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ESSAYS ON HUMAN EVOLUTION AND ECONOMIC GROWTH

BY

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ABSTRACT

This thesis explores a series of dynamic models incorporating evolutionary and economic dynamics and demonstrates the contribution that an evolutionary approach can make to economics.

After assessing the extent to which economic thinkers have incorporated evolutionary theory into their analysis, we show that a simulation of the model developed in Galor and Moav [Galor, Oded and Omer Moav (2002) Natural selection and the origin of economic growth. *Quarterly Journal of Economics* 117(4), 1133–1191] resembles the period of Malthusian stagnation before the Industrial Revolution and the take-off into a modern growth era. We investigate the stability of the modern growth era and show the economy can regress to a Malthusian state.

We then present an evolutionary theory of long-term economic growth in which the human ability to develop new ideas evolves over time. As more mutations occur in larger populations, population growth will increase the rate at which innovation-enhancing traits may emerge. Human evolution enhances population growth and it makes the population more robust to technological shocks. As the population grows, the scale effect of population size on the production of ideas becomes a relatively more important driver of technological progress than further increases in innovative potential.

In the following chapter, we propose the evolution by sexual selection of the male propensity to engage in conspicuous consumption contributed to modern rates of economic growth. We develop a model in which males engage in conspicuous consumption to send an honest signal of their quality to females. As males fund their conspicuous consumption through participation in the labour force, an increase in the prevalence of signalling males gives rise to an increase in economic activity.

Finally, we propose the recent rise in the fertility rate in developed countries may be the beginning of a broader increase in fertility towards above-replacement levels. Environmental shocks that reduced fertility over the past 200 years temporarily raised fertility heritability. As those with higher fertility are selected for, the “high-fertility genotypes” are expected to come to dominate the population. Even with low levels of genetically based variation in fertility, there can be a rapid return to a high-fertility state. In the longer term, this implies that the proportion of elderly in the population will be lower than projected.

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CHAPTER 1

INTRODUCTION

Our economic traits and preferences are shaped by evolutionary processes. This biological insight has spawned an interest in the interaction between human evolution and economic growth and development. Although often occurring over timeframes that are not relevant to many economic questions, evolutionary dynamics are important to long-term economic issues such as the emergence of economic growth.

Recently, some researchers have applied theoretical and molecular genetic approaches to macroeconomic questions and economic growth (Galor and Moav 2002; Zak and Park 2002; Clark 2007; Spolaore and Wacziarg 2009; Galor and Michalopoulos 2012; Ashraf and Galor 2013). This thesis builds on their work by presenting four essays on the role of the evolution of human traits and preferences in economic growth and development.

Chapter 2 establishes the context for these essays and comprises a review of the use of evolutionary biology in economics. Four major areas of study are identified: the evolution of preferences; the genetic basis of economic behaviour; the interplay of economic and evolutionary dynamics; and the genetic foundations of economic development. This research shows that previous work at the theoretical level is progressively supplemented by increasingly available and inexpensive genetic data, which further enables the use of evolutionary biology in economics. The essays in this thesis relate to the third of these research areas.

Chapter 3 focuses on the seminal theoretical paper linking human evolution and economic growth: Galor and Moav's (2002) *Natural Selection and the Origin of Economic Growth*. A simulation of Galor and Moav's model shows a pattern of population growth, technological progress and income growth that reflects the period of Malthusian stagnation before the Industrial Revolution, the economic take-off during the Industrial Revolution and the sustained economic growth in the modern era. However, the stability of the modern growth era can be affected by the presence of agents with a strong preference for quantity of children, which undermines human capital formation. This dynamic evolutionary analysis yields materially different predictions to those made without an evolutionary framework and presents a basic challenge to standard endogenous growth models.

In chapter 4, the economic consequences of a larger population generating more mutations and greater potential for evolutionary change are considered. By incorporating an evolving trait into the population that affects research productivity, which may be called "innovative potential", it is shown that population growth is proportional to both the population size and the innovative potential of that population. Further, as the innovative potential of the population increases, further increases in population size become a more important driver of the acceleration of population growth and technological progress than continuing increases in innovative potential.

This chapter raises two major questions: which traits associated with the expansion of the technological frontier have spread through populations in recent evolutionary history (that is, what is innovative potential?) and under what conditions does someone with those traits have higher fitness? Traits suggested in other work that may be tested under this framework include time preference, risk aversion, the preference for quantity or quality of children, a preference for hard work, and entrepreneurial spirit.

Chapter 5 examines one particular trait that may affect economic growth, the propensity of humans to engage in conspicuous consumption. Examination of the dynamic emergence of this trait shows that the trait can spread rapidly and drive a marked increased economic activity. This could explain the sudden appearance of conspicuous consumption goods in the historical record, the increasing technological progress after the emergence of this trait, and the level of work effort in modern society, which is maintained despite all resource needs for survival being met.

Work in evolutionary psychology has firmly established the evolutionary roots of conspicuous consumption, which leaves open questions for future research about when it emerged and through which pathways it most strongly operates. The effect of culture on the particular forms of conspicuous consumption may also provide scope for analysis given the broad arrays of conspicuous consumption that have been observed across millennia and societies.

In Chapter 6, human evolution is applied to a modern policy consideration, the population fertility rate. As fertility is a heritable trait, high-fertility genotypes will increase as a proportion of the population. It is plausible that this increase in fertility will occur within timeframes relevant to today's economic policy makers. To the extent that fertility data underpins projections of population structure, the future tax base, or education and health requirements, the evolutionary analysis suggests that current policy settings may not be calibrated to the challenges that will actually be faced. If the numerous causative factors of fertility shocks could be untangled, cross-country and cross-population differences in fertility patterns could provide an opportunity to examine this theory.

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CHAPTER 2

THE EVOLUTIONARY FOUNDATIONS OF ECONOMICS

“The Mecca of the economist lies in economic biology rather than in economic dynamics.”

Alfred Marshall, *Principles of Economics* (1920)

“Nothing in biology makes sense except in the light of evolution”

Theodosius Dobzhansky (1973)

2.1 Introduction

Human traits and preferences were shaped by natural selection. In that context, economics and evolutionary biology have been intertwined since the work of Thomas Malthus (1798) prepared the ground for Charles Darwin’s revolutionary development of the theory of evolution by natural selection (Darwin 1892).¹ Central contributors to the development of modern economics, such as Alfred Marshall (1920), recognised the relevance of biology and other natural sciences for economic analysis.² However, despite this early recognition, the use of evolutionary theory as a tool to analyse human preferences, economic growth and economic policy is a recent phenomenon.

¹ Hirshleifer (1977) noted that while Malthus’s influence on Darwin represents the influence of economics on biology, Malthus in turn had drawn his ideas from a biological generalisation of Benjamin Franklin.

² Marshall (1920) also wrote in the margin of Appendix C of *Principles of Economics*: “But economics has no near kinship with any physical science. It is a branch of biology broadly interpreted.”

In this paper we review the research at the interface between economics and evolutionary biology and the extent to which evolutionary thinking is influencing economics. Evolutionary biology has been used in four areas in economics: the evolution of preferences, the genetic basis of economic traits, the interaction of evolutionary and economic dynamics, and the genetic foundations of economic development. These four areas of interdisciplinary research are shown in the cells of Table 1. The left margin of the table indicates that one strand of research is mostly theoretical, exploring the structure of models with genetic foundations, whereas a second strand is more empirical, focusing on observable genetic and economic data. The top margin of the table shows that this research has been applied at the level of individual preferences in microeconomics and at the population level in macroeconomics and economic development. The cells in Table 1 provide the structure for the review that follows in the next four sections.

Table 1.1: Fields of research integrating economics and evolutionary biology

	Individual preferences and traits	Dynamic macroeconomic processes
Theoretical	Evolution of preferences (Section 2)	Interaction of evolutionary and economic dynamics (Section 4)
Empirical	Genetic basis of economic traits (Section 3)	Genetic foundations of economic development (Section 5)

The research on the evolution of preferences (Section 2) and the genetic basis of economic traits (Section 3) has been subject to previous reviews by Robson and Samuelson (2011a) and Benjamin et al. (2012a). Accordingly, we focus on the incorporation of these two fields into broader economic thought and the future opportunities in these areas. For our review of evolutionary economic dynamics

(Section 4) and the genetic foundations of economic development (Section 5), we present a more thorough analysis.

The subject matter of this paper needs to be distinguished from what is commonly called “evolutionary economics”. Evolutionary economics uses biological concepts, such as natural selection, and applies them to the dynamics of firms, business processes and institutions. The economy is seen as a complex adaptive system in which innovation and change are central considerations. The origin of evolutionary economics is often traced to Veblen (1898), and was revived by Alchian (1950) and later Nelson and Winter (1982), whose seminal work inspired a vast literature. The subject matter of this paper differs from evolutionary economics in that we focus on human biology rather than seeking to apply a biological analogy to higher levels such as firms. This paper is about the application of evolutionary biology to economic processes at the level of humans and their genes and their interactions at the population level.

2.2 The evolution of preferences

Human preferences play a central role in economic analysis. By understanding preferences, the response of individuals to economic incentives and the aggregate phenomena emerging in the population can be studied. Two early advocates of examining the evolutionary foundations of preferences were Becker (1976) and Hirshleifer (1977). Motivated by the publication of *Sociobiology* by E.O Wilson (1975), Becker and Hirshleifer saw the benefits of biological thinking in economics and parallels between the economic and biological ways of thought.

Becker (1976) argued that preferences could be explained by selection of traits with higher fitness. In illustration, he provided an explanation of the existence of altruistic behaviour, which by the usual definition of altruism harms the fitness of the altruist.

Extending his “rotten kid theorem” beyond the family, Becker argued that an altruist’s fitness may actually be strengthened if the altruist’s threat to transfer resources to harmed parties at a cost to the transgressor prevents the latter from harming people, including the altruist. This contrasts with explanations developed by biologists to explain the preference for altruism, such as kin selection (Hamilton 1964a, 1964b), reciprocal altruism (Trivers 1971), or group selection (Wynne-Edwards 1963).³ Becker’s model explains why altruism is not selected against, but it does not address how a preference for altruism could have evolved and spread through the population. Besides the evolution of preferences such as altruism, Hirshleifer (1977) saw sociobiological analysis as useful in examining the evolution of preferences, as well as understanding exchange and the division of labour, and in examining evolving as opposed to equilibrium socio-economic systems.

In this section, we review the work that has followed Becker and Hirshleifer’s initial advocacy of an evolutionary analysis of human preferences. In the first part, we deal with the objective of the economic agent, and in the second, the shape of the agent’s utility function.

2.2.1 Choosing the objective: consumption versus fitness

In evolutionary biology, fitness is an individual’s ultimate “objective”. Fitness, which is measured as the individual’s contribution of genes to the next generation, may be maximised by pursuing proximate objectives, with those proximate objectives shaped by evolution.⁴ By contrast, in economic models agents typically maximise utility from

³ The usefulness of group selection arguments remain a subject of debate [for example, (West et al. 2008; Eldakar and Wilson 2011)].

⁴ The distinction between proximate and ultimate evolutionary objectives was made by Mayr (1961) and Tinbergen (1963).

the consumption of a basket of goods and services. To reconcile the economic and evolutionary objectives, we need to ask if the proximate objective shaped by evolution is reflected in the utility function in economic models. In other words, does consumption maximises fitness? If it does not, consumption maximisation would not be selected for and other proximate objectives should be included in utility functions.

A seminal paper that illustrates this point is Rubin and Paul (1979) on the evolution of risk preferences. In their model, they defined utility as fitness, which depends on the number of females that a male attracts. This utility formulation explains changes in risk preferences of males as they age and gain additional income and resources. A male with a level of income below that required to attract a female will be risk seeking with respect to income, as a loss in income does not reduce his utility. A male with a level of income slightly above that required to attract a female will be risk averse, as a small drop in income will materially reduce his utility. This pattern would be repeated at higher levels of income wherever a threshold for additional mates is approached.

In another attempt to reconcile utility and fitness, Gandolfi et al. (2002) considered a framework in which a person maximises fitness by maximising long-term intergenerational wealth, which can be spent on children and their education as required. This model explains the low fertility in modern societies as a long-term fitness maximising strategy. It is not the number of children and their genes in the next generation that matters but the number of children over the entire future. However, the positive correlation between numbers of children across generations in developed countries (Rodgers et al. 2001; Murphy and Knudsen 2002) suggests that people with more children have higher fitness. More direct evidence that parents overinvest in the education of children to the detriment of their number comes from Kaplan et al. (1995), who found that men were not maximising their number of grandchildren.

