. Cipriani
Department of Economics and IZA, University of Verona, Verona, Italy
e-mail: giampietro.cipriani@univr.it

T. Fioroni (✉)
Department of Economics, University of Verona, Verona, Italy
e-mail: tamara.fioroni@univr.it

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almost everywhere government schemes or compulsory health insurance constitute the main health care financing arrangements, accounting for almost three-quarters of all health care spending on average, and for 15 per cent of government expenditure.

In the same period, life expectancy has dramatically increased. For instance, in the OECD countries it has increased by over ten years on average since 1970. This demographic change has taken place almost everywhere. In fact, world life expectancy at birth rose by 3.6 years between 2000–2005 and 2010–2015, or from 67.2 to 70.8 years, and it is projected to rise to 77 years in 2045–2050 (United Nations, 2017). In general, both developed and developing countries are experiencing increases in life expectancy: in the second half of the last century, developed regions experienced an increase of more than 10 years, whereas developing regions experienced an even more remarkable increase of about 20 years. A large literature has studied the effects of health spending on longevity. In general, countries with higher health spending have better health outcomes and tend to have a longer life expectancy even though, clearly, the efficient use of health resources is also important and must be taken into account. A recent analysis (OECD, 2017) on the relative contribution of factors within and beyond the health system to life expectancy gains between 1995 and 2015 in all 35 OECD countries, using panel data from OECD Health Statistics and the World Bank, estimates that a 10 per cent increase in health expenditures is associated with an increase in life expectancy by 3.5 months. Considering that health spending actually grew by 98 per cent from 1990 to 2010 it is easy to see that health spending was the major determinant to gains in life expectancy over the last two decades, followed by education and income (Fig. 7.1).

Together with life expectancy, another important demographic change has been, of course, the fall in fertility. In all European countries fertility is currently below the replacement level and, in most cases, it has been so for several decades. In fact, 46 per cent of the world's population lives in countries with a fertility level below 2.1 births per woman and this is expected to raise to 69 per cent by 2050 (United Nations, 2017). Globally, the total fertility rate (TFR) has fallen from 5 children per woman in 1950 to about 2.5 today. Much of the fertility decline in developed countries

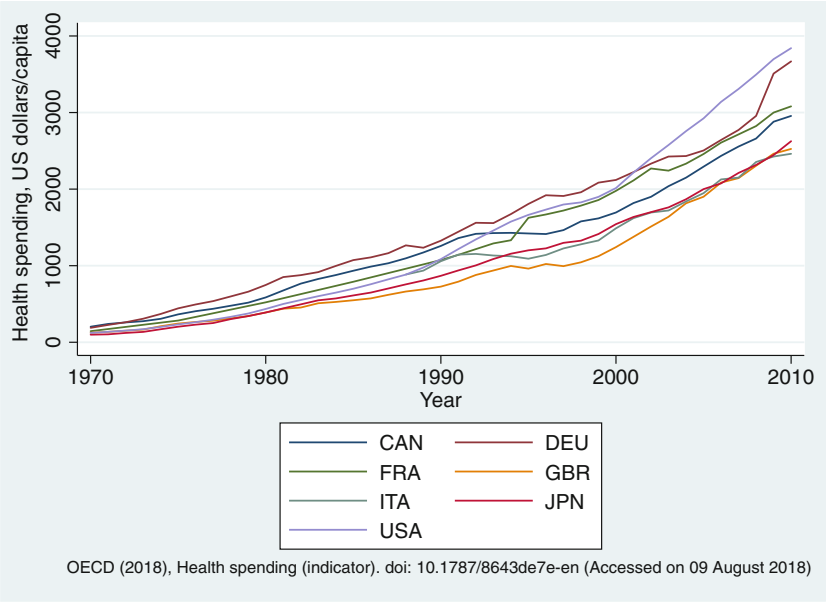

Fig. 7.1 Health spending (1970–2010)

began in the late nineteenth or early twentieth centuries and by 1950 this number was already 2.8, whereas in the developing countries this decline has been rather recent and dramatic, with the TFR falling from 6 to 2.7 in the second half of the last century (Fig. 7.2).

Falling fertility and increasing longevity determine a process of population aging which poses much stress on pension systems everywhere. In fact, in most countries the number of people older than 60 is growing rapidly both in absolute number and as a share of the total population. Whilst in developing countries this share is now 10 per cent but expected to double by the middle of this century, the share of 60+ in most developed countries is above 20 per cent already, with a peak of 30 per cent for Japan. This situation has induced many governments to enact reforms. These reforms seek to ensure the sustainability of the pay-as-you-go system by various measures that modify the contribution or the benefit rate and the retirement age. For example, in the past ten years, more than half of the OECD countries have reformed their pension policies in order

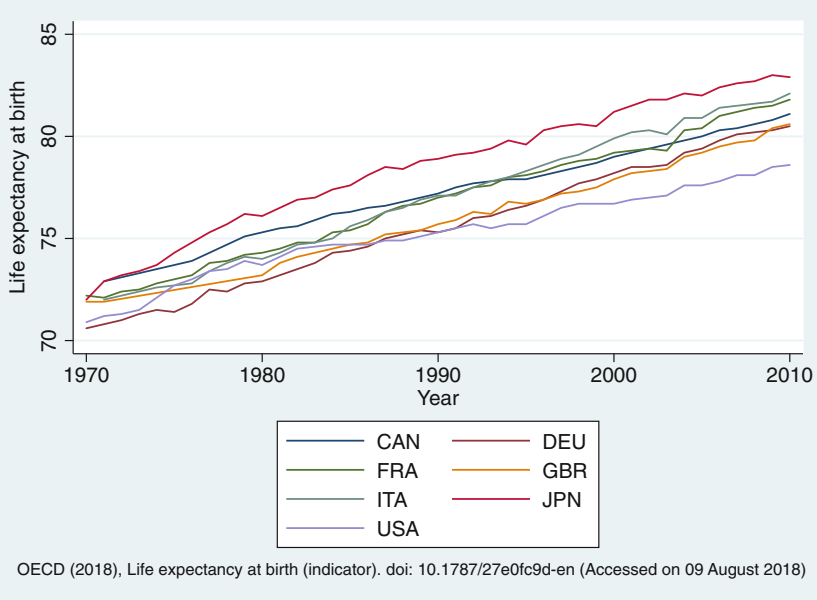

Fig. 7.2 Life expectancy at birth (1970–2010)

to extend the working lives and many countries, in the same period, have increased taxes and contributions for the financing of public pensions. Also, pension benefits were, or are planned to be, changed in 12 OECD countries (OECD, 2017).

In this chapter we build a model to study these issues. Our setting allows for a fully endogenous aging population, where fertility is chosen by optimizing agents in an overlapping generations framework. Individuals live for two periods with certainty and face a probability of surviving for an extra period. This survival probability depends, with decreasing returns, on public health expenditure. In particular, the government collects resources through income tax and allocates these resources between health spending and social security benefits. Individuals can invest resources in their children's human capital and therefore face the usual quality-quantity trade-off with respect to their fertility choices (Fig. 7.3).

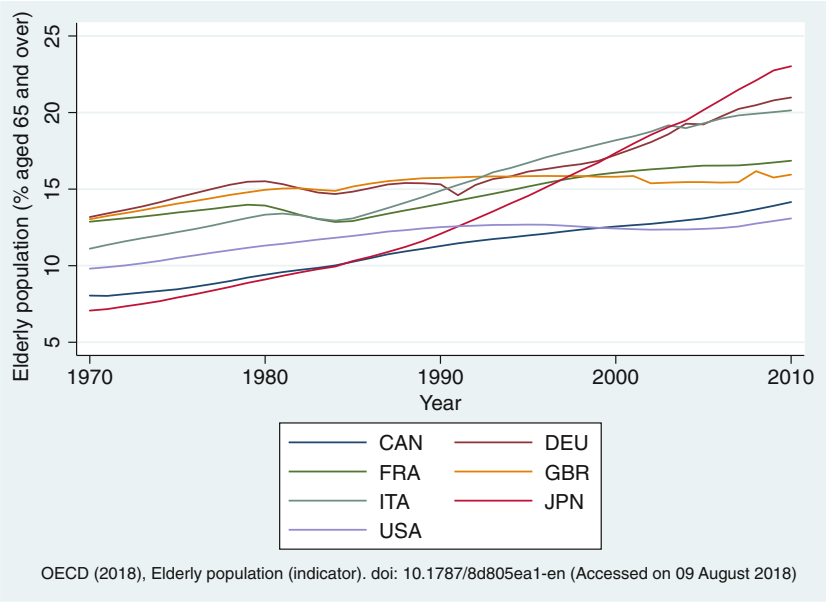

Fig. 7.3 Elderly population (1970–2010)

According to the existing literature (see for example de la Croix and Doepke, 2003; de la Croix and Doepke, 2004; Fioroni, 2010; Fiaschi and Fioroni, 2017) we show that there is a threshold level of income under which parents do not invest in their children's education, fertility is high and life expectancy is low. If income is above this threshold, parents begin to invest in their children's education and the quantity-quality trade-off starts operating and thus fertility decreases with respect to income. Therefore, depending on the initial level of income, different growth patterns may arise. If the initial level of income is very low, the economy is trapped in a low steady state with high fertility, low life expectancy and no private education. At intermediate levels of income multiple steady states can arise and finally, when the initial level of income is sufficiently high, the economy converges to a steady state characterized by a high level of education, high life expectancy and low fertility. We focus on the impact of government policies on these possible outcomes. In particular, we analyze two alternative policies: (a) a change in the income tax rate

and (b) a change in the allocation of public spending between social security benefits and health expenditures, without varying the tax rate. Regarding the first policy, we find that an increase in income tax has a non-monotonic impact on income. In particular, at low income levels, a higher tax rate, as long as it remains below a certain threshold, has a positive effect on economic growth, whereas at high levels of income, it always has a negative impact. The underlying intuition is that a higher income tax affects the evolution of income through different channels, leading to opposing effects whose extent depends on the initial level of income. In particular, a higher tax rate reduces disposable income and therefore has a negative impact on savings and education. Also, it has a direct positive impact on pension benefits leading to a negative displacement effect on savings. Finally, it also increases longevity, thus positively affecting savings and education and negatively affecting fertility. The overall effect of an increase in income tax, therefore, depends on the initial level of income and longevity. When they are very low, the decreasing returns of health expenditure imply a strong positive impact of a higher tax on longevity, leading to a positive impact on economic growth, as long as the increase in the tax rate is not too high. At high income levels, instead, longevity is very high, and therefore an increase in health spending has a weaker impact on longevity, and therefore the negative effect of a higher income tax rate prevails.

As far as the second policy is concerned, the model shows that an increase in the fraction of public spending devoted to health always increases income, thus ensuring a higher steady state in the low equilibrium or a transition to the high income equilibrium. Intuitively, an increase in the fraction of public spending devoted to health increases education and savings because of a higher longevity and reduces social security benefits, leading to higher savings and a lower number of children. These effects reinforce each other to increase per capita income. The overall effect on pension benefits is therefore ambiguous, given that the direct negative impact is ameliorated via the general equilibrium effect on income.

In the next section we give a brief review of the literature. Then, in Sect. 7.3 we set up the model and derive the equilibrium. In Sect. 7.4

we study government policies and the subsequent section is devoted to a simulation. A final section briefly presents the conclusions from this chapter.

7.2 Literature Review

There is a vast empirical literature on the relationship between health spending and longevity and also on the nexus between fertility and pensions.

On the first issue, the evidence suggests that the effects of health expenditures on longevity vary between countries due to different population characteristics and economic factors. However, in general health expenditures are found to be positively related to life expectancy in developed countries, whilst the relationship is less certain for developing countries. For a recent analysis of this heterogeneous effects see Obrizan and Wehby (2018) who find that the largest returns from health spending are in countries with lower life expectancy (and particularly for males). Interestingly, they also find that the effect of other variables (such as clean water and sanitation, labor force participation, urban population) was consistent with the previous literature, but much less important than health expenditure. Differentiating the effects of public and private health expenditure, Aísa et al. (2014) find that although the effect of aggregate health expenditure is not conclusive, public health expenditure, like in our model, always significantly affects longevity, even though the effect on longevity is lower after a certain level of health expenditure.

On the theoretical side, Chakraborty (2004) models endogenous lifetime through public investments in health into a standard OLG model. In his model, high mortality discourage savings and investment in education because individuals discount the future more heavily and poverty traps might emerge, since if the future stock of capital is low, future health and economic outcomes will also be poor. Similarly to his setting, we also find that if technological efficiency (represented by the shift parameter A in the production function) differs, one could have large and persistent differences in output per worker and longevity. However, differently from his approach, in our model we also include fertility choices so

that population aging is fully endogenous. Since Chakraborty's seminal paper, dozens of theoretical articles have followed the same approach in modeling life expectancy. Among these, recently Fanti and Gori (2014) also added fertility choice, thus creating a framework where population aging is fully endogenous. With respect to their approach, we do not consider child policies but we include a quality-quantity trade-off in the parental optimization problems in the form of human capital investment. Like in Chakraborty and Das (2005) and Fanti and Gori (2014) we obtain multiple development regimes, which depend on the initial conditions but also on government policies.

Concerning the second issue, there exists a well-established empirical and theoretical literature on fertility and pensions, which, on the one hand, has studied the mechanism by which a public pension system affects fertility, and on the other hand, the sustainability of a pension system in the presence of a declining population. For excellent overviews of this large literature, together with an empirical analysis and a theoretical model in the spirit of the seminal work by Cigno (1993), see Cigno and Werding (2007) and Fenge and Scheubel (2017). In summary, whilst some studies rely on cross-country variation (e.g. Ehrlich and Zhong, 1998; Boldrin and Montes, 2005) and others focus on specific countries or exogenous changes within a specific pension system (e.g. Cigno and Rosati, 1992), then all find that fertility is endogenous and jointly determined with saving and a more generous pension system depresses fertility and saving. The most recent developments in this theoretical literature have studied overlapping generations models with pension policies in aging economies (see Cipriani, 2014), also in models that consider government policies on the retirement age, like Cipriani and Pascucci (2018). In this chapter, however, to keep the model tractable we do not include retirement's choice and assume that all individuals retire at the end of the second period. Also, we study only the case of a pay-as-you-go pension system with defined contribution. There are many other papers, of course, that compare and contrast the consequences of different pension systems and investigate how institutional factors and behavioral responses may influence the impact of an increase in longevity or a fall in fertility on capital accumulation and income per capita.

Dedry et al. (2017), for instance, is an interesting recent example of this literature which shows that both the type of aging, i.e. declining fertility or increasing longevity, and the characteristics of pension system—in their case defined contributions versus defined benefits—affect welfare. Differently from these contributions, we set up a model where the government policy has two objectives: sustaining consumption in the old age by a pension system and investing in public health, which, based on the aforementioned empirical evidence, we assume has a positive effect on longevity.

In fact, relatively few theoretical papers, combine the health spending and endogenous longevity mechanism with the social security and endogenous fertility literature. Among these, the most recent and novel paper is Yew and Zhang (2018), which investigates health spending and social security in a dynastic model with endogenous fertility and longevity, with two-sided altruism like Blackburn and Cipriani (2005). They consider the length of old age as increasing in private health spending in middle age and old age and derive the socially optimal social security and health subsidy. However, there is no education and human capital in their model. Cipriani and Makris (2012), among others, consider a model with aging, education and PAYG pensions, however fertility is considered as exogenous and longevity depends directly on average human capital. In fact, we argue that the inclusion of education and human capital in an overlapping generations model with public health spending, social security and fully endogenous demographics is novel in its own right.

7.3 The Model

We consider an economy characterized by overlapping generations of people who potentially live for three periods, perfectly competitive firms and a government. Agents are identical in every aspect and each adult individual is endowed with h_t units of human capital and has a probability of surviving for a third period. They only make decisions in adulthood, choosing how many children to have, the investment in education for each child and their own consumption.

Let N_t denote the number of adults in period t, and n_t the number of children per adult. The size of adult population in the next period is, therefore, given by $N_{t+1} = n_t N_t$.

7.3.1 Consumers

In the first period of their life agents are children, acquire education and make no decisions; in adulthood individuals work full time, save, raise their offspring and invest in their children's education. Though certain to live through childhood and adulthood, agents are, however, subject to a probability p_t of surviving to old age. If they survive to the third period, agents consume their savings and benefit from a state-funded PAYG pension scheme.

Preferences of an adult agent (born in period $t-1$) are defined over consumption in adult age, c_t, the number of children n_t, the human capital of each child h_{t+1}, and, if they survive to the third period, consumption in old age c_{t+1}. Thus, their expected utility function is given by:

$$U^t = \ln c_t + \gamma \ln n_t h_{t+1} + \beta p_t \ln c_{t+1}, \tag{7.1}$$

where $\beta \in (0,1)$ is the weight attached to consumption in old age and $\gamma > 0$ captures the weight given to quantity and quality of children.

The individuals probability of surviving beyond the second period depends on public health expenditure G_t and is given by a non-decreasing concave function (see Bhattacharya and Qiao, 2007; Chakraborty, 2004):

$$p_t = p(G_t), \tag{7.2}$$

where $p'(G_t) > 0$, $p''(G_t) < 0$, $p(0) = 0$ and $\lim_{G\to\infty} p(G_t) = \bar{p}$.

Human capital of children in period $t+1$ is an increasing, strictly concave function of the investment in education e_t made by their parents, that is:

$$h_{t+1} = (1 + e_t)^{\delta}, \tag{7.3}$$

where $\delta \in (0,1)$. The assumption that when $e_t = 0$, $h_{t+1} = 1$ implies that nature equips each child with one efficiency unit of human capital. This means that if parents do not invest in their children's education, they will go on to supply unskilled labor in adulthood (see for example Hazan and Berdugo, 2002; Moav, 2005).

In the adult age, parents allocate their wage between consumption c_t, saving s_t, raising their children n_t, spending on children's education e_t and paying a tax τ. Thus, the budget constraint of an adult agent in period t is:

$$c_t = (1 - \tau - zn_t)w_t h_t - e_t n_t - s_t, \tag{7.4}$$

where $0 < z < 1$ is the fraction of the parental wage required to raise each child and $n_t \leq (1 - \tau)/z$. In the third period, agents consume their savings entirely and receive a pension benefit b_{t+1}. Thus the budget constraint in old age is given by:

$$c_{t+1} = \frac{R_{t+1}s_t}{p_t} + b_{t+1} \tag{7.5}$$

where R_{t+1}/p_t is the rate of return on savings, given that the savings of agents that do not survive to old age are redistributed to the surviving ones. Note that, for the sake of simplicity, we do not consider capital income taxation (see for example Bhattacharya and Qiao, 2007; Feldstein, 1985; Fenge and Scheubel, 2017; Cipriani, 2014; Fanti and Gori, 2008).

7.3.2 Production

Production occurs according to a constant returns to scale technology using physical capital and skilled labor as inputs. Assuming a Cobb-Douglas production function, output produced at time t is:

$$Y_t = AK_t^{\alpha} H_t^{1-\alpha}, \tag{7.6}$$

where $\alpha \in (0,1)$, $A > 0$ is the level of technology, K_t is the aggregate physical capital, H_t is the effective labor at time t, i.e. $H_t = h_t N_t$.

Firms operate in a perfectly competitive market. In each period t, firms choose the level of physical capital K_t and the efficiency units of labor H_t so as to maximize profits. Thus the wage rate, w_t, and the rate of return to capital, r_t, are given by:

$$w_t = A(1-\alpha)\left(\frac{k_t}{h_t}\right)^{\alpha} = (1-\alpha)\frac{y_t}{h_t}, \tag{7.7}$$

and

$$r_t = \alpha A\left(\frac{k_t}{h_t}\right)^{\alpha-1} = \alpha\frac{y_t}{k_t}, \tag{7.8}$$

where $y_t = Y_t/N_t$ and $k_t = K_t/N_t$.

The equilibrium condition in the capital market, under the assumption that physical capital depreciates completely after one period, is:

$$K_{t+1} = N_t s_t. \tag{7.9}$$

Thus, given that $N_{t+1} = n_t N_t$, per worker physical capital is:

$$k_{t+1} = \frac{s_t}{n_t} \tag{7.10}$$

7.3.3 Government

We assume that the government, in every period, operates under a balanced budget rule. Thus, the revenues from labor income taxation $\tau w_{t+1} h_{t+1}$ are used to finance public health spending G_{t+1} and retirement pensions b_{t+1} for those who survive to old age. The government budget constraint in period $t+1$ is therefore:

$$\tau w_{t+1} h_{t+1} N_{t+1} = G_{t+1} N_{t+1} + b_{t+1} p_t N_t. \tag{7.11}$$

We suppose that the government devotes a fraction $0 < \lambda < 1$ of tax revenues to investment in public expenditure and $1 - \lambda$ to the social security benefits, thus the internal allocation of government expenditure is:

$$G_{t+1} = \lambda \tau w_{t+1} h_{t+1}, \tag{7.12}$$

and

$$b_{t+1} = \frac{(1-\lambda)\tau w_{t+1} h_{t+1} n_t}{p_t} \tag{7.13}$$

Given Eqs. (7.7) and (7.12), the probability of surviving to the third period in Eq. (7.2) is, in equilibrium, a function of per capita income:

$$p_t = p(\lambda \tau (1-\alpha) y_t) = p(y_t). \tag{7.14}$$

7.3.4 Optimization

Each household chooses n_t, e_t and s_t so as to maximize the utility function (7.1) subject to (7.4) and (7.5) taking as given the wage, interest rate, pension benefit, public health spending and the tax rate. From Eqs. (7.7) and (7.8), after substituting for b_{t+1} from Eq. (7.13), an interior solution for the optimal level of education arises only if parents' income is sufficiently high, i.e. $y_t > \hat{y}$ (see, for example, de la Croix and Doepke, 2004; Fioroni, 2010; Fiaschi and Fioroni, 2017, 2019). Thus, first-order conditions imply:

$$e_t = \begin{cases} 0 & \text{if } y_t \leq \hat{y}, \\ \dfrac{\delta z (1-\alpha) y_t - 1}{1-\delta} & \text{if } y_t > \hat{y}, \end{cases} \tag{7.15}$$

$$n_t = \begin{cases} \dfrac{\alpha\gamma(1-\tau)y_t}{\alpha(1+\gamma+\beta p(y_t))zy_t - \gamma\tau(1-\lambda)k_{t+1}} & \text{if } y_t \leq \hat{y}, \\ \dfrac{\alpha\gamma(1-\tau)(1-\delta)(1-\alpha)y_t}{\left[\alpha(1+\gamma+\beta p(y_t))[z(1-\alpha)y_t - 1] \right. \\ \left. -\gamma\tau(1-\lambda)(1-\delta)(1-\alpha)k_{t+1}\right]} & \text{if } y_t > \hat{y}, \end{cases} \tag{7.16}$$

and:

$$s_t = \begin{cases} \dfrac{(1-\tau)(1-\alpha)y_t[\alpha\beta p(y_t)zy_t - \gamma\tau(1-\lambda)k_{t+1}]}{\alpha(1+\gamma+\beta p(y_t))zy_t - \gamma\tau(1-\lambda)k_{t+1}} & \text{if } y_t \leq \hat{y}, \\ \dfrac{(1-\alpha)(1-\tau)y_t[\alpha\beta p(y_t)(z(1-\alpha)y_t - 1) \\ -\gamma\tau(1-\alpha)(1-\delta)(1-\lambda)k_{t+1}]}{\alpha(1+\gamma+\beta p(y_t))[z(1-\alpha)y_t - 1] \\ -\gamma\tau(1-\lambda)(1-\delta)(1-\alpha)k_{t+1}} & \text{if } y_t > \hat{y}, \end{cases} \tag{7.17}$$

where:

$$\hat{y} = \frac{1}{\delta z(1-\alpha)} \tag{7.18}$$

From Eqs. (7.10), (7.16) and (7.17) per worker physical capital is given by:

$$k_{t+1} = \begin{cases} \dfrac{(1-\alpha)\alpha\beta p(y_t)zy_t}{\gamma\eta} & \text{if } y_t \leq \hat{y}, \\ \dfrac{\alpha\beta p(y_t)[z(1-\alpha)y_t - 1]}{\gamma(1-\delta)\eta} & \text{if } y_t > \hat{y}. \end{cases} \tag{7.19}$$

where $\eta \equiv \alpha + \tau(1-\lambda)(1-\alpha)$.

Substituting Eq. (7.19) into Eqs. (7.16) and (7.17) optimal fertility and saving in general equilibrium, are therefore given by:

$$n_t = \begin{cases} \dfrac{\gamma(1-\tau)\eta}{z(1+\gamma)\eta + z\alpha\beta p(y_t)} & \text{if } y_t \leq \hat{y}, \\ \dfrac{\gamma(1-\tau)(1-\delta)(1-\alpha)\eta y_t}{[zy_t(1-\alpha)-1][(1+\gamma)\eta + \alpha\beta p(y_t)]} & \text{if } y_t > \hat{y}, \end{cases} \tag{7.20}$$

and:

$$s_t = \frac{(1-\tau)(1-\alpha)\alpha\beta p(y_t) y_t}{(1+\gamma)\eta + \alpha\beta p(y_t)} \tag{7.21}$$

Equation (7.20) shows that the optimal number of children is, of course, positively related to parents' taste for children in the utility function, i.e. γ, negatively related to the cost of raising a child, z and negatively related to parents' income. In particular, when $y_t < \hat{y}$ income only indirectly affects fertility through adult survival whereas when $y_t > \hat{y}$ there is also a direct negative effect.

Thus, as income increases, population aging from above, i.e. from increasing life expectancy, is reinforced by aging from below, i.e. by decreasing fertility.

Equations (7.15)–(7.17) and (7.10) allow us to characterize the dynamic of income per worker in period $t+1$ as follows:

$$y_{t+1} = \begin{cases} A\Psi(p(y_t)y_t)^{\alpha} & \text{if } 0 \leq y_t \leq \hat{y}, \\ A\Delta[p(y_t)]^{\alpha}[z(1-\alpha)y_t - 1]^{\alpha+\delta(1-\alpha)} & \text{if } y_t > \hat{y} \end{cases} \tag{7.22}$$

where:

$$\Psi = \left[\frac{(1-\alpha)\alpha\beta z}{\gamma\eta}\right]^{\alpha}, \tag{7.23}$$

and:

$$\Delta = \left[\frac{\alpha\beta}{\gamma(1-\delta)\eta}\right]^{\alpha}\left(\frac{\delta}{1-\delta}\right)^{\delta(1-\alpha)}. \tag{7.24}$$

Simple calculations show that y_{t+1} is increasing and continuous and $\lim_{y_t\to\hat{y}^-}\partial y_{t+1}/\partial y_t < \lim_{y_t\to\hat{y}^+}\partial y_{t+1}/\partial y_t$ (for technical details see Appendix 1).

From Eqs. (7.13), (7.20) and (7.22) we can observe that the overall impact of higher income per worker in period t on pension payout is ambiguous. In fact, on the one hand it has a positive effect because it increases income per worker in period $t+1$, but, on the other hand, it also has a negative effect because it increases the pension system dependency ratio i.e. $p(y_t)/n_t$. However, in the simulations we always found that with our set of parameters, the relationship between pension and income is positive.

7.3.5 The Dynamic System

In this section we characterize the dynamics of income per capita. For the sake of analytical tractability we need to specify the adult survival function. In particular, following Chakraborty and Das (2005) we choose the following function:

$$p_t = \begin{cases} x y_t^{\epsilon} & \text{if } 0 \le y_t \le \bar{y}, \\ \bar{p} & \text{if } y_t > \bar{y}, \end{cases} \tag{7.25}$$

where $x = [\lambda\tau(1-\alpha)]^{\epsilon}$ and:

$$\bar{y} = \frac{\bar{p}^{1/\epsilon}}{\lambda\tau(1-\alpha)}. \tag{7.26}$$

Based on the stylized fact of a positive correlation between schooling and life expectancy (see, among others, Lorentzen et al., 2008), we assume that adult survival reaches its maximum value when income is sufficiently high to allow parents to invest in education. Thus:

Assumption 7.1 *We assume that $\bar{y} > \hat{y}$, that is:*

$$\tau < \frac{\delta z \bar{p}^{1/\epsilon}}{\lambda}. \tag{7.27}$$

Note that this assumption implies an upper limit to the share of tax revenues devoted to public health expenditure $\lambda\tau$.

From Eq. (7.25) and under Assumption 7.1 the dynamics of income per worker is, therefore, given by:

$$y_{t+1} = \begin{cases} A\Psi(xy_t^{1+\epsilon})^{\alpha} & \text{if } 0 \leq y_t \leq \hat{y}, \\ A\Delta(xy_t^{\epsilon})^{\alpha}[z(1-\alpha)y_t - 1]^{\alpha+\delta(1-\alpha)} & \text{if } \hat{y} < y_t < \bar{y}, \\ A\Delta\bar{p}^{\alpha}[z(1-\alpha)y_t - 1]^{\alpha+\delta(1-\alpha)} & \text{if } y_t > \bar{y} \end{cases} \tag{7.28}$$

which is concave in the interval $0 < y_t < \bar{y}$ if $\epsilon < (1-\alpha)(1-\delta)/\alpha$ and always concave for $y_t > \bar{y}$ (see Appendix 1).

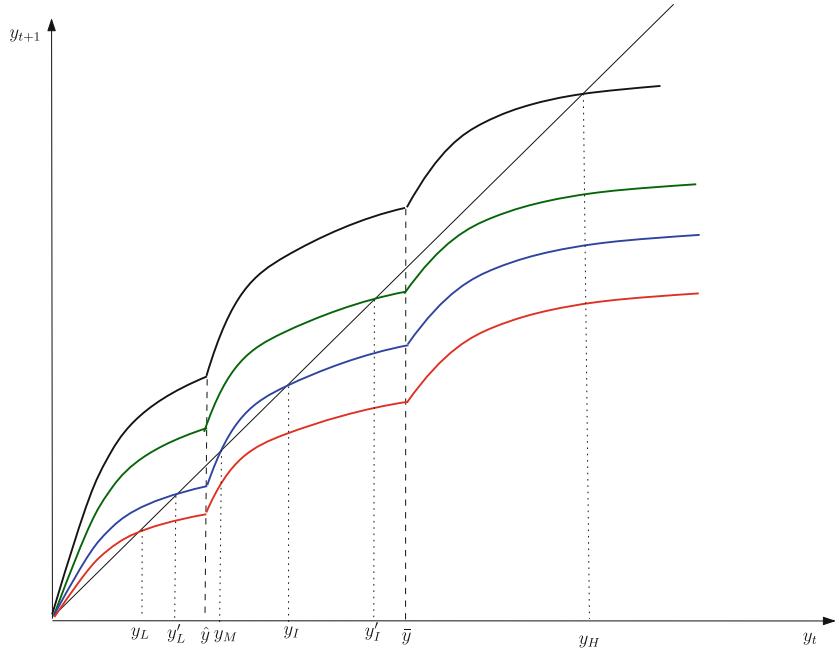

Fig. 7.4 Dynamics of income per worker

Figure 7.4 depicts the dynamic system of income per worker. Depending on the level of technology, different scenarios can arise (see Appendix 1 for technical details). In particular, we identify three critical levels of technology:

$$\hat{A} = \left[\left(\frac{E}{z(1-\alpha)} \right)^{E} \left(\frac{1}{E-1} \right)^{E-1} \right]^{\alpha+\delta(1-\alpha)} \frac{1}{\Delta x^{\alpha}}, \tag{7.29}$$

$$\tilde{A} = \frac{\hat{y}^{1-(1+\epsilon)\alpha}}{x^{\alpha}\Psi}, \tag{7.30}$$

and:

$$\bar{A} = \frac{\bar{y}}{\Delta \bar{p}^{\alpha}[z(1-\alpha)\bar{y}-1]^{\alpha+\delta(1-\alpha)}}, \tag{7.31}$$

where $E = (1-\alpha\epsilon)/[\alpha+\delta(1-\alpha)]$ and if $\bar{A} > \tilde{A} > \hat{A}$ we can classify the dynamic system into four cases (see Appendix 1). Proposition 7.1 characterizes these cases.

Proposition 7.1 *If the level of technology is very low, i.e.* $A < \hat{A}$*, the dynamic system has a unique and stable equilibrium* y_L *with no education, where:*

$$y_L = (A\Psi x^{\alpha})^{\frac{1}{1-\alpha(1+\epsilon)}}. \tag{7.32}$$

If $\hat{A} < A < \tilde{A}$ *economy features multiple steady states: one stable equilibrium with no education* y'_L*, one unstable* y_M *and one stable equilibrium* y_I *with a positive investment in education.*

If $\tilde{A} < A < \bar{A}$ *the dynamic system shows a unique and stable steady state* y'_I *with positive education, low fertility and intermediate adult survival.*

Finally, if technology is sufficiently high, i.e. $A > \bar{A}$*, the economy converges to a unique and stable equilibrium* y_H *characterized by a high level of education, high adult survival and low fertility.*

Thus, when the level of technology is very low, i.e. $A < \hat{A}$, parents' income is not sufficiently high so as to ensure a positive investment in their children's education and therefore the economy converges to a unique and stable steady state with no education, high fertility and a low level of adult survival. On the other hand, when $\hat{A} < A < \tilde{A}$ the initial level of per capita income determines the characteristics of the long-run equilibrium. A poor economy, where per capita income is below y_M converges to a zero-education, high-fertility and low adult survival equilibrium, whereas an economy where income is above y_M converges to a high-education, low-fertility and high (intermediate) adult survival equilibrium.

Finally, when the level of technology is very high, parents' income is sufficiently high as to allow human capital accumulation and therefore the economy converges to a unique stable steady state with a high level of human capital and adult survival and a low level of fertility.

From Eqs. (7.29)–(7.31) we can observe that the three levels of A are affected by both wage income tax τ and the allocation of public spending between health spending λ and social security benefits $1 - \lambda$. Thus, different combinations of A, τ and λ, can lead to various scenarios. We discuss this point, focusing on the impact of government policy, in the next section.

7.4 Policy Effects

7.4.1 Labor Income Tax

From Eq. (7.28) we find that the impact of income tax on the evolution of income per capita depends on both the level of taxation and the level of income. In particular, firstly, from Eq. (7.26), we observe that a higher tax rate decreases the threshold level of income such that longevity reaches its maximum, i.e. $\bar{y}$, and thus, through this channel, has a positive effect on the evolution of income. Secondly, from Eq. (7.28) when income is below $\bar{y}$, τ has a positive effect on y_{t+1} if $\tau < \hat{\tau}$ (see Appendix 2[1]), whereas, when income is above $\bar{y}$, a higher tax rate always decreases y_{t+1}.

The intuition behind this result is that a higher income tax rate affects the evolution of income through different channels, leading to opposing effects. In particular, a first direct impact of a higher tax rate, is a reduction of agents' disposable income, which in turns negatively affects savings and education. Secondly, it has a direct positive impact on pension benefits, leading to a negative displacement effect on savings. Thirdly it increases longevity, thus positively affecting savings and education and negatively affecting fertility. At low income and longevity levels, the decreasing returns of health expenditure on life expectancy implies a strong positive impact of a higher income tax on longevity, leading to an overall positive impact on income, as long as the increase in the tax rate is modest. By contrast, at high income and life expectancy levels, the impact of health

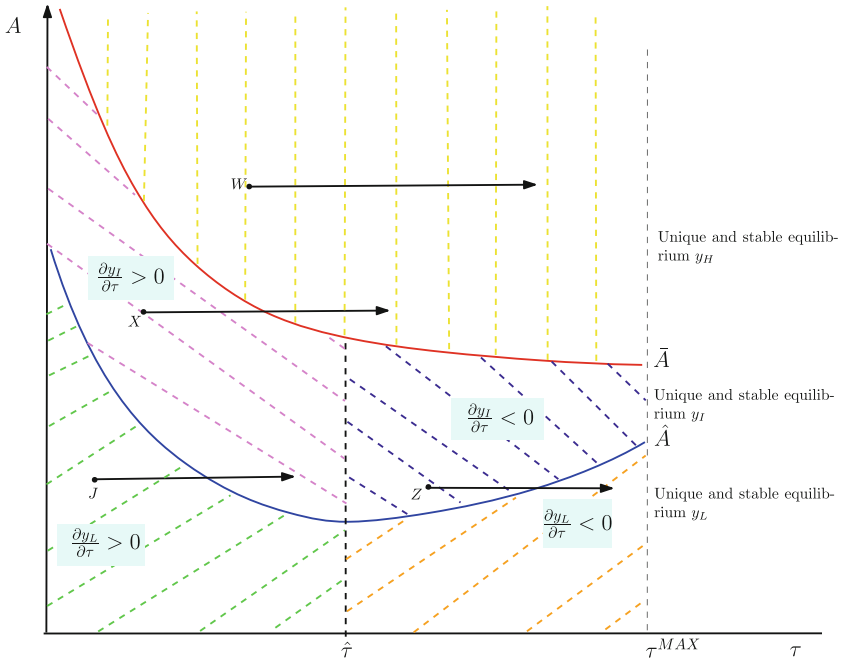

Fig. 7.5 The impact of τ on income

spending on longevity is low and therefore the negative effect of a higher tax rate prevails. Given these results, and the impact of different level of technology on the evolution of income as shown in Proposition 7.1, in Fig. 7.5 we analyze how different combinations of technology and income tax can lead to various scenarios.[2]

From Eqs. (7.29) and (7.31) we get that in the range $0 < \tau < \tau^{MAX}$, $\hat{A}$ decreases with respect to τ if $\tau < \hat{\tau}$ and then increases (see Appendix 1).[3] The impact of a higher wage income tax on $\bar{A}$ is, in general, ambiguous. However, in the simulations we always find that with our set of parameters, the relationship between $\bar{A}$ and τ is negative in the range $0 < \tau \leq \tau^{MAX}$ (see Fig. 7.5).

Consider the case of an economy in which the level of technology is very low, i.e. $A < \hat{A}$, and which, therefore, shows the unique and stable low income equilibrium y_L. From Eq. (7.32) and as mentioned above, if

τ increases, but remains below $\hat{\tau}$, the equilibrium y_L increases, while if τ rises above $\hat{\tau}$ the equilibrium y_L decreases. Thus, in a poor economy, when τ increases, but remains below $\hat{\tau}$, the equilibrium y_L can increase above $\hat{y}$ and therefore, as shown by the point J in Fig. 7.5, the economy can move from y_L to the steady state y_I (see also Fig. 7.6 in Sect. 7.5). Thus, in a poor economy, an increase in the tax rate, provided that it remains below a certain level, positively affects the equilibrium level of income.

When the economy shows the unique and stable equilibrium y_I, i.e. $\tilde{A} < A < \bar{A}$, the relationship between the equilibrium y_I and τ, as for the equilibrium y_L, is positive if $\tau < \hat{\tau}$ and negative if $\tau > \hat{\tau}$ (for technical details see Appendix 2). However, in this latter case, the overall impact of a higher τ is ambiguous, because although, on the one hand, τ negatively affects y_I, on the other hand the threshold $\bar{y}$ declines. Thus, if the level of technology is relatively low, when τ increases above $\hat{\tau}$ the equilibrium y_I decreases and the economy can move from a scenario characterized by the equilibrium y_I to the low income equilibrium y_L (see point Z in Fig. 7.5). However, if technology is sufficiently high, although the level of taxation increases above $\hat{\tau}$, the increase in longevity, thanks to a higher public spending on health, may lead to the transition to the high income equilibrium y_H (see Fig. 7.7 in Sect. 7.5). Finally, when there is a unique and stable high income equilibrium y_H, i.e. $A > \bar{A}$, if τ increases, the equilibrium y_H declines but it does not disappear (see point W in Fig. 7.5). In this case, in fact, longevity is at its maximum level $\bar{p}$ and therefore the increases in the tax rate only have a negative impact on disposable income. However, although the equilibrium y_H decreases, it does not disappear because the threshold level of income which allows longevity to reach its maximum level, i.e. $\bar{y}$, decreases (see Fig. 7.8 in the Sect. 7.5).

We now move on to see the impact of an increase in wage income tax on fertility and pension benefits. A higher wage income tax rate affects fertility through three different channels. Firstly, it decreases the after tax income and therefore has a negative income effect on fertility. Secondly, it increases adult survival, thus decreasing fertility. Thirdly, *ceteris paribus*, it increases the pension payout and therefore has a positive income effect.

From Eq. (7.20) we find that in the range $0 \leq y_t \leq \bar{y}$, increasing the wage income tax, with $\tau < \hat{\tau}$, has a negative effect on fertility, whereas if $\tau > \hat{\tau}$ this impact is in general ambiguous but is negative under a certain restriction on the parameters (see Appendix 2). When $y_t \geq \bar{y}$ a higher wage income tax always decreases fertility.

As far as the impact of the tax on pension payouts is concerned, from Eq. (7.13), we see that the income tax affects the pension benefit directly and indirectly through income and the old age dependency ratio i.e. p_t/n_t. In particular, the effect of a rise in the wage income tax on the pension benefit is summarized by the following total derivative:

$$\frac{db_{t+1}}{d\tau} = \overbrace{\frac{\partial b_{t+1}}{\partial \tau}}^{+} + \overbrace{\frac{\partial b_{t+1}}{\partial y_{t+1}}}^{+} \overbrace{\frac{\partial y_{t+1}}{\partial \tau}}^{+,-} + \overbrace{\frac{\partial b_{t+1}}{\partial n_t/p_t}}^{+} \overbrace{\frac{\partial (n_t/p_t)}{\partial \tau}}^{-}. \tag{7.33}$$

Thus, when the wage income tax is raised, pension benefits increase due to a direct positive effect, and decrease because of the increase in the old age dependency ratio, while the impact of income depends on the level of taxation and on the initial level of income. In particular, as we specify above, for $0 \leq y_t \leq \bar{y}$, if τ increases, but remains below $\hat{\tau}$, income rises and therefore it positively affects the pension benefit, whereas when τ increases above $\hat{\tau}$, income decreases and therefore has a negative effect on the pension payout. When income is sufficiently high, i.e. $y_t > \bar{y}$, income declines and therefore it negatively affects the pension benefit.

The overall effect is therefore ambiguous and depends on the initial condition and on the level of taxation. For this reason, in the Sect. 7.5 we employ a numerical simulation to study the relationship between pension benefits and the wage income taxation.

7.4.2 Social Security and Public Health Spending

From Eq. (7.28) we get that a reallocation of public funds from social security benefits to public spending in health positively affects the evolution of income per worker, i.e. $\partial y_{t+1}/\partial \lambda > 0$. Moreover from

Eqs. (7.29), (7.30) and (7.31) we can observe that as λ increases $\hat{A}$, $\tilde{A}$ and $\bar{A}$ decrease. Thus an increase in the fraction of public spending on health positively affects the steady state level of income per worker and can allow the transition from a stagnant equilibrium with zero education, high fertility and low adult survival to a high income equilibrium. The basic intuition behind this results is that λ has positive effects on the dynamics of income per worker both directly and indirectly through adult longevity. In fact, on the one hand, increasing public spending in health, *ceteris paribus*, has a direct negative effect on pension benefits and therefore on income for the old generation. Thus, a lower social security benefit, as we can observe from Eqs. (7.21) and (7.20), leads agents to increase savings and to have a lower number of children. On the other hand, a reallocation from social security benefits to public investment in health increases adult longevity which in turn, as is evident from Eqs. (7.20) and (7.21), reinforces these effects.

From Eq. (7.13) we can observe that the effect on pension benefits of a shift of tax revenues from social security benefits to public health spending is in general ambiguous. In particular, the overall impact of a rise in λ on pension payout is given by the following total derivative:

$$\frac{db_{t+1}}{d\lambda} = \overbrace{\frac{\partial b_{t+1}}{\partial \lambda}}^{-} + \overbrace{\frac{\partial b_{t+1}}{\partial y_{t+1}}}^{+}\overbrace{\frac{\partial y_{t+1}}{\partial \lambda}}^{+} + \overbrace{\frac{\partial b_{t+1}}{\partial n_t/p_t}}^{+}\overbrace{\frac{\partial (n_t/p_t)}{\partial \lambda}}^{-}, \tag{7.34}$$

where we can observe that an increase in the fraction of public spending devoted to health, on the one hand, reduces pension payouts though a direct negative effect and an increase in the old age dependency ratio, on the other hand it increases the pension benefits because the income per capita rises. The overall effect is thus ambiguous. For this reason, in the next section using a set of parameter values, we will work out a simulation of the model.

7.5 Simulations

In this section we simulate the main results derived in the previous section under some reasonable values of the parameters. The purpose of this simulation is to illustrate the analytical results especially in the cases when they are ambiguous.

In summary, with regard to the change in the labor income tax in Sect. 7.4.1 we derive that: (i) the equilibria y_L and y_I increase with respect to τ if $\tau < \hat{\tau}$ and then decline after that. Thus, as shown in Fig. 7.5, if τ increases but remains below $\hat{\tau}$ the economy can move from the equilibrium y_L to the equilibrium y_I and from the equilibrium y_I to the equilibrium y_H; (ii) the equilibrium y_H decreases with respect to τ but it does not disappear; (iii) the impact of τ on fertility is negative if $\tau < \hat{\tau}$ and ambiguous if $\tau > \hat{\tau}$, (iv) the effect of τ on pensions is ambiguous.

With regard to the change in the fraction of public spending devoted to health, in Sect. 7.4.2 we obtained that, *ceteris paribus*, when λ increases: (i) income per capita increases and therefore a higher λ can allow the transition from y_L to y_I and from y_I to y_H; (ii) fertility declines; (iii) the impact on pension benefits is ambiguous.

The parameters values are $\alpha = 1/3$, $\beta = 0.3$, $\delta = 0.3$, $\gamma = 0.6$, $\bar{p} = 1$, $\epsilon = 0.3$, $z = 0.4$. The value of α, the elasticity of output with respect to capital, is defined is defined from the empirical regularity that the capital share in added value is about 1/3 (OECD, 2011). The parameter z, the child rearing cost, is in line with the empirical literature on the children's resource share (see for example Apps and Rees, 2001). In the literature, the quarterly individual discount factor β is set equal to 0.99. Hence for the entire life span, it is evaluated as 0.99^{120}, roughly 0.3 (see de la Croix and Doepke, 2003; Cipriani and Pascucci, 2018). The maximum value of adult survival $\bar{p}$ and the elasticity of the adult survival with respect to income ϵ are such to satisfy the conditions on parameters assumed in the previous section. In any case, a sensitivity analysis available under request shows that the results are quite robust to alternative range of values of the parameters.

In the simulations that focus on the impact of the wage income tax we fixed $\lambda = 0.2$; this implies that $\tau^{MAX} = 0.38$.

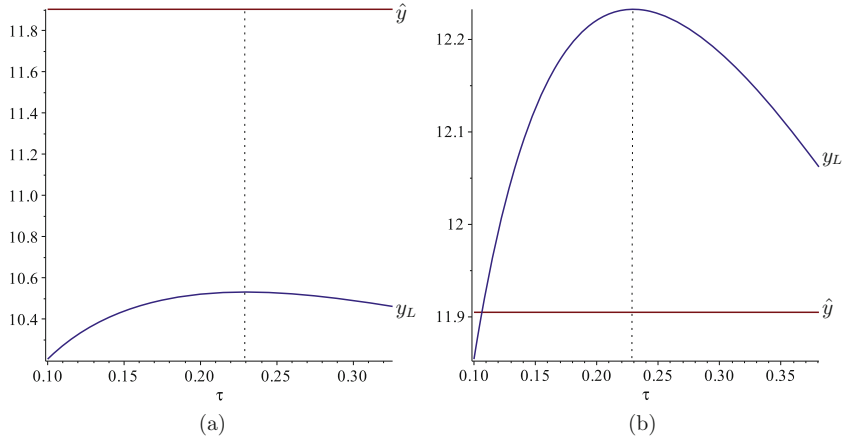

Fig. 7.6 The impact of a higher τ on the equilibrium y_L, case $A < \hat{A}$. **(a)** Low A. **(b)** High A

Figure 7.6 confirms the non-monotonic relationship between the low income equilibrium y_L and the wage income tax: increasing if $\tau < \hat{\tau}$ and then declining after the level $\hat{\tau}$. Moreover, Fig. 7.6b shows that when the level of A is sufficiently high, y_L increases above the threshold $\hat{y}$ and therefore the economy moves to the equilibrium y_I.

Figure 7.7 shows that when the level of A is sufficiently high, when τ increases the economy moves from the equilibrium y_I to the equilibrium y_H because of a decrease of the threshold $\bar{y}$.

Figure 7.8 shows, in accordance with the theory, that when τ increases the equilibrium y_H declines but it does not disappear.

Figure 7.9 confirms the negative relationship between fertility and income and shows that with our set of parameters, the relationship between fertility and the wage income tax is negative for each level of per capita income because the negative effect due to a lower after tax income and a higher adult survival always prevails on the positive effect from pensions.

Figure 7.10 shows that the impact of the tax rate on pensions, for each level of income y_t, depends on the value of λ. In particular, as shown in Fig. 7.10a if λ is sufficiently low, the relationship between pension benefits

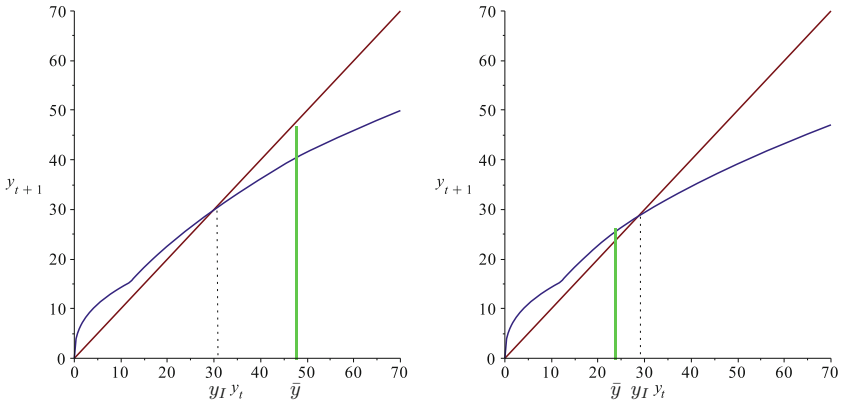

Fig. 7.7 The impact of a higher τ when A is sufficiently high, case: $\tilde{A} < A < \bar{A}$

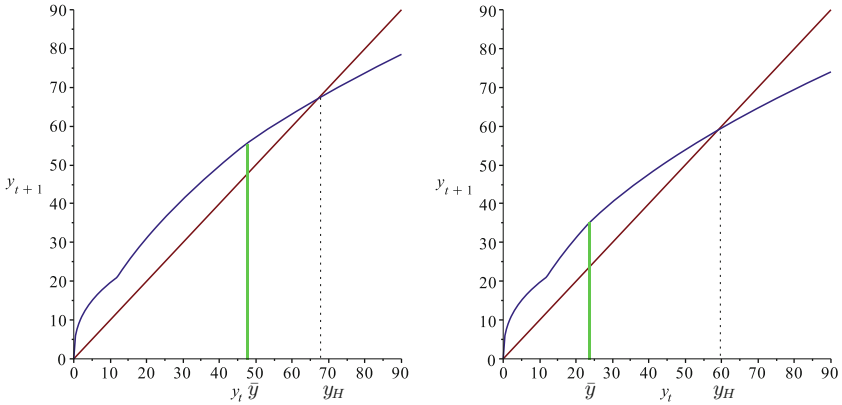

Fig. 7.8 The impact of a higher τ, case: $A > \bar{A}$

and wage income tax may be non-monotonic: initially increasing with τ and then declining after a certain level. On the other hand, as shown in Fig. 7.10b when the level of λ is sufficiently high, pension benefits are lower, the relationship between τ and b_{t+1} is always positive The intuition behind this result is that with a higher value of λ, $\hat{\tau}$ increases and $\bar{y}$ declines. This implies, therefore, a higher indirect positive impact of τ through income on pension benefits, as we can see from Eq. (7.33), which thus leads to the prevalence of the total positive impact.

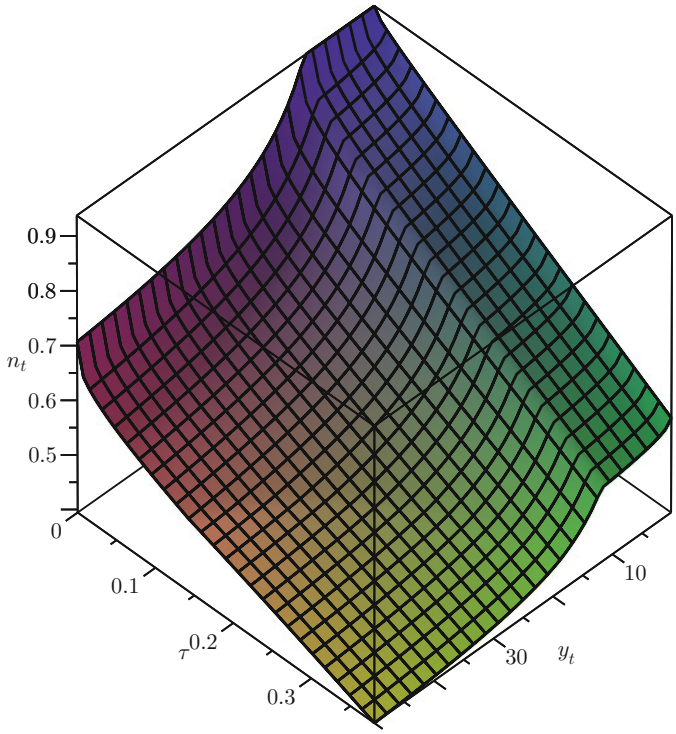

Fig. 7.9 Fertility, income and wage income tax

As far as the impact of a reallocation of public funds from social security benefits to public spending in health is concerned, Fig. 7.11 shows that, in accordance with the theoretical results, increasing λ positively affects the evolution of income per worker, allowing the transition from the equilibrium y_I to the equilibrium y_H. In particular, in the figure we fixed $\tau = 0.25$, this implies that λ can change from 0.1 to 0.3, thus we simulate for $\lambda = 0.1$ and $\lambda = 0.3$.

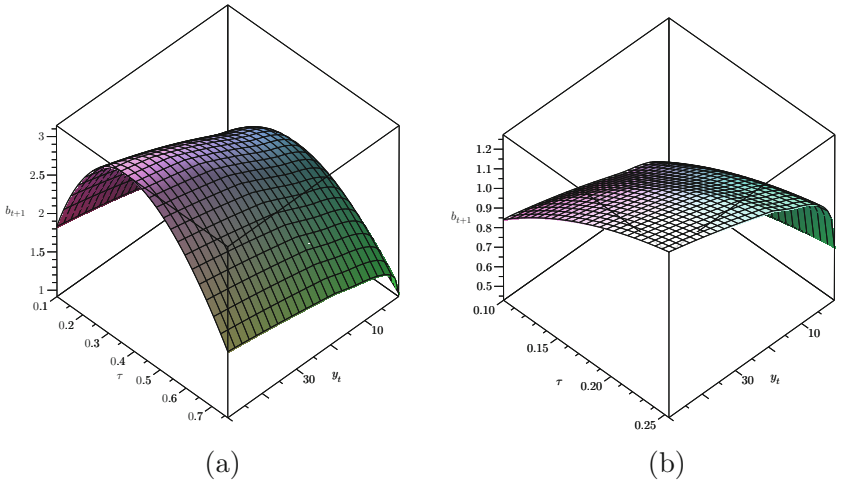

Fig. 7.10 Pension, income and wage income tax. **(a)** $\lambda = 0.1$. **(b)** $\lambda = 0.3$

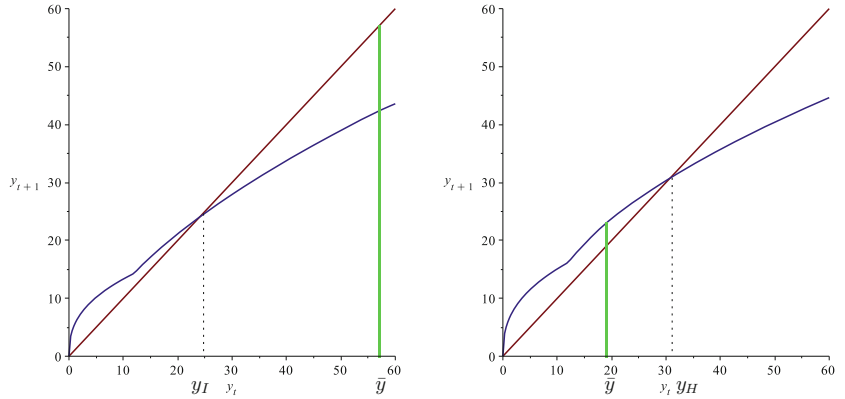

Fig. 7.11 The impact of a higher λ

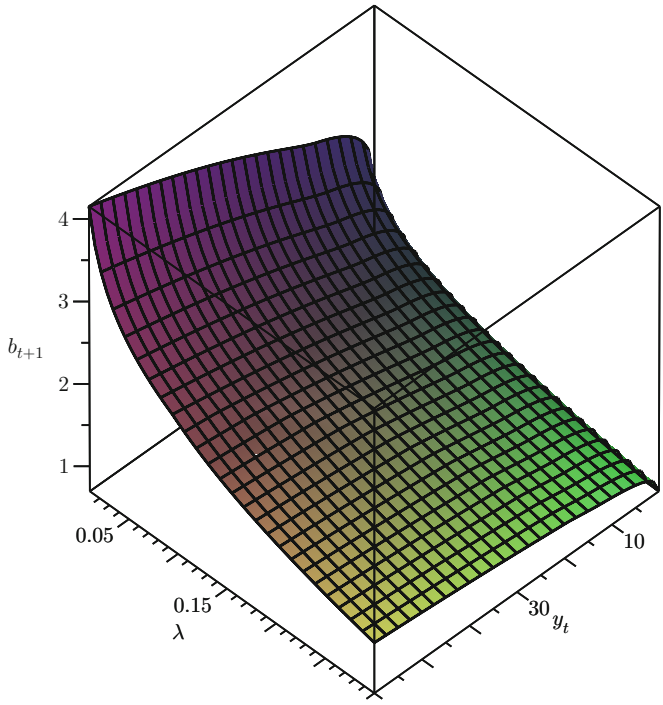

Fig. 7.12 Pension, income and λ

Figure 7.12 shows that reallocating public funds from social security benefits to public investment in health always decreases the pension benefit because the direct negative effect and the indirect negative effect through the old age dependency ratio prevail on the positive effect arising from a higher income per capita.

7.6 Concluding Remarks

Significant gains in life expectancy have been achieved almost everywhere in the world in the last decades. This demographic change has been accompanied by a fall in fertility and the resulting aging population has put strain on pension systems. These dynamics are expected to continue

across the globe during most of this century, with the median age increasing everywhere except in Africa. Aging will become particularly important in Europe, where it is expected that by 2060 the fraction of people over 65 will be almost twice as large as the fraction under 15 (United Nations, 2017). At the same time, there has been a significant increase in health expenditures, especially in high income countries where health costs have been rising faster than GDP over the past 40 years and are projected to continue to rise at rates that exceed the growth of GDP per capita, leading to higher shares of government outlays spent on health (Jenkner and Leive, 2010). The empirical literature has shown that health expenditures are positively associated with life expectancy, especially for countries with lower life expectancy (Obrizan and Wehby, 2018), even though there are marked differences in health system efficiency across the OECD countries (Barthold et al., 2014). In this chapter, we presented a model of endogenous aging with public expenditure on health and pensions and private expenditure on education. We found that, under certain conditions, there is a low income steady state and a high income steady state. The low development regime traps the economy in a situation with low life expectancy and high fertility. Depending on initial conditions, the economy may converge instead toward a high development regime, where income is higher and there is population aging. Government policies on health and pensions can move an economy from one regime to the other. In particular, while there is a non-monotonic relationship between the tax rate and equilibrium income, an increase in the fraction of public expenditure devoted to health increases the steady state level of income, even though pension benefits may decrease and the problem of population aging may become worse.

The model could be extended in a number of interesting ways. On the pensions side, one could consider a model with retirement, where agents can work for part of their third period of life. This is a particularly interesting extension in a context of increasing longevity and has been recently studied by Cipriani and Pascucci (2018) in a model with exogenous longevity and Cipriani (2018) in a model with exogenous fertility.

Also, in our framework we only consider private education expenditure, even though only 30 per cent of total expenditure at tertiary level is paid directly by households. A more comprehensive model should also consider the share of public funding, and also the fact that better-educated people have lower morbidity rates and greater life expectancy (Cutler and Lleras-Muney, 2012). Finally, we have ignored the fact that each individual can influence his own life expectancy through some private health expenditure and also that health expenditures can obviously have other important effects than those on the length of life, such as improving the quality of life and therefore directly entering the utility function or increasing the effectiveness of human capital in the production function or the human capital production function itself. We leave these extensions for further research.