This low fertility at the cost of fitness may be caused by the fact that today humans live in an environment that has changed dramatically, offering little time for selection to act on relevant traits.⁵ As evolution shapes traits through proximate mechanisms, a change in environment can result in pursuit of a proximate objective failing to maximise fitness (Bowlby 1969; Tooby and Cosmides 1992; Irons 1998). For example, the taste for fat and sweetness, which increased fitness when calories were scarce in the Malthusian environment, is leading to overconsumption of high calorie foods in modern times (Breslin 2013). Therefore, the traits under selection in past environments need to be considered to determine preferences, as proposed by Jones (2000), Miller (2003) and Burnham (2013). Alternatively, the evolutionary system could be considered dynamically, allowing people to adapt to the new environment and for utility maximising behaviour to move towards maximization of fitness.

It is also possible that consumption is fitness maximising through its role as a signal of quality.⁶ De Fraja (2009) showed that if male conspicuous consumption serves as a signal that females prefer when choosing mates, a utility function in which a male maximises consumption is equivalent to fitness maximisation. Other papers make an implicit assumption that consumption maximises fitness [for example, Hansson and Stuart (1990)], without stating how the agents allocate resources between consumption and reproduction. This approach may be justifiable as consumption could include

⁵ In Galor and Moav's (2002) model, the lower fitness of quality-preferring types in the modern growth era is due to this type of overinvestment.

⁶ Veblen (1899) coined the term conspicuous consumption for the wasteful signalling of wealth or other qualities. Amotz Zahavi (1975) argued that waste makes a signal reliable as only a high quality individual can carry the "handicap" imposed by the waste. Biologists debated whether Zahavi's concept was plausible [Maynard Smith (1976) concluded it was not], until Grafen (1990a, 1990b) showed that the condition for a handicap to be a reliable signal was that high and low-quality agents must face different marginal costs of signalling. This mechanism is the same as that in Spence's (1973) job market signalling model.

allocation of resources to the production of children. Alternatively, the findings of De Fraja could be used to justify this assumption.

2.2.2 The shape of the utility function: time preference and risk preference

Utility functions may have different shapes, with two properties – time preference and risk preference – attracting special attention. Time preference, the relative valuation that people place on goods based on the time they are consumed, has received significant analysis from an evolutionary perspective (Hansson and Stuart 1990; Rogers 1994; Sozou 1998; Dasgupta and Maskin 2005; Robson and Samuelson 2007; Robson and Szentes 2008; Robson and Samuelson 2009; Netzer 2009). Time preference has strong parallels with life history theory, which examines the effect of natural selection on the timing of the stages of life of an organism, such as development, maturation, reproduction, investment in offspring, senescence and death.

The evolution of time preference was considered as early as Fisher (1930), who pointed out that it should approximate the rate of population increase, although he noted that this would yield a rate of time preference below that observed. Hansson and Stuart (1990) agreed that the intergenerational discount rate would reflect long-term population growth. Rogers (1994) studied the optimal transfer of resources from a mother to her daughter. He concluded that the discount factor is one half per generation and the long-term real interest rate should equal approximately two per cent per year. Robson and Szentes (2008), however, argued that Rogers's analysis was based on the assumptions of identical offspring and a single same-age transfer between mother and daughter. Without these assumptions, the rate of time preference would depend upon the survival function faced at each age and thus no particular rate of time preference could be derived. While these approaches generate a low rate of time preference,

Robson and Samuelson (2009) proposed that aggregate risk that affects the viability of the entire population gives rise to a higher optimal discount rate than agent-specific risk. Ultimately, however, evolutionary theory has provided little guidance for parametric improvement of existing utility functions, such as determining what is the appropriate rate of time preference or level of risk aversion. This may be because experimental evidence relating to risk or time preference is a better source.

The dominant approach behind these results is to derive the utility function that would maximise fitness in a given environment. This generally results in a pattern of exponential discounting that may be considered ‘rational’ in the sense that it leads to consistent choices over time. One notable exception is work by Sozou (1998), who provided an evolutionary argument for hyperbolic discounting, which generates inconsistent choices over time (Strotz 1955; Ainslie 1975). Sozou showed that people can update their estimate of the probability of an underlying hazard, with the induced reduction in the discount rate generating a hyperbolic pattern of discounting. Where time-inconsistent behaviour is observed in experimental settings that do not involve a hazard [such as in Tversky et al. (1990)], the hyperbolic behaviour may be an evolutionary relic from more hazardous times. Robson and Samuelson (2007) also demonstrated an evolutionary basis to hyperbolic discounting using life-history theory.

Alternative evolutionary approaches provide scope for other behaviour such as loss aversion (Kahneman and Tversky 1984) and preference reversals (Lichtenstein and Slovic 1971) that do not relate to intertemporal choice. The remainder of this section will consider these types of behaviour.

The first step is to understand the agent’s evolutionary objective. For example, in Rubin and Paul (1979), agents appear risk averse above certain incomes and risk seeking

below them only through misspecification of the agents' objectives, for in the domain of attracting mates or fitness, the behaviour is risk neutral.

These evolutionary objectives can vary with context. The modular theory of intelligence is based on the concept that the human mind does not act as a single, centralised processing unit, but rather is comprised of relatively independent modules that solve problems in different domains (Cosmides and Tooby 1992). Accordingly, different decision rules will be applied in different contexts, such as whether the decision relates to mating, child rearing, status or social interaction. Kenrick et al. (2009) proposed that this modularity can be used to explain many of the departures from rationality reported in the behavioural economics literature. Such decision rules would have had positive fitness consequences for most of human history, and Kenrick et al. describe them as “deeply rational”.

Another approach deals with bounded rationality or the use of heuristics (rules of thumb).⁷ Rayo and Becker (2007a, 2007b) demonstrated how peer comparison and habit formation could arise by considering happiness as an imperfect gauge by which economic agents make decisions. If agents had superior sensory capabilities and their happiness response was perfectly attuned to their choices, the evolved utility function would simply map happiness onto fitness. But if agents are constrained in the manner that they feel happiness, such direct mapping may not be possible.

Rayo and Becker (2007a, 2007b) considered agents who cannot tell close together choices apart due to limits to the sensitivity and bounds of happiness.⁸ These physiological constraints might be likened to a voltmeter, which must first be calibrated

⁷ One excellent analysis of decision making in this framework is by Gigerenzer (2000).

⁸ Similar constraints were used by Robson and Samuelson (2011b) in providing an evolutionary explanation why people's decision utilities and experienced utilities vary.

to the problem at hand to give an accurate reading (Robson 2001), or the human eye, which adjusts to the general luminosity of the surroundings (Frederick and Loewenstein 1999). If an agent cannot discriminate between choices, it may be possible to achieve greater sensitivity through evolving an amplified happiness response. But if there is a bound on happiness, amplification may push certain choices outside of the viable range. Rayo and Becker showed that under these constraints, agents will maximise the strength of the signal where it matters most, considering currently available opportunities. Hence, utility will depend on the relative outcome of decisions, with information conveyed to the brain in terms of contrast between outcomes. Their approach captures the empirical observations of the short-lived effect of a change in income on happiness, and peoples' strong positional concerns. A general increase in income across society does not increase happiness [consistent with Easterlin (1974)]. This formulation is also consistent with a positive correlation between income and happiness in cross-section data as people with a higher income are more likely to have received a recent positive income shock.

Evolutionary theory may also provide insight into the heterogeneity of preferences, such as variation in time preference (Warner and Pleeter 2001; Frederick et al. 2002) and risk aversion (Cohen and Einav 2007; Barseghyan et al. 2011). The heritability of economic traits points to the influence of genetic factors [for example, as demonstrated by Cesarini et al. (2009)]. Saint-Paul (2007) considered the interaction of genetically heterogeneous agents in a trading situation. He found that genetic heterogeneity could be maintained where trade allowed for comparative advantage.

A further extension of research in this area examines a broader range of economic preferences, particularly those that are not features of typical utility functions. Time preference and risk preference have attracted much attention, whereas preferences such

as the human desire to cooperate, innovate or signal have received less interest, particularly in the economic literature. As a striking example, the propensity to exchange among non-kin [an area of analysis suggested by Hirshleifer (1977)], a hallmark of humans when compared to other species (Kaplan et al. 2012), is the foundation to much economic activity and to concepts such as comparative advantage. Yet despite the “propensity to truck, barter, and exchange one thing for another” being noted by Adam Smith (1776), the evolutionary examination of this preference is rarely considered in the analysis of economic preferences.

2.3 The genetic basis of economic traits

An important empirical finding of behavioural genetics is that all human behaviour is heritable (Turkheimer 2000). It follows that a proportion of the variation in phenotypic (observable) behavioural traits can be attributed to genetic variation among individuals. This finding also applies to economic behaviour, with a genetic basis to economic behaviour demonstrated across a range of studies (Benjamin et al. 2012a, 2012b). The empirical analysis of molecular genetic information as it relates to economic traits has become known as *genoeconomics* (Benjamin et al. 2008).

The recent growth of *genoeconomics* builds on past work on the heritability of economic traits, particularly through twin and adoption studies. In twin studies, a higher correlation in traits between identical twins than for fraternal ones provides an estimate of heritability. In adoption studies traits of adopted children are compared with those of their adoptive and natural parents. These studies have produced estimates of heritability of savings behaviour of 0.33 (Cronqvist and Siegel 2015) (that is, 33 per cent of the variation in savings behaviour is attributable to variation in additive genetic factors), of risk preference of 0.2 to 0.57 (Cesarini et al. 2009; Le et al. 2012; Zhong et al. 2012)

and of intelligence of 0.5 to 0.8 (Johnson et al. 2010). Estimates of the heritability of income include those from Taubman (1976), who estimated a heritability of 0.18 to 0.41 in white male twins, and Benjamin et al. (2012a), who found heritability of permanent income of 0.37 to 0.58 for men and 0.28 and 0.46 for women. Sacerdote (2007) produced a series of estimates of the heritability of several measures of educational attainment, which ranged between 0.34 and 0.46.

Early behavioural genetic studies were candidate gene studies, where a gene of interest is hypothesised to affect an economic trait based on that gene's biological function. However, candidate gene studies have a poor record of replication. For example, Chabris et al. (2012) sought to replicate published associations between general intelligence and genetic variants. They found a significant relationship in only one of 32 tests, compared to the expected 10 to 15 given the power of the tests. Benjamin et al. (2012a) pointed out that the failure of candidate gene studies may be due to small sample sizes, with only one study reviewed in their paper using more than 500 people; the use of ex-post hypotheses that are formed after discovery of a statistical relationship; and publication bias, which is the tendency that only positive findings will be published.

As genomic techniques became cheaper, genome wide association studies (GWAS) became feasible. These studies take an array of hundreds of thousands to millions of single-nucleotide polymorphisms (SNPs) and search for associations between the sampled SNPs and a range of tested phenotypic outcomes. One limitation of GWAS is that the effect of most SNPs is low and typically explains less than one per cent of the phenotypic variation, even for traits with a large genetic component such as height (Lango Allen et al. 2010). Consequently, large sample sizes are required so that the significance level can be set high enough to avoid false positives, but still have enough power to identify SNPs that affect the trait of interest. A significance level of 5×10^{-8} is

commonly adopted, which reflects the 1,000,000 SNPs in a typical array (Risch and Merikangas 1996). However, even large samples may fail to produce consistent results. Beauchamp et al. (2011) searched for genetic variants associated with educational achievement in one sample of 7,500 people. They were unable to replicate their initial findings in a second sample using 9,500 people.

Accordingly, to this time, most of the heritability observed in twin and adoption studies has not been explained by the identification of the relevant SNPs. This has been termed the missing heritability problem (McCarthy and Hirschhorn 2008). The complexity of gene-gene and gene-environment interactions, the small effect of any particular gene and the difficulty of obtaining genotypic data across all members of the relevant sample create practical constraints to the use of genetic data.

The difficulty in finding SNPs with a significant association with a trait of interest stimulated the development of an alternative approach examining the combined contribution of the genotyped SNPs. Benjamin et al. (2012b) used a technique [developed in Yang et al. (2010)] that they called genomic-relatedness-matrix restricted maximum likelihood (GREML) to estimate the proportion of variance in economic and political preferences and in educational attainment that could be explained by the combined genetic variation within an SNP array. While the GREML approach is a noisy measure that gives a lower bound estimate of heritability, genetic variation was found to explain at least 20 per cent of the variation in trust. No significant relationship was found for the other three economic traits tested: risk, patience and fairness. The analysis of political and economic preferences using GREML indicates that although the genetic effects are highly polygenic, genetic information in the form of SNP data will be able to predict a substantial proportion of phenotypic variation. This finding supports other studies that demonstrated that genotyped SNPs explain a substantial proportion of the

variance in traits such as height (Yang et al. 2010), intelligence (Davies et al. 2011) and personality (Vinkhuyzen et al. 2012). The question then becomes when data sets may become large enough to identify the SNPs that affect economic preferences.