Appendix 1: Dynamics of Income Per Capita

We present some general properties of y_{t+1} with respect to y_t derived from Eq. (7.22).

- y_{t+1} is strictly increasing and continuous in the interval $(0, +\infty)$:

$$\lim_{y_t \to \hat{y}^-} y_{t+1} = \lim_{y_t \to \hat{y}^+} y_{t+1} = \left\{ \frac{\alpha\beta p(\hat{y})}{\gamma[\alpha + \tau(1-\alpha)(1-\lambda)]} \right\}^{\alpha} \tag{7.35}$$

- The function $\partial y_{t+1}/\partial y_t$ is discontinuous at $\hat{y}$, in particular:

$$\lim_{y_t \to \hat{y}^-} \frac{\partial y_{t+1}}{\partial y_t} - \lim_{y_t \to \hat{y}^+} \frac{\partial y_{t+1}}{\partial y_t} = -Ap(\hat{y})^{\alpha}$$
$$A \left\{ \frac{\alpha\beta}{\gamma\delta[\alpha + \tau(1-\alpha)(1-\lambda)]} \right\}^{\alpha} \frac{\delta^2 z(1-\alpha)}{1-\delta} < 0 \tag{7.36}$$

When we specify adult survival, from Eq. (7.28) we get that:

- $\partial y_{t+1}/\partial y > 0$ in the interval $(0, +\infty)$, that is:

$$\frac{\partial y_{t+1}}{\partial y_t} = \begin{cases} A\alpha\Psi(1+\epsilon)x^{\alpha}y_t^{\alpha(1+\epsilon)-1} & \text{if } 0 \leq y_t \leq \hat{y}, \\ A\Delta(xy_t^{\epsilon})^{\alpha}[z(1-\alpha)y_t - 1]^{\alpha+\delta(1-\alpha)} & \\ \quad \left(\frac{\alpha\epsilon}{y_t} + \frac{[\alpha+\delta(1-\alpha)]z(1-\alpha)}{z(1-\alpha)y_t - 1}\right) & \text{if } \hat{y} < y_t < \bar{y}, \\ A\Delta\bar{p}^{\alpha}[z(1-\alpha)y_t - 1]^{\alpha+\delta(1-\alpha)-1}z(1-\alpha) & \\ \quad [\alpha + \delta(1-\alpha)] & \text{if } y_t > \bar{y} \end{cases} \tag{7.37}$$

- The function $\partial y_{t+1}/\partial y_t$ is discontinuous at $\hat{y}$ and $\bar{y}$, in particular:

$$\begin{cases} \lim_{y_t \to \hat{y}^-} \frac{\partial y_{t+1}}{\partial y_t} < \lim_{y_t \to \hat{y}^+} \frac{\partial y_{t+1}}{\partial y_t} \\ \lim_{y_t \to \bar{y}^-} \frac{\partial y_{t+1}}{\partial y_t} > \lim_{y_t \to \bar{y}^+} \frac{\partial y_{t+1}}{\partial y_t} \end{cases} \tag{7.38}$$

- From Eq. (7.37) it is easy to note that in the range $0 \leq y_t \leq \hat{y}$, $\partial^2 y_{t+1}/\partial y_t^2 < 0$ if:

$$\epsilon < \frac{1-\alpha}{\alpha} \tag{7.39}$$

In the interval $\hat{y} \leq y_t \leq \bar{y}$:

$$\frac{\partial^2 y_{t+1}}{\partial y_t^2} = \chi\left\{[z(1-\alpha)y_t]^2(2-E-Q) + 2z(1-\alpha)y_t(E-1) - E\right\}, \tag{7.40}$$

where: $\chi = y_{t+1}\alpha\epsilon z(1-\alpha)[\alpha+\delta(1-\alpha)]/y_t^2 z(1-\alpha)[z(1-\alpha)y_t - 1]^2$, $E = (1-\alpha\epsilon)/[\alpha+\delta(1-\alpha)]$ and $Q = (1-\alpha)(1-\delta)/\alpha\epsilon$.

It is easy to note that if $E > 1$, then $Q > 1$ and therefore $E+Q > 2$. In this case, therefore, $\partial^2 y_{t+1}/\partial y_t^2 < 0$ if:

$$T(y_t) = -[z(1-\alpha)y_t]^2(E+Q-2) + 2z(1-\alpha)y_t(E-1) - E < 0. \tag{7.41}$$

This is a concave downward function and thus if the maximum value of this function is negative then this function is always negative. In particular, given the maximum point:

$$y^{MAX} = \frac{E-1}{(E+Q-2)z(1-\alpha)}, \tag{7.42}$$

we get that the maximum value of the function is always negative, that is:

$$T_{y_t=y^{MAX}} = \frac{1-EQ}{E+Q-2} = \frac{1}{-\delta(1-\alpha)-\alpha(1+\epsilon)} < 0. \tag{7.43}$$

In short $\partial^2 y_{t+1}/\partial y_t^2 < 0$ if $E > 1$, that is:

$$\epsilon < \frac{(1-\alpha)(1-\delta)}{\alpha}, \tag{7.44}$$

note that if this condition is satisfied then so is (7.39).

Finally when $y_t > \bar{y}$, from Eq. (7.37) it is easy to note that $\partial^2 y_{t+1}/\partial y_t^2 < 0$.

Equilibria

Assuming that the condition in Eq. (7.44) holds then:

- In the range $0 \leq y_t \leq \hat{y}$ the economy can reach the steady state:

$$y_L = (A\Psi x^{\alpha})^{\frac{1}{1-\alpha(1+\epsilon)}} \tag{7.45}$$

The necessary and sufficient condition for the existence of such equilibrium is that $y_L < \hat{y}$ (or $y_{t+1}|_{y_t=\hat{y}} < \hat{y}$). This holds if $A \leq \tilde{A}$, where:

$$\tilde{A} = \frac{\hat{y}^{1-(1+\epsilon)\alpha}}{x^{\alpha}\Psi} = \frac{\hat{y}^{1-\alpha\epsilon}}{\Delta x^{\alpha}[z(1-\alpha)\hat{y}-1]^{\alpha+\delta(1-\alpha)}} \tag{7.46}$$

In summary there exists one stable equilibrium in the range $y_t \in (0, \hat{y}]$ if:

$$A \leq \tilde{A} \tag{7.47}$$

- In the range $y_t \in (\hat{y}, \bar{y}]$ different scenarios can arise.
  In particular, $y_{t+1}|_{y_t=\bar{y}} > \bar{y}$ if $A \geq \bar{A}$ with:

$$\bar{A} = \frac{\bar{y}}{\Delta \bar{p}^{\alpha}[z(1-\alpha)\bar{y}-1]^{\alpha+\delta(1-\alpha)}}. \tag{7.48}$$

In order to reduce the number of feasible scenarios for the evolution of the economy we suppose that $\bar{A} > \tilde{A}$. This is true if the following sufficient condition holds:

$$\tau < \tau^{MAX} = \bar{p}^{1/\epsilon}\frac{\delta z(1-\delta)^{1/(E-1)}}{\lambda}. \tag{7.49}$$

Thus in the range $\hat{y} \leq y_t \leq \bar{y}$, if $A > \bar{A}$ there is no steady state; if $\tilde{A} < A < \bar{A}$ there is one stable equilibrium and finally when $A < \tilde{A} < \bar{A}$ there are two steady states if:

$$T[z(1-\alpha)y_t - 1] = y_t^E, \tag{7.50}$$

where $T = (A\Delta x^{\alpha})^{\frac{1}{\alpha+\delta(1-\alpha)}}$. Note that in all other cases there is no steady state. Both the LHS and RHS of (7.50) increase with respect y_t, in particular:

$$\begin{aligned} \frac{\partial LHS(y_t)}{\partial y_t} &> 0; \\ \frac{\partial^2 LHS(y_t)}{\partial y_t^2} &= 0 \\ \lim_{y \to 0} LHS(y_t) &= -T \\ \frac{\partial RHS(y_t)}{\partial y_t} &> 0; \\ \lim_{y_t \to 0} RHS(y_t) &= 0; \end{aligned} \tag{7.51}$$

It easy to note that the sign of $\partial^2 RHS(y_t)/\partial y_t^2$ can be either negative if $E < 1$ or positive if $E > 1$.

However, as specified above in Eq. (7.44) we assume $E > 1$. In this case, therefore, Eq. (7.50) can show no or two steady states. In particular by defining the maximum point of the function $LHS(y_t) - RHS(y_t)$ as y^{MAX} there are two steady states if:

$$LHS(y_t) - RHS(y_t)|_{y_t = y^{MAX}} > 0, \tag{7.52}$$

and:

$$y^{MAX} > \hat{y}. \tag{7.53}$$

The maximum point of $LHS(y_t) - RHS(y_t) > 0$ is:

$$y^{MAX} = \left[\frac{Tz(1-\alpha)}{E}\right]^{\frac{1}{E-1}} \tag{7.54}$$

Some calculations show that (7.52) holds if $A > \hat{A}$ where:

$$\hat{A} = \left[\left(\frac{E}{z(1-\alpha)} \right)^{E} \left(\frac{1}{E-1} \right)^{E-1} \right]^{\alpha+\delta(1-\alpha)} \frac{1}{\Delta x^{\alpha}}, \tag{7.55}$$

and $y^{MAX} > \hat{y}$ if $A > A_2$ where:

$$A_2 = \left[\left(\frac{1}{\delta z(1-\alpha)} \right)^{E-1} \left(\frac{E}{z(1-\alpha)} \right) \right]^{\alpha+\delta(1-\alpha)} \frac{1}{\Delta x^{\alpha}} \tag{7.56}$$

In summary, assuming that the parameters' condition in Eq. (7.44) holds, then when $\hat{y} < y_t < \bar{y}$ the economy shows two steady states if $A > max\{\hat{A}, A_2\}$.

From Eqs. (7.46), (7.55) and (7.56) we get that $\hat{A} > A_2$ and $\tilde{A} > A_2$ if:

$$E < \frac{1}{1-\delta} \Rightarrow \epsilon > \frac{(1-\alpha)(1-\delta)-\delta}{(1-\delta)\alpha} \tag{7.57}$$

We assume that $\tilde{A} > \hat{A}$, that is:

$$\left(\frac{E-1}{\delta E} \right)^{E} > \frac{(1-\delta)(E-1)}{\delta}. \tag{7.58}$$

- When $y_t \geq \bar{y}$ there is one stable equilibrium if $A > \bar{A}$. If $A < \bar{A}$ there are no or two steady states.

Appendix 2: Policy Effect

Income

From Eq. (7.26) we can observe that $\partial \bar{y}/\partial \tau < 0$ and thus through this channel positively affects the evolution of income. From Eq. (7.28), in the

range $0 \leq y_t \leq \bar{y}$, $\partial y_{t+1}/\partial \tau > 0$ if $\partial(\Psi x^{\alpha})/\partial \tau > 0$ and $\partial(\Delta x^{\alpha})/\partial \tau > 0$. In particular, given $\eta = \alpha + \tau(1-\lambda)(1-\alpha)$ and $x = [\lambda\tau(1-\alpha)]^{\epsilon}$, from Eqs. (7.23) and (7.24) both $\partial(\Psi x^{\alpha})/\partial \tau > 0$ and $\partial(\Delta x^{\alpha})/\partial \tau > 0$ if $\partial(\tau^{\epsilon}/\eta)/\partial \tau > 0$ which holds if:

$$\tau < \hat{\tau} = \frac{\alpha\epsilon}{(1-\alpha)(1-\lambda)(1-\epsilon)}. \tag{7.59}$$

Thus, $\partial y_{t+1}/\partial y_t > 0$ if $\tau < \hat{\tau}$ and $\partial y_{t+1}/\partial y_t < 0$if $\tau > \hat{\tau}$. This implies that both $\partial y_L/\partial \tau > 0$ and $\partial y_I/\partial \tau > 0$ if $\tau < \hat{\tau}$. See also Eq. (7.32) to back this result with respect to the equilibrium y_L. When $y_t \geq \bar{y}$ y_{t+1} is always decreasing in τ.

With regard to the impact of the wage income tax on $\hat{A}$ and $\tilde{A}$ from Eqs. (7.29) and (7.30) we can observe that both $\partial \hat{A}/\partial \tau > 0$ and $\partial \tilde{A}/\partial \tau > 0$ if $\tau > \hat{\tau}$. From Eq. (7.31) we see that the impact of a higher wage income tax on $\bar{A}$ is ambiguous. However, with our set of parameters, the relationship between $\bar{A}$ and τ is always negative in the range $0 < \tau \leq \tau^{MAX}$.

Fertility

With respect to the impact of a higher wage income tax on fertility from Eq. (7.20) we derive that $\partial n_t/\partial \tau < 0$ if:

$$-(1+\gamma) - \alpha\beta\frac{p(y_t)}{\eta}\left[1 + (1-\tau)\frac{\partial(p(y_t)/\eta)/\partial \tau}{p(y_t)/\eta}\right] < 0. \tag{7.60}$$

From Eq. (7.25), note that when $y_t > \bar{y}$, this is always satisfied because $\partial(p(y_t)/\eta)/\partial \tau = 0$.
When $y_t < \bar{y}$ the term inside the brackets is given by:

$$1 + (1-\tau)\frac{[\alpha\epsilon - (1-\lambda)(1-\alpha)(1-\epsilon)\tau]}{\tau[\alpha + \tau(1-\lambda)(1-\alpha)]} \tag{7.61}$$

which is positive since

$$\tau^2(1-\lambda)(1-\alpha)(2-\epsilon)+\tau(1-\epsilon)[\alpha-(1-\lambda)(1-\alpha)]+\alpha\epsilon > 0 \qquad (7.62)$$

if the sufficient condition $\lambda > (1-2\alpha)/(1-\alpha)$ holds. Thus under this parametric restriction, we can conclude that, a higher wage income tax always decreases fertility.

Notes

1. In particular:

$$\hat{\tau} = \frac{\alpha\epsilon}{(1-\lambda)(1-\alpha)(1-\epsilon)} \qquad (7.63)$$

2. For the sake of simplicity we do not consider the case of multiple steady states, that is $\hat{A} < A < \tilde{A}$.
3. In particular, $\tau < \tau^{MAX}$ is a sufficient condition to ensure $\bar{A} > \tilde{A}$ (see Appendix 1), where:

$$\tau^{MAX} = \bar{p}^{1/\epsilon}\frac{\delta z(1-\delta)^{1/(E-1)}}{\lambda}. \qquad (7.64)$$

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8

Health and Knowledge Externalities: Implications for Growth and Public Policy

Pierre-Richard Agénor

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8.1 Introduction

A large strand of the literature on economic growth focuses on human capital, which is often defined to include the knowledge, skills (or more generally abilities), and health that individuals accumulate or achieve in

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P.-R. Agénor (✉)
School of Social Studies, University of Manchester, Manchester, UK
e-mail: Pierre-richard.Agenor@manchester.ac.uk

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the course of their lives.[1] A key premise of that literature is that education and good health combine to enhance worker productivity and promote growth. Investments in health, in particular, can influence the pace of economic growth via their effects on a variety of health outcomes and health-related factors, including labor market participation and labor productivity, life expectancy, savings, and fertility decisions (Bloom and Canning, 2009). Conversely, poor health may impede not only physical strength but also mental abilities, incentives to invest in education (which affects an individual's future earning capacity), and the ability of parents to provide child care. As a result, it may not only be a *cause* of persistent poverty, but also an *outcome* of poverty. There is much evidence to support this two-way causality; Lorentzen et al. (2008), for instance, found a bidirectional link between life expectancy and income.

An extensive analytical and empirical literature has also focused on the possible interactions between components of human capital, especially education and health, and how they affect growth. Benos and Zotou (2014) for instance, in a comprehensive meta-regression analysis, found that the growth effect of education is not homogeneous across studies, but varies according to several factors, including differences in the data used to measure education and model specification. More importantly from the perspective of this contribution, they point out that heterogeneity may be due to the fact that education is conditional on health outcomes, and that these effects vary across countries.[2] Conversely, increasing education levels—above and beyond their effect on income—can also improve health outcomes. This implies that, in general, education, health and growth are all determined simultaneously, as documented empirically by Finlay (2007) in a cross-country study.

At the same time, there is significant evidence suggesting that late life health is the outcome of a cumulative process of exposure to health risks in childhood, especially infectious diseases in the first years of life. By determining health outcomes later in life, health in childhood may therefore play a critical role in the determination of health and socioeconomic status in adulthood (see Case et al., 2005; Smith, 2009). There is therefore *health persistence*, which represents an important source of dynamics in a growth context. The link between childhood health and

health in adulthood can operate in the opposite direction as well. Indeed, there is evidence suggesting that cognitive and physical impairments of children may begin *in utero*, due to inadequate nutrition and poor health of the mother—illustrated most dramatically through mother-to-child transmission of HIV.[3] According to estimates reported by Bloom and Canning (2005), for instance, an estimated 30 million infants are born each year in developing countries with impaired growth due to poor nutrition during fetal life. More generally, the health of parents may also affect the health of their children, after they are born, to the extent that it determines (as noted earlier) their own physical and mental ability to provide child care.[4]

This chapter examines the interactions between education and health, as two key components of human capital, and their impact on economic growth, in an overlapping generations (OLG) model.[5] In the model, education and health outcomes are jointly determined, taking into account the possible externalities alluded to earlier. At the same time, the key difference between these components is that education (or knowledge) can be accumulated without bounds, whereas health status cannot.[6] In addition, the chapter accounts for the fact that (as also noted earlier) parents' health affects directly the health of their children (intergenerational transmission), and that health outcomes in childhood may affect health outcomes in adulthood (intragenerational transmission). As a result, health status displays persistence, as in Osang and Sarkar (2008), de la Croix and Licandro (2013), and Agénor (2015) for instance. In addition, in the model the provision of education or health services, while complementary to each other at the microeconomic level, requires the use of public resources. At the macroeconomic level, there is therefore an inherent trade-off between the provision of these two types of services. Understanding the nature of these trade-offs, and the role that externalities may play, is thus critical for public policy.

The remainder of the chapter is organized as follows. Section 8.2 provides a discussion of the interactions between education and health. Section 8.3 presents a 3-period OLG model that captures the key linkages between education and health as well as the persistence in health. In the model both components of human capital, like conventional economic

goods, require a variety of inputs to be produced. Section 8.4 solves for the optimal household decision rules and derives the balanced growth path. Section 8.5 studies the impact of public policy on education and health outcomes, as well as economic growth. Section 8.6 endogenizes the adult survival rate and considers the extent to which multiple growth paths may emerge.[7] The issue of whether an increase in public spending in health or education may allow a country to escape from a low-growth equilibrium is also addressed. The last section of the chapter offers some concluding remarks.

8.2 Background

As noted earlier, health and education are largely interlinked in their contribution to growth because they both contribute to human capital accumulation. This section provides a more detailed review of the recent evidence on the two-way interactions between health and education.[8] The causal link from health to education is first discussed whereas the reverse link is taken up next.

8.2.1 Impact of Health on Education

It is now well recognized that health can have a sizable effect on education and the accumulation of knowledge.[9] Indeed, good health and nutrition are essential prerequisites for effective learning by children (see Glewwe and Miguel, 2008). In a study based on Ecuadorian data, Paxson and Schady (2007) found that health measures such as height for age and weight for age are positively related to language development (a measure of cognitive ability). Healthier children do better in school and this in turn may promote health-related knowledge (see Behrman, 2009).

Improving the health of individuals also increases the effectiveness of education. In Bangladesh, the Food for Education program, which provided a free monthly ration of food grains to poor families in rural areas if their children attended school, was highly successful in increasing school enrollment (particularly for girls), promoting attendance, and

reducing dropout rates (see Ahmed and Arends-Kuenning, 2006). In a study focusing on rural Guatemala, Maluccio et al. (2009) found that improving nutrition during early childhood had a substantial impact on adult educational outcomes. Field et al. (2009), in a study of Tanzania, found that intensive iodine supplementation *in utero* had a large impact on cognition and human capital—particularly for girls. For their part, Bharadwaj et al. (2013) found that in Chile, children who received extra medical care at birth had not only lower mortality rates but subsequently also higher test scores and grades in school.

Conversely, inadequate nutrition, which often takes the form of deficiencies in micronutrients, reduces the ability to learn and study. Zinc deficiency, in particular, impairs brain and motor functions. Poor nutritional status can therefore adversely affect children's cognitive development, and this may translate into poor educational attainment, as documented in Behrman (1996, 2009), Miguel (2005), Schultz (2005), and Bundy et al. (2006).[10] For instance, as documented by Ampaabeng and Tan (2013), differences in intelligence test scores in Ghana can be robustly explained by the differential impact of a famine that occurred in 1983 in different parts of the country. Moreover, the impact was most severe for children under two years of age during the famine. Poor health, in the form of respiratory infections for instance, is also an important underlying factor for low school enrollment, absenteeism, and high dropout rates. Inadequate diets may have adverse effects on mental health as well, and therefore the ability to raise children (Mental Health Foundation, 2006).

The role of early childhood health on schooling outcomes is also well illustrated in studies focusing on the incidence of malaria. Bundy et al. (2006) found that in Tanzania, the use of insecticide-treated bed nets reduced the incidence of malaria and increased attendance rates in schools. Using data on the malaria-eradication campaigns in the United States (circa 1920) and in Brazil, Colombia, and Mexico (circa 1955), Bleakley (2010a) found that, relative to nonmalarious areas, cohorts born after eradication of the disease had higher income as adults than the preceding generation—presumably as a result of higher human capital. Conversely, McCarthy et al. (2000) found that malaria morbidity, viewed

as a proxy for the overall incidence of malaria among children, had a negative effect on secondary enrollment ratios. Related results are obtained by Thuilliez (2009), using cross-country regressions.

Similarly, early vaccination appears to have a significant effect on subsequent learning outcomes. In western Kenya, deworming treatment improved primary school participation by 9.3 percent, with an estimated 0.14 additional years of education per pupil treated (see Miguel and Kremer, 2004). In the same vein, Bleakley (2007) found that deworming of children in the American South had an effect on their educational achievements while in school, whereas Bloom et al. (2005) found that children vaccinated against a range of diseases (including measles, polio, and tuberculosis) as infants in the Philippines performed better in language and IQ scores at the age of ten, compared to unvaccinated children—even within similar social groups. Bundy et al. (2006), in their overview of experience on the content and consequences of school health programs (which include for instance treatment for intestinal worm infections), emphasized that these programs can raise productivity in adult life not only through higher levels of cognitive ability but also through their effect on school participation and years of schooling attained.

Another channel through which health can improve education outcomes and spur growth is through higher life expectancy and changes in time allocation within households. Increases in life expectancy tend to raise the incentive to invest in education (in addition to increasing the propensity to save, as discussed later) because the returns to schooling are expected to accrue over longer periods. Thus, at the individual level, to the extent that spending on health lengthens planning horizons, it may also raise the returns (as measured by the discounted present value of wages) of greater resources devoted to education. In a study of Sri Lanka between the period 1946 and 1953, Jayachandran and Lleras-Muney (2009) found that a reduction in maternal mortality risk increases both female life expectancy and female literacy. In a study of Brazil, Soares (2006) also found that higher longevity is associated with improved schooling outcomes. These results are consistent with the view that longer life expectancy encourages investment in education.

The evidence also suggests that intrafamily allocations regarding school and work time of children tend to be adjusted in the face of disease within the family; in turn, these adjustments may influence education outcomes and thus the rate of economic growth. As discussed by Corrigan et al. (2005), for instance, when parents become ill, children may be pulled out of school to care for them, take on other responsibilities in the household, or work to support their siblings. Hamoudi and Birdsall (2004) provided evidence that AIDS reduced schooling rates in sub-Saharan Africa. These results are consistent with the view that the risk that children may be infected by AIDS tends to deter parents from investing in their education, as argued by Bell et al. (2006). Put differently, an environment where there is great uncertainty about child survival may create a precautionary demand for children, with less education being provided to each of them. In turn, the lack of skilled labor may hamper economic growth, as illustrated by Arndt (2006) in his study of AIDS and growth in Mozambique.

Health in childhood may affect health and income in adulthood through education.[11] Pain, fatigue, and malnutrition—in addition to being a primary cause of child mortality, as documented by Pelletier et al. (2003)—can reduce the ability to concentrate and to learn. Illness can crowd out other activities that might be beneficial to child development. Some health conditions, such as attention-deficit hyperactivity disorder or deafness for instance, can also have a direct, negative impact on cognitive or verbal ability, respectively. Studies have shown indeed that education levels in adulthood are to a large extent already determined during childhood. Measures of child development, such as cognitive and verbal ability, predict fairly well measures of education outcomes in adulthood, such as earnings and employment (see Currie, 2000).[12] For instance, in a study of German data, Salm and Schunk (2008) found that gaps in child development between socioeconomic groups can be explained by differences in child health—specifically, 18.4 percent of the gap in cognitive ability and 64.8 percent of the gap in verbal ability between children of college educated parents and less educated parents can be attributed to poor initial health conditions.[13]

8.2.2 Impact of Education on Health

A significant body of research, at both the micro and macro levels, has also shown that better education can improve health outcomes. The positive effect of education on health (just like the effect of health on education) works to a significant extent through productivity and income; but there are other channels as well.

Several studies have found that where mothers are better educated, infant mortality rates are lower.[14] This is likely because better-educated women tend, on average, to be more aware or to have more knowledge about the health risks that their children face.[15] For developing countries in general, Smith and Haddad (2000) estimated that improvements in female secondary school enrollment rates are responsible for 43 percent of the 15.5 percentage point reduction in the child underweight rate recorded during the period 1970–1995.[16] In the cross-section regressions reported by McGuire (2006), average years of female schooling have a statistically significant impact on under-five mortality rates. For sub-Saharan Africa alone, it has been estimated that five additional years of education for women could reduce infant mortality rates by up to 40 percent (see Summers, 1994). For Niger specifically, researchers have found that infant mortality rates are lower by 30 percent when mothers have a primary education level, and by 50 percent when they have completed secondary education. In a study of Uganda, Keats (2018) found that women with more schooling have higher early child health investments and have less chronically malnourished children. In the same vein, Paxson and Schady (2007), in a study of Ecuador, found that the cognitive development of children aged 3–6 years is positively associated with the level of education of their mother. Of course, third factors could be at play as well; more educated women normally earn more (thereby allowing them to spend more on the health of their children) and are more likely to live in urban areas, where access to health facilities, or nutritional supplements, is easier. But in many instances the positive effect of education on health persists even after controlling for income, location and other socio-economic factors. Indeed, Wagstaff and Claeson (2004) found that an increase in female education reduces infant mortality and

raises the survival rate for children, even after controlling for income effects.

The diversity of factors through which parental schooling can affect child health outcomes is well illustrated in a study of Pakistan by Aslam and Aslam and Kingdon (2012). They considered several mechanisms through which parental schooling may promote better health outcomes (height and weight) for their children: educated parents' greater household income, exposure to media, literacy, labor market participation, health knowledge, and the extent of maternal empowerment within the home. They found that while father's education is positively associated with the immunization decision, mother's education is more critically associated with longer term health outcomes.[17]

Finally, there is also evidence suggesting that better educated individuals are more able to adopt healthy lifestyles and inspire their children to follow the same type of behavior (see Grossman and Kaestner, 1997; Silles, 2009; Mullahy and Robert, 2010). For instance, Cutler and Lleras-Muney (2008) found that, controlling for several factors, better educated people in the United Kingdom and the United States are less likely to be obese, less likely to smoke, and less likely to be heavy drinkers. The broader evidence that they review also suggests that increasing levels of education lead to different thinking and decision-making patterns. Conversely, a low level of education may also lead to maternal malnutrition, with dire consequences for children: inadequate intakes of nutrients during pregnancy can have irreversible effects on children's brain development, as noted earlier.

The foregoing discussion suggests that the causality between health and education can go in both directions, and that taking into account these interactions is essential to study their joint effect on economic growth. Some of the evidence reported earlier can indeed be interpreted from the perspective of bidirectional causality. The results of Kohler and Soldo (2004) for instance, who found in a study of Mexico that individuals with low levels of education have higher mortality rates than better-educated individuals, may also be due to the fact that the level of education varies positively with health status. The next section presents a formal analysis of the interactions between education and health and their impact on economic growth.

8.3 The Model

Consider an OLG economy where a single good is produced and individuals live (at most) for three periods: childhood, adulthood and old age. They accumulate knowledge in the first period, supply labor in the second, and retire in the third. The good can be either consumed in the period it is produced or stored to yield capital at the beginning of the following period. All individuals are endowed with one unit of time in each period of life. Schooling is mandatory; children therefore devote all their time to education and depend on their parents for consumption. In middle age, individuals become parents and allocate their time between child rearing and the labor market. In old age, all time is devoted leisure. The only source of income is therefore wages in the second period of life, which serves to finance consumption in adulthood and old age. Savings can be held only in the form of physical capital. Agents have no other endowments, except for an initial stock of physical capital, K_0 at time $t = 0$, which is held by an initial generation of retirees.

Reproduction is asexual. In adulthood each individual bears $n \geq 1$ children, who are born with the same innate abilities. Keeping children healthy and fostering their education involves a cost, in terms of the parent's time.[18] Children mature safely into adulthood. At the end of the second period of life, there is a non-zero probability of dying. For children, education and health status depend on the time parents allocate to rearing their offspring, on the parent's level of education or health, as well as access to public services. Health status in adulthood depends solely on the individual's health in childhood. There is therefore state dependence in health outcomes. This specification is consistent with the evidence discussed earlier on intragenerational health persistence.

In addition to individuals, the economy is populated by firms and an infinitely-lived government. Firms produce marketed goods using private capital and effective labor. The government spends on education, health, and some unproductive activities. All government services are provided free of charge. Only the wage income of adults is subject to taxation. The government cannot borrow and therefore must run a balanced budget in

each period. Finally, all markets clear and there are no debts or bequests between generations.

8.3.1 Individuals

At the beginning of their adult life in t, each individual born at $t-1$ bears 1 child. Population is thus constant. Raising a child involves a time cost; each parent devotes $\varepsilon_t^R \in (0,1)$ units of time to that activity, namely for home schooling and to take care of the child's health (breast feeding, taking children to medical facilities for vaccines, and so on). Adults also allocate time, in proportion ε_t^W, to working. The individual's time constraint is thus

$$\varepsilon_t^W + \varepsilon_t^R = 1. \tag{8.1}$$

By implication, although access to "out of home" health and education services *per se* are free, child rearing involves a cost in terms of foregone wage income and consumption.