Genoeconomics may improve economic models by providing direct measures of behavioural parameters and allowing the use of genes as control variables or instruments in empirical studies. Identification of biological pathways as the basis of economic traits would give the analysis of the evolution of preferences more substantial grounding and provide additional foundation to theoretical analysis. In particular, this may assist in the dynamic analysis of how preferences evolved. Genomic information may also benefit social programs and public health policy through identifying heterogeneity between people. Benjamin et al. (2012a) provided an example of targeting supplementary reading programs at those whom genetic screening has identified as being at increased risk of dyslexia. However, the use of heritability in policy development has been subject to criticism, which is also likely to be made of genoeconomic research. In an influential paper, Goldberger (1979) questioned the value of information on heritability in social policy [largely reflected in Manski (2011)]. Goldberger argued that information on the heritability of poor eyesight has no effect on the policy decision of whether or not to provide eyeglasses. However, knowing the genetic cause allows a more effective targeting of screening programs and early intervention.

To reach a point where genoeconomics can make these contributions, datasets large enough to provide the requisite power for analysis are required. To achieve this, there may be value in pooling datasets, which requires consistent measurement of phenotypes

across studies. The Social Science Genetic Association Consortium⁹ aims to achieve this through the establishment of common surveys for traits such as risk and time preference, trust, education and wellbeing. In one output from this pooling, members of the consortium conducted a GWAS using a sample of 101,069 individuals, and a replication sample of 25,490, and found SNPs that explained approximately 2 per cent in the variance in educational attainment and cognitive function. In the future, full genome testing may be routine and allow even larger samples as sequencing costs continue to decrease rapidly (Wetterstrand 2013). This was illustrated in one recent analysis of height using a sample of over 250,000 people, which found 697 genetic variants that, in combination, explained 20 per cent of the heritability for adult height (Wood et al. 2014). Once large enough samples are collected for economic traits and behaviours, the quantity of data will be difficult to ignore.

Until genoconomics progresses to this point, however, there are alternative means to incorporate genetic information into economic analysis. In the absence of molecular data, family history can provide control variables relating to heritable traits and capture much of the phenotypic variation due to genotype. For example, estimates of height using a 54-loci genomic profile explained only 4 to 6 per cent of the sex and age adjusted variance in height in a population, whereas parental height explained approximately 40 per cent (Aulchenko et al. 2009). Larger genetic sample sizes may eventually yield more accurate prediction than family history, but that is not yet the case.

Another area with potential for future analysis is epigenetic transmission of traits. Epigenetics is the study of heritable chemical changes in gene expression that are not

⁹ For information on the SSGAC, visit <http://www.ssgac.org>

caused by changes in the DNA sequence (Berger et al. 2009).¹⁰ These changes include modification of histones, which are proteins around which DNA is wrapped, DNA methylation in which a methyl group is added to DNA nucleotides, and RNA modification.

Epigenetic changes are influenced by environmental factors such as abuse during childhood and poverty. They have been proposed to affect physical and mental health in later life (Hochberg et al. 2011; Hoffmann and Spengler 2014) and have also been proposed to affect subsequent generations. Intergenerational transmission may occur through the parents' behaviour affecting their offspring, or due to environmental effects on the embryo and its germline. As human eggs form in the female embryo, environmental stresses on a pregnant woman can act directly on the eggs of her daughter, which will eventually develop into grandchildren. Of interest from an evolutionary perspective is the potential for intergenerational transmission of epigenetic changes beyond the people or germlines exposed to the environmental stress. One famous example of intergenerational transmission of epigenetic changes comes from a study of three cohorts born in the Överkalix parish in northern Sweden, where diabetes mortality was higher if the paternal grandfather experienced food scarcity during certain stages of development (Kaati et al. 2002). Similarly, the children of men who were prenatally undernourished in the 1944-45 Dutch famine were heavier and more obese than those in the cohort who were not undernourished (Veenendaal et al. 2013).

If epigenetic changes can be transmitted across multiple generations, they could provide variation for natural selection to act upon and thereby allow faster pathways for individuals to adapt to changing environments. However, the development of the field

¹⁰ The definition of epigenetics is subject to debate and includes definitions that do not require the changes to be heritable (Ledford 2008).

of epigenetics does not present an alternative to classic gene based approaches. First, epigenetic changes are likely to be induced by the organism's genes, and some instances of proposed epigenetic transmission may simply reflect unidentified genetic mutations (Heard and Martienssen 2014). Second, there is limited evidence that epigenetic changes in humans are transmitted with high fidelity across more than a couple of generations. In mammals, the embryo and germline undergo a round of epigenetic reprogramming in which most parental epigenetic marks are erased, although a limited number of marks escape the reprogramming (Daxinger and Whitelaw 2012). Third, for most examples of intergenerational epigenetic transmission, no biochemical mechanism by which the epigenetic change occurred or was transmitted has been identified (Kaati et al. 2002; Heard and Martienssen 2014).

However, epigenetic changes may still be a relevant economic consideration, even if transmission is behavioural or rapidly decays across generations. The transmission of environmental stresses across a few generations is of interest for economic and social policy. But the lack of identified mechanisms means that it is not currently feasible to include epigenetic marks in any analysis. Controlling for parental and possibly even grandparental traits and experiences is one alternative to capture the effects of interest.

2.4 The interaction of economic and evolutionary dynamics

Given that most economic change occurs over shorter periods than human evolutionary change, taking economic preferences as fixed through time seems a reasonable assumption. However, over the longer timeframes that are relevant for economic growth and development, the evolution of traits and preferences needs to be considered. This is particularly the case given the increasing evidence of the accelerating pace of evolution and changes in gene frequencies in human populations since spread of agriculture

(Voight et al. 2006; Hawks et al. 2007; Cochran and Harpending 2009; Fu et al. 2013). Any economic analysis over tens or hundreds of generations should incorporate evolutionary change. Wilson (1970) argued that there could be significant alteration in intellectual and emotional traits in humans in less than 10 generations, with considerable evidence of this occurring in recent human evolutionary history (Stearns et al. 2010; Milot et al. 2011; Courtiol et al. 2012).

2.4.1 Human evolution and economic growth

While economists such as Hansson and Stuart (1990) noted that human populations evolving in different environments may vary in their evolved economic traits, the dynamic analysis of economic preferences and economic growth received limited attention until Galor and Moav (2002) considered whether human evolution was a factor underlying the transition from Malthusian conditions to modern levels of economic growth. In a unified growth framework (Galor and Weil 2000; Galor 2011), Galor and Moav developed a model in which the population comprises two types, with each type varying genetically in the relative weight they place on the ‘quality’ or quantity of their children. Quality-preferring types invest more in the education of their children than those who prefer a large number of children. Accordingly, quality-preferring types have a higher level of human capital and fitness advantage. In the Malthusian state, technological progress is slow, as the quality-preferring types comprise only a small proportion of the population. As the prevalence of the quality-preferring type increases, so does the average level of education in the population, driving increased technological progress. Ultimately, technological progress increases to a rate where even the quantity-preferring types will educate their children, sending the economy into a new high growth state.

Simulation of the Galor and Moav model by Collins et al. (2014; Chapter 3) showed that the economic take-off could occur within a few generations, which reflects the nature of the take-off observed around the time of the Industrial Revolution in parts of Europe. After the take-off, the quality-preferring types decline in prevalence because they overinvest in the education of their children relative to the level that maximises fitness. Still, the new high-growth state is maintained by the continuing investment in education by the quantity-preferring types.¹¹

Galor and Michalopoulos (2012) utilised a similar framework, but their trait of interest is entrepreneurial spirit, proxied by the degree of novelty or risk seeking. They proposed that in the early stages of development, risk tolerant individuals had an evolutionary advantage. As they expanded to form a larger portion of the population, the risk tolerant types drove technological progress through their entrepreneurial activity, ultimately triggering a take-off in economic growth. After the take-off, risk averse individuals have a fitness advantage and increase in prevalence. Galor and Michalopoulos proposed that a reduction in the proportion of risk tolerant individuals in developed countries might lie behind the process of convergence between developed and less developed countries. The economy may even be vulnerable to a return to the Malthusian state through the reproductive success of increasingly risk averse people.

A core feature of the papers by Galor and Moav (2002) and Galor and Michalopoulos (2012) is the manner in which the evolutionary processes operate. In the Galor and Moav model, the quality-preferring types lose their evolutionary advantage after the economic take-off. The result is that the population before and after the take-

¹¹ The stability of the modern growth state is subject to the assumption of only two types of parents and a return to Malthusian conditions cannot be ruled out in the presence of a third strongly quantity-preferring type.

off has the same composition. In fact, the evolutionary dynamic may not be required if there is another source of technological progress, such as a scale effect [which reflects the model of Galor and Weil (2000)]. Similarly, in the Galor and Michalopoulos model, the evolutionary advantage switches between risk tolerant and risk averse individuals at the time of the economic take-off, meaning only a temporary change in population composition.

A different approach to human evolution and economic growth was taken by Collins et al. (2013; Chapter 4), who extended Kremer's (1993) model of population growth and technological progress to incorporate the evolution of "innovative potential". Kremer combined the concepts that a Malthusian population's size is constrained by its level of technology and that more people lead to more ideas, to show that population growth is proportional to its size. Collins et al. extended this framework by proposing that population growth is proportional to human innovative potential because more people means more mutations and greater potential for evolutionary change (Fisher 1930). Adding the evolution of innovative potential to the analysis makes the population more robust to technological shocks. As innovative potential increases, growth of the now more innovative population becomes the predominant source of economic growth. The population composition therefore changes substantially and permanently.

Taking an empirical approach, Clark (2007) proposed that the inheritance of fitness enhancing traits such as thrift, prudence and hard work was a factor behind the emergence of the Industrial Revolution in pre-1800 Great Britain. Building on Clark and Hamilton (2006), Clark used data from parish wills to show that the wealthy had more reproductive success than the poor. To the extent that the traits of the rich such as thrift, prudence and hard work were passed from parent to child, they would spread through the population and provide a basis for the acceleration in economic growth.

Clark was equivocal as to whether the transmitted traits were cultural or genetic, although in subsequent work Clark (2008) argued for a genetic inheritance.

Clark's finding of higher fecundity of the rich is suggestive of the role of sexual selection in humans. Fitness depends on an individual's ability to attract a mate. Conflicts arise among males for access to females and females become choosy and discriminate against unwanted males. Sexual selection can result in fast evolutionary changes as it has a direct impact on reproductive success and fitness (Brown et al. 2009). Wade and Shuster (2004) estimated that sexual selection accounts for approximately half of total selection in *Homo sapiens*, while Miller (2001) suggested that sexual selection shaped the human mind.

Zak and Park (2002, 2006) incorporated sexual selection into an age-structured model in which agent cognitive ability and beauty (*sic*) is genetically determined. The agents in Zak and Park's model do not maximise biological fitness directly, as they trade-off marriage, children and consumption of goods. As such, an agent with lower preference for consumption relative to children would have a fitness advantage. In Zak and Park's baseline scenario, sexual selection increases intelligence, human capital and beauty. The base-line simulation generated one per cent growth in human capital per generation over 40 generations, which Zak and Park suggest is a reasonable approximation of the last 800 years.

A recent application of sexual selection to the analysis of economic growth is by Collins et al. (2015; Chapter 5), who hypothesised that sexual selection and the resulting propensity to engage in conspicuous consumption contributes to economic growth. Collins et al. posited that men who signal their quality through conspicuous consumption have higher reproductive success, as conspicuous consumption provides a

signal of their quality to potential mates. The creative and productive activities required to fund conspicuous consumption generate economic growth. This analysis may provide an explanation for several phenomena, including the sudden appearance of goods associated with conspicuous consumption in the historical record, increasing rates of technological progress following the appearance of those goods, and continuing investment in work effort in modern economies where additional resources are not required for subsistence.

2.4.2 Evolution of economic traits

Traits may not always be able to be determined by a maximisation exercise of the nature undertaken in the previous analysis of the evolution of preferences. This possibility is illustrated by Frank (1988), who argued that the path dependence of evolution led to emotions playing a role in creating a credible threat of retaliation when engaging in trade. He proposed that when we are considering whether to retaliate against a party who has cheated us, we do not engage in a rational cost-benefit analysis of whether the gain in reputation in the future is worth the retaliation cost today. We instead have an emotional response to cheating, which impels us to retaliate. The mix of emotions with high discount rates applied to future reputation gains is a stable evolutionary bootstrap resulting from the path by which these respective traits evolved.

Another study investigating the dynamic evolution of an economic trait was done by Saint-Paul (2007), who analysed the role of trade in human evolution. Saint-Paul describes a population that engages in two activities – fight and defence. Applying a haploid structure, each person has a gene that determines fighting productivity and a

gene determining productivity in defence.¹² Each gene can be of either high productivity (H) or low productivity (L), leading to four possible genotypes: HH, HL, LH and LL. Without trade, each person must be self-reliant, and the low productivity alleles (variants of genes) are eventually eliminated from the population. With trade, a person can specialise in a high productivity activity. However, the specialized phenotypes HL and LH have a selective disadvantage because they may produce LL children with lower fitness if they mate. Therefore, with trade, selection will eventually produce a population consisting only of HH and HL or HH and LH phenotypes. These outcomes differ from the concept of comparative advantage in economics. The genetic analysis tells us that when there is trade, only those who have maximum productivity in at least one activity will be present in the equilibrium population. Trade may make an unproductive (LL) person better off in the short-term, but over the long-term, their unproductive alleles will be eliminated – totally in the case of no trade and from at least one locus in the case of trade.¹³

A recent analysis of the dynamics of an economically relevant preference is by Collins and Richards (2014; Chapter 6), who considered the evolution of fertility preferences after a fertility shock. They proposed that the genes associated with higher fertility will spread through the population after a negative fertility shock because individuals with high fertility have a fitness advantage. In fact, in several countries fertility has rebounded from the low rates that prevailed in the second half of the twentieth century.

¹² Humans are diploid with two sets of chromosomes, one from each parent, whereas a haploid organism has only one set of chromosomes. It is common to treat humans as haploid in studying the evolution of social behaviour as it avoids complications such as diploid reproduction, multi-gene traits, interactions between genes and phenotypic expression (Grafen 1991).

¹³ Ofek (2001) proposed that the evolution of the human brain was driven by trade.

One area of analysis relevant to evolutionary dynamics is gene-culture coevolution (often called dual inheritance theory), as proposed by Campbell (1965) and Cavalli-Sforza and Feldman (1973). The domestication of cattle and other milk-producing livestock is often viewed as the classical example of gene-culture coevolution. The domestication of these livestock was closely tied to the development of lactose tolerance in populations that undertook this domestication, which in turn increased the incentives to maintain cattle (Simoons 1969, 1970). However, it has been questioned whether it is appropriate to incorporate cultural change into an evolutionary or Darwinian framework (Claidière and André 2011), with most of the gene-culture coevolution literature relying on ad hoc models with particular assumptions. El Mouden et al. (2014) proposed a formal framework for gene-culture coevolution that highlighted difficulties in considering culture in an evolutionary frame.

Heterogeneity of traits is often incorporated in gene-culture coevolution through the use of agent-based models, which analyse the evolution of preferences using evolutionary game theory. Gene-culture coevolution is typically path dependent, with much of the interest on the initial conditions that allow a trait to arise, in addition to the stability of the trait once it moves toward fixation in the population. For example, after examining the robustness and stability of the tit-for-tat strategy, Axelrod and Hamilton (1981) turned to its initial viability and examined how the strategy may have spread in the population. Seabright (2004) considered how traits that support cooperation and trust developed in small bands of foragers before the dawn of agriculture. He then investigates the development of the economic institutions that allow the interaction of thousands of strangers with these traits.

Spolaore and Wacziarg (2013) suggested that of three evolutionary mechanisms – biological (genetic and/or epigenetic), cultural, and gene-culture coevolution – recent

research on nature versus nurture may make it meaningless to separate the first two. Yet, cultural transmission is a markedly different mechanism as it occurs horizontally as well as vertically and may occur between unrelated parties, such as through socialisation in groups. Whether the traits are transmitted genetically or culturally is important in understanding the dynamic process of development, plus the policy implications that flow from the analysis are likely to vary with the nature of transmission.

What is considered genetic or cultural is subject to some ambiguity. Where the trait of interest is transmitted vertically from parent to child, the reason for the label of genetic or cultural transmission is often not provided or the author may adopt an equivocal stance as to the nature of the transmission. As an example, Brown et al. (1982) noted that their general model on the evolution of social behaviour by reciprocation could be interpreted to involve a number of vertical transmission mechanisms including genetic, learning and cultural transmission. Galor and Moav (2002) labelled the preference for quality or quantity of children as genetic, although they note in a footnote that it may be cultural. Similarly, Clark (2007) noted transmission of traits of the wealthy in pre-Industrial Revolution England may have been either genetic or cultural. Conversely, Fernandez and Fogli's (2009) analysis of transmission of labour force participation and fertility and Algan and Cahuc's (2010) examination of the inheritance of trust, while described as cultural, could equally have been discussed as having a genetic component.

However, examination of genetic transmission at the molecular level makes it possible to disentangle genetic and cultural pathways. For example, the gene-culture coevolution that occurred at the time of the domestication of milk producing livestock left a genetic fingerprint in alleles that allow the adult digestion of lactose. Much of the literature on gene-culture coevolution explicitly considers their interaction. Although many other

economic traits are polygenic and likely subject to complex multi-gene and gene-environment interactions, there is some optimism that the relevant SNPs will be identified (Yang et al. 2010; Davies et al. 2011; Vinkhuyzen et al. 2012).

Bisin and Verdier (2001) considered the distinction between genetic and cultural transmission by analysing a process whereby children acquire a cultural trait from their parents or society. In their analysis, they showed that cultural transmission mechanisms can result in a heterogeneous spread of traits in the population, unlike the convergence on a single trait that occurs in most evolutionary analysis of economic preferences. However, heritability estimates of behavioural traits suggest a genetic component to the variation.

One of the growing research opportunities in the area of evolutionary and economic dynamics may arise from the use of the time series data being developed by evolutionary anthropologists and economic historians. For example, the data collected by Clark and Hamilton (Clark and Hamilton 2006; Clark 2007) from their analysis of English parish wills could form the basis of a population genetic analysis of the evolution of the English population in the period leading up to the Industrial Revolution. Subsequent work by Clark et al. (2014) on social mobility using surnames also points to potential areas for evolutionary inquiry.

2.5 The genetic foundations of economic development

The increasing availability of genomic data that allows comparison of gene frequencies across populations provides an opportunity to study differences in economic development. The research in this area is important in view of the persistence of technological and income differences across populations (Comin et al. 2010; Putterman and Weil 2010; Michalopoulos and Papaioannou 2011; Easterly and Levine 2012). To

the extent that genetic factors cause these income differences, there is potential for genetic research to contribute to the understanding of economic development. To date, this research has focussed on relative gene frequencies rather than directly inherited genetic traits, largely reflecting the nature of the data available for analysis at this time (see Section 3).

Spolaore and Wacziarg (2009) examined the relationship between economic development and genetic distance, which is a measure of the time since two populations have a common ancestor.¹⁴ They used genetic data for 42 populations from Cavalli-Sforza et al. (1994) based on the ethnic composition of 137 countries. Spolaore and Wacziarg found that the logarithm of income was negatively correlated with average genetic distance from the United States population (the technological frontier). Genetic distance accounted for 39 per cent of the variation in income in the sample. They also calculated genetic distance between 9,316 pairs of countries in a world sample and 325 pairs in a European sample. Using these paired samples, genetic distance accounts for less variation in income than using genetic distance from the United States, although genetic distance remained significant. Similarly, Bai and Kung (2011) found that the relative genetic distance of the population of Chinese provinces from that of Taiwan was positively correlated with differences in income.

Spolaore and Wacziarg (2009) emphasised that their research does not necessarily imply that differences between populations at the molecular level affect income or productivity. The measure of genetic distance is based on 120 neutral alleles that are not

¹⁴ Spolaore and Wacziarg use F_{ST} genetic distance, which is the probability that an allele at a given locus selected at random from two populations will be different.

considered to be under natural selection.¹⁵ Instead, Spolaore and Wacziarg believed that genetic distance captures barriers to diffusion of technology and economic development. Societies that are more closely related are able to learn from each other more easily than societies that have diverged across many generations. From this perspective, genetic distance is a summary statistic that captures divergence “in the whole set of implicit beliefs, customs, habits, biases, conventions, etc. that are transmitted across generations – biologically and/or culturally – with high persistence.” Desmet et al. (2011), who showed that genetic distance reflects similarity in answers to questions in the World Values Survey, interpret their results in the same way.

Spolaore and Wacziarg (2009) observed that the effect of genetic distance on economic development decreases from 1500 through to 1820, spikes around 1870, and then resumes the decline. This is in accordance with their interpretation of the effect of genetic distance being a barrier to diffusion of technology from the world’s technological frontier. The spike in the effect of genetic distance reflects the sudden growth in technology in one part of the world during the nineteenth century, followed by a decline in income differences when technology spread. Spolaore and Wacziarg (2011) extended this analysis by examining the rate of technological take-up as it relates to genetic distance. As predicted, greater genetic distance was associated with slower adaptation of technology in countries of greater genetic distance from the frontier.

Guiso et al. (2009) investigated the relationship between genetic distance and trust. In an analysis of factors affecting trust between European countries, they found that increasing genetic distance by one standard deviation reduces bilateral trust by 1.8 standard deviations. In contrast, Giuliano et al. (2014) found that the negative

¹⁵ Neutral alleles are used in population measures such as genetic distance so that selection of the alleles does not distort attempts to track evolutionary history (Cavalli-Sforza et al. 1994).

correlation between genetic distance and trade flows merely reflects the common effect on geography on the two. The relationship between genetic distance and income identified by Spolaore and Wacziarg (2009) was, however, robust to inclusion of controls for geography and transportation costs.

Ashraf and Galor (2013a) proposed a more direct genetic relationship in their hypothesis that genetic diversity affects economic development. Genetic diversity is a measure of diversity within a population, while genetic distance reflects diversity between populations.¹⁶ Genetic diversity within populations is affected by what is known as the serial-founder effect. As humans moved out of Africa, genetic diversity was lost along the path of migration because individuals in founder populations carry only a subset of the genetic diversity of the parent population. Thus, diversity tends to decline moving from Africa to Europe to the Americas.

Ashraf and Galor (2013a) proposed that genetic diversity promotes economic development through the wider mix of traits that can advance and implement new technologies. They showed that genetic diversity is a significant predictor for scientific output, with a one per cent increase in diversity linked to an increase of 0.02 scientific articles per person per year. They also suggest that genetic diversity provides for faster adaptive change. For example, populations with more genetic diversity might be better able to respond to environmental changes. This reflects the argument put forward by Saint-Paul (2007).

Conversely, Ashraf and Galor (2013a) also noted that genetic diversity may impede economic development as it increases the chance of conflict within a society and

¹⁶ Ashraf and Galor use expected heterozygosity as their measure of genetic diversity, which is the probability that two randomly selected people differ with respect to a given gene, averaged over the measured genes.

generally reduces the level of social order. They proposed that this detrimental effect occurs because genetic diversity is associated with a lower average degree of relatedness between people, which kin selection theory suggests affects cooperation (Hamilton 1964a). In another paper, Ashraf and Galor (2013b) suggested that ethnolinguistic heterogeneity caused by genetic diversity can be another source of distrust.

These countervailing factors could result in a hump-shaped relationship between genetic diversity and economic development. Ashraf and Galor (2013a) tested this hypothesis with genetic data from the Human Genome Diversity Cell Line Panel (Cann et al. 2002), which comprises 53 ethnic groups believed to be native to an area and relatively isolated from gene flow from other groups. Using population density as the measure of economic development for the period around 1500, they confirmed the presence of a hump-shaped relationship between genetic diversity and development. Ashraf and Galor also developed an index of predicted genetic diversity based on migratory distance for 145 countries. Using this measure of diversity also produced a hump-shaped relationship between diversity and economic development for the period around 1500. Examining the mix of ethnicities in a country, Ashraf and Galor replicated this result for the year 2000.

However, more work is required on the biological foundations of the observed relationships before they will be accepted as being more than suggestive correlations. For instance, it has not yet been established that humans possess the ability to detect differences in relatedness within populations at the level required. Further, measures of genetic distance and genetic diversity are typically based on non-protein coding regions of the genome that are not phenotypically expressed. One possibility is that these non-protein coding regions proxy phenotypically expressed genetic characteristics. It

has also been found that other economically relevant traits have been under selection since humans migrated from Africa. For example, the dopamine receptor gene, DRD4, which affects financial risk taking in men (Dreber et al. 2009), has had the allele associated with greater risk tolerance under strong positive selection since its emergence 30,000 to 50,000 years ago (Ding et al. 2002; Matthews and Butler 2011).

Spolaore and Wacziarg (2013) recently suggested that the mechanisms by which intergenerationally transmitted traits affect development could be divided into direct effects on economic performance on the one hand, and barriers to the spread of technological on the other hand. Galor and Moav (2002), Clark (2007) and Ashraf and Galor (2013a) considered direct effects, whereas Spolaore and Wacziarg (2009) examined barrier effects. Spolaore and Wacziarg suggested that it is harder to study direct effects on economic performance than barrier effects, as an economic event such as the Industrial Revolution is a unique and complex phenomenon. In contrast, the diffusion of technology has many opportunities for comparative study. However, a failure to study the direct effects may result in misidentification of barriers. Further, if the mechanism behind barrier effects is intergenerationally transmitted traits, analysis of both dimensions will likely be required to understand how the barrier effects operate and whether policy measures may assist in overcoming them.