Assuming that consumption of children is subsumed in their parent's consumption, an individual's expected lifetime utility at the beginning of period t is specified as

$$U = \ln c_t^{t-1} + \eta_E \ln e_t^C + \eta_H \ln h_t^C + p\frac{\ln c_{t+1}^{t-1}}{1+\rho}, \tag{8.2}$$

where c_{t+j}^{t-1} denotes consumption in period $t+j$, with $j = 0, 1$, $\rho > 0$ is the discount rate, and $p \in (0,1)$ the probability of survival from adulthood to old age, which is taken as constant for the moment. Children's education, e_t^C, and health, h_t^C, matter to parents. Coefficients η_E and η_H are both positive and measure the individual's relative preference for children's education and health, respectively.[19]

The period-specific budget constraints are

$$c_t^{t-1} + s_t = (1-\tau)a_t \varepsilon_t^W w_t, \tag{8.3}$$

$$c_{t+1}^{t-1} = (1 + r_{t+1})s_t / p, \tag{8.4}$$

where a_t is individual labor productivity, w_t the wage rate, s_t saving, r_{t+1} the rental rate of capital, and $\tau \in (0,1)$ the tax rate. Equation (8.4) indicates that individuals consume at period $t+1$ with probability p.[20]

Combining these two equations yields the consolidated budget constraint

$$c_t^{t-1} + \frac{p c_{t+1}^{t-1}}{1 + r_{t+1}} = (1-\tau)a_t \varepsilon_t^W w_t. \tag{8.5}$$

Each individual maximizes (8.2), subject to (8.5), with respect to c_t^{t-1}, c_{t+1}^{t-1} and ε_t^R, with ε_t^W solved for residually from (8.1). In a second step, parents allocate rearing time between education and health, in fixed proportions $\chi \in (0,1)$ and $1-\chi$, respectively. Thus, along the lines suggested by Guryan et al. (2008), time spent with children is an investment in their education and health outcomes.

8.3.2 Firms

There is a continuum of identical firms, indexed by $i \in (0,1)$. They produce a single nonstorable good, which is used either for consumption or investment. Production requires the use of effective labor and physical capital, which firms rent from the currently old agents.

Assuming a Cobb-Douglas technology, the production function of firm i takes therefore the form

$$Y_t^i = (A_t \varepsilon_t^{W,i} N_t^i)^{\beta} \left(\frac{K_t^i}{\bar{N}^{\phi_N}}\right)^{1-\beta}, \tag{8.6}$$

where K_t^i denotes the firm-specific stock of physical capital, A_t average, economy-wide labor productivity, N_t^i the number of adult workers employed by firm i, $\varepsilon_t^{W,i}$ the time allocated by each individual to work at firm i, and $\beta \in (0,1)$. Thus, production exhibits constant returns to scale in firm-specific inputs, effective labor $A_t \varepsilon_t^{W,i} N_t^i$ and capital K_t^i. However, there is a population externality; the greater the size of the adult population, $\bar{N}$, the lower the productivity of each firm's capital stock. This congestion effect reflects the possibility that if more workers use fixed physical assets (such as roads or electricity) it becomes more difficult for each firm to use them (due to traffic jams, which prevent trucks from moving around to deliver goods, or to frequent power outages, which limit the use of machines and equipment). The magnitude of this congestion effect is measured by the parameter $\phi_N \geq 0$.

Markets for both physical capital and labor are competitive. Each firm's objective is to maximize profits, Π_t^i, with respect to raw labor and capital, taking A_t, $\varepsilon_t^{W,i}$ and input prices as given:

$$\max_{N_t^i, K_t^i} \Pi_t^i = Y_t^i - r_t K_t^i - w_t A_t \varepsilon_t^{W,i} N_t^i.$$

Profit maximization yields

$$w_t = \beta Y_t^i / A_t \varepsilon_t^{W,i} N_t^i, \quad r_t = (1-\beta) Y_t^i / K_t^i, \tag{8.7}$$

which implies that inputs are paid at their marginal product.

Given that all firms are identical, in a symmetric equilibrium $N_t^i = N_t = \bar{N}$ and $K_t^i = K_t, \forall i$. Thus, these conditions become

$$w_t = \beta Y_t / A_t \varepsilon_t^{W,i} \bar{N}, \quad r_t = (1-\beta) Y_t / K_t. \tag{8.8}$$

Average productivity is given by

$$A_t = E_t h_t, \tag{8.9}$$

where E_t is the average stock of knowledge and h_t average adult health status. Thus, both education and health affect individual productivity. For tractability, a simple multiplicative form is used.[21]

Because the number of firms is normalized to 1, aggregate output is given by, using (8.9),

$$Y_t = \int_0^1 Y_t^i = \bar{N}^{\beta-\phi_N(1-\beta)}\left(\frac{E_t}{K_t}\right)^{\beta} h_t^{\beta} K_t.$$

To eliminate the scale effect associated with population requires setting $\beta - \phi_N(1-\beta) = 0$, or equivalently $\phi_N = \beta/(1-\beta)$. Consequently, using (8.1),

$$Y_t = (1-\varepsilon_t^R)^{\beta} x_t^{\beta} h_t^{\beta} K_t, \tag{8.10}$$

where $x_t = E_t/K_t$ is the knowledge-physical capital ratio (or, for short, the knowledge-capital ratio). Given that, as shown later, both x_t and h_t, as well as ε_t^R, are constant in the steady state, the model is linear in the physical capital stock and exhibits therefore endogenous growth.

8.3.3 Schooling

The schooling technology depends on several inputs. First, it depends on the time allocated to education in childhood; as noted earlier, children must allocate all of their time to education, and this is normalized to unity. Second, knowledge accumulation is affected by the time allocated by parents to child rearing. As noted earlier, a sequential process is considered: parents determine first the total amount of time allocated to rearing their children, ε_t^R, and then subdivide that time into a fraction $\chi \in (0,1)$ allocated to home schooling and $1-\chi$ to health care.

Third, knowledge accumulation depends on government spending on education, G_t^E, per child. Given that each individual has only one child, the total number of children is simply equal to the adult population, $\bar{N}$. Fourth, it depends on the level of education of the parent. Because individuals are identical within a generation, parental education is taken

to be equal to the average stock of knowledge of the current generation. Finally, to capture the health externality discussed earlier, schooling depends on how healthy the individual is in childhood.

Normalizing the adult population to unity, knowledge acquired in childhood, e_t^C, is thus given by

$$e_t^C = (G_t^E)^{\nu_1} E_t^{1-\nu_1} (\chi \varepsilon_t^R)^{\nu_2} (h_t^C)^{\nu_3}, \tag{8.11}$$

where $\nu_1 \in (0, 1)$ and $\nu_2, \nu_3 > 0$. For tractability, constant returns to scale are imposed on the education technology with respect to public spending per child and average parental knowledge. In addition, as in Hazan and Zoabi (2006) for instance, education and health are gross complements in the production of knowledge ($\partial^2 e_t^C / \partial E_t \partial h_t^C > 0$).

In adulthood, individuals do not engage in additional learning.[22] Assuming for simplicity no depreciation and full persistence in learning, the knowledge that each individual has in the second period of life, e_{t+1}, is therefore

$$e_{t+1} = e_t^C. \tag{8.12}$$

Substituting (8.11) in (8.12) yields

$$e_{t+1} = \left(\frac{G_t^E}{E_t}\right)^{\nu_1} E_t (\chi \varepsilon_t^R)^{\nu_2} (h_t^C)^{\nu_3}, \tag{8.13}$$

Thus, because a parent's education affects his children's learning ability, there is serial dependence in knowledge. In addition, knowledge in adulthood also depends on health status in childhood.

8.3.4 Health Status

The health status of a child, h_t^C, depends on the amount of time allocated by each parent to rearing them, the average parent's level of education and health, E_t and h_t, respectively, and government expenditure on health, G_t^H, per child. This last effect captures for instance the impact of public

spending on nutritional programs in schools and vaccination campaigns, which reduce children's vulnerability to disease and improves their health. Thus, health status in childhood is given by

$$h_t^C = (\frac{G_t^H}{K_t^{\phi_H}})^{\theta_1}[(1-\chi)\varepsilon_t^R]^{\theta_2} E_t^{\theta_3} h_t^{\theta_4}, \tag{8.14}$$

where all coefficients are positive. The externality associated with parental education is captured by θ_3, whereas the external (intergenerational) effect associated with parental health is captured by θ_4. With $\theta_4 < 1$, parental health exerts a diminishing marginal effect on a child's health. In addition, the supply of public health services is congested by the stock of physical capital, with a congestion parameter $\phi_H > 0$.[23] As before, this congestion effect could represent the effect of an intensive use of a fixed stock of public physical assets (such as roads or electricity) to produce goods, which makes it more difficult to access health facilities. Alternatively, the scaling of G_t^H by $K_t^{\phi_H}$ can be viewed as capturing the fact that greater economic activity (as proxied by the capital stock) has potentially adverse effects on children's well-being (as a result of air pollution for instance), which in turn mitigates the benefits of public spending on health.[24]

Equation (8.15) can be rewritten as

$$h_t^C = (\frac{G_t^H}{K_t})^{\theta_1}[(1-\chi)\varepsilon_t^R]^{\theta_2}(\frac{E_t}{K_t})^{\theta_3} h_t^{\theta_4} K_t^{\theta_1+\theta_3-\phi_H\theta_1}.$$

To ensure that health status is stationary, the restriction $\phi_H = (\theta_1 + \theta_3)/\theta_1$ is also imposed. The above expression therefore becomes

$$h_t^C = (\frac{G_t^H}{K_t})^{\theta_1}[(1-\chi)\varepsilon_t^R]^{\theta_2}(\frac{E_t}{K_t})^{\theta_3} h_t^{\theta_4}. \tag{8.15}$$

To capture the idea (discussed in the introduction) that cognitive deficits in early life may be impossible to reverse, and that health does not deteriorate over time, the health status of adults is assumed to depend

only on their health status in childhood:

$$h_{t+1} = h_t^C. \tag{8.16}$$

Substituting (8.15) in (8.16) yields

$$h_{t+1} = \left(\frac{G_t^H}{K_t}\right)^{\theta_1}[(1-\chi)\varepsilon_t^R]^{\theta_2} x_t^{\theta_3} h_t^{\theta_4}. \tag{8.17}$$

In the steady state, the public health spending-capital ratio, time allocated to child rearing, and the knowledge-capital ratio are all constant; health status is thus stationary as well. Knowledge, by contrast, grows without bounds. This is the fundamental difference, alluded to earlier, between education and health as sources of human capital.

8.3.5 Government

The government taxes only adults at the constant rate $\tau \in (0,1)$ and spends a total of G_t^E on education, G_t^H on health, and G_t^U on other (unproductive) items. It cannot issue bonds and must therefore run a balanced budget:

$$G_t = G_t^E + G_t^H + G_t^U = \bar{N}\tau A_t \varepsilon_t^W w_t, \tag{8.18}$$

Shares of spending are constant fractions of revenues:

$$G_t^h = \upsilon_h \bar{N}\tau A_t \varepsilon_t^W w_t, \quad h = E, H, U \tag{8.19}$$

where $\upsilon_h \in (0,1)$. Combining (8.18) and (8.19) therefore yields

$$\sum \upsilon_h = 1. \tag{8.20}$$

In sum, the model captures the possible bidirectional externalities associated with health and education, discussed in the previous section, through the parameters ν_3 and θ_3. If $\nu_3 = 0$, health generates no benefit

in terms of childhood education, whereas if $\theta_3 = 0$ knowledge has no benefit in terms of health outcomes. Through the parameter θ_4, the model captures also intergenerational persistence in health.

8.3.6 Market Clearing and Equilibrium

Given the assumption of full depreciation of the stock of physical capital, the asset market-clearing condition requires tomorrow's capital stock to be equal to today's aggregate savings:

$$K_{t+1} = \bar{N} s_t. \tag{8.21}$$

The following definition may therefore be proposed:

Definition 8.1 A competitive equilibrium for this economy is a sequence of prices $\{w_t, r_t\}_{t=0}^{\infty}$, income and time allocations $\{c_t^{t-1}, c_{t+1}^{t-1}, s_t, \varepsilon_t^R\}_{t=0}^{\infty}$, physical capital stock $\{K_{t+1}\}_{t=0}^{\infty}$, knowledge stock $\{E_{t+1}\}_{t=0}^{\infty}$, health status of children and adults $\{h_t^C, h_t\}_{t=0}^{\infty}$, a constant tax rate $\tau$and constant spending shares υ_E, υ_H such that, given the initial stocks K_0, $E_0 > 0$,and health status h_0, individuals maximize utility, firms maximize profits, markets clear, and the government budget is balanced.

In equilibrium, individual productivity must also be equal to the economy-wide average productivity, so that $a_t = A_t$, and similarly for knowledge, so that $e_t = E_t$. The following definition characterizes the balanced growth path:

Definition 8.2 A balanced growth equilibrium is a competitive equilibrium in which c_t^{t-1}, c_t^{t-1}, A_t, E_t, K_t, Y_t and w_t all grow at the constant endogenous rate $1 + \gamma$, the rate of return on private capital is constant, and health status is constant.

8.4 Steady-State Growth

The solution of the individual's maximization problem is provided in the Appendix. It shows that in equilibrium,

$$\tilde{\varepsilon}^R = \frac{(\eta_E \nu_2 + \eta_H \theta_2)(1-\sigma)}{1+(\eta_E \nu_2 + \eta_H \theta_2)(1-\sigma)} < 1, \qquad \tilde{\varepsilon}^W = 1 - \tilde{\varepsilon}^R, \tag{8.22}$$

where σ is the marginal propensity to save, defined as

$$\sigma = \frac{p}{(1+\rho)+p} < 1. \tag{8.23}$$

From these solutions, it can be shown that an increase in the survival probability, p, raises the savings rate, s, lowers time allocated to child rearing, $\tilde{\varepsilon}^R$, and raises time allocated to market work, $\tilde{\varepsilon}^W$. The first result is fairly standard and consistent with the empirical evidence on longevity.[25] Through a life-cycle effect, a higher adult survival rate dictates a need for higher savings to finance consumption in old age, and thereby has a positive effect, *ceteris paribus*, on the incentive to save in adulthood, that is, the savings *rate*. At the same time, an increase in the survival rate leads, *ceteris paribus*, to less total time allocated to caring for children, as in Zhang and Zhang (2005), for instance. Thus, parents also increase the *level* of their savings by allocating more time to market work.

The dynamic system driving the economy is also derived in the Appendix, in terms of two variables: health status in adulthood, h_t, and the knowledge-capital ratio, $x_t = E_t/K_t$. Specifically, the model can be condensed into a first-order linear difference equation system in $\hat{h}_t = \ln h_t$ and $\hat{x}_t = \ln x_t$ which (ignoring constant terms) can be written as

$$\begin{bmatrix} \hat{x}_{t+1} \\ \hat{h}_{t+1} \end{bmatrix} = \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix} \begin{bmatrix} \hat{x}_t \\ \hat{h}_t \end{bmatrix}, \tag{8.24}$$

where

$$a_{11} = 1 - \beta + \phi_1,$$

$$a_{12} = \phi_2 - \beta,$$

$$a_{21} = \beta\theta_1 + \theta_3 > 0, \quad a_{22} = \beta\theta_1 + \theta_4 > 0,$$

and

$$\phi_1 = -(1-\beta)(\nu_1 + \theta_1\nu_3) + (\theta_1 + \theta_3)\nu_3 \gtrless 0,$$

$$\phi_2 = \beta(\nu_1 + \theta_1\nu_3) + \theta_4\nu_3 > 0.$$

As also shown in the Appendix, the balanced-growth rate of output per worker is given by

$$1 + \gamma = \beta\sigma(1-\tau)(1-\tilde{\varepsilon}^R)^{\beta}\tilde{x}^{\beta}\tilde{h}^{\beta}, \tag{8.25}$$

where $\tilde{x}$ and $\tilde{h}$ are the steady-state values of x_t and h_t, which are solutions of the system

$$\tilde{x} = \left\{ \frac{\Lambda_1}{\beta\sigma(1-\tau)} (\tilde{\varepsilon}^R)^{\nu_2+\theta_2\nu_3} (1-\tilde{\varepsilon}^R)^{\beta[(\nu_1+\theta_1\nu_3)-1]} \tilde{h}^{\phi_2-\beta} \right\}^{1/(\beta-\phi_1)}, \tag{8.26}$$

$$\tilde{h} = [\Lambda_2(1-\tilde{\varepsilon}^R)^{\beta\theta_1}(\tilde{\varepsilon}^R)^{\theta_2}\tilde{x}^{\beta\theta_1+\theta_3}]^{1/[1-(\beta\theta_1+\theta_4)]}, \tag{8.27}$$

where

$$\Lambda_1 = (\upsilon_E\tau\beta)^{\nu_1}(\chi)^{\nu_2}(\upsilon_H\tau\beta)^{\theta_1\nu_3}(1-\chi)^{\theta_2\nu_3}, \tag{8.28}$$

$$\Lambda_2 = (\upsilon_H\tau\beta)^{\theta_1}(1-\chi)^{\theta_2}, \tag{8.29}$$

In what follows it will be assumed that $\theta_4 \in (0,1)$ is not too large, to ensure that $1-(\beta\theta_1+\theta_4) > 0$.[26] But to make further progress, alternative cases regarding the externality parameters θ_3 and ν_3 must be considered.

Case 1. If there are *no externalities* of any sort, that is, $\theta_3 = \nu_3 = 0$, then $\phi_1 = -(1-\beta)\nu_1 < 0$, so that $\beta - \phi_1 > 0$ and, given that $\nu_1 \in (0,1)$, $\phi_2 - \beta = \beta(\nu_1 - 1) < 0$.

Case 2. If there is *only an education externality for health*, that is, $\nu_3 = 0$ and $\theta_3 > 0$, then again $\phi_1 = -(1-\beta)\nu_1 < 0$, $\beta - \phi_1 > 0$, and $\phi_2 - \beta = \beta(\nu_1 - 1) < 0$.

Case 3. If there is *only a health externality for education*, that is, $\nu_3 > 0$ and $\theta_3 = 0$, then $\phi_1 = -(1-\beta)(\nu_1+\theta_1\nu_3)+\theta_1\nu_3\beta(\nu_1+\theta_1\nu_3)-\nu_1 \gtrless 0$, $\beta - \phi_1 = -\beta[(\nu_1 + \theta_1\nu_3) - 1] + \nu_1 \gtrless 0$, and $\phi_2 - \beta = \beta[(\nu_1 + \theta_1\nu_3) - 1] + \theta_4\nu_3 \gtrless 0$.

Case 4. If *both types of externalities* are present, that is, $\nu_3, \theta_3 > 0$, then in order to have $\beta - \phi_1 > 0$, it must be that $(\theta_1 + \theta_3)\nu_3 < \beta + (1-\beta)(\nu_1 + \theta_1\nu_3)$, whereas for $\phi_2 - \beta < 0$ it must be that $\nu_3 < \beta(1-\nu_1)/(\beta\theta_1 + \theta_4)$.

In what follows, the focus will be on the two opposite cases 1 and 4, assuming in the latter that the combination of ν_3 and θ_3 is such that both restrictions are satisfied.[27] To establish the signs of a_{11} and a_{12}, note that in Case 1,

$$a_{11} = 1 - \beta + \phi_1 = (1-\beta)(1-\nu_1) > 0,$$

$$a_{12} = \phi_2 - \beta = \beta(\nu_1 - 1) < 0,$$

whereas in Case 4, given that again $\beta - \phi_1 > 0$, then $-\beta + \phi_1 < 0$, and thus, $a_{11} = 1 - \beta + \phi_1 \gtrless 0$. And given that $\phi_2 - \beta < 0$ then $a_{12} < 0$.[28]

Equations (8.26) and (8.27) define the steady-state relationships between x_t and h_t. In both Cases 1 and 4, $\beta - \phi_1 > 0$ and $\phi_2 - \beta < 0$. Equation (8.26) defines a convex curve depicted as XX in Fig. 8.1, whose slope is negative and given by $(\phi_2 - \beta)/(\beta - \phi_1) < 0$. Similarly, Eq. (8.27) defines a curve depicted as HH, whose slope is positive and

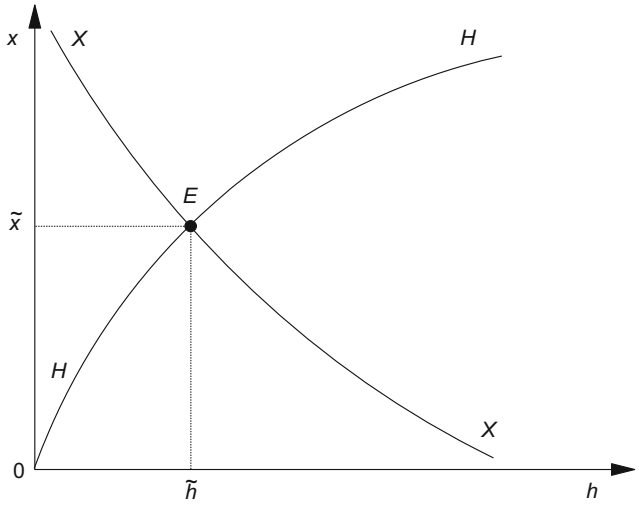

Fig. 8.1 Balanced growth equilibrium

given by $[1 - (\beta\theta_1 + \theta_4)]/(\beta\theta_1 + \theta_3) > 0$.[29] It is immediately clear from the shape of these curves that there is a unique equilibrium, located at point E. The knowledge-physical capital ratio and health status in adulthood are thus both constant in the steady state. As shown in the Appendix, for empirically plausible values of the parameters β, θ_4, ν_1 and θ_1, the equilibrium is stable in Case 1 (where there are no externalities, or, more generally, when these externalities are weak), as well as in Case 4.[30]

Before studying the effects of public policy in this setting, it is worth noting the conflicting effects of time allocated to child rearing, $\tilde{\varepsilon}^R$, on the steady-state solutions (8.26) and (8.27), and thus on the steady-state growth rate (8.25). On the one hand, increased time devoted to child rearing improves health and education (both in childhood and adulthood), which raise productivity, but on the other it reduces time allocated to market work. This trade-off becomes even more palpable when considering, as is done later, the case of an endogenous survival rate.

8.5 Public Policy

Equations (8.22) and (8.23), as well as (8.25)–(8.27) can be used to study the impact of changes in the shares of government spending on time allocation and growth, assuming that these increases are either budget neutral and financed by a cut in unproductive spending ($d\upsilon_h + d\upsilon_U = 0$, $h = E, H$) or instead by a cut in the other component of productive spending ($d\upsilon_E + d\upsilon_H = 0$).[31] In the latter case, there is a trade-off between the two components of expenditure, which can be internalized by solving for the growth-maximizing share of one of them. These issues are considered in turn, with a focus on steady-state effects rather than transitional dynamics.

8.5.1 Changes in Government Spending

Consider first a budget-neutral increase in public spending in education, financed by a cut in unproductive expenditure ($d\upsilon_E + d\upsilon_U = 0$). The results are illustrated in Fig. 8.2. Curve XX shifts to the right, whereas curve HH does not change. The equilibrium moves from point E to point E', implying that the outcome is both an improvement in health status and a higher knowledge-physical capital ratio. Consequently, the steady-state growth rate, as can be inferred from (8.25), increases unambiguously. The stronger the externalities associated with education and health, the stronger these effects are. Even though higher output means higher savings and investment, the increase in the stock of knowledge is always larger than the increase in the stock of physical capital. As a result, the knowledge-capital ratio always increases.

Consider now a budget-neutral increase in health spending, again financed by a cut in unproductive expenditure ($d\upsilon_H + d\upsilon_U = 0$). The results are illustrated in Fig. 8.3. Curve XX shifts to the right, whereas curve HH shifts down. However, there are now two cases to consider, depending on the magnitude of the shift in these curves. In both cases, while health status always improves, the net effect on education outcomes is ambiguous.

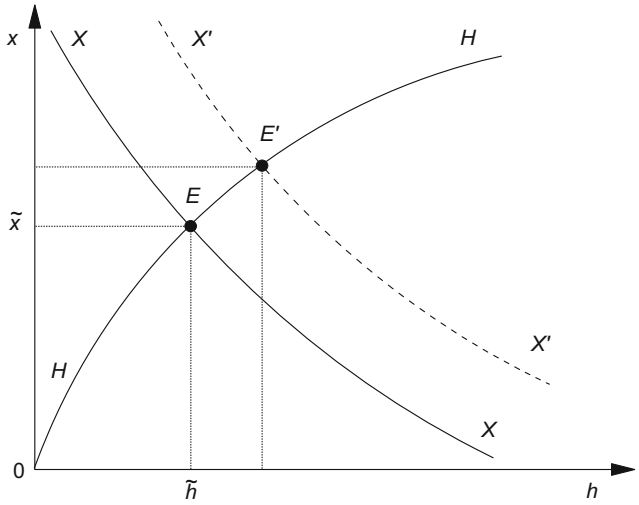

Fig. 8.2 Increase in government spending on education

Indeed, Scenario A depicts the case where XX shifts strongly, to $X'X'$, relatively to HH. The new equilibrium is at E', characterized (as in the case of higher spending on education), by an improvement in both health status and education. However, the figure also illustrates the case where XX shifts relatively little, to $X''X''$, so that the new equilibrium is at E''. In this scenario, which is likely to occur when the externality of health for education is low, health status improves but (relative) education outcomes deteriorate. Similarly, Scenario B corresponds to the case where HH can either shift a little (to $H'H'$, so that the new equilibrium is at E') or a lot (to $H''H''$, so that the new equilibrium is at E''). In the first scenario, both education and health outcomes improve, whereas in the second the only benefits are in terms of health status. When the effects on education outcomes are negative, the net effect on growth is ambiguous—even if changes in government spending are financed by cuts in unproductive spending. As shown in the Appendix, outcome E'' corresponds to the case where the health externality for education is weak, whereas outcome E' corresponds to the case where that externality is sufficiently strong. In addition, outcomes C in Scenario A, and outcomes C and C'' in Scenario

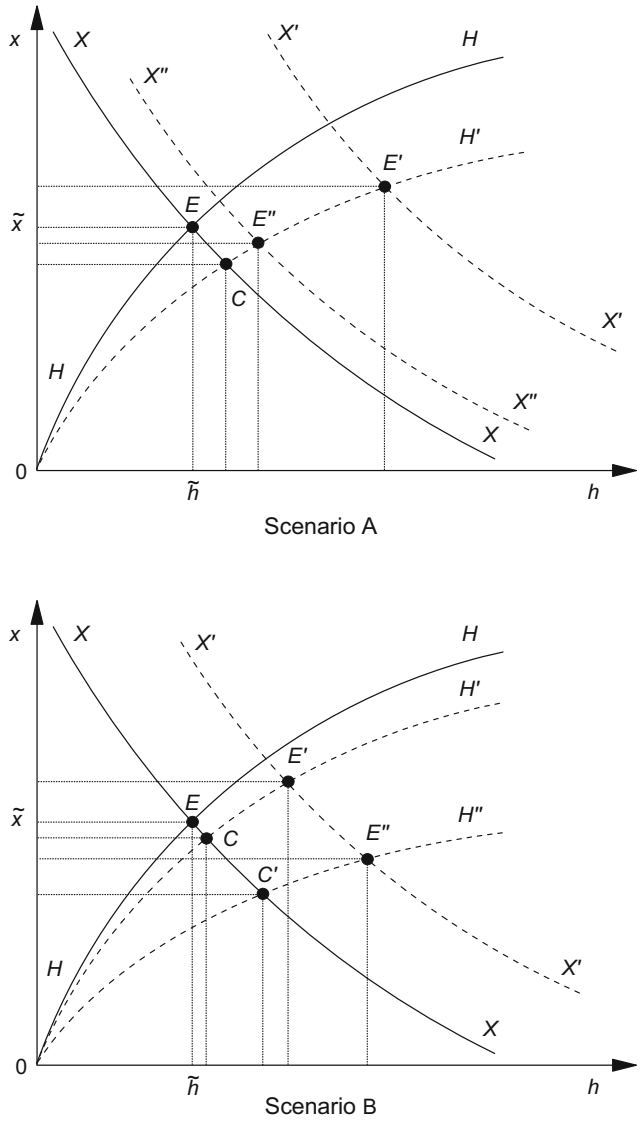

Fig. 8.3 Increase in government spending on health

B, correspond to the case where XX does not change at all, which is what occurs when there is no health externality for education, that is, $\nu_3 = 0$.

Intuitively, the reason why the effect on education outcomes (or, more precisely, the human-physical capital ratio) is ambiguous when health spending is increased is as follows. The direct effect of higher spending on health is an improvement in health status and productivity, which raises output, and therefore government spending across the board. This has a positive effect on both education and health outcomes. However, at the same time the increase in income raises savings and investment, and therefore the stock of physical capital as well. When the health externality for education is weak, the latter effect dominates, so that the human-physical capital ratio falls. By contrast, when the health externality for education is sufficiently strong, the increase in knowledge dominates, and the human-physical capital ratio increases. Thus, whether the net effect on the steady-state growth rate of output is negative or positive cannot be determined a priori. But the stronger the direct effect of health spending on health status, or the stronger the health externality, the more likely it is that an increase in government spending on health will lead to higher growth.

8.5.2 Growth-Maximizing Policy

Consider now the case where there is a trade-off in spending, that is, $d\upsilon_E + d\upsilon_H = 0$. In such conditions, even in the absence of externalities, the government faces a choice regarding how best to allocate its tax revenues. Specifically, suppose that that the government internalizes this trade-off by choosing a spending allocation that maximizes growth, rather than welfare.[32]

From (8.25) to (8.27), the growth-maximizing share of government spending on education is given by setting $d\ln(1+\gamma)/d\upsilon_E = 0$, that is,

$$\left.\frac{d\ln(1+\gamma)}{d\upsilon_E}\right|_{d\upsilon_E+d\upsilon_H=0} = \beta \left.\frac{d\ln\tilde{x}}{d\upsilon_E}\right|_{d\upsilon_E+d\upsilon_H=0} + \beta \left.\frac{d\ln\tilde{h}}{d\upsilon_E}\right|_{d\upsilon_E+d\upsilon_H=0} = 0.$$

As shown in the Appendix, the growth-maximizing share of spending on education is given by

$$\upsilon_E^* = \frac{b_{13}(1-b_{21})}{(b_{13}+b_{14})(1-b_{21})+b_{24}(1-b_{12})} < 1, \tag{8.30}$$

where, in both Cases 1 and 4,

$$b_{12} = -\frac{\phi_2-\beta}{\beta-\phi_1} > 0, \quad b_{13} = \frac{\nu_1}{\beta-\phi_1} > 0, \quad b_{14} = \frac{\theta_1\nu_3}{\beta-\phi_1} > 0,$$

$$b_{21} = -\frac{\beta\theta_1+\theta_3}{1-(\beta\theta_1+\theta_4)} < 0, \quad b_{24} = \frac{\theta_1}{1-(\beta\theta_1+\theta_4)} > 0,$$

which imply that

$$1-b_{12} = \frac{\phi_2-\phi_1}{\beta-\phi_1}, \quad b_{13}+b_{14} = \frac{\nu_1+\theta_1\nu_3}{\beta-\phi_1} > 0, \quad 1-b_{21} = \frac{1+\theta_3-\theta_4}{1-(\beta\theta_1+\theta_4)} > 0.$$

Formula (8.30) is quite complicated in general—even without education and health externalities, because the direct effects (as measured by ν_1 and θ_1) matter. To make further progress in assessing the role of externalities, a simple numerical exercise can be performed. Parameter values are set at $\beta = 0.65$ (a standard value), $\theta_4 = 0.6$ (to ensure that $1-(\beta\theta_1+\theta_4) > 0$), $\nu_1 = 0.55$ (as in Osang and Sarkar, 2008), $\theta_1 = 0.55$ (for symmetry), and $\nu_3 = \theta_3 = 0$ initially. Thus, as implied by (8.30), with no externalities of any sort, $\upsilon_E^* = 0.421$. Using the same values, with $\nu_3 = 0$ and $\theta_3 = 0.4$ yields $\upsilon_E^* = 0.593$, whereas with $\nu_3 = 0.4$ and $\theta_3 = 0$ the result is $\upsilon_E^* = 0.296$. With $\nu_3 = \theta_3 = 0.4$, then $\upsilon_E^* = 0.457$. More generally, Fig. 8.4 illustrates how the optimal share of spending on education changes when ν_3 and θ_3 vary between 0 and 1. What these results indicate is that the stronger the externality of education for health is, the larger the share of spending on education should be (or the lower the share of health spending should be). Conversely, the stronger the health externality for knowledge accumulation, the lower should be the share of spending on education. At the same time, the

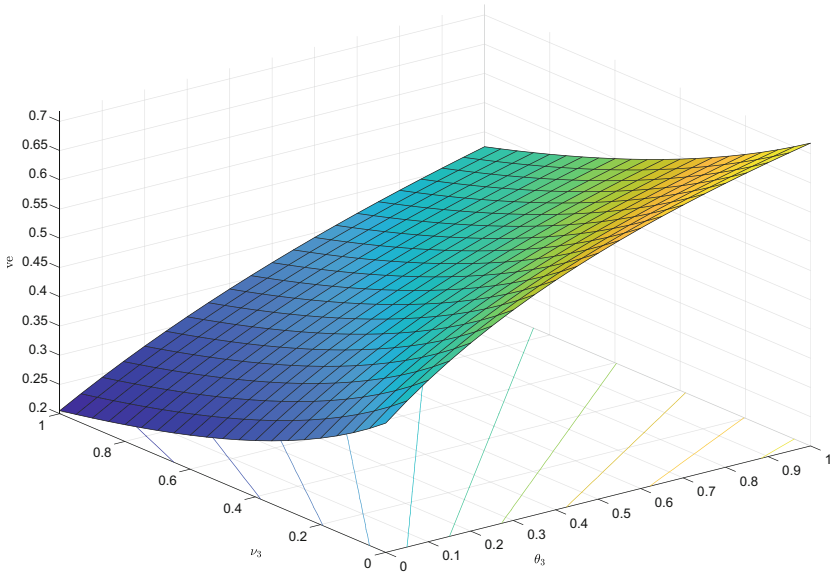

Fig. 8.4 Externalities and growth-maximizing share of spending on education

knowledge externality for health has a particularly strong impact on the growth-maximizing allocation; as θ_3 increases (for any given value of ν_3), the growth-maximizing share of spending on education increases rapidly—much more so than in the reverse scenario where ν_3 increases (for any given value of θ_3). Even when the externalities are equally strong, the effect is not symmetric; it is still optimal to spend a bit more on education. These results are also consistent with the fact that changes in education expenditure have unambiguous effects (as discussed earlier) on the knowledge-capital ratio and the rate of economic growth, in contrast with changes in health expenditure.

8.6 Endogenous Survival Rate

In the foregoing analysis the survival rate, p, was assumed exogenous. Suppose now, as in Finlay (2006), Tang and Zhang (2007), Osang and Sarkar (2008), and Agénor (2015) for instance, that life expectancy is endogenous and related directly to health status.[33] To capture this link,

one approach is to relate the survival rate directly to the individual's *own* health status. In solving their optimization problem, parents would then internalize the implications of their time allocation decisions. An alternative approach is to assume that the survival probability of any particular individual depends on *average* health status in the economy—which, in equilibrium, is of course the same for all individuals. Thus, when choosing their consumption and time allocation, agents would continue to take p as given and the solutions derived earlier continue to apply.

Suppose then that the adult survival rate is a piece-wise function defined as

$$p_t = \begin{cases} p_m < p_M \text{ for } h_t < h_L, \\ f(h_t) \text{ for } h_L \leq h_t < h_H, \\ p_M < 1 \text{ for } h_t \geq h_H, \end{cases} \tag{8.31}$$

where $f' > 0$ and $f'' < 0$. Thus, if health status is below h_L, the likelihood of surviving to old age is p_m. In the context of poor countries, this could reflect the fact that at first, improvements in health status do not translate into higher survival rates. As health status improves above that threshold, the relationship between p_t and h_t is positive and concave over the range (h_L, h_H).[34] It becomes constant again at p_M and $p_m < p_M < 1$ for values of health status above h_H. Put differently, beyond a certain point, further changes in health status have no effect on the probability to survive—perhaps reflecting the fact that there always remains a risk of accidental death.[35]

The implication of this analysis is as follows. Consider first the case where initially health status is at or below h_L, so that p is constant. Suppose that an ambitious increase in spending on health, financed by a cut in unproductive spending, leads to an improvement in health status—despite possibly having ambiguous effects on education outcomes, as discussed earlier—and that this increase is large enough to move the economy into the intermediate range (h_L, h_H). The survival rate therefore increases, which raises the savings rate and time allocated to market work, thereby promoting growth. However, in this setting rearing time is productive; it benefits both education and health outcomes. A reduction

in rearing time may therefore have adverse effects on these variables, despite higher government spending. Moreover, these effects may be magnified if externalities are strong. As a result, the net effect on growth can be ambiguous—even if the increase in public spending on health is offset by a cut in unproductive spending.[36] Conversely, it is also possible that the net effect on growth is positive; a health subsidy can help move the economy from a low-growth equilibrium to an equilibrium with a higher saving rate, higher life expectancy, and faster growth. If the direct effect on health status is positive, a strong externality of health on education would increase the likelihood of a transition from stagnation to growth. This result is thus consistent with those of Tang and Zhang (2007), albeit in a model where health and education externalities are not accounted for, and Hazan and Zoabi (2006), who emphasize (as is the case here) the importance of a sufficiently high degree of complementarity between health and education in the production of knowledge.[37] In addition, even if an increase in spending on health is financed by a cut in education expenditure, it is still possible for the net effect on growth to be positive if the health externality for education is strong.

Finally, it is worth noting that, with p constant, the values of ν_2 and θ_2 (which measure the effect of rearing time on education and health outcomes, respectively) do not matter for stability, as shown in the Appendix. However, when p is endogenously related to health status, h, it affects the savings rate and thus time allocated to child rearing, $\tilde{\varepsilon}^R$, which therefore becomes endogenous—and so does time allocated to market work. Consequently, the stability conditions discussed in the Appendix would be more complicated. In addition, this would affect the slopes of XX and HH in Figs. 8.1, 8.2 and 8.3, as well as the impact of changes in government spending on growth, as discussed earlier. Finally, with an endogenous survival rate, the solution of the growth-maximizing problem would also become highly nonlinear, thereby precluding the derivation of an explicit expenditure allocation rule, as in (8.30).

8.7 Concluding Remarks

Education and health are two important dimensions of human capital. The purpose of this chapter was to review the evidence on the interactions between these two dimensions, present an endogenous growth model that captures their interactions, and study the impact of public policy in that setting. A key feature of the model is that health is distinct from knowledge as a source of human capital because it cannot grow without bounds. In addition, consistent with the evidence, the model accounts for the possibility that causality can go both ways: policies that impact educational attainment may have a large effect on health outcomes, and vice versa. It also accounts for the well-documented facts that parental health effects the health of children at birth, and that health in late life is the outcome of a cumulative process of exposure to health risks in childhood.

The analysis showed that growth dynamics depend in critical ways on the externalities associated with knowledge and health. Depending on the strength of these externalities, an increase in government spending on health (financed by a cut in unproductive spending) may have ambiguous effects on economic growth. It was also shown that trade-offs between education and health spending can be internalized by setting the composition of expenditure so as to maximize the growth rate. All else equal, the stronger the health (education) externality in education (health), the smaller (larger) the share of spending on education should be. With an endogenous adult survival rate, multiple growth paths may emerge. A reallocation of public spending from education to health may shift the economy from a low-growth equilibrium to a high-growth path. However, if the time allocation effect associated with an endogenous increase in the survival probability—a reduction in time allocated to child rearing, due to life-cycle considerations, which on the one hand leads to an increase in time allocated to market work, but on the other may adversely affect education and health outcomes, and thus productivity—it is theoretically possible that an increase in government spending on health may have an adverse effect on growth.

The analysis presented in this chapter can be extended in several directions. First, at the empirical level, it would be useful to conduct a comprehensive cross-country econometric analysis of a simultaneous determination of schooling or education levels, health outcomes and economic growth. This would allow an assessment of the magnitude of the externalities associated with education and health, and provide some of the key parameters needed for a full-blown calibration of the model, in order to study numerically its properties.

Second, the fertility rate could be endogenized, to assess how changes in health outcomes can affect the decision to have children. Based on the results in Agénor (2015), one can infer what is likely to happen in that case: an increase in the survival rate (due to an improvement in health status, itself related to higher spending on health, as discussed earlier), would reduce the fertility rate and total time allocated to child rearing. The effect on unit rearing time, however, is likely to be ambiguous. Intuitively, the reduction in the fertility rate allows parents to allocate more time to each of them to improve their health—even though total time devoted to child rearing falls—in effect, substituting quality to quantity. Because changes in rearing time have persistent effects on health and education, they would also alter in significant ways the dynamics of the economy and whether multiple equilibria may emerge.

Finally, the analysis could be extended by introducing a gender dimension. This would allow, in particular, to study how the level of knowledge of each parent (which may differ due to discrimination, both at home and in the market place, against women) affects education and health outcomes for their children. For instance, Breierova and Duflo (2004), in a study of Indonesia, found that female and male education seem equally important factors in reducing child mortality. However, as noted earlier, Aslam and Kingdon (2012) in a study of Pakistan found that while a father's education is positively associated with the immunization decision, a mother's education is more critically associated with longer term health outcomes. Accounting for a gender dimension would help to consider how a broader set of policies can affect education and health outcomes, as well as, ultimately, economic growth.

Notes

1. Two comprehensive composite measures of human capital have been published recently. The first, by the Institute for Health Metrics and Evaluation, covers 195 countries, whereas the second, by the World Bank, covers 157 countries. See https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(18)31941-X/fulltext and http://www.worldbank.org/en/publication/wdr2019.
2. Among available studies, Baldacci et al. (2004), using cross-country regressions, found that health outcomes (as proxied by the under-five child mortality rate) have a statistically significant effect on school enrollment rates.
3. See Case et al. (2005), Paxson and Schady (2007), Smith (2009), and surveys by Behrman (2009) and Currie (2009), and Bleakley (2010b). Agénor et al. (2014) discuss the recent literature on both issues from a gender perspective.
4. See for instance the results of Powdthavee and Vignoles (2008) for Britain.
5. Tang and Zhang (2007) develop an OLG model with education and health but do not account for direct interactions between them. Tamura (2006) and Ricci and Zachariadis (2013) develop OLG models where schooling exerts external effects on health, in the form of a negative effect on adult mortality in the first case and a positive effect on longevity in the second. In the models of Galor and Mayer-Foulkes (2004) and Hazan and Zoabi (2006), health is, in addition to education, an input in the production of human capital. However, none of these contributions fully examines bidirectional effects, and the role of public policy, as is done here. Finally, Agénor (2011) do account for bidirectional effects in a continuous time, infinite-horizon setting, but in their model health is not stationary.
6. The requirement that health status be stationary is consistent with the specification in Osang and Sarkar (2008) and Agénor (2015).
7. The assumption that the survival rate is constant initially is for expositional reasons. It helps to clarify the role of externalities and the fundamental trade-off between spending on education and spending on health.
8. This section draws in part on Groot and van den Brink (2007), Agénor (2012, Chapter 3), and Grossman (2015).

9. See Bleakley (2010b) for an overview of the evidence on the impact of health and education.
10. Research at the National institute of Health in the United States has also shown that the children of mothers who did not eat food with ample omega-3 fatty acids had a lower IQ than children who did.
11. See Gertler and Zeitlin (1996, 2002), Mayer-Foulkes (2005), Miguel (2005), and surveys by Behrman (1996) and Currie (2009).
12. At the same time, child development may also be related to a child's socioeconomic background (see Taylor et al., 2004). If so then children from disadvantaged families may fall behind early in life and may be unable to catch up later.
13. See also Oreopoulos et al. (2008), who found in a study for Canada that poor infant health is a strong predictor of future education outcomes.
14. See Grossman and Kaestner (1997), Glewwe (1999, 2002), Chou et al. (2010), and the cross-country regressions of Baldacci et al. (2004) and Wagstaff and Claeson (2004).
15. As noted by Kohler and Soldo (2004) for instance, it is useful to separate two potential channels that may relate parents' education to their children's health and offsprings' late life health outcomes. The first is the father's education, which likely operates through economic circumstances (because fathers may be those who were the primary suppliers of economic resources in the family). The second is the mother's education, which operates through knowledge about health care and health behavior that are essential determinants of children's health outcomes.
16. Evidence that education affects health outcomes is also available for industrial countries; see for instance Cutler et al. (2006) and Altindag et al. (2011) for the United States.
17. The gender dimension of the interactions between education and health is further discussed in the concluding remarks.
18. For simplicity, the direct cost of schooling and the cost of keeping children healthy (medicines, and so on) are abstracted from.
19. If parents care equally about the health and education of their child, $\eta_E = \eta_H$.
20. Alternatively, it could be assumed that the saving left by individuals who do not survive to old age is confiscated by the government, which transfers them in lump-sum fashion to surviving members of the same cohort. The effective rate of return to saving would thus be $(1+r_{t+1})/p$,

which would yield an equation similar to (8.4). See Agénor (2012, Chapter 3) for a simple derivation.

21. A more general specification would be to set $A_t = E_t^{\chi} h_t^{1-\chi}$, where $\chi \in (0, 1)$.
22. This assumption is consistent with the evidence for Sub-Saharan Africa for instance, which suggests that only 6.8 percent of youth engage in tertiary education, compared to a world average of 30 percent (United Nations, 2016, p. 46).
23. See Osang and Sarkar (2008) and Agénor (2015). Of course, a similar argument could apply for the production of education services in (8.11). However, unlike health, knowledge does grow without bounds and the specification adopted in that equation is sufficient to ensure constant growth in the steady state.
24. Activity in that case could of course be measured by the level of final output, but given the linear relationship between Y_t and K_t implied by (8.10) the use of the latter is mainly a matter of convenience.
25. See for instance Blackburn and Cipriani (2002) and Zhang and Zhang (2005).
26. Using $\theta_1 = 0.55$, as in Osang and Sarkar (2008, Table 4) for instance, and a standard value of $\beta = 0.65$, this condition implies that θ_4 cannot be higher than 0.64.
27. Note that Case 2 is qualitatively very similar to Case 1. An exhaustive analysis of all cases would require a numerical calibration.
28. There are also intermediate cases, where one type of externality is high and the other low, which are ignored for the moment to facilitate the exposition of the graphical analysis.
29. Curve HH can be either concave or convex, depending on whether $[1 - (\beta\theta_1 + \theta_4)]/(\beta\theta_1 + \theta_3) \gtrless 1$. For illustrative purposes, it is shown as concave in Fig. 8.1. The difference between Cases 1 and 4, of course, is that the slopes of the two curves would be different, depending on the values of ν_3 and θ_3. However, this difference is inconsequential for a qualitative analysis.
30. Note that if $\phi_2 = \beta$ then $a_{12} = 0$ and system (8.24) is recursive; the dynamics are in terms of $\hat{x}_t$ only. Then stability requires $a_{11} = 1 - \beta + \phi_1 < 1$, or $\phi_1 < \beta$. If $\nu_3 = 0$, then this condition becomes $\beta(\nu_1 - 1) < 1$ which is always satisfied.
31. A variety of other experiments could also be conducted, such as for instance a change in parental time allocated between the health and edu-

cation needs of their children, that is, a change in χ. These experiments are left to the interested reader.

32. The focus on growth could be because the economy considered is poor and the priority is to raise living standards. More formally, differences between the growth- and welfare-maximizing solutions can lead to relatively small differences in growth rates, and possibly welfare levels. See Misch et al. (2013) for a discussion.
33. Some other contributions which focus on knowledge accumulation, such as Blackburn and Cipriani (2002), Cervellati and Sunde (2005), Castelló-Climent and Doménech (2008), for instance, have assumed that life expectancy is related to education. This can be justified by arguing that, as noted earlier, improved knowledge can lead to changes in lifestyle that may translate into better health outcomes. In the present setting, a more general approach, of course, would be to consider jointly education and health status as determinants of life expectancy. However, this would complicate significantly the analysis and would detract from the main contribution of this chapter.
34. A simple functional form for f could be the exponential function, that is, $p_t = 1 - 1/\exp(h_t)$.
35. As noted in Agénor (2015), in the model health status can be interpreted as a broad measure of health, such as the body mass index (BMI). From that perspective, the thresholds h_L and h_H can be thought of as the lower and upper bounds of the *BMI Chart*, which are commonly used to measure the ranges for *underweight* (up to h_L in the model), *healthy weight* (between h_L and h_H), and *overweight and obesity* (above h_H), based on a person's height. The last threshold is, in practice, further decomposed into separate thresholds for overweight and obesity but this does not matter from the perspective of this discussion.
36. Based on the previous discussion, if the increase in public spending on health is financed by a cut in spending on education, the possibility of an adverse effect on growth would be magnified.
37. Hazan and Zoabi (2006), however, focus on private expenditure on health and education, not public spending.

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9

Population and the Environment: The Role of Fertility, Education and Life Expectancy

Fabio Mariani, Agustín Pérez-Barahona, and Natacha Raffin

JEL Classification J11; O44; Q56

9.1 Introduction

This chapter is concerned with the interplay between population and environmental quality. In particular, we set up a theoretical framework suitable to jointly analyze two major endogenous forces of demographic change, namely fertility and life expectancy, and relate them to human capital and environmental conditions in a dynamic perspective. This complements the existing literature on environmental matters, which

F. Mariani (✉)
IRES/LIDAM, UCLouvain, Louvain-la-Neuve, Belgium
IZA, Bonn, Germany
e-mail: fabio.mariani@uclouvain.be

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has, in most cases, separately studied the implications of longevity and fertility—thus missing some interesting elements of analysis.

To do so, we build an extension of our previous work (Mariani et al., 2010), which allows us to reproduce and revisit some key results of the previous literature and gain new insight on the consequences of policy measures such as environmental taxes and educational subsidies on environmental dynamics and economic development. Among others, we highlight (1) the potential perverse effect of environmental regulation, (2) the possibility of relying on educational subsidies as an effective policy tool to alleviate the pressure on natural resources, and (3) the existence of poverty traps related to human capital and environmental quality.

A key starting point of our work is that environmental care betrays some concern for the future, be it one's own or that of forthcoming generations. Yet, the way people value future is crucially affected, among others, by their life expectancy: a higher longevity—or better health—makes people more sympathetic to future generations and/or their future selves, thus pushing them to invest more in environmental quality and in the quantity and/or quality of their offspring. These investment decisions drive the future dynamics of the economy, affecting its demographic and economic trajectory, and therefore the state of the environment. The causal link between life expectancy and socio-economic and demographic outcomes (environmental quality, human capital accumulation and population growth) may, however, also go the other way around, with the latter being a major factor affecting health and longevity. This intricate interplay, and the way it can be shaped by policy, lie at the center of our analysis.

A. Pérez-Barahona
THEMA, University of Cergy-Pontoise, Cergy-Pontoise, France
Ecole Polytechnique, Palaiseau, France
e-mail: agustin.perez-barahona@u-cergy.fr

N. Raffin
Normandie University, UNIROUEN, CREAM and EconomiX, University Paris Nanterre, Nanterre, France
e-mail: natacha.raffin@univ-rouen.fr

This chapter is organized as follows. After this Introduction, Sect. 9.2 reviews some of the most relevant research in the field. Section 9.3 presents and develops our model, whose dynamics and the related policy implications are analyzed in Sect. 9.4. Finally, Sect. 9.5 provide some concluding remarks.

9.2 Literature Review

Many economists have pointed out the importance of studying population growth and, in particular, how societies form their fertility decisions, as main determinants of environmental quality and natural resources. For instance, Dasgupta (1993) suggests the possibility of important childbearing externalities on common property assets such as natural resources. Very much in the same spirit as our contribution, Dasgupta (1995) also highlights that population growth can be both a cause and a consequence of underdevelopment and environmental degradation, thus recommending not to regard population as an exogenously given feature of the economy. This calls for the need of an appropriate analytical framework to study the interaction between endogenous population growth, environmental conditions and economic growth, and identify appropriate policy interventions.

In this context, Harford (1998) provides a stylized, two-period model of endogenous fertility, where population growth deteriorates the environment. Aggregate consumption induces pollution which, in turn, reduces individual welfare. With endogenous fertility, the usual Pigouvian tax on pollutant activities is not enough to decentralize the social optimum, since individuals should not only pay a tax covering the pollution cost generated by their own consumption, but also compensate for the externality created by their children. Harford's model, however, has important limitations since it overlooks the issues of economic growth and sustainability, and does not consider the quantity-quality trade-off inherent to parental decisions.[1]

A more recent paper by Schou (2002) proposes an alternative analytical framework, based on an infinite-time model with the quantity-quality trade-off. Like Harford (1998), he shows that implementing the social

optimum requires to tax fertility, in the presence of population externalities. By assuming that agents are perfectly altruistic, however, the model underestimates the effect of intergenerational externalities on environmental conditions related to fertility choices.

The hypothesis of perfect altruism is relaxed by de la Croix and Gosseries (2011), who propose an overlapping generations model showing how considering endogenous fertility is crucial to gauge the implications of pollution control. Their OLG framework allows for "warm glow" altruism and the quantity-quality trade-off , and consider Pigouvian taxes or tradable quotas in order to control pollution. The key finding of that paper is that pollution control induces a natalist bias, and therefore a pernicious effect on the environment, when fertility is endogenous. In fact, for a given technology, environmental taxes pushes agents to shift away from production to tax-free activities such as procreation, thus increasing the demographic pressure on the environment and gradually impoverishing successive generations.[2]

In a recent study, Gerlagh et al. (2018) consider an OLG model with dynastic altruism, whose problem is fairly similar to Schou (2002). They consider that pollution, modeled as a stock that accumulates over time, damages the productive potential of the economy—rather than hurting utility. They also assume a non-linear technology for human capital accumulation in the fashion of de la Croix and Doepke (2003), and deliver a message largely consistent with the previous literature: family planning should be part of environmental and climate policies.

While considering fertility as exogenous, another branch of the literature focuses specifically on the interaction between environmental conditions and life expectancy—motivated by the intriguing two-way causal relationship between those two variables.[3] Initially, this literature studied environmental care under uncertain lifetime without considering, however, that environmental conditions can in turn affect longevity (see for instance Ono and Maeda, 2001). On the opposite, Jouvet et al. (2010) explicitly consider the state of the environment as a decisive factor influencing mortality, but ignore the role of life expectancy in determining the environmental choices made by economic agents, namely their investment in environmental maintenance.

Building on this earlier literature, we have been the first (Mariani et al., 2010) to consider both directions of the aforementioned two-way causal relationship, and explore the implications of the interplay between life expectancy and environmental quality. In our paper, we integrated the well-established effect of environmental conditions on life expectancy with the idea that a higher longevity makes people more sympathetic to future generations and/or their future selves, thus making longer-lived agents more inclined to invest more in environmental quality. A major contribution of our analysis is that it can explain the existence of multiple equilibria: some countries might be caught in a low-life-expectancy/low-environmental-quality trap while the others reach a long-run equilibrium characterized by better living conditions and longer life expectancy.[4] This outcome is consistent with stylized facts relating life expectancy and environmental performance measures. In particular, using data on life expectancy and the EPI indicator, we have been able to show that environmental quality and life expectancy are bimodally distributed among countries.[5] In Mariani et al. (2010), we further show that our findings are robust to the introduction of growth dynamics based on physical or human capital accumulation, but do not explore the implications of endogenous fertility.

In a similar vein, Constant (2019) develops an OLG model where human capital is the source of endogenous growth and inequality among individuals, and emphasizes the role of heterogeneous effects of pollution on agents' longevity in determining multiple balanced growth paths. In this framework, she defines a new "kind" of environmental poverty trap also characterized by increasing inequality, and shows that taxing pollution can help the economy escape the environmental trap. Like Mariani et al. (2010), however, Constant (2019) abstracts from endogenous fertility decisions.

Introducing endogenous fertility choices in Mariani et al. (2010) constitutes the starting point of the present chapter. This allows us to bridge the gap between the two branches of literature mentioned above: that concerned with the interplay between life expectancy and the environment (to which our previous paper belongs) and the one dealing more specifically with the effect of population growth on the environment. In fact, with the possible exception of Jouvet et al. (2011),

the existing literature has proposed a separate treatment of the impact of fertility and life expectancy on the environment.[6] We believe that our original framework, by dealing with human capital driven growth (and thus the quality-quantity trade-off), can be fruitfully extended in order to analyze the complex fertility-longevity-environment nexus.

9.3 The Model

We set up a simple model in which agents allocate their income between consumption, environmental maintenance and children's education.

9.3.1 Demography, Preferences and Technology

We consider an infinite-horizon economy populated by overlapping generations of agents living for three periods: childhood, adulthood, and old age. Time is discrete and indexed by t. All decisions are taken in the adult period of life. Individuals live safely through the first two periods, while survival to the third period is subject to uncertainty. We also deal away with heterogeneity by assuming that agents are identical within each generation.

Adult agents maximize the following utility function

$$U_t = \ln c_t + \pi_t(\alpha \ln n_t h_{t+1} + \gamma \ln m_t), \tag{9.1}$$

where c_t denotes own consumption, m_t is the expenditure in environmental maintenance, while n_t and h_{t+1} refer to the quantity and quality (human capital) of children, respectively. In particular, n_t denotes the number of children per parent. The survival probability is $\pi_t \in (0, 1)$, whereas $\alpha > 0$ and $\gamma > 0$ represent the weight that agents give to children and environmental quality. The overall importance of those two arguments of utility is mediated by the survival probability as we assume that, contrary to current consumption, children and environmental quality are enjoyed in the third period of the agents' life. Note also that—for

the sake of simplicity—environmental quality is introduced in a joy-of-giving fashion: agents derive utility directly from the actions they take in order to improve the environment, rather than from future environmental quality itself.[7] In this sense, γ may be interpreted as a measure of some kind of environmental sensitivity.

The individual budget constraint is given by

$$(1-\tau)(1-\phi n_t)w_t h_t = c_t + m_t + (1-\sigma)n_t e_t. \tag{9.2}$$

On the left-hand side of the above equation, we have the agents' disposable income. In particular, the unit wage w_t multiplied by human capital h_t and working time $(1-\phi n_t)$ (where $\phi \in (0,1)$ is the time cost of raising each child) gives the gross income, while $\tau \in (0,1)$ is the tax rate on production that, as will become clearer below, can account for some kind of environmental regulation. On the right-hand side, we have total expenditure, where e_t is per-child educational expenditure, and $\sigma \in (0,1)$ stands for an educational subsidy.

Our agents face technological constraints. Following de la Croix and Doepke (2003), human capital accumulates according to

$$h_{t+1} = \delta(\theta + e_t)^{\beta} h_t^{1-\beta}, \tag{9.3}$$

where $\delta > 0$ is a productivity parameter, $\theta > 0$ prevents human capital from falling to zero in the absence of educational investment, whereas $\beta \in (0,1)$ regulates the importance of nature (parental human capital h_t) *vs.* nurture (education e_t) in human capital formation.

Output is produced using human capital (of adult individuals) only, such that it is given by

$$Y_t = (1-\tau)(1-\phi n_t)wh_t P_t, \tag{9.4}$$

where P_t is the size of the active (i.e. adult) population at time t, and w is a productivity parameter that, for simplicity, we assume to stay constant over time. In this stylized framework, the tax rate τ can be interpreted as an environmental policy instrument: by imposing cleaner production

standards, the government reduces the size of output that can be obtained for a given value of inputs, and thus the available income of workers employed in production.[8] Because of the structure of the production function, which is linear in labor, the profit-maximizing wage rate is

$$w_t = w. \tag{9.5}$$

As far as environmental quality X is concerned, it evolves over time according to the following dynamic equation:

$$X_t = (1-\eta)X_{t-1} + (\mu m_t - \xi c_t - \kappa(1-\tau)(1-\phi n_t)wh_t)P_t. \tag{9.6}$$

In this formulation we can see that environmental quality may be improved through maintenance, while it is harmed by two potential sources of pollution: consumption and production. The impact of these three variables depends on population size P_t, and is further mediated by the parameters $\mu, \xi, \kappa > 0$. As far as production is concerned, its environmental impact is attenuated by τ. We also assume that $\eta \in (0,1)$, such that environmental quality can be regarded as a stock (see among others, Mariani et al., 2010; Palivos and Varvarigos, 2017; Gerlagh et al., 2018).[9]

Note also that we make the simplifying assumption that people do not consume as old and as children. Introducing this possibility in the model would bring about further analytic complications, without significantly altering the qualitative results of our analysis.

Finally, adult population varies over time according to

$$P_{t+1} = n_t P_t. \tag{9.7}$$

Throughout this chapter we will often use the term "population" to refer to adult population, since adults are the only economic agents who take relevant economic decisions. At any date t, however, the true size of population (which sums over the three overlapping generations) is equal to $P_t(1 + n_t + \pi_t/n_{t-1})$.