Research into the genetic foundations of economic development will thrive when human genomes across times and populations become available. These genomic data will provide a time series in which selection on specific genes might be observed. With information on the function of those genes, it will be possible to hypothesise as to the selective pressures faced by humans and which traits might be more conducive to technological advance and economic development. When combined with genoeconomic research that indicates how the genes under selection affect economic preferences, we

may be in a better position to identify the direct and barrier effects of genetically transmitted traits and preferences affecting economic development.

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CHAPTER 3

ECONOMIC GROWTH AND EVOLUTION: PARENTAL PREFERENCE FOR QUALITY AND QUANTITY OF OFFSPRING

3.1 Introduction

Over the past thirty years, there has been an increasing scientific interest in using evolutionary theory to explain human economic behaviour. Since the advocacy of this approach by Becker (1976) and Hirshleifer (1977), Darwinian (1859) thinking has been used to explain the evolution of human risk preference (Rubin and Paul II 1979), time preference (Hansson and Stuart 1990; Rogers 1994; Robson and Samuelson 2007; Robson and Szentes 2008), and the shape of utility functions (Netzer 2009).¹⁷ More recently, evolutionary theory has been applied to the emergence of modern economic growth.

Galor and Moav (2002) developed a unified growth model in which natural selection favours traits that affect the economic environment. This model was the first to use frequency changes of heritable traits to explain the shift of human populations from Malthusian stagnation to modern economic growth. Galor and Moav proposed a gene-encoded preference for quality or quantity of children, which is similar to r/K selection in behavioural ecology (Planka 1970). The quantity-quality trade-off has been hypothesised as an economic factor by, among others, Becker (1960), and Becker and

¹⁷ Alchian (1950) and Nelson and Winter (1982) applied evolutionary concepts to the theory of the firm and industrial organization.

Lewis (1974). Becker et al. (1990) considered the link between the quantity-quality trade-off and economic growth.¹⁸

In the Galor and Moav model, individuals who invest a lot in the education of their children have a fitness advantage in the early stages of economic development. Fitness, as the term is used in biology, is the proportional contribution of a genotype to the gene pool of the next generation. As technological progress depends on human capital and the returns to education increase with technological progress, this positive feedback ultimately results in an escape from Malthusian stagnation. Galor and Moav noted that natural selection might favour other growth promoting traits. For example, Galor and Michalopoulos (2012) suggested that entrepreneurial spirit creates a selective advantage in the early stages of economic development, while less entrepreneurially spirited individuals do well in mature economies. The positive feedback between entrepreneurial spirit and economic development lifts the economy out of Malthusian stagnation.

Galor and Moav investigated the dynamics of their model analytically using phase diagrams. In this paper, their model is analysed numerically by simulation. The method is similar to the one that Lagerlof (2006) used to simulate the model of Galor and Weil (2000).¹⁹ The advantage of simulation is that it allows exploration of a richer specification of models for which there exists no closed-form solution. In particular, it will be possible to consider the addition of a strongly quantity-preferring genotype to the population and to demonstrate that, given the absence of a scale effect of population

¹⁸ Increasing technological progress and variation in heritable preferences underlies the trade-off in the Galor and Moav model, while a substitution effect due to higher wages drives the trade-off proposed by Becker et al. (1990)

¹⁹ The trigger for the take-off in the Galor and Weil (2000) model is increasing technological progress with increasing population, while the Galor and Moav (2002) model relies on investment in education by the quality-preferring types in the population.

in the model, the economy can regress to a Malthusian state under this change in the initial distribution of genotypes.

3.2 Background

Besides Galor and Moav (2002), several other authors have applied evolutionary theory in the analysis of economic growth and the transition from the Malthusian state to modern rates of growth. In their seminal paper on the evolution of preferences for saving and labour supply, Hansson and Stuart (1990) proposed that human preferences depend on the availability of resources. Harsh natural environments select for genotypes that have a stronger preference for saving, leading to an equilibrium with low population density and high per-capita capital. Selected traits include a preference for work and accumulation of physical capital. This might explain why humans left the Malthusian state first in regions with harsh winters.

Clark (2007) suggested that selection for certain heritable characteristics accounted for the Industrial Revolution. While open as to whether these traits were transmitted genetically or culturally, he found higher reproductive success among wealthy males in England between 1250 and 1800.²⁰ He hypothesised that individuals with favoured traits such as a propensity to hard work and saving increased in frequency during this time. This change in population composition could then have provided the basis for the Industrial Revolution.

The increasing availability of population genetic data, such as Cavalli-Sforza et al. (1994), has led to more research. Spolaore and Wacziarg (2009) linked differences in economic development with the genetic distance between populations, which depends

²⁰ Clark's proposal followed from work published by Clark and Hamilton (2006) on the reproductive success of the wealthy in England.

on the time elapsed since two populations shared a common ancestor. They proposed that genetic distance increases income differences because it may act as a barrier to the diffusion of technological development between populations. As genetic distance is based on neutral genes that are not subject to selection pressure, their hypothesis does not necessarily rely on any genetically determined difference in traits between populations, although genetic distance may serve as a proxy for vertically transmitted characteristics that affect the diffusion of development.

Recently, Ashraf and Galor (2013) proposed that the geographic distance of a population from Africa has affected the level of growth and development across regions. They found that populations with elevated or reduced genetic diversity experienced the lowest level of economic development in pre-industrial times, and that this pattern has persisted following the Industrial Revolution.²¹ Indigenous populations of the Americas have the lowest level of genetic diversity due to the founder effect, whereas Africans have the highest.²² They suggested that the hump-shaped relationship between genetic diversity and economic development is due to a trade-off between the costs and benefits of genetic diversity. A high level of genetic diversity expands the production possibilities through complementarities in knowledge production, but reduces the efficiency of the aggregate production process as lower levels of trust and coordination between dissimilar individuals reduce cooperation and create the potential for socioeconomic disruption. As for the measure of genetic distance used by Spolaore and Wacziarg (2009), the measure of genetic diversity used by Ashraf and Galor is

²¹ Genetic diversity was measured using expected heterozygosity, an index of the probability that two individuals, selected at random from the relevant population, are genetically different from one another.

²² The founder effect is the loss of genetic diversity that occurs when a small subset of a larger population establishes a new population.

based on non-protein coding regions of the genome and accordingly, their hypothesis does not rely on genetically determined differences in traits between populations.

3.3 The Galor and Moav model

Galor and Moav (2002) developed an overlapping generations model, with each agent living for two periods (childhood and adulthood). In childhood, agents are passive and they receive education. During adulthood, agents decide on how much time to dedicate to work or childrearing and they choose the number of children and their education. Reproduction is asexual by a single parent.

Production in the economy occurs with inputs of labour, H_t , and a limited resource, X , which may be called land. H_t measures the aggregate quantity of efficiency units of labour at time t . Aggregate output, Y_t , is given by a constant returns to scale technology:

$$Y_t = H_t^{1-\alpha} (A_t X)^\alpha \quad \alpha \in (0,1) \quad (3.1)$$

The level of technology, A_t , is determined endogenously in the model. $1-\alpha$ is the elasticity of output with respect to labour input.

Assuming there are no property rights over land, the return to land is zero and the wage per efficiency unit of labour, w_t , is the output per unit of labour, x_t .

$$w_t = x_t^\alpha \quad (3.2)$$

where $x_t = \frac{A_t X}{H_t}$

The population consists of two genotypes ($i = a, b$) with different preferences between the quality and quantity of their children. Genotype a is a quality-preferring genotype, while genotype b has a relative preference for quantity. The utility function is:

$$u_t^i = (1-\gamma) \ln c_t^i + \gamma (\ln n_t^i + \beta^i \ln h_{t+1}^i) \quad (3.3)$$

$$\gamma \in (0,1); \quad \beta^i \in (0,1]; \quad i \in a, b$$

where c_t^i is the consumption of an individual with genotype i in period t , n_t^i is the number of children, and h_{t+1}^i is the level of human capital of each child. The parameter γ measures the relative weight of children in the utility function and the parameter β^i determines the weight that a genotype i individual gives to the quality of children. Both parameters are inherited without change by the subsequent generations.

In adulthood, agents have one unit of time that they allocate between childrearing and participation in the labour market. Potential income, z_t^i , is the maximum income that could be earned if the agent's entire time endowment was devoted to labour force participation. Since the wage rate is expressed per efficiency unit of labour, potential income is:

$$z_t^i = w_t h_t^i = x_t^\alpha h_t^i \quad (3.4)$$

A parent incurs a base time cost, τ , for each child, with an additional time cost to educate the child to the level of education e_{t+1}^i . The total cost of raising a family with n children is $n_t^i(\tau + e_{t+1}^i)$ and the time left for working is $1 - n_t^i(\tau + e_{t+1}^i)$. Thus, the budget constraint faced in adulthood is:

$$c_t^i \leq w_t h_t^i [1 - n_t^i(\tau + e_{t+1}^i)] \quad (3.5)$$

Human capital, which determines an agent's efficiency units of labour during adulthood, is a function of education and the technological environment. Education increases human capital, while technological progress reduces the usefulness of existing human capital. The function for human capital and the conditions it must satisfy are as follows:

$$\begin{aligned} h_{t+1}^i &= h(e_{t+1}^i, g_{t+1}) & g_{t+1} &\equiv (A_{t+1} - A_t) / A_t & h_e(e_{t+1}^i, g_{t+1}) &> 0 \\ h_{ee}(e_{t+1}^i, g_{t+1}) &< 0 & h_g(e_{t+1}^i, g_{t+1}) &< 0 & h_{gg}(e_{t+1}^i, g_{t+1}) &> 0 \\ h_{eg}(e_{t+1}^i, g_{t+1}) &> 0 & h(0, 0) &= 1 & \lim_{g \rightarrow \infty} h(0, g_{t+1}) &= 0 \end{aligned} \quad (3.6)$$

Human capital increases at a diminishing rate with education (e_{t+1}^i) and is eroded at a decreasing rate by technological progress (g_{t+1}). Technological progress strengthens the effect of education on human capital. Human capital is normalised to one in the absence of education and technological progress.

Substituting equations (3.5) and (3.6) into equation (3.3), a genotype i parent of generation t faces the following optimisation problem:

$$\begin{aligned} \{n_t^i, e_{t+1}^i\} = \operatorname{argmax} \bigg\{ & (1-\gamma) \ln w_t h_t^i \left[1 - n_t^i (\tau + e_{t+1}^i) \right] \\ & + \gamma \left[\ln n_t^i + \beta^i \ln h(e_{t+1}^i, g_{t+1}) \right] \bigg\} \end{aligned} \quad (3.7)$$

subject to income being enough to meet the subsistence level of consumption \tilde{c} .

$$w_t h_t^i \left[1 - n_t^i (\tau + e_{t+1}^i) \right] \geq \tilde{c} \quad (3.8)$$

$$(n_t^i, e_{t+1}^i) \geq 0$$

The fertility of a genotype i individual varies across three scenarios. These are where the subsistence constraint does not bind, where it binds, and where potential income is insufficient to meet the subsistence level of consumption. Taking the first order condition of equation (3.7) with respect to n_t^i determines fertility when the constraint does not bind. Solving equation (3.8) as an equality gives fertility where the constraint binds. No children are born when the parent is reduced to the subsistence level of consumption. These three scenarios are shown in equation (3.9):

$$n_t^i = \begin{cases} \frac{\gamma}{\tau + e_{t+1}^i} & \text{if } z_t^i \geq \tilde{z} \\ \frac{1 - \tilde{c} / z_t^i}{\tau + e_{t+1}^i} & \text{if } \tilde{c} \leq z_t^i \leq \tilde{z} \\ 0 & \text{if } z_t^i \leq \tilde{c} \end{cases} \quad (3.9)$$

where $\tilde{z} \equiv \frac{\tilde{c}}{(1-\gamma)}$

Equation (3.9) indicates that the number of children depends positively on potential income and negatively on the time cost of childrearing. Above the critical value \tilde{z} , only the time cost of childrearing matters. No children are born when the parent is reduced to the subsistence level of consumption.

Taking the first-order condition of equation (3.7) with respect to the second choice variable e_{t+1} gives:

$$\beta^i h_e(e_{t+1}^i, g_{t+1}) - \frac{h(e_{t+1}^i, g_{t+1})}{(\tau + e_{t+1}^i)} \begin{cases} = 0 & \text{if } e > 0 \\ \leq 0 & \text{if } e = 0 \end{cases} \quad (3.10)$$

The first term represents the utility benefit of a marginal increase in investment in the quality of children. The utility benefit of education depends positively on the partial derivative of the human capital function h_e and the weight given to the quality of children in the utility function β^i . The second term is the utility benefit of a marginal increase in investment in the quantity of children. Optimal behaviour requires that the marginal benefit of education equals the marginal benefit of additional children if the parent chooses a positive level of education.

The following condition ensures that the level of education is positive for those with the highest valuation for quality ($\beta^i = 1$) when technological progress is zero:

$$h_e(0,0) > \frac{1}{\tau} \quad (3.11)$$

If equation (3.11) is not satisfied, no agents will educate their children, leading to a permanent Malthusian state.

The average level of education in the population, e_t , is:

$$e_t = q_t e_t^a + (1 - q_t) e_t^b \quad (3.12)$$

$$q_t = \frac{L_t^a}{L_t^a + L_t^b} = \frac{L_t^a}{L_t}$$

q_t indicates the proportion of genotype a in the population, with L_t^a and L_t^b the number of genotype a and b individuals and L_t the total population. It is assumed that the rate of technological progress, g_{t+1} , which determines economic growth, is an increasing and concave function of the average level of education:

$$g_{t+1} \equiv \frac{A_{t+1} - A_t}{A_t} = \varphi(e_t) \quad (3.13)$$

$$\varphi_e > 0; \quad \varphi_{ee} < 0; \quad \varphi(0) = 0$$

Finally, the number of efficiency units of labour supplied by the population is:

$$H_t = L_t^a f_t^a h_t^a + L_t^b f_t^b h_t^b = L_t \left[q_t f_t^a h_t^a + (1 - q_t) f_t^b h_t^b \right] \quad (3.14)$$

where f_t^i is the fraction of time used by genotype i for labour:

$$f_t^i = \begin{cases} 1 - \gamma & \text{if } z_t^i \geq \tilde{z} \\ \frac{\tilde{c}}{z_t^i} & \text{if } \tilde{c} \leq z_t^i \leq \tilde{z} \end{cases} \quad (3.15)$$

Equation (3.15) reflects the growing allocation of time to child rearing when potential income increases. When income reaches the critical value \tilde{z} , the fraction of time used for child rearing reaches a maximum of γ , leaving the fraction $1 - \gamma$ for labour.

Using equations (3.14) and (3.15), the aggregate labour supply is:

$$H_t = \begin{cases} L_t(1-\gamma)[q_t h_t^a + (1-q_t)h_t^b] & \text{if } z_t^a \geq \tilde{z} \text{ and } z_t^b \geq \tilde{z} \\ L_t[q_t(1-\gamma)h_t^a + (1-q_t)(\tilde{c}/z_t^b)h_t^b] & \text{if } z_t^a \geq \tilde{z} \text{ and } \tilde{c} \leq z_t^b \leq \tilde{z} \\ L_t[q_t(\tilde{c}/z_t^a)h_t^a + (1-q_t)(\tilde{c}/z_t^b)h_t^b] & \text{if } \tilde{c} \leq z_t^a \leq \tilde{z} \text{ and } \tilde{c} \leq z_t^b \leq \tilde{z} \end{cases}$$

$$\equiv H(L_t^a, L_t^b, e_t^a, e_t^b, g_t, z_t^a, z_t^b) \quad (3.16)$$

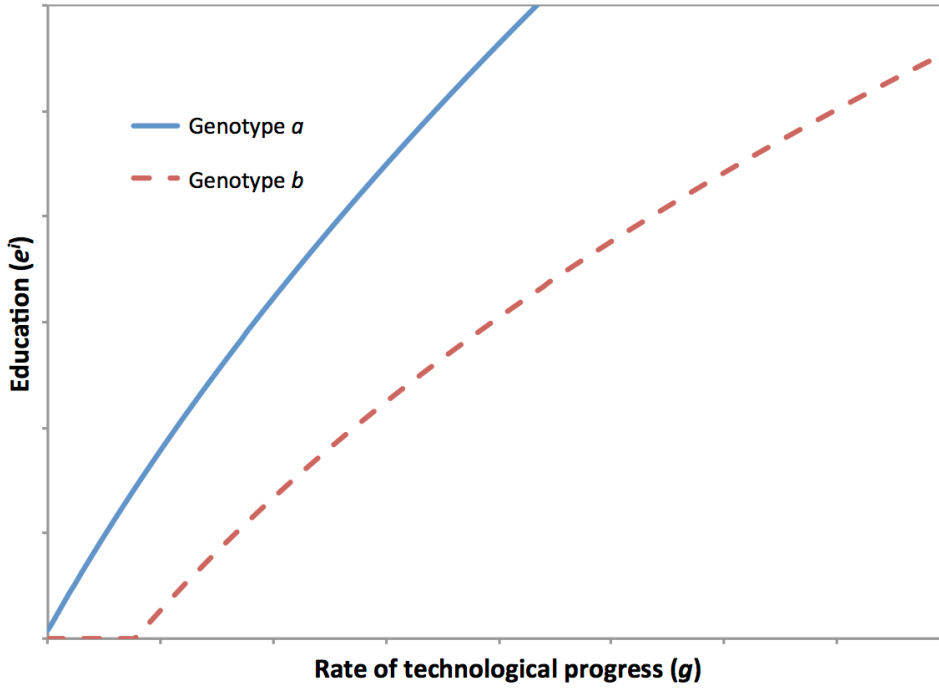
3.4 Response curves

Despite each genotype having a fixed preference for quality, this does not result in a fixed level of investment in education over time as the return to education changes with the rate of technological progress. However, the agents' education response curve to the rate of technological progress is fixed. Figure 3.1 shows how much time each genotype invests into education at a given rate of technological progress, with the quality-preferring genotype a investing more in education at all rates of technological progress. The shape of the response curves is based on simulations of the model in section 3.8. The inequality (3.11) guarantees that quality-preferring genotype a parents always choose a positive level of education for their children. It is also possible to derive the slope of the education response curve by applying the implicit function rule to equation (3.10).²³

²³ Using $F(e, g) = \beta h_e - \frac{h(e, g)}{\tau + e} = 0$

$$\frac{de}{dg} = -\frac{F_g}{F_e} = -\frac{\beta^i h_{eg}(\tau + e_{t+1}^i) - h_g}{\beta^i h_{ee}(\tau + e_{t+1}^i) - h_e + \frac{h}{(\tau + e_{t+1}^i)}}$$

Figure 3.1: Education response curves



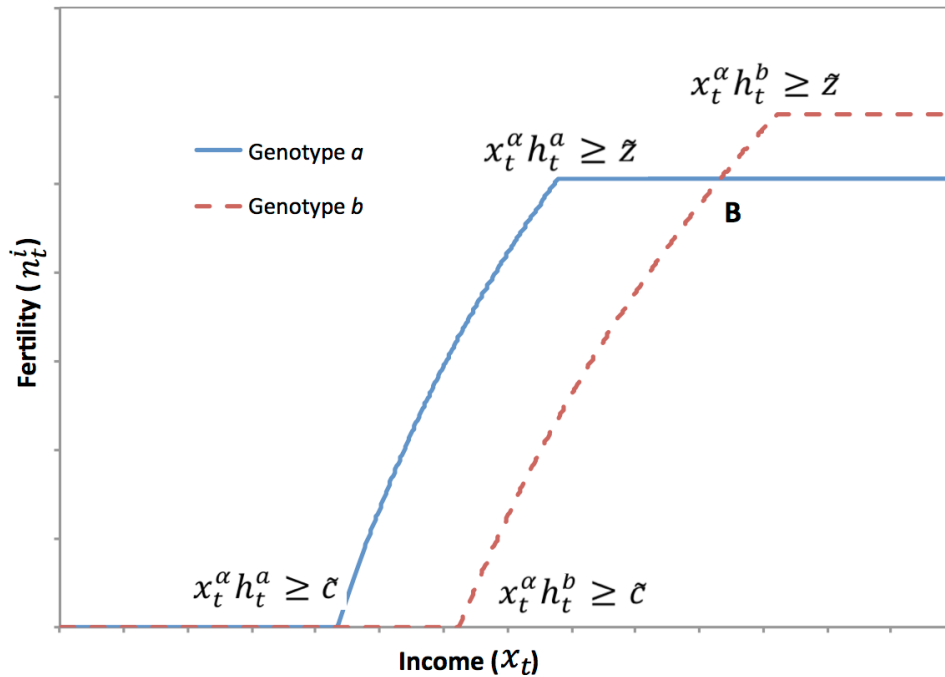
In the Galor and Moav model, the fitness of a genotype depends solely on the number of offspring, which determines its prevalence in the population over time. Which genotype has more children in turn depends on the rate of technological progress and economic growth. In the Malthusian state, the quality-preferring genotype has more children because education increases human capital and potential income. The rise in the prevalence of the quality-preferring genotype underpins slow technological progress in the Malthusian state. The rate of technological progress gradually increases until a threshold is reached at which it becomes worthwhile for the quantity-preferring genotype to invest in education. This threshold is given by point A in Figure 3.1. This positive feedback leads to an acceleration of technological progress and economic growth, putting an end to Malthusian stagnation.

The education response curve slopes upward if: $-\beta^i h_{ee} (\tau + e_{t+1}^i) + h_e > \frac{h}{(\tau + e_{t+1}^i)}$

When potential income exceeds the critical value \tilde{z} for both genotypes, they both will invest the same proportion of time into raising children. However, since $h_g < 0$, technological progress degrades human capital, which makes it costly for the quality-preferring genotype to maintain the high level of human capital of its children. For this reason, the quantity-preferring genotype gains a fitness advantage during the period of economic growth that follows the Malthusian state.

Figure 3.2 shows the number of children of each genotype as a function of output per efficiency unit of labour, x_t , which determines the wage per unit of human capital. The fertility response curves are based on the optimum conditions in equation (3.9) and the definition of potential income in equation (3.4). The quality-preferring genotype can procreate at a lower level of x_t than the quantity-preferring genotype. Figure 3.2 illustrates the reversal in relative fitness of genotypes that occurs during economic development, at point B.

Figure 3.2: Fertility response curves



The higher fitness of the quality-preferring genotype in the Malthusian state and the quantity-preferring type in the modern growth state is akin to the classical r/K selection theory in evolutionary biology. Individuals that use the r strategy produce many offspring, each of which has a low probability of surviving to adulthood, while K strategists produce fewer offspring in which they invest more heavily, giving them a higher probability of surviving to adulthood.²⁴ r strategists exploit less crowded ecological niches, while K strategists are favoured in more crowded environments. This behaviour occurs in the Galor and Moav model. In the Malthusian state, where resources are scarce and the economy is effectively crowded, the quality-preferring genotype has higher fitness. In the modern growth regime, the economy has become uncrowded, giving higher fitness to the quantity-preferring genotype.

The education and fertility response functions in Figures 3.1 and 3.2 allow each genotype to vary the level of education of their children and their number in response to technological progress. However, they do not have this flexibility in other dimensions. In particular, neither genotype fine-tunes their response to economic growth to optimise fitness. In the modern growth era, quality-preferring parents engage in a self-defeating strategy of overeducating their children. Additional flexibility in the education response could materially affect model predictions.

²⁴ In relation to other species, human reproductive strategy of even the quantity-preferring type would be described as strongly K . There is considerable debate in the literature as to the appropriateness of applying r/K selection theory within the human species [for example, see Graves (2002)].

3.5 Functional forms

To simulate the model, functional forms for h_{t+1}^i and g_{t+1} are needed. The following function for h_{t+1}^i matches most of the requirements given in equation (3.6):

$$h_{t+1}^i = h(e_{t+1}^i, g_{t+1}) = \frac{me_{t+1} + a}{e_{t+1} + rg_{t+1} + a} \quad (3.17)$$

This function does not fulfil the condition that $h_{eg} > 0$ for all values of e_{t+1}^i and g_{t+1} , but this is only a sufficient and not a necessary condition. Simulating the model of Galor and Weil, Lagerlof (2006) uses a similar functional form, with $m = r = 1$. Defining $a = \rho\tau$; $\rho \in (0,1)$, Lagerlof interpreted the parameter ρ as the portion of fixed time cost of childrearing that contributes towards the development of the base level of human capital.

The parameter m is included in equation (3.17) to allow the condition in equation (3.11), which ensures education by the quality-preferring genotype when there is zero economic growth, to be met. Using Lagerlof's definition of $a = \rho\tau$, m must be bigger than 1.

$$m > \frac{a}{\tau} + 1 = \rho + 1 \quad (3.18)$$

The parameter r is selected to produce modern rates of education and economic growth.²⁵

²⁵ The parameter a could also be used for this purpose, but reducing a tends to increase perturbations and increase the instability of the model.