9.3.2 Optimal Choices

Taking π_t as given, agents choose c_t, e_t, n_t and m_t so as to maximize their utility function (9.1) subject to Eqs. (9.2) and (9.3). From the first order conditions for e_t, n_t and m_t, we obtain:

$$e_t = \begin{cases} 0 & \text{if } h_t \leq \dfrac{(1-\sigma)\theta}{\beta\phi(1-\tau)w} \\ \dfrac{\beta\phi(1-\tau)wh_t - \theta(1-\sigma)}{(1-\beta)(1-\sigma)} & \text{if } h_t > \dfrac{(1-\sigma)\theta}{\beta\phi(1-\tau)w} \end{cases}, \tag{9.8}$$

$$n_t = \begin{cases} \dfrac{\alpha\pi_t}{\phi(1+(\alpha+\gamma)\pi_t)} & \text{if } h_t \leq \dfrac{(1-\sigma)\theta}{\beta\phi(1-\tau)w} \\ \dfrac{\alpha(1-\beta)(1-\tau)\pi_t wh_t}{(1+(\alpha+\gamma)\pi_t)(\phi(1-\tau)wh_t - \theta(1-\sigma))} & \text{if } h_t > \dfrac{(1-\sigma)\theta}{\beta\phi(1-\tau)w} \end{cases}, \tag{9.9}$$

$$m_t = \frac{\gamma(1-\tau)\pi_t wh_t}{1+(\alpha+\gamma)\pi_t}. \tag{9.10}$$

Residually, we can also obtain the value of consumption by replacing the above optimal choices into the budget constraint.[10]

From Eqs. (9.8) and (9.9), we can see that fertility is at its maximum as long as human capital is low enough, and parents choose not to educate their children ($e_t = 0$). If we restrict our attention to interior solutions ($e_t > 0$), we can look at some of the main determinants of the optimal choices.

Concerning the role of human capital, we have that $\partial e_t/\partial h_t > 0$ and $\partial n_t/\partial h_t < 0$, while $\partial m_t/\partial h_t > 0$. More educated parents, because of the higher opportunity cost of children, have lower fertility but invest more in the education of their offspring. Being richer, they also devote more resources to environmental maintenance—which has only a monetary cost and does not detract from their time endowment.

A higher probability to survive to the third period, by boosting their preference for the future, pushes parents to make more children ($\partial n_t/\partial \pi_t > 0$) and take better care of the environment ($\partial m_t/\partial \pi_t > 0$),

while—different from Mariani et al. (2010)—it does not affect their investment in education ($\partial e_t/\partial \pi_t = 0$), because parents are also concerned about the quantity (and not only the quality) of their offspring.[11]

As far as policy parameters are concerned, we see that τ—which can be regarded as an environmental tax, as it hinges on polluting production—has some interesting effects on optimal choices. By making workers poorer, it discourages investment in education ($\partial e_t/\partial \tau < 0$), which may benefit the environment by reducing future production. However, it also hampers investment in maintenance ($\partial m_t/\partial \tau < 0$) and stimulates fertility ($\partial n_t/\partial \tau > 0$) by redirecting resources from the production of output to the production of children. By consequence, taxing pollution activities may have perverse effects on population growth, which may harm the environment in a dynamic perspective. This result echoes de la Croix and Gosseries (2011), who show how policies intended to control pollution through taxes make people more inclined to engage in tax-free activities such as procreation: this natalist bias may end up deteriorating the environment, through an increased demographic pressure on natural resources.

Finally, educational subsidies do not affect optimal maintenance but favor quality over quantity of children ($\partial e_t/\partial \sigma > 0$, $\partial n_t/\partial \tau < 0$). This has potentially ambiguous effect on the environment, since a higher σ increases production (and thus pollution) per capita, but slows down population growth.

9.4 Dynamics

We now turn to the analysis of the dynamic behavior of our economy.

Once we substitute optimal choices—as given by expressions (9.8), (9.9) and (9.10)—into Eqs. (9.3), (9.6) and (9.7), we obtain a dynamic system of three equations, which governs the evolution of our economy over time.[12]

In order to simplify our analysis, we restrict our parameters to those values that allow us to (1) rule out multiple equilibria, when the survival probability is constant, and (2) focus on a balanced growth path with

strictly increasing human capital. The implied parametric condition requires productivity to be high enough.

Assumption 9.1

$$w > \frac{1-\sigma}{\delta^{1/\beta}\beta\phi(1-\tau)}. \tag{9.11}$$

Under this condition, the long-run growth rate of human capital is given by

$$g_h^\star = \delta\left(\frac{\beta\phi(1-\tau)w}{(1-\beta)(1-\sigma)}\right)^\beta - 1. \tag{9.12}$$

It can be immediately seen that the parameters σ, β, w and ϕ all have a positive effect on the growth rate of the economy.[13] In particular, a higher time-cost of children, by pushing parents to prioritize quality over quantity, fosters human capital accumulation and growth. On the other hand, τ hampers production and thus slows down growth in the long run.

We also assume, for the moment, that $\pi_t = \pi$ (the case of endogenous life expectancy will be explored in Sect. 9.4.2). Along the balanced growth path population grows at a rate given by

$$\lim_{h\to\infty} n_t = \frac{\alpha(1-\beta)\pi}{\phi(1+(\alpha+\gamma)\pi)}. \tag{9.13}$$

Note that, while α and π have a positive effect on population growth, parameters such as ϕ, β and γ—by making parents less interested in reproduction (and more inclined to invest in children's education and environmental maintenance, respectively) tend to reduce demographic growth.

Since we cannot fully characterize analytically the dynamical system, in what follows we resort to numerical simulations in order to describe the dynamics of the model. Although largely arbitrary, the parameterization upon which our numerical examples are based is intended to have

plausible implications. In particular, we will rely on a common set of parameter values and play with (1) policy parameters (τ and σ) and (2) initial conditions on environmental quality and human capital, so as to discuss some interesting implications of our analysis.

In our benchmark parameterization we set $w = 30$, $\delta = 6/5$, $\beta = 1/5$, $\phi = 1/5$, $\eta = 1/5$, $\mu = 1/100$, $\gamma = 1/4$, $\xi = 0$, $\alpha = 1/2$, $\pi_t = p = 4/5$, $\theta = 1$, $\kappa = 1/1000$.[14] We have chosen a subset of our parameters with the objective of stabilizing population in the long run, i.e. $\lim_{h\to\infty} n_t = 1$. For what concerns initial conditions, we have chosen to start with $h_0 = 1/2$, $P_0 = 1000$ and $X_0 = 800$.

9.4.1 Policy Intervention

We start by considering the possible dynamic effects of the two policy parameters of our model, τ and σ.

9.4.1.1 Environmental Regulation

We first focus on the role of environmental regulation. From Sect. 9.3.2, we already know that a higher τ decreases the opportunity cost of having children, thus encouraging fertility. In order to assess the dynamic implication of pollution controls, we compare the simulated time paths of h_t, P_t and X_t under two alternative scenarios: in the first one (that we take as a benchmark) we set $\tau = 0$ and $\sigma = 0$, while in the second one we introduce environmental taxation, by setting $\tau = 1/5$.

Let us start by focusing on the benchmark simulation, as described by the solid lines in Fig. 9.1. We can observe that, as our parameterization satisfies Assumption 9.1, human capital converges to a positive growth rate in the long run. Moreover, population growth vanishes to zero on the BGP. Finally, as far as environmental quality is concerned, we have selected a parameter configuration compatible with a kind of environmental Kuznets curve.[15] Our notion of environmental quality, as summarized by Eq. (9.6) (with $\eta \in (0, 1)$), implies that the state of the environment tends to deteriorate, in the absence of maintenance

activities. This is what happens in the first few periods. As the economy grows sufficiently rich, however, demographic pressure weakens and agents devote an increasing quantity of resources to environmental care. Under the chosen parameterization, this translates into an improving quality of the environment.

If we introduce pollution controls (under the form of production taxes), we see that it hampers human capital accumulation and increases, in each period, population size. As far as the aggregate impact of τ on the environment is concerned, it depends on three distinct effects. On the one hand, by hindering human capital formation, τ decreases economic activity, which is in itself a potential source of pollution. It also directly reduces production and its impact on the environment, as implied by Eqs. (9.4) and (9.6). On the other hand, however, a higher τ makes parent more inclined to invest in the quantity (rather than quality) of their offspring, thus increasing the demographic pressure on the environment. In our numerical example, this negative effect prevails—as can be seen from the third panel of Fig. 9.1. This illustrates the possible dynamic consequences of the perverse effect of environmental policy, which have been also highlighted—although in a different framework—by de la Croix and Gosseries (2011).

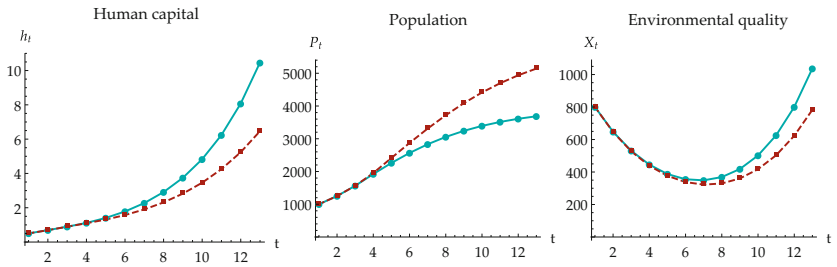

Fig. 9.1 Pollution control: simulated time paths for $\tau = 0$ (solid) and $\tau = 1/5$ (dashed)

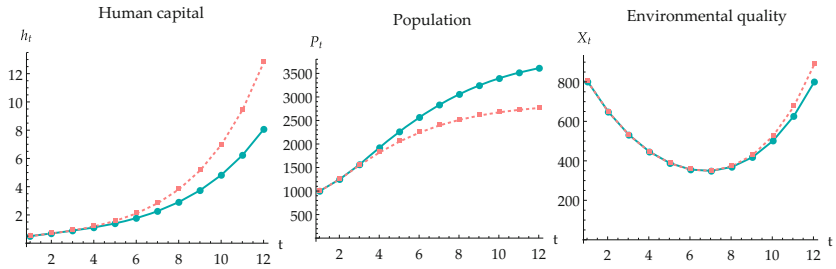

Fig. 9.2 Educational subsidies: simulated time paths for $\sigma = 0$ (solid) and $\sigma = 1/5$ (dashed)

9.4.1.2 Educational Subsidies

We now turn to the analysis of the dynamic consequences of subsidies. Figure 9.2 contrasts the benchmark ($\tau = 0$, $\sigma = 0$) with the case in which $\sigma = 1/5$ (and $\tau = 0$). The results are not surprising, in light of Sect. 9.3.2: subsidizing education bends the quality-quantity trade-off towards education, so that growth is faster along the balanced growth path, and population stabilizes at a smaller size. Overall, the chosen parameterization shows a beneficial effect of subsidies on environmental quality, which tends to amplify when the economy gets richer.[16]

These findings seem to suggest that educational subsidies may be used as an effective environmental policy. In particular, if the objective of the policy-maker is to reduce demographic pressure on natural resources, subsidizing education may be a viable alternative to controversial policies like taxing birth, even in the presence of a polluting effect of human capital-driven growth.[17] Such normative implication highlights the importance of considering endogenous fertility: in the presence of a quality-quantity tradeoff, subsidizing human capital accumulation would still bring about more environmental degradation through faster growth, but at the same time alleviates the demographic pressure on the environment through reduced fertility (with the latter effect prevailing, in our example).

9.4.2 Endogenous Life Expectancy: Poverty Traps

In our past research (Mariani et al., 2010), we highlighted the possibility of environmental poverty traps related to endogenous life expectancy. In particular, we showed that if life expectancy (and, more in general, health) depends on environmental quality and/or human capital, multiple equilibria may arise and economies characterized by different initial conditions may converge either to a good equilibrium (characterized by high life expectancy, high human capital and good environmental quality) or to a worse one (with short life expectancy, modest levels of development and a very deteriorated environment).

In what follows we see how things change if, different to our previous study, we introduce endogenous fertility. In the same fashion as Mariani et al. (2010), we now make the survival probability depend on human capital and the state of the environment. In particular,

$$\pi_t = \begin{cases} \underline{\pi} & \text{if } X_t + \chi h_t < J \\ \overline{\pi} & \text{if } X_t + \chi h_t \geq J \end{cases}, \tag{9.14}$$

where $\chi, J > 0$. The survival probability is introduced as a binary variable for analytical simplicity, and in order to ensure comparability with Mariani et al. (2010).[18] Equation (9.14) also allows for some substitutability (accounted for by χ) between human capital and environmental quality as determinants of longevity. There is in fact ample empirical evidence that a healthy environment is key to having a long life expectancy (among others, Pope, 2000; Pope et al., 2004; Evans and Smith, 2005). As far as the role of human capital is concerned, its theoretical importance has been highlighted by Blackburn and Cipriani (2002) and de la Croix and Licandro (2013, 2015) among others, while its empirical relevance has been assessed by several studies, such as Lleras-Muney (2005).[19]

In our numerical simulation, the new parameters introduced in Eq. (9.14) are set to the following values: $\underline{\pi} = 1/2$, $\overline{\pi} = 4/5$ and $J = 600$. The value of χ, as well as initial conditions, will be chosen so as to illustrate different types of poverty traps.

9.4.2.1 Environmentally Induced Poverty Traps

We start by considering environmentally induced poverty traps. To do so, we deal away with human capital as a determinant of longevity by setting $\chi = 0$. Since life expectancy depends on environmental quality only, two economies that differ only with respect to the initial state of their environment may be driven to different equilibria. This non-ergodic behavior is related to π_t being a key determinant of the optimal choices (namely fertility and maintenance) that govern the time evolution of the economic system.

Figure 9.3 describes two otherwise identical economies, which start from different levels of environmental quality ($X_0 = 550$ and $X_0 = 1300$, respectively) that place them on the two sides of the threshold J, so that the two economies are initially characterized by different survival probabilities. We can see that, as π_t does not affect parental investment in education, the trajectory of h_t is the same for both economies. The two economies, however, display a divergent behavior in terms of population and environmental quality. In particular, the economy that starts from better initial conditions manages to keep the state of its environment always above the threshold J, even after an initial decrease. This implies that this "luckier" economy can sustain a larger population in the long run, and also experience an improvement in environmental conditions, driven by the larger investments in maintenance related to a longer life expectancy.

A similar situation is depicted in Fig. 9.4. The only difference, with respect to Fig. 9.3, is that now the "worse" economy starts from $X_0 = 900$ (rather than $X_0 = 550$). This means that, for some periods, it behaves exactly as the "better" one in terms of human capital, population and survival probability. Path dependency, however, implies that its environmental quality keeps lagging behind that of the other economy, and eventually falls below the threshold J. At this point, life expectancy drops in the economy with worse initial conditions, and the two economies diverge.[20]

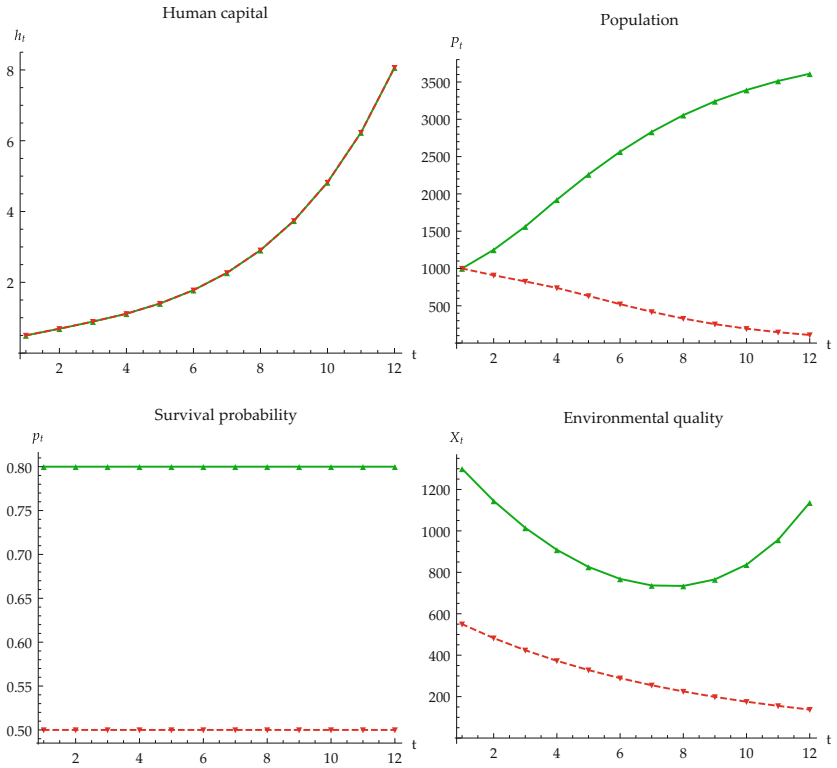

Fig. 9.3 Multiple equilibria: simulated time paths when $X_0 = 1300$ (solid, green) and $X_0 = 550$ (dashed, red)

9.4.2.2 Human Capital Related Poverty Traps

We now want to put the spotlight on divergence as determined by differential initial levels of development (rather than environmental quality).

To do so, we reactivate the channel through which human capital affects life expectancy and set $\chi = 120$. In Fig. 9.5, we present the simulated time paths of two economies that start from the same level of environmental quality ($X_0 = 300$), but differ in their initial human capital ($h_0 = 1/5$ and $h_0 = 4/3$, respectively).

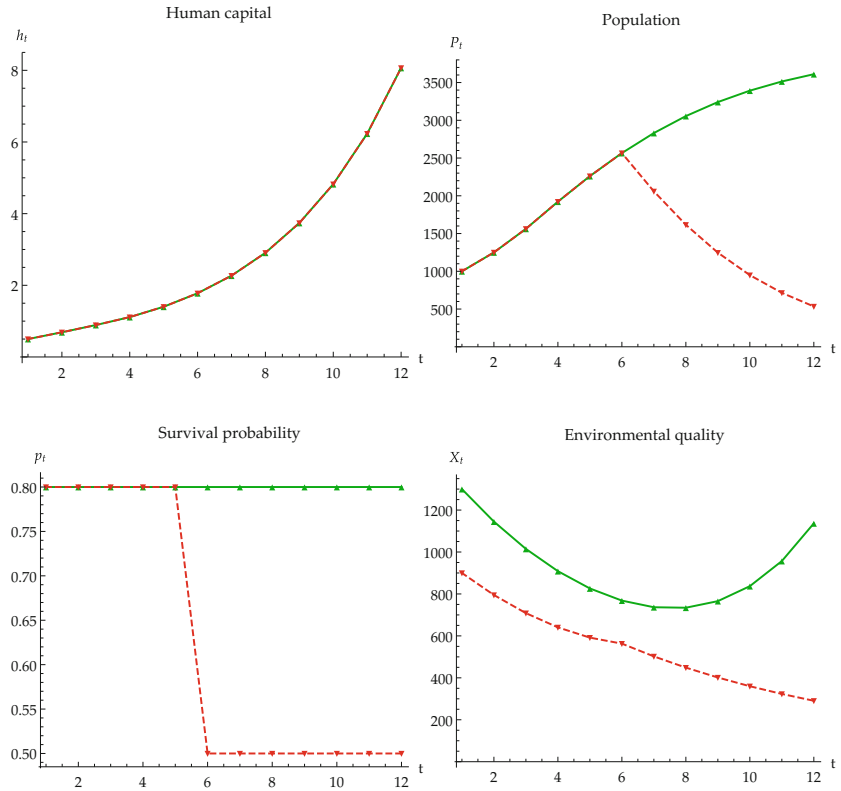

Fig. 9.4 Multiple equilibria: simulated time paths when $X_0 = 1300$ (solid, green) and $X_0 = 900$ (dashed, red)

Here we can notice that the initially less developed economy remains stuck in a poverty trap defined by slower human capital accumulation, shorter life expectancy, declining population and diminishing environmental quality. The other economy starts off with a higher human capital: at the beginning this is not enough to raise the survival probability, and just translates into a faster demographic decline (since more educated parents prefer quality to quantity of children), without allowing the richer country to do better than the poorer one in terms of pollution. Eventually, however, the richer economy reaches a level of human capital that brings

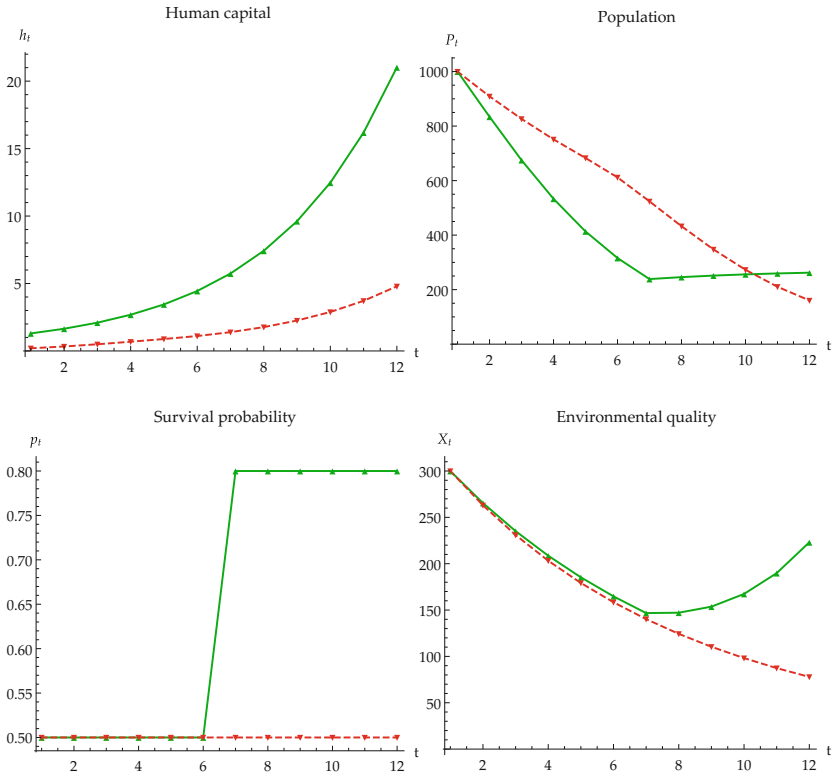

Fig. 9.5 Multiple equilibria: simulated time paths when $h_0 = 1/5$ (solid, green) and $h_0 = 4/3$ (dashed, red)

about an increase in life expectancy: at that point the demographic and environmental trends are reversed, with both population and environmental quality that start increasing, thus generating a divergent trajectory with respect to the other economy.

This case is interesting since, different from Fig. 9.4, the survival probability is shown to increase over time—because of the beneficial effect of human capital accumulation, which more than compensates the impact of a deteriorating environment on longevity.

9.4.2.3 Escaping the Trap

One may wonder, at this stage, under which circumstances an economy starting from unfavorable initial conditions can avoid being stuck in a poverty trap, and which role can be played by policy intervention.

Without presenting additional simulations, we can briefly discuss a few channels through which an initially-trapped economy may ultimately converge to the equilibrium characterized by a longer life-expectancy, a higher level of economic development, and possibly a better environmental quality. Technically, this would be possible through (1) a permanent reduction of the threshold value of J in Eq. (9.14) (so that the economy finds itself automatically out of the trap), or (2) an enhanced dynamic evolution of environmental quality and/or human capital. The first mechanism, as suggested by Mariani et al. (2010) may correspond to exogenous improvements in medicine and/or technological transfers in the fields of health and environmental care. Perhaps more interestingly, the policy instruments τ and σ may also be used in order to sort an economy out of the trap. In particular, a sufficiently large increase in σ, and possibly a reduction in τ may drive the economy towards the superior equilibrium, by pushing parents to invest more in education, rather than in the quantity of their children. These policy measures would significantly alleviate the demographic pressure on the environment, and stimulate human capital accumulation that can in turn stimulate environmental maintenance.[21]

The above mechanisms, however, may work in the opposite direction. Even temporary shocks (such as natural disasters or episodes of acute environmental stress and pollution) may be sufficient to throw an economy back into a poverty trap.[22] The same applies to policy intervention. In particular, the possible perverse effects of environmental taxes may even be deeper and very long-lasting, if an economy is at risk of being pulled back into a trap.

9.4.2.4 The Role of Endogenous Fertility

Overall, considering endogenous fertility helps us to improve our understanding of the dynamic challenges implied by the management of environmental quality. In fact, introducing a quality-quantity trade-off into the framework proposed by Mariani et al. (2010) allows us to deal with population as a dynamic variable, and better understand the interplay between demographic and environmental variables along the growth process of the economy. By doing so, we have been able—for instance—to draw new policy implications and gauge the trade-offs associated with normative intervention (as explained in Sect. 9.4.1). Our dynamic analysis also reveals that a better initial environmental quality allows the economy to sustain a larger population, thus making the demographic constraint less binding. Finally, the dynamic simulations developed in Sect. 9.4.2 suggest that, in the presence of multiple equilibria, the "superior" steady state is not necessarily associated with a smaller population—an interesting result that depends on endogenous fertility and education choices.

9.5 Conclusions

In this chapter, we have presented a growth model in which education, fertility, environmental quality and life expectancy can all be endogenously determined. Such model provides a general framework of analysis, which can encompasses both original features and some results already known in the literature. In particular, we put together two key elements often studied separately in the literature: the two-way causality between life expectancy and the environment, and the link between fertility decisions and environmental quality.

In particular, our model highlights (1) the potentially perverse effect of environmental policies, (2) the role of demographic dynamics in explaining (at least partially) the environmental Kuznets curve, (3) the opportunity of using educational subsidies as an effective policy tool

to alleviate the pressure on natural resources, and (iv) the possibility of poverty traps related to human capital and environmental quality.

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Appendix 1: Derivation of Optimal Choices, as in Eqs. (9.8)–(9.10)

After replacing (9.2) and (9.3) into the utility function (9.1), we can solve the optimization program by relying on the following Lagrangian:

$$\begin{aligned}\mathcal{L} = &\ln[(1-\tau)(1-\phi n_t)wh_t - c_t + m_t + (1-\sigma)n_t e_t] \\ &+\pi_t \alpha[\ln(\delta(\theta + e_t)^{\beta} h_t^{1-\beta}) + \gamma \ln m_t]. \end{aligned} \tag{9.15}$$

We then have to solve the system composed by the following first-order conditions:

$$\frac{\partial \mathcal{L}}{\partial m_t} = 0 \;\Leftrightarrow\; \frac{1}{(1-\tau)(1-\phi n_t)wh_t - c_t + m_t + (1-\sigma)n_t e_t} = \frac{\gamma \pi_t}{m_t}, \tag{9.16}$$

$$\frac{\partial \mathcal{L}}{\partial e_t} = 0 \;\Leftrightarrow\; \frac{(1-\sigma)n_t}{(1-\tau)(1-\phi n_t)wh_t - c_t + m_t + (1-\sigma)n_t e_t} = \frac{\alpha\beta\pi_t}{(\theta + e_t)}, \tag{9.17}$$

$$\frac{\partial \mathcal{L}}{\partial n_t} = 0 \;\Leftrightarrow\; \frac{(1-\tau)\phi wh_t + (1-\sigma)e_t}{(1-\tau)(1-\phi n_t)wh_t - c_t + m_t + (1-\sigma)n_t e_t} = \frac{\alpha \pi_t}{n_t}. \tag{9.18}$$

Solving the system, we obtain

$$e_t = \frac{\beta(1-\tau)\phi w h_t - (1-\sigma)\theta}{(1-\sigma)(1-\beta)}, \tag{9.19}$$

and we can see that we have an interior solution for education (i.e. $e_t > 0$), only if

$$h_t > \frac{(1-\sigma)\theta}{\beta(1-\tau)\phi w}.$$

Otherwise, we are in a corner solution, with agents choosing not to invest in education ($e_t = 0$).

In the lower part of Eqs. (9.8)–(9.10), we report the optimal choices associated to an interior solution for e_t, which can be immediately obtained from the first-order conditions specified above. In the case of corner solution, we must replace $e_t = 0$ in the agents' budget constraint, and re-optimize with respect to n_t and m_t only. The implied solution are reported in the upper part of Eqs. (9.8)–(9.10).

Appendix 2: Derivation of the Long-Run Growth Rate of Human Capital

The rate of growth of human capital g_h is obtained as

$$g_h = \frac{h_{t+1} - h_t}{h_t} = \delta \left[\frac{\beta}{1-\beta} \left(\frac{\phi(1-\tau)w}{(1-\sigma)} + \frac{\theta}{h_t} \right) \right]^{\beta} - 1. \tag{9.20}$$

In the long run, we have that $h_t \to \infty$ and therefore $\theta/h_t \to 0$. It follows that

$$g_h^{\star} = \delta \left(\frac{\beta\phi(1-\tau)w}{(1-\beta)(1-\sigma)} \right)^{\beta} - 1. \tag{9.21}$$

Notes

1. Becker (1960) first introduced the idea that parents may substitute quality for quantity, choosing to have less but better educated children. See also Becker and Lewis (1973), de la Croix and Doepke (2003) and Doepke (2004), among others.
2. One possible solution to this problem could consist in capping population.
3. In particular, several studies in medicine and epidemiology have documented how environmental quality is a significant factor affecting health and, in particular, longevity. See for instance Pope (2000), Pope et al. (2004), and Evans and Smith (2005).
4. In the "environmental" literature pioneered by John and Pecchenino (1994), environmental quality and longevity have typically been identified as important elements to explain underdevelopment traps (see also Ikefuji and Horii, 2007). The role of life expectancy, however, has typically been overlooked—different to some papers such as Blackburn and Cipriani (2002) or Chakraborty (2004) that, although not focused on environmental problems, have put the spotlight on longevity as a possible source of multiple equilibria.
5. The bimodal distribution of life expectancy and the EPI has been further confirmed by Dao and Edenhofer (2018), who use updated data and identify more than two convergence clubs.
6. In their paper, Jouvet et al. (2011) consider a possible trade-off between longevity and fertility and focus on population density as a threat to environmental quality. They do not consider, however, the possibility that agents directly affect the stock of the available resources through their choices, and abstract from the quality-quantity trade-off when studying fertility decisions.
7. This is reminiscent of "warm glow" specification of altruism due to Andreoni (1989).
8. This way of modeling environmental regulation does not require us to write down the government budget constraint.
9. The case of $\eta = 1$, which is more appropriate to study pollution as a flow, rather than environmental quality as such, will be explored in further research.
10. More details on the derivation of optimal choices are provided in Appendix 1.

11. In de la Croix and Doepke (2003), who also have both quantity and quality of children in the parents' utility function, the discount rate affects fertility but not education. Here, we have the same result for the survival probability, which contributes to define the rate of preference for the future.
12. Notice that Eq. (9.3) can be solved independently of (9.6) and (9.7), if life expectancy is exogenous. By consequence, dynamics are one-dimensional. When, instead, life expectancy is endogenously determined by the stock of human capital and/or environmental conditions (see Sect. 9.4.2), the analysis of the dynamic system is much more complicate, since the three equations become interdependent.
13. The derivation of Eq. (9.12) is presented in Appendix 2, where it becomes clear why some parameters (such as θ) do not influence growth in the long run.
14. By setting $\xi = 0$, we deal away with consumption-generated pollution. This is not of primary importance, as we want to focus on the effect of environmental taxes hitting production. Introducing consumption taxes, and making environmental quality depend on consumption has only quantitative implications.
15. As a sample of the literature concerned with the EKC, see for instance Dinda (2004) or Kijima et al. (2010). The specificity of our analysis consists in relating the non-monotonic evolution of the environment to demographic dynamics.
16. Figure 9.2 suggests, however that such beneficial effect is of limited size (and smaller, in modulus, than the impact of τ depicted in Fig. 9.1). In fact, the increased output due to faster human capital accumulation almost offsets the alleviation of demographic pressure.
17. As pointed out before, several papers suggest that family planning should be part of climate policies (for instance, Harford, 1998; Schou, 2002; or Gerlagh et al., 2018).
18. Moreover, this kind of step function captures the idea that a substantial improvement in life expectancy can be achieved only if environmental quality reaches some minimal level. A step function is, in a sense, an extreme case of a convex–concave relationship, which is a rather appropriate description of the cumulative effects of environmental degradation on human health.
19. Apart from income-related effects, human capital might translate into higher life expectancy because better educated people have access to more

accurate information about health and are less inclined to take up health-threatening behaviors.

20. The possibility of pollution-driven mortality resurgence is highlighted by McMichael et al. (2004) among others, and is usually exemplified by the trajectories of several ex-USSR republics—see for instance Feachem (1994) and Jedrychowski (1995).
21. Growth can, however, also bring about more pollution. Here, we stay consistent with the findings of our dynamic simulations, according to which human capital has a net beneficial effect.
22. See Mariani et al. (2010) for a more detailed analysis.

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10

HIV/AIDS, Demography and Development: Individual Choices Versus Public Policies in SSA

Luca Gori, Piero Manfredi, and Mauro Sodini

JEL Classification J11, J13, O1, O41

10.1 Introduction

For more than three decades since its debut in 1980, the HIV/AIDS pandemic has ravaged Sub-Saharan Africa (SSA) with a devastating mortality burden especially frightening young adults of both sexes. However, in the last few years the epidemic seems to have finally entered a declining phase (UNAIDS, 2018) thanks to the massive efforts aiming to (1) expand HIV

L. Gori (✉)
Department of Law, University of Pisa, Pisa, Italy
e-mail: luca.gori@unipi.it

P. Manfredi · M. Sodini
Department of Economics and Management, University of Pisa, Pisa, Italy
e-mail: piero.manfredi@unipi.it; mauro.sodini@unipi.it

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testing to make seropositive people (i.e., people in the HIV stage) aware of their serostatus, (2) increase the number of seropositive and sick people (i.e., people with full blown AIDS stage) accessing effective antiretroviral therapies (ART), and (3) maintain high rates of population awareness on AIDS risks, by continuing and extending ongoing prevention and screening campaigns. For example, in 2016 the proportion of people accessing ART amongst those living with HIV in the overall SSA region reached the level of 54% (though with large inter- and intra-country variation) compared to essentially none 10 years before. This is a great success that, compared to 2004–2005, representing the epoch of the AIDS-related mortality peak in SSA, has contributed to nearly halve AIDS mortality over a span of one decade (UNAIDS, 2017). Indeed, this has radically changed overall perspectives of AIDS control in SSA, as summarised in the so-called 90-90-90 plan launched by the UN in 2014 (UNAIDS, 2017), upon which the slogan "ending AIDS by 2030" was coined (UNAIDS, 2017)—referring to a major reduction in incidence and obviously not to disease eradication.

Clearly, going beyond the current—already burning—debate about the feasibility of the 90-90-90 plan (Levi et al., 2016), the success achieved so far should not let us forget that AIDS currently remains the second main cause of mortality in SSA (WHO, 2017), with a huge number of seropositive individuals unaware of their serostatus (UNAIDS, 2017) and therefore able to continue to transmit the infection to others for a long time, suggesting that the battle is only initiated. In relation to this, several challenges have arisen, whose outcomes are surrounded by large uncertainties. In our opinion, these include (amongst others) three strictly inter-related areas at the interplay of epidemiology and public health on one hand, and demo-economics on the other hand, deserving maximal attention by scholars and policy makers. The first one deals with the ultimate impact of HIV on the fertility transition in SSA and its consequences for economic development. The second regards the dramatic amount of resources that will have to be mobilised at both the national and international levels for successfully winning the AIDS battle, and its ambiguous effect on economic development (WHO, 2018). The third one—perhaps the true major challenge—deals with the emergence of resistance to HIV treatment (Hamers et al., 2018), whose effects are

at present largely uncertain. These effects, which might be reasonably kept under control in industrialised countries, where HIV epidemics are mild, might reveal devastating in SSA by dramatically worsening the first two threats and eventually questioning the current optimism in the fight against AIDS (Hamers et al., 2018, Phillips et al., 2017).

Amongst the three challenges, the first one on the eventual impact of HIV on the fertility transition in SSA and economic development has already been the object of a sustained debate amongst economists, with two dramatically opposite positions. On one extreme, Young (2005, 2007) concluded that AIDS is contributing to lowering fertility in sub-Saharan Africa, and although representing a humanitarian disaster, does not cause an economic disaster. Rather, AIDS will allow future SSA generations to enjoy higher welfare than current generations thanks to the increase in the capital- and output-labour ratios because of AIDS mortality's impact on labour supply that will dominate the main negative effect of HIV namely, the disruption of human capital. At the other extreme, Kalemli-Ozcan and Turan (2011) and Kalemli-Ozcan (2012) were first in corroborating an earlier intuition by Kalemli-Ozcan (2002) that HIV has the potential to reverse the fertility transition in SSA. In particular Kalemli-Ozcan and Turan (2011) revisited Young's (2005, 2007) analyses using the same data and concluded that "the effect of HIV prevalence on fertility turns out to be positive". Moreover, Kalemli-Ozcan (2012, p. 891) empirically showed the potential for HIV to reverse the fertility transition in SSA via both a positive direct effect on the quantity of children and a negative effect on the quality of children because of the existence of a negative correlation between HIV prevalence and school enrolment, and explained this phenomenon with the upward pressure that the mortality upturn due to AIDS causes on the precautionary demand for children (quantity) and to the downward pressure on the demand for their education (quality). Within these two extreme positions there exists an entire spectrum of intermediate ones, which are reviewed in Gori et al. (2017).

On the previous topic, we have investigated (Gori et al., 2017) the possible consequences of the HIV epidemic on the fertility transition in SSA by using a novel model integrating an explicit dynamics of a fatal epi-

demics along the formulation firstly proposed in Chakraborty et al. (2010, 2016) into a basic Unified Growth Theory (UGT) general equilibrium model (Galor and Weil, 2000; Galor, 2011) including only human capital accumulation, to capture the main feature of the HIV context in SSA, and endogenous fertility and child and adult survival. Child and adult mortality schedules were made dependent on both human capital accumulation, to mirror the pre-AIDS mortality transition, and the HIV prevalence to capture the additional effects of HIV/AIDS-related mortality. The aim was to track the ultimate consequences—at the general equilibrium level—of the fall in education and human capital accumulation following the HIV-related blow-up of mortality amongst young adults on the quantity-quality switch, and therefore the fertility transition, in SSA. By choosing a parameterisation of SSA mortality allowing an appropriate balance between the time scales of the demographic transition and AIDS spread, we indeed concluded that an HIV-induced stall (or even a reversal) of the fertility transition is a potentially robust phenomenon that, even under reasonably optimistic hypotheses on the timing and effects of AIDS control, might delay the trajectory of fertility in SSA of several decades thus compromising economic development in the region. These results bring theoretical evidence to the findings supporting the fertility "reversal" hypothesis, formulated first by Kalemli-Ozcan (2012) and Kalemli-Ozcan and Turan (2011) (see Gori et al., 2017, and references therein).

The possibility that the fertility transition in SSA might be paralysed by HIV makes it imperative to rapidly bring the epidemics under full control, thereby yielding to the second challenge above. Indeed, successfully fighting this battle will in turn call for unprecedented further mobilisation of resources specifically devoted to fight HIV/AIDS in the short as well as in the medium and long terms. Recent estimates (Collier and Sterck, 2018) indicate that, given current high costs of a single therapy, the overall economic cost of generalised lifelong HIV treatment in the poorest SSA countries would be in the range of 80% of current GDP! The magnitude of this figure can hardly be covered by a massive expansion of international donations, representing the main funding source of the intervention against HIV carried out in SSA so far (UNAIDS, 2017). As was noted by Resch et al. (2015) and Remme

et al. (2016)—based on current perspectives—, international aids might not be able to expand significantly beyond current levels. On the other hand, in the light of the dramatic extent of foreign financing of the AIDS response, which has reached levels above 90% of total AIDS funding in low income countries in 2013, there have been concerns about the fact that an HIV response completely dependent on external resources might have potentially destabilising effects on the afflicted countries (Mohiddin and Johnston, 2006; UNAIDS, 2013).

The previous considerations therefore highlight not only the need for massive resources deployment but also the delicate balance between foreign aid and the ability, for HIV afflicted SSA countries, to develop an autonomous endogenous response to AIDS. This is a major challenge whose response depend from an endless list of factors including primarily: (1) the magnitude of the HIV epidemic, (2) the stage of economic development in the afflicted country and its perspectives for GDP growth in the medium term, (3) the threats from other diseases, first of all malaria and TB, that in turn require allocation of large quotas of GDP, (4) the ability to develop new financial tools to face the HIV challenge (Resch et al., 2015; Atun et al., 2016a; Remme et al., 2016). These complicate questions have already raised a tough debate in the areas of public health and health economics (Katz et al., 2014; Resch et al., 2015; Atun et al., 2016b; Remme et al., 2016).

On this topic, in a different work (Gori et al., 2018) we have initiated an investigation of the macro-economic dynamic implications of the affordability (by the government of HIV-afflicted countries) of control programmes mainly relying on public domestic expenditure. In that article, also motivated by the need to provide a well-funded formulation of intervention against HIV (taken as fully exogenous in Gori et al. (2017) we used a simplified OLG macroeconomic framework à la Diamond (1965) with endogenous population and endogenous HIV transmission. About the latter, we represented the intervention against HIV by modelling the probability of transmission of HIV infection per single sexual contact as the sum of an endogenous component, representing public expenditure (financed at a balanced budget) specifically targeted to fight against HIV, and of an exogenous component reflecting international donations.

The main aim of the present work is to go one step further compared to both Gori et al. (2017) and Gori et al. (2018) by superimposing to the standard Diamond setup also private education and human capital accumulation, *thereby enabling us to tackle the issue of HIV spread and control in a model with the quantity-quality trade off and consequently capable to also offer predictions on the fertility issue as well*. For the macroeconomic component of the model we adopt a UGT-like structure along the lines of the contribution of Yakita (2010) with both physical and human capital accumulation. This macroeconomic setup is combined with an endogenous representation of the transmission and control of HIV/AIDS following the pioneering works of Chakraborty et al. (2010, 2016), who were first in proposing an explicit representation of the transmission process into an OLG model. However, unlike Chakraborty and co-authors, whose agents optimally chose their private health preventive expenditure against HIV, in our formulation the transmission probability is determined by (1) the public interventions managed by the government of the afflicted country, (2) foreign aid donations, which historically have represented the main channel of interventions carried out so far in SSA, (3) a component tuning the positive impact of private education on the awareness of being at risk and ultimately capable to enhance the ongoing policies. Foreign aids are assumed to be scaled by the two main factors that should drove international programmes in SSA, namely the rate of HIV prevalence and per capita income of the afflicted country, although it is documented that GDP has been the main driver of the intervention while prevalence seemingly played a secondary role (Haacker, 2009; UNAIDS, 2017). Compared to the formulation adopted in Chakraborty et al. (2016), which is clearly well suited for settings where financial markets are adequately developed, we believe that ours can better fit the current context of HIV in SSA, which is—with the sole exception of South Africa—the poorest region worldwide with inadequate health infrastructures, severe shortages of physicians and medicines, low education and poor health (UNDP, 2018) causing the highest mortality setting worldwide even in the absence of HIV.

Our formulation inherits the growth structure from Yakita (2010) showing the two development regimes characterising UGT growth mod-

els (e.g., Galor and Weil, 2000; Kalemli-Ozcan, 2002; de la Croix and Doepke, 2003, 2004; Galor, 2011), that is an initial exogenous growth phase (representing the paradigm of the poverty trap), where private education does not play any role and there exists only a precautionary demand for children, and a subsequent endogenous growth phase where education promotes the quantity-quality switch, which in turn plays a critical role in both human capital accumulation and economic development. However, the inclusion of the endogenous dynamics of HIV generates a rich interplay amongst the macroeconomic set up, the diseases and its possible intervention strategies, which have the potential to affect in a complicate manner the possibility to switch between the two development regimes as well as to promote significant quantitative differences within each of them. Additionally, thanks to the flexibility of our formulation of control strategies (public policy versus donors versus private education) the model allows to pinpoint a rich number of endogenous policy options that can be used by the afflicted country in the battle against the HIV disease, which however can have profoundly different impacts on the development regimes depending on the specific mix of the interventions adopted.

On one hand, high levels of HIV prevalence have the potential to relegate the economy in its poverty trap, where the lack of ability to sustain education makes unavoidable the need for international foreign aid donations. Obviously, in low-resource settings, that is countries already entrapped in poverty, AIDS can dramatically worsen the conditions for escaping underdevelopment. On the other hand, lower levels of HIV prevalence and/or the ability to effectively control the epidemics can promote the escape from the poverty trap, entering a regime where the development of education and the accumulation of human capital make available further tools for successfully fighting the disease, eventually entering a virtuous circle where HIV-afflicted countries can afford to successfully fight the battle exclusively with internal resources. Unlike Young (2005), this model shows that the increase in adult mortality due to HIV/AIDS can represent a serious threat to economic development by its upward pressure on the precautionary demand for children and downward pressure on education, as first stressed by Kalemli-Ozcan

(2002), which in turn can dramatically reduce physical and human capital accumulation.

The rest of the article proceeds as follows. Section 10.2 reviews the main facts about the demographic and economic impact of the HIV/AIDS epidemic in SSA, with a special emphasis on the critical issue of funding HIV control programmes. Section 10.3 presents the macro-economic dynamic framework. Section 10.4 studies the dynamics of the model. Section 10.5 reports and discusses the main results of the work. Section 10.6 outlines the conclusions.

10.2 HIV/AIDS in SSA: Natural History, Mortality, Fertility and the Demographic Transition

10.2.1 Global Facts About HIV/AIDS

Several scholars have identified the still dramatic levels of mortality, with the highest toll from communicable diseases worldwide, and the underlying poor health conditions, as the major issue responsible for underdevelopment in SSA (e.g., Bloom and Canning, 2004; Lorentzen et al., 2008). Notwithstanding these tragic circumstances, SSA countries were able to achieve continued (though slow) progresses in life expectancy until the early 1980 i.e., prior the HIV/AIDS initiated to ravage the region yielding a tragedy of major proportions.

While in several other regions of the world HIV epidemics remained confined for years in special and therefore relatively small risk groups such as men having sex with men, intravenous drug users, and blood transfused, since the very beginning of the pandemics the spread of HIV in SSA was mainly driven by heterosexual transmission in the young adult population thereby favouring high rates of disease spread as well as large rates of vertical transmission to new-born children from infected mothers. Fast diffusion was further enhanced by a number of factors highly specific to the context of SSA including high rates of sexual partner change, high rates of prostitute attendance, almost no use of protections during sexual

intercourse, large human mobility due to seasonal works, sporadic and largely ineffective awareness campaigns—with a few notable exceptions—such as the Ugandan case (Green et al., 2006)—up to the phenomenon of AIDS denialism, which in some cases, as that of South-Africa, was unbelievably promoted at the highest policy level, namely by the country prime minister (Fassin and Schneider, 2003, Simelela and Venter, 2014). These circumstances allowed HIV to reach large prevalence levels in several SSA countries, thereby causing AIDS to rapidly emerge—and to remain for about 25 years—the leading cause of mortality (UNAIDS, 2017).

The updated UNAIDS central estimates (UNAIDS, 2017) document that HIV/AIDS—by far the major pandemic of the current epoch, with 76 million people infected by HIV and 35 million died from AIDS related illnesses (primarily Tuberculosis) since the onset of the epidemic in the late seventies up to 2016—has devastated SSA. Still in 2016, out of a total of 36.7 million people living with HIV worldwide, with an incidence of about 1.8 million new HIV infections and 1 million deaths from AIDS-related illnesses, SSA—which in the same period was homing 13% of the world population—accounted for 25.5 million people living with HIV, i.e. 70% of total HIV prevalence worldwide, for 64.5% of new infections, for 73% of total estimated deaths from AIDS, and for 85.5% new infections among children (UNAIDS, 2017). However, these figures represent a dramatic decline (−42%) compared to the disease peak phase occurred between 2001 and 2005, when the number of AIDS-related deaths in SSA was in the region of 1.7 million per year. The factors underlining this decline are complicate and include awareness campaigns—that essentially represented the only available weapon against the infection until 2005—aimed to reduce risky behaviour, possible saturation effects in the at-risk population—and the growing access of SSA population to antiretroviral treatments. Considering that essentially no treatments were available in SSA still in 2005, progresses have been dramatic: in 2016 about 50% of those living with HIV in SSA were accessing to therapies though with large regional disparities (about 60% in southern and eastern Africa vs. only 35% in western and central Africa).

10.2.2 Country-Level Epidemics

A major distinctive trait of HIV/AIDS in SSA, as by the way all over the world, is the marked heterogeneity in prevalence amongst different communities as clear from the temporal trends of country-level prevalence of HIV infection in the most afflicted countries (Fig. 10.1). For example, in Zimbabwe, that possibly suffered the most aggressive HIV epidemics worldwide with a peak in the region of 29% in the 1997–1998, the epidemics already entered in a declining phase since the 2000, thus well before the introduction of therapies. This has been primarily attributed to the success in the awareness campaigns conducted therein, which were subsequently reinforced by the spread of therapies (Halperin et al., 2013). However, in the recent years the prevalence curve seems stalling at levels still exceptionally high (more than 15%) suggesting that the battle is far from being won. A similar trend was observed in Botswana, and at lower prevalence regimes in Malawi and Namibia. Somewhat differently, in Swaziland (27.5% prevalence at 2014), Lesotho (23% prevalence at 2014) and South-Africa (about 19% prevalence at 2014), HIV prevalence has reached a plateau between 2000 and 2005, remaining essentially constant or even slightly increasing thereafter. These slightly increasing trends

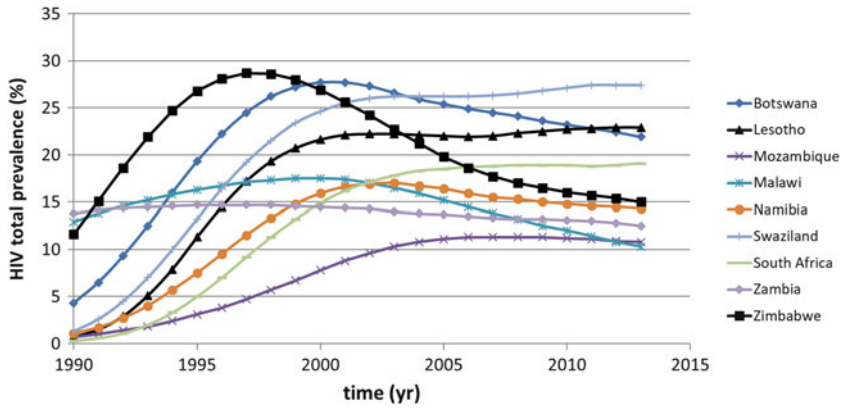

Fig. 10.1 HIV in SSA. Temporal trends of HIV prevalence in the overall (male + female) adult population (15–49 years) in most afflicted countries

also reflect the positive effects of therapies, which extend the duration of sojourn in the seropositive (i.e., HIV) phase, thereby increasing the size of the HIV population. Nonetheless, this effect is also illustrative of the complexities of intervention against HIV: any success of therapies in delaying the transition to full-blown AIDS disease has the effect of increasing the size of the HIV population. As the latter includes individuals in good health state and infective, that is capable to retransmit the infection to others, efforts to keep awareness high and ensure safe sexual behaviour will require a lot of effort in the very long-term.

10.2.3 HIV/AIDS and Life Expectancy

The growth in HIV prevalence curves, followed with a delay of some years, by the corresponding growth in full-blown AIDS prevalence curves, and ensuing rapid death (in the absence of therapies) was mirrored by a dramatic impact on life expectancy all over SSA but especially in countries with large HIV epidemics. Figure 10.2 reports UN (2017) medium variant estimates and projections for the post-1950 trend in the life expectancy at age 15 (e_{15}) in the SSA countries suffering the most severe HIV epidemics (as reported in Fig. 10.1). Life expectancy at age 15 was chosen as representative of the expected lifespan for individuals entering young adulthood in SSA. As previously stated, HIV/AIDS was able to dramatically reverse the long-lasting—though slow—increasing trend in life expectancy showed everywhere in SSA. In the worst epidemics, in Zimbabwe, the life expectancy at age 15 that was estimated in the range of 54 years between 1985–1990 i.e., before the onset of HIV/AIDS, fell between 2000 and 2005 (that is, as expected, a few years after the predicted peak in prevalence reported in Fig. 10.1) to a level only slightly in excess of 30 years. Though the decline in HIV prevalence observed in Zimbabwe in subsequent years is predicted to allow life expectancy to return to the pre-AIDS levels already by 2015–2020, things are dramatically different in other contexts. For example, in Lesotho and Swaziland e_{15} is projected by the UN medium variant to remain substantially below its pre-AIDS level for decades, and to return to it only between 2060 and 2070 (Fig. 10.2). Which will be

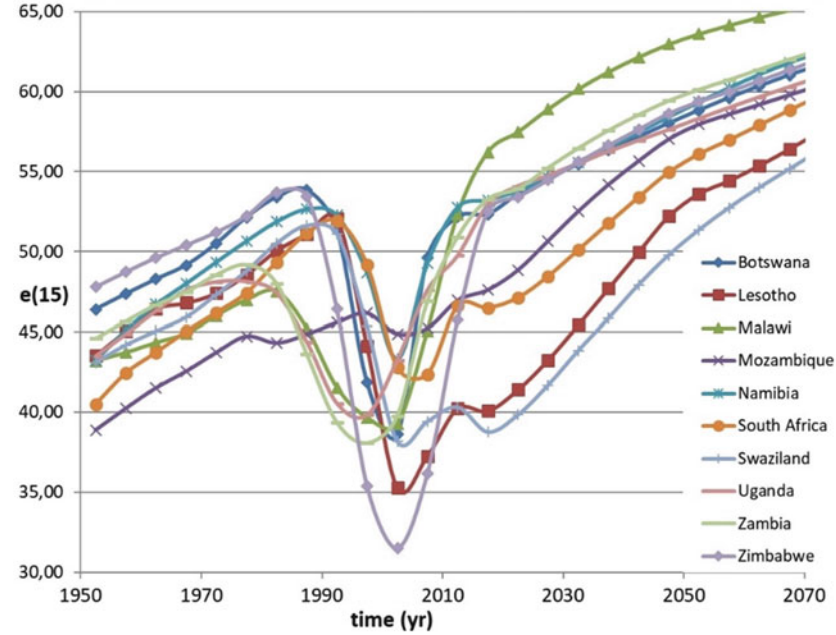

Fig. 10.2 Temporal trends of overall (male + female) life expectancy at age 15 (e_{15}) in most afflicted SSA countries. Source: UN medium variant (UN, 2017)

the ultimate consequences of the persistent fall in life expectancy for young adults on economic decisions (e.g., investment in children and their education) is presently unclear. Surely, mortality decline has represented the major trigger of the fertility transition in both conventional demographic explanations (Bongaarts and Casterline, 2012; Livi-Bacci, 2017), emphasising fertility decline as a direct homeostatic response to increasing survival, and modern demo-economic theories (Galor, 2011), emphasising the endogenous nature of mortality decline which promoted investments in education, thus favouring the switch in children's demand from "quantity" (Malthusian growth regime) to "quality" (modern growth regime). Focusing on the role played by the exceptionally high mortality of adults in SSA, Lorentzen et al. (2008) showed that it was the main cause of the collapsed economic growth and development in the region

via shortening time horizons, increasing risky behaviours and eventually raising fertility.

10.2.4 HIV/AIDS and the Fertility Transition

The issue of the possible effects of HIV/AIDS on fertility decisions in SSA raised in the previous section, is currently the object of a tough debate in the recent empirical economic literature.[1] The resulting conclusions are however dramatically variable, with some studies suggesting that AIDS mortality is already having a substantial positive effect on fertility in SSA and others predicting that AIDS is not having any effects or even lowering fertility in SSA (an extensive literature review on the subject is reported in Gori et al., 2017).

As remarked by influential scholars (Bongaarts and Casterline, 2012), the fertility transition in SSA has been dramatically delayed (the most dramatic evidence is represented by the case of the Democratic Republic of the Congo, Romaniuk, 2011) compared to other world regions started from similar initial conditions, such as Asia and the Latin America (Bongaarts and Casterline, 2012). In this general context, a worrying news has been represented by the generalised slowing down of fertility decline in the early 2000's (Schoumaker, 2009) and especially by the symptoms of stalling or even relapsing fertility in the group of countries suffering the most severe HIV/AIDS epidemics, which notably were SSA leaders in fertility decline at the onset of the HIV epidemics, according to UN data (UN, 2017). Additionally, in South Africa, where fertility was decreasing since already 1950, the decline was dramatically slowed down after HIV prevalence became substantial in the 1980's. This is a remarkable fact as, on the contrary, in SSA countries suffering intermediate HIV epidemics (e.g., Malawi, Zambia, Mozambique and Kenya), fertility decline slowed down during 1995–2005 but accelerated thereafter (UN, 2017), while no slowing down was observed in countries suffering mild epidemics.

Though these are just temporal associations based on aggregate data, the possibility that the persistent mortality blow-up due to AIDS might cause a paralysis or even a reversal in the fertility transition in SSA might have such dramatic implications for the development of the region so

to represent, in our opinion, one of the major conundra for current economic development studies.

10.3 The Model

This section builds on a modified version with HIV spread of the growth model of Yakita (2010) with physical and human capital accumulation. The OLG (general equilibrium) closed economy is populated by a continuum of finite-lived (perfectly) rational and identical individuals of size N_t per generation ($t = 0, 1, 2, \ldots$ is the time index). The length of each generation is conventionally set at 25 years. The life of the typical agent belonging to generation t is divided into childhood, young adulthood and old age. As a child, an individual does not make any economic decisions and directly consumes resources in the parent's household, while also receiving education from his parents. We consequently avoid including AIDS-related child mortality in the model as justified by the fact that the AIDS-mortality burden amongst children (following by vertically transmitted AIDS) is steadily decreasing thanks to pre-natal treatments, and anyhow second-order compared to AIDS-related adult mortality in SSA (UNAIDS, 2018). Therefore, we only consider the children surviving at the entry of adulthood (n_t) as the key variable of the model (Galor and Weil, 1996; Gori and Sodini, 2019). As a young adult, an individual is economically and sexually active. He works, saves, gives birth and takes care of children, and may also acquire HIV infection. The HIV spread follows the rule developed by Chakraborty et al. (2016), which will be clarified below. In addition, he invests the amount e_t per child for educational purposes of their children. When young, an individual is endowed with 1 unit of time. We assume that raising children is a purely time-consuming activity (see Guryan et al., 2008 for empirical evidence). The child rearing technology requires a positive exogenous fraction $z > 0$ of the parent's time endowment to raise a child (representing parent's foregone earnings), i.e. the time required to care for children cannot be spent working. As n_t represents the number of surviving children at time t, zn_t is the time needed to care for n_t descendants of a parent that belongs to generation t. This implies that the opportunity cost of children is

proportional to the wage rate per effective labour (w_t), which is provided to firms for production purposes. Therefore, the budget constraint when young reads as follows:

$$s_t + e_t n_t = (1 - \tau)(1 - z n_t) w_t h_t, \tag{10.1}$$

where s_t is saving, $e_t n_t$ is the total private expenditure that parents devote to educate their own n_t children, $0 \leq \tau < 1$ is the constant labour income tax rate levied by the government to finance a public expenditure specifically devoted to fight HIV, and $w_t h_t$ is the total labour income of an individual of the working-age generation who has h_t efficient units of human capital. Of course, the condition $n_t < 1/z$ should always hold to guarantee economic feasibility.

By following Yakita (2010), the labour productivity of a young adult individual is given by the stock of human capital endowed at the beginning of the working period. The stock of human capital of an individual belonging to the next (working-age) generation (h_{t+1}) depends on the educational expenditure per child provided by the current working-age generation to their children (e_t) and the existing stock of human capital (h_t). The human capital accumulation rule therefore is the following:

$$h_{t+1} = \varepsilon (e_t + \theta h_t)^{\delta} \overline{h}_t^{1-\delta}, \quad \varepsilon, \theta > 0, \quad 0 < \delta \leq 1, \tag{10.2}$$

where $\overline{h}_t$ is the average or economy-wide stock of human capital reflecting the spillovers from the society at time t, ε is a scaling parameter, δ is the constant elasticity of the overall private component $e_t + \theta h_t$ in human capital accumulation. This term $e_t + \theta h_t$ implies perfect substitutability in the process of human capital accumulation between the expenditure in education provided by parents to children and the ability of children to absorb knowledge by looking at their own parents at work (parental background). This ability is tuned by the positive parameter $\theta > 0$ (see Yakita, 2010 for details).