A simple functional form for equation (3.13) is the power function:

$$g_{t+1} = ke_t^d; \quad 0 < d < 1; \quad k > 0 \quad (3.19)$$

Using equations (3.10) and (3.17), the level of education that each genotype gives to its children is:

$$e_{(t+1)}^i = \max \left\{ 0, \frac{1}{2m} \left[\left(m(B^i - 1)(rg_{t+1} + a) - a(B^i + 1) \right) + \sqrt{\left(m(B^i - 1)(rg_{t+1} + a) - a(B^i + 1) \right)^2 + 4m \left((mB^i\tau - a)(rg_{t+1} + a) - aB^i\tau \right)} \right] \right\} \quad (3.20)$$

$$\equiv \varphi^i(e_t)$$

This equation indicates that education in period $t+1$ is a function of the rate of technological progress in period $t+1$, which in turn is a function of the average level of education in period t . This link between education in one period and the next is crucial for the transition out of the Malthusian state.

3.6 The dynamical system

The dynamics of the system can be captured in a system of six difference equations that describe the behaviour of the endogenous variables A_t , g_t , e_t^a , e_t^b , L_t^a and L_t^b . Before defining these equations, z_t^i and n_t^i must be expressed in terms of the endogenous variables.

Collecting equations (3.2), (3.4) and (3.17) and given $H_t \equiv H(L_t^a, L_t^b, e_t^a, e_t^b, g_t, z_t^a, z_t^b)$, potential income per worker equals:

$$z_t^i = w_t h_t^i = \left(\frac{A_t X}{H_t} \right)^\alpha \left(\frac{me_t^i + a}{e_t^i + rg_t + a} \right) \equiv z(A_t, L_t^a, L_t^b, e_t^a, e_t^b, g_t) \quad (3.21)$$

Equations (9), (20) and (21) yield the number of children:

$$n_t^i = \begin{cases} \frac{\gamma}{[\tau + \varphi^i(e_t)]} & \text{if } z_t^i \geq \tilde{z} \\ \frac{1 - \tilde{c} / z_t^i}{[\tau + \varphi^i(e_t)]} & \text{if } \tilde{c} \leq z_t^i \leq \tilde{z} \\ 0 & \text{if } z_t^i \leq \tilde{c} \end{cases} \equiv \eta(A_t, L_t^a, L_t^b, e_t^a, e_t^b, g_t) \quad (3.22)$$

The dynamical system for the six endogenous variables is:

$$A_{t+1} = [1 + g(e_t)] A_t$$

$$g_{t+1} = g(e_t)$$

$$e_{t+1}^i = \varphi^i(e_t); \quad i \in a, b$$

$$L_{t+1}^i = \eta(A_t, L_t^a, L_t^b, e_t^a, e_t^b, g_t) L_t^i; \quad i \in a, b$$

3.7 Parameter values

Table 3.1 lists the numerical values given to each parameter for the base case model. The preference parameter of the quality-preferring genotype, β^a , is set equal to 1. The preference parameter of the quantity-preferring type, β^b , must be high enough to allow for an exit from the Malthusian state. For any value of β^b below 0.894, the economy remains in the Malthusian state because technological progress never reaches a level high enough to induce the quantity-preferring genotype to invest in education. $\beta^b = 0.9$ is chosen because it produces a realistic level of education in the modern growth era.

Table 3.1: Parameter values

		Description	Value
Parameters	$1 - \alpha$	Output elasticity of labour	0.6
	γ	Weight on children in utility function	0.259
	τ	Fixed time cost of children	0.20
	β^a	Preference for quality of genotype a	1
	β^b	Preference for quality of genotype b	0.9
	m	Weighting of education in production of human capital	2
	a	Portion of fixed cost time of raising child towards human capital	0.99τ
	X	Land	1
	\tilde{c}	Subsistence consumption constraint	1
	k	Growth function parameter	8.88
	d	Growth function parameter	0.5
	r	Responsiveness of human capital to economic growth	0.108
Initial values	L_0^a	Initial population of genotype a	0.007
	L_0^b	Initial population of genotype b	0.7
	e_0^a	Initial education of genotype a	0
	e_0^b	Initial education of genotype b	0
	A_0	Initial technology	1
	g_0	Initial rate of technological progress	0

The output elasticity of labour, $1-\alpha$, equals the labour share in national income if input factors are paid their marginal products. Clark (2010) provides estimates of the share in income from the Middle Ages to modern times. The labour share increased from a low of 0.478 in the thirteenth century to above 0.6 in the early nineteenth century and to over 0.75 in the late twentieth century. We use a mid-point value of 0.6.

Population perturbations in the Malthusian state and during the transition period limit the potential values for the fixed time cost of children, τ . We can determine the range of τ for which population perturbations can be minimised by considering a population comprising solely genotype b . If $\tilde{c} \leq z_t^b \leq \tilde{z}$, as would be the case during the Malthusian era, and setting \tilde{c} , A_t , and X equal to 1, the population equation simplifies to the following first-order difference equation.

$$L_{t+1} = \frac{1}{\tau} L_t (1 - L_t^\alpha) \quad (3.23)$$

Setting $L_t = L_{t+1}$, the equilibrium for the population is $L^* = (1 - \tau)^{1/\alpha}$. Taking the derivative of equation (3.23) and substituting the equilibrium condition yields:

$$\frac{\partial L_{t+1}}{\partial L_t} = \frac{1}{\tau} - \frac{1 + \alpha}{\tau} (1 - \tau) \quad (3.24)$$

The population equilibrium is unstable if $\partial L_{t+1} / \partial L_t > |1|$, with stability depending on the value of the fixed time cost of child rearing. For $\alpha = 0.4$, the equilibrium is unstable if $\tau \leq 0.1666$. Thus, for any value of the fixed time cost of children below this value, we can expect significant population perturbations. Subsequent testing demonstrated that even for values of τ slightly above that threshold, the presence of genotype a in the population results in ongoing perturbations. Setting $\tau = 0.20$ prevents extinctions and maintains reasonable population dynamics.²⁶

An estimate for education expenditure, e , in the high-growth regime can be derived from OECD statistics. In 2009, education expenditures averaged 5.8 per cent of GDP across OECD countries (OECD 2009 Table B.2.4). The model is calibrated to obtain an

²⁶ Haveman and Wolfe (1995) estimated expenditure on children as a proportion of GDP as approximately 0.15.

education level of genotype b individuals of 0.059, which is the OECD estimate for the United Kingdom. Given the predominance of genotype b , this is also the population average education in the modern growth regime.

The parameter γ , which is the same for both genotypes, determines the relative weight of children in the utility function. As modern fertility in developed countries is generally below replacement, γ is set to achieve zero population growth in the high-growth era, i.e. each parent has a single child. Setting $\gamma = 0.259$ and using the earlier values for τ and e yields $n = \gamma/(\tau + e) = 0.259/(0.20 + 0.059) = 1$.

Income per worker grew 2.3 per cent per year in the United Kingdom from 1950 to 2008 [Average annual growth 1960-2008 in Clark (2010), Table 33]. Assuming 20 years per generation and using continuous compounding, the rate of technological progress g equals 216 per cent per generation in the modern growth era.²⁷ With this g value and letting $d = 0.5$, the parameter k equals 8.88 in growth equation (3.19).

The parameters a and m enter equation (3.17), which determines human capital h_t^i . Population perturbations increase if a is much less than 1 and m far above the level required for the quality-preferring type to educate their children when there is no economic growth. We set $a = 0.99\tau$ and $m = 2$, satisfying inequality (3.18) and minimising perturbations. The selection of $r = 0.108$ yields the chosen equilibrium values of education and economic growth in the modern growth era.

Finally, initial values must be chosen. The initial education is zero and hence initial economic growth is zero. Initial technology, A , and land, X , are set equal to 1. At time

²⁷ $g^* = e^{\left(\frac{0.023*20}{\alpha}\right)} - 1 = e^{\left(\frac{0.023*20}{0.4}\right)} - 1$

zero, the number of genotype a and b individuals is assumed to be $L_0^a=0.007$ and $L_0^b=0.7$, with genotype a comprising one per cent of the population. This is close to the equilibrium population in the first period. Using equation (3.21), the level of income in the first period is approximately 1.25, which is above subsistence but such that the subsistence constraint still binds [as it is below $\tilde{z} = 1/(1 - \gamma) = 1.35$].

3.8 Simulation results

The model explains the transition from Malthusian stagnation to modern economic growth, which occurred during the Industrial Revolution in the late 18th and early 19th centuries. Initiating the simulation shortly before the beginning of the second millennium, the take-off occurs after about 45 generations or 900 years. The length of time to the take-off depends on the initial proportion of genotype a and b individuals. The transition phase from Malthusian stagnation to modern growth lasts about six generations or 120 years. During the transition phase, the rate of technological progress surges from less than 1 per cent per annum to 5.7 per cent and income growth rises to the modern growth rate of 2.3 per cent per annum. Population growth increases until the time of the take-off and then reverses, dropping to zero during the transition phase. Figure 3.3 displays the behaviour of the annual growth rates of technology, income and population, and Figure 3.4 shows the log-levels of these variables. For population growth, income growth and fertility, we present the results as five-generation moving averages as short-run population perturbations require smoothing of the graphs to show the important trends and relative values in an effective visual manner.

Figure 3.3: Annual growth rate of technology, population and income

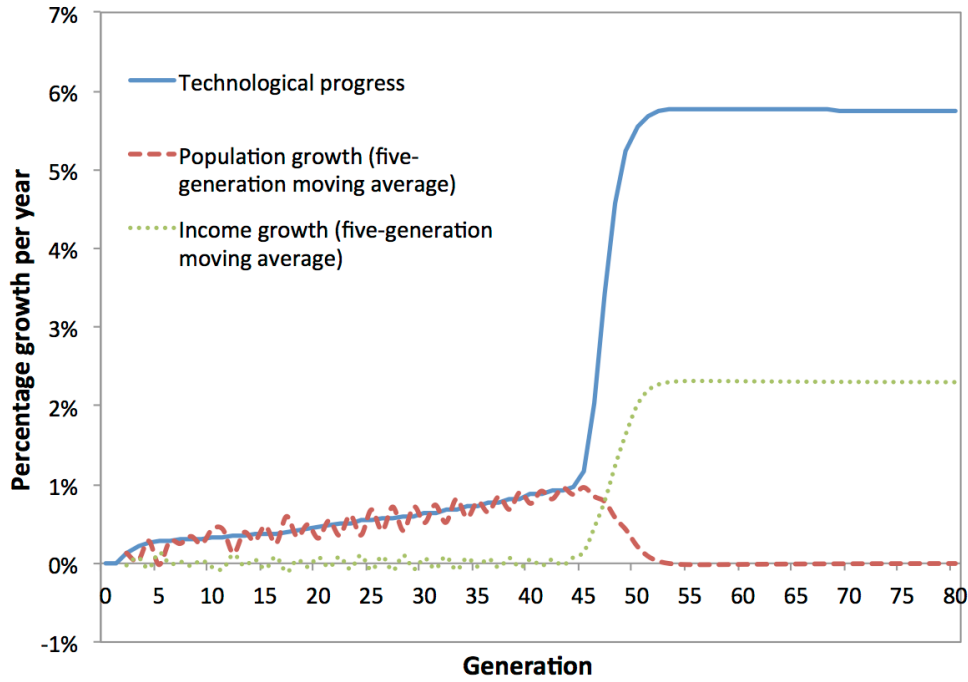
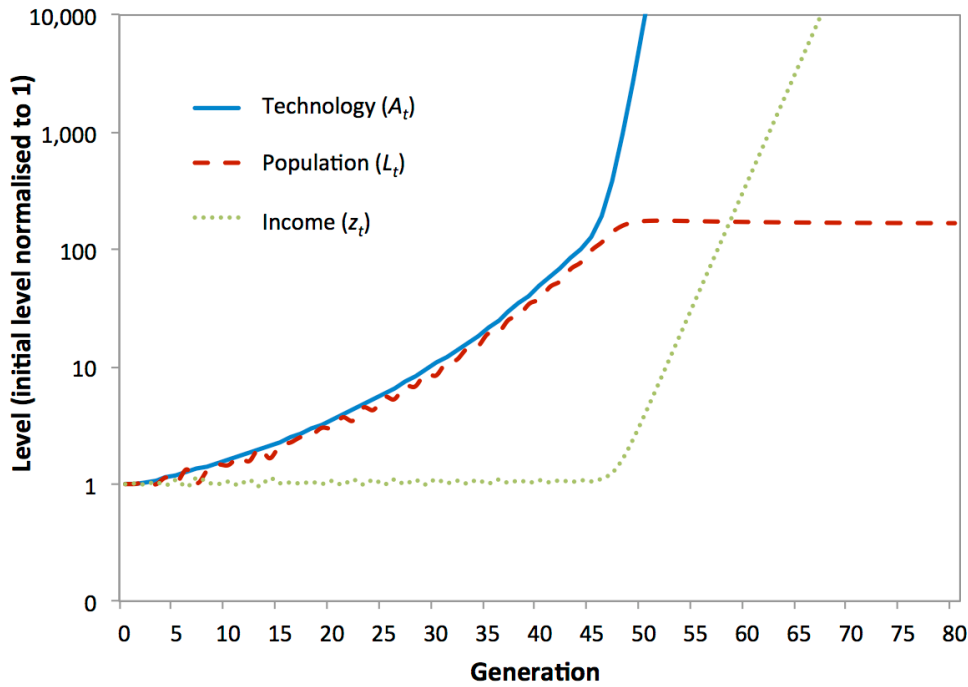