The survival probability of a young adult agent belonging to generation t, $0 < \beta_t \leq 1$, is endogenous and determined by his individual state of health. The existence of HIV/AIDS makes the individual health status

worse off so that adult survival negatively depends on the prevalence rate of HIV infection i_t, representing the proportion of agents infected at time t. We assume that this probability has the following simplified form:

$$\beta_t = \beta_A (1 - i_t), \tag{10.3}$$

where $\beta_A < 1$ represents (exogenous) survival from causes of death different from AIDS. This formulation is obviously over-simplified, but it aims to reflect the well documented facts that prior to the HIV/AIDS crisis: (1) life expectancies at all ages were increasing everywhere in SSA (UN, 2017), despite the dramatic burden of mortality claimed by other communicable diseases such as malaria and tuberculosis; (2) HIV/AIDS has represented for three decades the most important cause of death in SSA. The purpose of the formulation of endogenous survival implicit in Eq. (10.3) is to reflect the role of premature death of adults due to HIV/AIDS as the dominant effect of the epidemic at the macroeconomic level. We are aware that there are several other important effects of HIV, first the burden of child mortality through vertical transmission. Although dramatically relevant from a humanitarian point of view, this factor does not affect the structure of the model as it deals with non-economically active agents (children).

Young adults may acquire HIV infection by sexual transmission only. The probability p_t that an HIV-susceptible agent will become infected is defined as follows (Chakraborty et al., 2016):

$$p_t = 1 - (1 - i_t \pi_t)^{\mu}. \tag{10.4}$$

where $0 < \pi_t \leq 1$ is the probability of acquiring the infection per sexual partnership and $\mu > 0$ is the average number of sexual encounters that a young agent has during his entire adulthood. If the population is large, the prevalence rate at time t amongst young adults converges to the probability that a young adult agent can be HIV-infected, i.e. $i_t = p_{t-1}$ for any t. In this model, the probability π_t of acquiring HIV per sexual partnership negatively is assumed to depend on three main factors, which are: (1) private education (e_t) that contributes to raise individual awareness against the risk of HIV infection and therefore promotes subsequent

actions such as the use of protections during sexual intercourse, (2) public interventions, which are endogenously managed by the government of the afflicted country, that are provided in the model on a per worker basis (g_t),[2] and (3) foreign aid donations that are instead provided on a per young basis (d_t). The last component is assumed to depend on two main factors that should drive international programmes in SSA, namely the rate of HIV prevalence and the GDP of the afflicted country (Haacker, 2009), which is measured on a per young basis for analytical convenience. We will turn to the specifics of all these three components affecting π_t later in this chapter. Then,

$$\pi_t = \frac{\pi_A}{1 + \pi_B (q e_t^* + g_t + d_t)^{\omega}}. \tag{10.5}$$

where $\omega > 0$ is a parameter that controls the degree of effectiveness of the overall amount of resources aimed at fighting the HIV disease, $0 < \pi_A \leq 1$ ($\pi_A > 1/\mu$), $\pi_B > 0$ and $e_t^* := e_t / w_t h_t$ is the expenditure per child over the individual total income and $q > 0$ represents the relative weight of the educational component in reducing the probability of acquiring HIV per sexual partnership.

If an individual survives at the onset of old age, his material consumption is based on accumulated saving. The existence of a perfect market for annuities allows an agent to make his own saving being intermediated through mutual funds. This assumption follows a recent established literature on endogenous lifetime in growth models dealing with economic development issues (Chakraborty, 2004; Fanti and Gori, 2014; Chakraborty et al., 2016), and implies that the savings of those who prematurely die (due to AIDS in this context) are allocated amongst those who are still alive.[3] Therefore, old-age material consumption (c_{t+1}) is constrained by the capitalised amount of resources saved when young divided by the survival probability of a young person of generation t, that is:

$$c_{t+1} = \frac{R_{t+1}^e}{\beta_t} s_t, \tag{10.6}$$

where R^e_{t+1} is the interest factor that individuals of generation t expect will prevail at time $t+1$.

The individual representative of generation t has preferences towards the number of children when young and material consumption and the quality of children (represented by the human capital accumulated through education) when he will be old. Therefore, the expected lifetime utility function has the following form:

$$U_t = \ln(n_t) + \beta_t \left[\ln(c_{t+1}) + \gamma \ln(h_{t+1})\right], \tag{10.7}$$

where $\gamma > 0$ measures the relative degree of altruism towards (the quality of) children and β_t, which resembles the subjective discount factor, is given by the expression in (10.3) and then it is negatively affected by the prevalence rate of the HIV disease, i_t. In other words, an increase in i_t causes an increase in adult mortality (i.e., a reduction in the intertemporal discount factor) and this in turn implies that individuals of generation t reduce the relative importance (in the utility function) of consuming material goods and enjoying having educated children when old because they live shorter. By taking as given factor prices, the tax rate and the prevalence rate of HIV, the representative agent maximises the expected lifetime utility function in (10.7) with respect to material consumption (c_{t+1}), the number of children (n_t) and the amount of educational expenditure per child (e_t) subject to the lifetime budget constraint

$$\frac{c_{t+1}\beta_t}{R^e_{t+1}} + e_t n_t = (1-\tau)(1-zn_t) w_t h_t, \tag{10.8}$$

obtained by combining (10.1) and (10.6). Then, he gets the following optimal values for the saving rate s_t, the number (precautionary demand) of children n_t and the quality (education) of children e_t[4]:

$$s_t = \frac{\beta_t (1-\tau) w_t h_t}{1+\beta_t}, \tag{10.9}$$

$$n_t = \begin{cases} \frac{1}{z(1+\beta_t)} & if \quad w_t \le \overline{w}_t \\ \frac{(1-\beta_t\delta\gamma)(1-\tau)w_t}{(1+\beta_t)[z(1-\tau)w_t-\theta]} & if \quad w_t > \overline{w}_t \end{cases}, \tag{10.10}$$

and

$$e_t = \begin{cases} 0 & if \quad w_t \leq \overline{w}_t \\ \frac{h_t[z(1-\tau)\beta_t\delta\gamma w_t - \theta]}{1-\beta_t\delta\gamma} & if \quad w > \overline{w}_t \end{cases}, \tag{10.11}$$

where $\overline{w}_t := \frac{\theta}{z(1-\tau)\beta_t\delta\gamma}$ is the threshold value of the wage rate per effective labour above which the representative individual has an incentive to invest in child quality. We note that the condition $w_t > \overline{w}_t$ also guarantees a positive value of the precautionary demand for children. For details regarding the discussion of marginal benefits and costs of investing in child quality see Yakita (2010). A specific feature of our model in comparison with the one developed by Yakita (2010) is the role played by the survival probability β_t on the critical value $\overline{w}_t$ that discriminates between the corner solution (a state in which parents do not want to invest in child quality) and the interior solution (a state in which parents are likely to spend on education of their children rather than having more children). As β_t negatively depends on i_t, it is straightforward to see that an increase in the rate of HIV prevalence reduces the survival probability of adults by increasing $\overline{w}_t$ thus making less convenient investing in education, as the marginal cost of educating children increases. This model therefore includes the quantity-quality trade-off as well as the substitutability between education provided by parents and intergenerational human capital transmission within the family in the process of human capital accumulation in presence of an endogenous mechanism of HIV transmission. These are reasonable ingredients to characterise SSA economies.

At time t, the government provides the amount G_t of public expenditure to fight HIV. The term G_t therefore includes any type of relevant actions such as building health infrastructures, providing effective antiretroviral therapies (ART) for seropositive and AIDS-sick individuals, physicians and medicines, promoting awareness on the risk of infections and therefore the adoption of safe sexual practices such as e.g. the use of condoms and so on, with the purpose to reduce the probability of acquiring HIV per sexual partnership.[5] These expenditures are devoted to young-adult people, which are the ones who are HIV-susceptible and

may then acquire HIV through sexual transmission. The HIV spending is financed by the government of the afflicted country at a balanced budget through labour income taxation. As the tax rate is $0 \leq \tau < 1$, the total amount of tax revenues is $\tau w_t L_t$, where L_t is the quantity of effective labour employed. Defining now $g_t \coloneqq G_t/L_t$ as the amount of public expenditure per worker, the government accounting rule at a balanced budget is the following:

$$g_t = \tau w_t. \tag{10.12}$$

In addition to the public (internal) HIV-related expenditure, there exist external resources provided by international organisations (e.g., UN and Bill & Melinda Gates Foundation) to high HIV-prevalent SSA countries. These resources (defined on a per young basis) depend on the GDP of the afflicted country (UNAIDS, 2018) and the rate of HIV prevalence according to the following rule:

$$d_t = \begin{cases} ai_t - by_t & if \;\; i_t > (b/a)\, y_t \\ 0 \quad if \;\; i_t \leq (b/a)\, y_t \end{cases}, \tag{10.13}$$

where a, $b > 0$ are the share of external resources devoted to each single HIV-afflicted individual at time t (based on the rate of HIV prevalence) and the share of external resources per head devoted to each young living at time t (based on income), respectively. The rule in (10.13) implies that international organisations increase foreign aid in SSA as HIV prevalence increases and reduce it as the HIV-afflicted country gets richer. When the HIV prevalence in one country is large (resp. small) enough, external donations are based on a mix between the two quotas discussed above (resp. become null). In the last case, the fight against the HIV/AIDS disease is entirely based on internal resources of the afflicted country, which then becomes fully autonomous in the battle against HIV, according to the auspices raised by the deputy director of UNAIDS (2013).

As individuals are identical, the condition $h_t = \overline{h}_t$ holds in a symmetric equilibrium. Therefore, depending on the level of education coming from the interior or corner solution in (10.11) of the individual optimisation

problem, human capital accumulation (10.2) becomes the following:

$$h_{t+1} = \begin{cases} \varepsilon\theta^{\delta} h_t & if \quad w_t \leq \overline{w}_t \\ \varepsilon(\beta_t \delta\gamma)^{\delta} \left[\frac{z(1-\tau)w_t - \theta}{1-\beta_t\delta\gamma}\right]^{\delta} h_t & if \quad w > \overline{w}_t \end{cases}. \tag{10.14}$$

When the wage is small enough ($w_t \leq \overline{w}_t$) parents choose to do not provide education to their children. In this case, the human capital growth rate is constant and depends only on parental background. It may grow indefinitely, shrink or follow a stationary pathway depending on whether the term $\varepsilon\theta^{\delta}$ is larger than, smaller than or equal to 1, respectively. In contrast, when the wage is large enough ($w_t > \overline{w}_t$) there exists a child quantity-quality trade-off as parents want to provide education to their children and the growth rate of human capital depends on the wage rate per effective labour.

Regarding the production side of the economy, firms are identical and act competitively on the market. The time-t representative firm produces a homogeneous good Y_t by combining physical capital K_t and the amount L_t of effective labour. These inputs are combined by using a Cobb-Douglas technology with constant returns to scale, that is:

$$Y_t = K_t^{\alpha} L_t^{1-\alpha}, \quad 0 < \alpha < 1. \tag{10.15}$$

The temporary equilibrium condition in the labour market at time t is determined by equating labour demand and labour supply, that is $L_t = (1 - zn_t)h_t N_t$. By assuming full depreciation of capital, a unit price of output and taking factor prices as given, profit maximisation by the representative firm implies that the wage rate and the interest factor are equal to the marginal product of efficient labour and the marginal product of capital, respectively, that is:

$$w_t = (1-\alpha)\, v_t^{\alpha}(1 - zn_t)^{-\alpha}, \tag{10.16}$$

and

$$R_t = \alpha v_t^{\alpha-1}(1 - zn_t)^{1-\alpha}, \tag{10.17}$$

where $v_t := k_t/h_t$ is the physical to human capital ratio, $k_t := K_t/N_t$ is the stock of physical capital per young person and GDP per young person is defined as $y_t := Y_t/N_t = v_t^{\alpha}(1 - zn_t)^{1-\alpha}$.

Equilibrium in the capital market is determined by equating aggregate saving by the representative individual and aggregate investment by the representative firm, that is $S_t = I_t$. The inter-temporal equilibrium condition in the capital market requires that the aggregate capital stock installed at time $t + 1$ is equal to the gross aggregate investment at time t. As physical capital fully depreciates at the end of each period, we get $K_{t+1} = I_t = S_t = s_t N_t$. Knowing that $K_{t+1} := k_{t+1}N_{t+1}$ and $N_{t+1} = n_t N_t$, equilibrium is characterised as follows:

$$k_{t+1} = \frac{s_t}{n_t}. \tag{10.18}$$

For the sake of simplicity, from the next section we set the constant β_A (representing survival from causes of death different from AIDS) to 1, as in this context it merely represents a scaling parameter without relevant qualitative effects.

10.4 Main Theoretical Predictions

Depending on the size of the wage rate w_t compared to the threshold $\overline{w}_t$, there exist two different development regimes, as in Yakita (2010). If individuals are relatively poor ($w_t \leq \overline{w}_t$), the economy is entrapped in a phase of underdevelopment. If they become richer ($w_t > \overline{w}_t$), the economy may follow a sustained development trajectory.

(1) [Underdevelopment, $w_t \leq \overline{w}_t$]. The individual optimal choices are described by the saving function defined in (10.9) as well as by the values of the first expression of n_t and e_t in (10.10) and (10.11), respectively. In this regime, individuals do not invest in education and there exists only a precautionary demand for children. By using the same strategy of Yakita (2010), it is possible to study the evolution of

the physical to human capital ratio and the HIV prevalence through the following map M_1 defined in the variables v_t and i_t, that is

$$M_1 : \begin{cases} v_{t+1} = \frac{(1-\alpha)(1-\tau)z}{\varepsilon\theta^{\delta}} v_t^{\alpha}(1-i_t)^{1-\alpha}(2-i_t)^{-\alpha} \\ i_{t+1} = 1 - \left[1 - i_t \frac{\pi_A}{1+\pi_B(g_t+d_t)^{\omega}}\right]^{\mu} \end{cases}, \tag{10.19}$$

where $g_t = \tau w_t = \tau\,(1-\alpha)\, v_t^{\alpha}\left(\frac{2-i_t}{1-i_t}\right)^{\alpha}$ is the amount of internal public spending against HIV on a per worker basis and foreign aid d_t is defined in (10.13). If foreign aids and/or the internal public health are strong enough to eliminate HIV in the phase of underdevelopment, the model essentially falls within the one-dimensional system studied by Yakita (2010), in which the evolution of all the variables can be characterised by studying the dynamics of v_t.

Unfortunately, studying system (10.19) is not an easy task as it cannot be dealt with in a neat analytical form. Indeed, it can exhibit a plethora of stationary states with positive values of the HIV prevalence ($i_t > 0$) in addition to the one characterised by the absence of HIV/AIDS ($i_t = 0$). In this regard, we must distinguish between the case in which international organisations devote resources in the fight against HIV, $v_t < \hat{v}_t$, to the case in which foreign aids are absent, $v_t \geq \hat{v}_t$, where $\hat{v}_t := \left(\frac{ai_t}{b}\right)^{\frac{1}{\alpha}}\left(\frac{1-i_t}{2-i_t}\right)^{\frac{\alpha-1}{\alpha}}$ represents the threshold value of the physical to human capital ratio that discriminates between international intervention and no international intervention, according to the rule specified in (10.13). We recall that the existence of (one or more) stationary states in the underdevelopment regime is possible only whether the condition $v_t \leq \overline{v}_t := \left[\frac{\theta}{z(1-\tau)\delta\gamma(1-\alpha)(1-i_t)}\right]^{\frac{1}{\alpha}}\left(\frac{1-i_t}{2-i_t}\right)$ is satisfied, where $\overline{v}_t$ is evaluated at its stationary state value. This threshold is obtained by replacing n_t with its optimal value, through the first expression of Eq. (10.10), in the equation of the marginal product of efficient labour in (10.16) and using $\overline{w}_t$. In this regard, we recall that, apart from the infectious dynamics, a stationary state in this model can be related to unbounded growth of physical and human capital if $\varepsilon\theta^{\delta} > 1$ or to a

situation where the human capital continuously reduces and the physical capital tends towards its stationary state level if $\varepsilon\theta^{\delta} < 1$ (see Yakita, 2010, for details). If the physical to human capital ratio is larger than the critical value $\overline{v}_t$, there exist no stationary states with a positive value of v in the underdevelopment regime. The stability properties of the stationary states of the dynamic system (10.19) are studied by resorting to numerical analyses given the complexity of the expressions of the map.

(2) [Beyond the development trap, $w_t > \overline{w}_t$]. In this case, the individual optimal choices are described by the saving function defined in (10.9) as well as by the values of the second expression of n_t and e_t in (10.10) and (10.11), respectively. In this regime, individuals are rich enough to invest in education and therefore they decide to trade off child quantity against child quality. Then, equilibrium dynamics of v_t and i_t are described by the following map M_2:

$$M_2 : \begin{cases} v_{t+1} = \dfrac{\frac{1-i_t}{2-i_t}\frac{1-\alpha}{(1-zn_t)^{\alpha}}}{\varepsilon\left[\frac{(1-i_t)\gamma\delta}{1-(1-i_t)\gamma\delta}\right]^{\delta}\left[z(1-\tau)(1-\alpha)\left(\frac{v_t}{1-zn_t}\right)^{\alpha}-\theta\right]^{\delta} n_t} v_t^{\alpha} \\ i_{t+1} = 1 - \left[1 - i_t \frac{\pi_A}{1+\pi_B (e_t^{*}+g_t+d_t)^{\omega}}\right]^{\mu} \end{cases}, \tag{10.20}$$

where $e_t = \frac{h_t[z(1-\tau)\beta_t\delta\gamma w_t-\theta]}{1-\beta_t\delta\gamma}$, $g_t = \tau w_t = \tau(1-\alpha) v_t^{\alpha}(1-zn_t)^{-\alpha}$, d_t is defined in (10.13) and n_t is determined as the *unique* solution of $n_t = \frac{[1-(1-i_t)\delta\gamma](1-\tau)(1-\alpha)v_t^{\alpha}(1-zn_t)^{-\alpha}}{(2-i_t)\left[z(1-\tau)(1-\alpha)v_t^{\alpha}(1-zn_t)^{-\alpha}-\theta\right]}$. Given the complexity of the dynamic system (10.20) and the problems related to the analytical tractability of characterising the possible switches between the two distinct regimes governed by maps M_1 and M_2, in what follows we will resort to numerical simulations to concentrate on some demo-economic results of the model. In doing this, we will follow the methodology introduced by Chakraborty et al. (2010, 2016) by fixing the main parameters of the problem at economically meaningful values for SSA countries and then studying the effects of both internal and external health policies on the HIV prevalence, the economic dynamics and their (macroeconomic) interplay.

10.5 Dynamics of the Model and Discussion of the Main Results

This section builds on a simulative exercise aiming at studying the economic and epidemiological dynamics characterised by Eqs. (10.19) and (10.20). The parameters of the model are chosen as follows: $\alpha = 0.37$ (Gollin, 2002), $\delta = 0.535$, $\theta = 0.011$, $\varepsilon = 22.3$ (these values are similar to those used by Yakita, 2010), $\gamma = 1.52$, $z = 0.12$ (assigned to obtain a fertility rate in line with SSA), $\pi_A = 0.0123$, $\pi_B = 35$, $\mu = 100$, $q = 0.1$ and $\omega = 1$ (assigned to get an equilibrium prevalence rate of 35% in the case of an uncontrolled HIV in the phase of underdevelopment).

First, we considered an economy without the HIV disease that moves away (at time $t = 3$)—thanks to human capital accumulation—from the underdevelopment regime, where parents did not invest in education and there existed only a precautionary demand for children, to a sustained development regime, where there is a child quantity-quality trade off. We have considered a parameterisation excluding the existence of poverty traps due to the initial conditions (history). This is because the main aim of the work is to stress the role of HIV in determining a fall back in poverty. The economy begins with a situation where fertility is constant and fixed at 4.16 children per woman in a phase of underdevelopment, ending up in context of sustained development where each parent has 2.85 children on average. In this last regime, education helps reducing the precautionary demand for children by changing the marginal conditions for having an extra child. At $t = 6$, the HIV disease begins ($i_6 = 0.01$) and the prevalence rate would reach 35% (in line with the values of some high-prevalent SSA countries) in the case of an uncontrolled spread. HIV is responsible for the falling back of the economy into a phase of underdevelopment due to a lowering life expectancy leading to a lower saving and higher fertility due to increased mortality by also reducing education to zero (child quality-quantity trade off). It is important to note that education has partially slowed down the development of HIV (clearly, education becomes zero when one falls back into the early stage of economic development).

An important role in addressing the humanitarian emergency due to HIV infection has been played by international aids from both private and public institutions (donors). In our simulative exercise, we calibrated these interventions by linking them positively to the prevalence rate and negatively to the income produced by the afflicted country (measured on a per young basis) according to the rule: the higher income, the lower international donations. Foreign aids start at $t = 48$ when the HIV prevalence is almost $i_{48} = 0.23$ succeeding in bringing the amount of infected people in the afflicted country below the threshold of 1% and letting the economy move on a sustained development path with positive levels of education and lower fertility. Once donors are reset to zero because the prevalence rate has gone below the critical threshold ($i_t = (b/a)y_t$, which was set at 1% with $a = 4$ and $b = 0.17$), we explored the possibility of an endogenous intervention by the government of the afflicted country aiming at replacing external aids with an internal policy financed by levying taxes on labour income ($\tau = 0.1$). We note that by setting the tax rate τ at too high a level would greatly reduce individual disposable income and saving, which in turn reduce physical capital accumulation by also stimulating the precautionary demand for children. Differently, setting the tax rate at too low a level would be ineffective and would make the disease spreading again. Therefore, the public expenditure against HIV initially causes a reduction in the demand for education (child quality) and increases fertility (child quantity) but at lower birth rates than those observed when the donors were active. This is because the direct negative effect on income due to labour income taxation is high and dominates the indirect positive effect of a lower HIV on mortality reduction. However, this taxation can be decreased over time, thus making the endogenous policy dynamically reactive and linked to the prevalence rates observed. Also, in this phase education plays a positive role and contributes to reducing both the number of children and HIV, but with a much lower intensity than in a pre-AIDS setting. We note, however, that long-term fertility is higher than the values observed in the (pre-AIDS) early stages of demographic transition. AIDS therefore represents a major cause depressing the development pathway of SSA countries (Figs. 10.3 and 10.4).

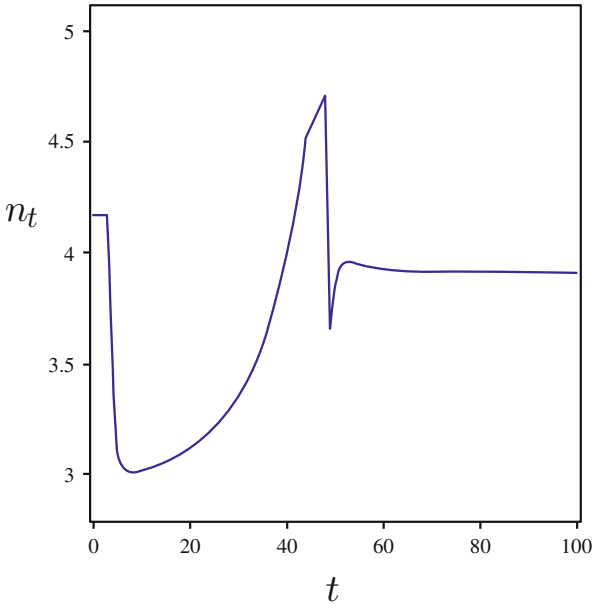

Fig. 10.3 Fertility

10.6 Conclusions

For more than three decades, HIV/AIDS has ravaged SSA and still now is claiming dramatic toll of deaths. Nonetheless, the increasing diffusion of anti-retroviral therapies and awareness campaigns have finally downturned the epidemic curve and brought optimistic perspectives on the control of the disease, as summarised in the global 90-90-90 plan (UNAIDS, 2018) aiming to bringing AIDS under full control by 2030. However, the battle against HIV in SSA is far from its end and several challenges are still open. Amongst these, we believe that there are at least three major threats that are critical for their potential effects to economic development and therefore should deserve major attention at the highest policy level, that is (1) the potential for an HIV-induced paralysis of the fertility transition, (2) the need for a dramatic expansion of the amount of resources devoted to funding the interventions against

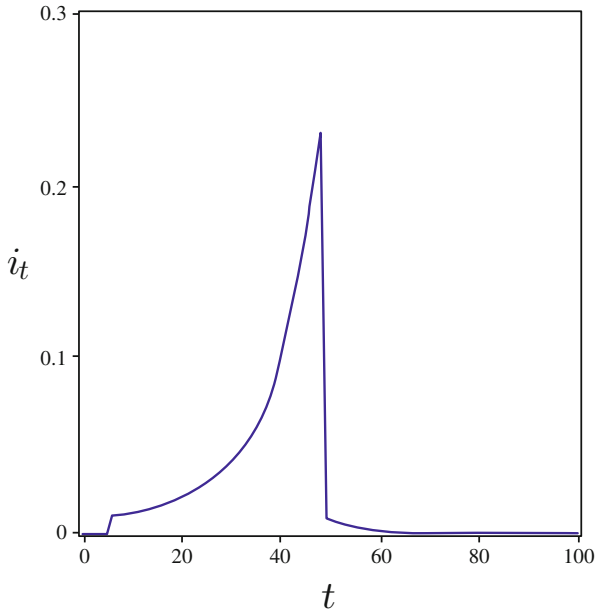

Fig. 10.4 HIV prevalence

HIV, and (3) the unclear magnitude of the emerging phenomenon of resistance to treatments. This work developed a macroeconomic dynamic framework capable to account for both the first two previously mentioned factors within a UGT-like growth set up. The model includes an explicit HIV transmission mechanism—as first proposed by Chakraborty et al. (2010, 2016)—and different funding rules of the interventions against HIV, covering almost all the kinds of interventions used in actual SSA countries. We believe that the explicit inclusion of the epidemic dynamics goes far enough beyond the possibility offered by models simply treating HIV as an exogenous mortality shock, as it allows to pinpoint the dynamic interplay between the time scales of the epidemics with those of fertility decline and, ultimately, of economic development.

Our main conclusions dramatically contrast Young's (2005) results, according to which AIDS is a humanitarian disaster but not an economic disaster. Indeed, we found that HIV has the potential to constrain an

economy in an underdevelopment regime with high HIV-prevalence rates and high fertility, or to worsen the conditions for being entrapped in poverty by reversing the long-term trend of fertility decline. The issue of the AIDS-induced fertility reversal is currently a controversial one. In Gori et al. (2017), we have surveyed several empirical contributions on the subject, showing a wide degree of disagreement in the responses, with a number of papers suggesting that HIV has a potential not only for stalling but also reversing the fertility transition in SSA, as first suggested in the seminal works of Kalemli-Ozcan and Turan (2011) and Kalemli-Ozcan (2012), and a similar number of papers reaching opposite conclusions. However, we found that countries suffering a combination of the following ingredients: (1) a stall in the fertility transition, (2) a large HIV epidemic, (3) negative perspectives on life expectancies requiring long intervals of time to restore the pre-AIDS levels, as is the case for example of Lesotho, should represent the highest priority for international interventions in SSA. We feel that this message is not so far at the top of the debate on the topic. On top of this, the present work has added the issue of the financing of the battle against HIV/AIDS. The model clearly enlightens the conditions under which an international intervention is necessary versus the circumstances that may allow afflicted countries to support the AIDS battle by using internal resources without compromising the escape form the poverty trap. In this last case, private education might play a key role in the battle against HIV by enhancing the internal public policy, therefore setting in motion a virtuous circle capable to simultaneously control epidemics and fertility.

We did not explicitly include in the model the third challenge, namely the resistance issue. This was due to the lack of clear evidence on the magnitude of the phenomenon in SSA which prevented a meaningful parameterisation of it. Nonetheless, the widespread diffusion of resistance would simply worsen the control conditions of HIV/AIDS predicted by our model and ultimately the perspectives for economic development.

The list of possible future developments is clearly endless. A point of major interest in our research agenda regards the implications (in terms of both survival and education) for the children of adults prematurely dying of AIDS. In this regard, a naturally raising question is whether

orphans suffer higher mortality and lower education opportunities than non-orphans (particularly in high-prevalence HIV settings) and then how to build a proper model for this, for instance along the lines of the approach proposed by Bell and Gersbach (2013). In general, this is a complicate problem for the model considered in our chapter that cannot be solved simply by adding (endogenous) child mortality, as this is a general shortcoming of OLG settings regardless of whether fertility is endogenous (for instance, the UGT set up) or exogenous (Diamond, 1965). In fact, both frameworks did not account for an economically active childhood generation. In our opinion, a solution to the problem would require to fully account for the heterogeneity between HIV/AIDS-sick versus non-sick parents and then accounting the possible different options available to, or behaviours of, children. This would imply at least the building of a heterogeneous-agents OLG settings, with heterogeneous utility functions in turn implying to also consider the effects of the peculiar socio-cultural SSA settings, such as for example the presence or not of extended households, which can offer care to orphans.

Conflict of Interest The authors declare that they have no conflict of interest.

Notes

1. On the demographic side, it should be recalled the work of Schoumaker (2009) pointing out that part of the fertility stall observed in SSA since the 2000 might partly be spurious due to data quality issues. Though clearly this is an important problem to be tackled in the future, we believe that the policy prescriptions of the present work are at all unaffected.
2. We chose to define the public expenditure against HIV on a per worker basis rather than in per young terms (in line with the definitions of the other main variables used later in this work) for reasons of analytical tractability.
3. We are aware that the assumption of perfect annuities does not perfectly describe the behaviour of intergenerational transmission of inheritance under uncertain survival in contexts such as SSA countries, as it is clearly well suited for settings where financial markets are adequately developed. However, the use of the alternative hypothesis of accidental bequests does

not allow to characterise the behaviour of individual fertility and thus the dynamics of the physical to human capital ratio and the rate of HIV prevalence.

4. The first order conditions define the optimal solution of the problem if and only if $\beta_t \delta \gamma < 1$ (this condition is assumed to hold throughout the work). As β_t beta depends on the prevalence rate of HIV, which in turn may vary depending on internal and external resources aimed at fighting the disease, in our analysis we will use the most restricting assumption $\delta \gamma < 1$.
5. The issue of contraception, which has been the object of a recent interest in development studies about the fertility transition in industrialised countries (Bhattacharya and Chakraborty, 2017; Prettner and Strulik, 2017) is of interest in relation to the interplay between HIV/AIDS and the fertility transition in SSA. Indeed, SSA is also characterised as the only region in the world where low levels of contraception are persisting (Greenwood et al., 2013) thus representing a main factor of the permanence of high fertility (Cleland, 2009). Therefore, the awareness of the risk of HIV might have an important by product in terms of fertility control.

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