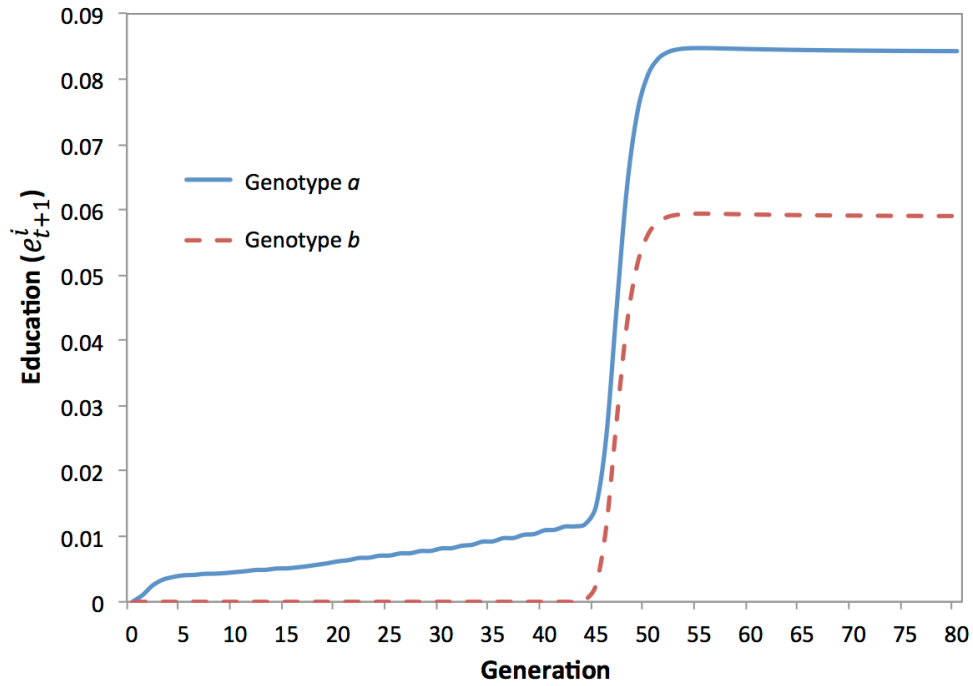
Figure 3.4: Level of technology, population and income



A sudden increase in the education of genotype b individuals prompts the take-off in economic growth that leads out of the Malthusian state. As genotype b forms the majority of the population at all times, the average level of education in the population,

which determines technological progress in equation (3.13), is approximately that of genotype b . Figure 3.5 shows the proportion of income spent on education by each genotype.

Figure 3.5: Education



Figures 3.6 and 3.7, which relate to the fertility rate of each genotype and the genetic composition of the population, convey the fitness of the competing genotypes. Genotype a has a fitness advantage through the Malthusian era. The increase in the prevalence of genotype a fosters slow technological progress during the Malthusian era until a threshold is reached that makes it worthwhile for genotype b to invest in education.

After the economic take-off, genotype a parents begin to overinvest into education of their children to an extent that hampers their fitness. For this reason, the fitness of genotype b is higher than that of genotype a from the beginning of the transition and the prevalence of genotype a starts to decrease. Genotype a 's prevalence peaks at under 5 per cent during the transition period. Thus, although the interaction between the two

genotypes is crucial for the growth dynamics of the economy, a preference for quality always remains a rare trait.

Figure 3.6: Fertility rate (five-generation moving average)

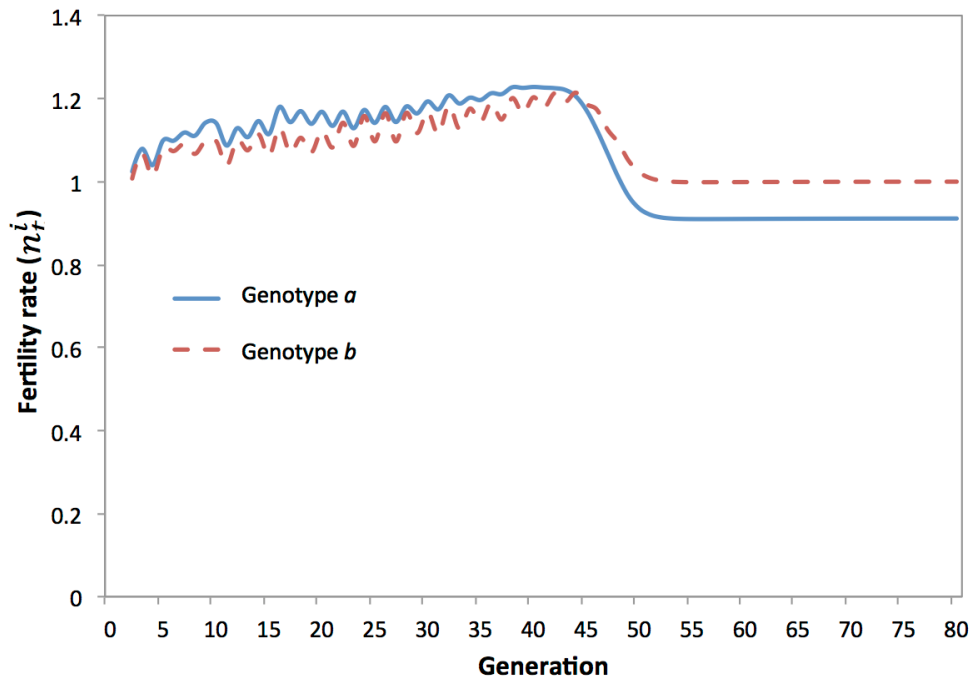
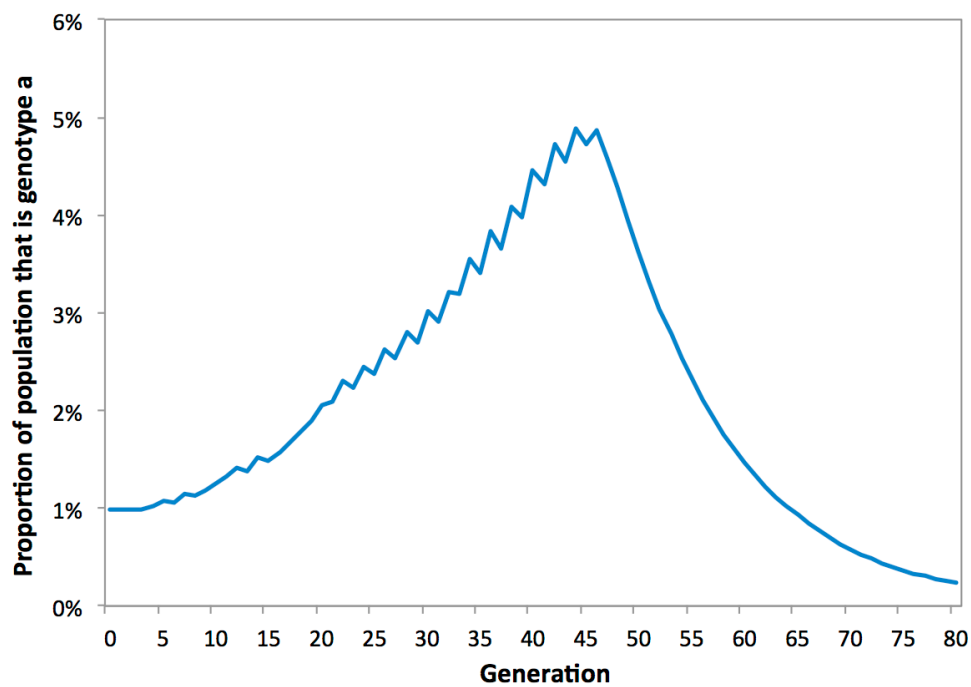


Figure 3.7: Proportion of population that is genotype *a*



During the Malthusian era, quality-preferring parents have both more and better-educated children, while quantity-preferring parents have fewer and less educated children. Therefore, no quantity-quality trade-off is apparent *at the population level*. While each individual makes a trade-off between quality and quantity, individuals who invest more in education also have higher fertility due to their own higher quality and income. From the beginning of the transition out of the Malthusian state, genotype *a* parents have better educated children, but fewer of them, while genotype *b* parents have more children with less education. From this point, the quantity-quality trade-off can be observed at the population level.

One empirical issue with the simulation concerns the timing of the demographic revolution. The simulated population stabilises during the transition phase, whereas high rates of population growth persisted in industrial countries to the end of the 19th century. No set of parameters was found that would delay the demographic revolution in the simulation. One interpretation of the model that may reconcile the timing of the demographic revolution would be to consider the time cost of children and their education as part of the national income. In that case, income is already rising with fertility before the simulated transition out of Malthusian stagnation.²⁸

3.9 Sensitivity of the modern growth regime to the introduction of a strongly quantity-preferring genotype

In the above simulation exercise, there are only two genotypes, with preferences for quality and quantity of children calibrated to achieve a transition of population, technology and income that reflects the Industrial Revolution. In this section, we show how the presence of a strongly quantity-preferring genotype affects the model

²⁸ We owe this interpretation to a comment by Oded Galor on an earlier draft of this paper.

dynamics. The main finding is that, in the absence of a scale effect in the model, a growing economy may regress to Malthusian conditions if a genotype that values education less than the other two genotypes is present in the population at the beginning of the simulated time period or is introduced exogenously during the simulation period. This finding is informative of the potential implications of migration (a new allele entering the population of interest from another population) or mutation (a spontaneous change in genotype).

Using the same functional forms as before, we simulate the model with three genotypes: the two genotypes a and b from the first simulation, plus a third strongly quantity-preferring genotype c . The quantity-quality preference parameters for the three genotypes are: $\beta^a = 1.0$, $\beta^b = 0.9$ and $\beta^c = 0.75$. All other parameters of the model are the same as in Table 3.1, except the initial levels of the subpopulations, which are $L_0^a = 0.007$, $L_0^b = 0.7$ and $L_0^c = 0.007$. Thus, both the quality-preferring genotype a and the new strongly quantity-preferring genotype c are around one per cent of the population at the beginning of the simulation.

Modifying equation (3.16), the aggregate labour supply is now:

$$H_t = \begin{cases} L_t(1-\gamma)(q_t^a h_t^a + q_t^b h_t^b + q_t^c h_t^c) & \text{if } z_t^a \geq \tilde{z}, z_t^b \geq \tilde{z} \text{ and } z_t^c \geq \tilde{z} \\ L_t \left[(1-\gamma)(q_t^a h_t^a + q_t^b h_t^b) + q_t^c (\tilde{c}/z_t^c) h_t^c \right] & \text{if } z_t^a \geq \tilde{z}, z_t^b \geq \tilde{z} \text{ and } \tilde{c} \leq z_t^c \leq \tilde{z} \\ L_t \left[(1-\gamma)q_t^a h_t^a + q_t^b (\tilde{c}/z_t^b) h_t^b + q_t^c (\tilde{c}/z_t^c) h_t^c \right] & \text{if } z_t^a \geq \tilde{z}, \tilde{c} \leq z_t^b \leq \tilde{z} \text{ and } \tilde{c} \leq z_t^c \leq \tilde{z} \\ L_t \left[q_t^a (\tilde{c}/z_t^a) h_t^a + q_t^b (\tilde{c}/z_t^b) h_t^b + q_t^c (\tilde{c}/z_t^c) h_t^c \right] & \text{if } \tilde{c} \leq z_t^a \leq \tilde{z}, \tilde{c} \leq z_t^b \leq \tilde{z} \text{ and } \tilde{c} \leq z_t^c \leq \tilde{z} \end{cases}$$

$$\equiv H(L_t^a, L_t^b, e_t^a, e_t^b, e_t^c, g_t, z_t^a, z_t^b, z_t^c) \quad (3.25)$$

q_t^a , q_t^b and q_t^c indicates the proportion of genotype a , b , and c in the population respectively.

Figures 3.8 and 3.9, which display the growth rates and log levels of technology, population and income, show that the first 60 generations of the simulation of the extended model including the strongly quantity-preferring genotype are similar to the baseline simulation in Section 3.8. The transition out of the Malthusian state occurs quite quickly, within six generations after the generation 45. Population growth again peaks early during the transition phase and the population then stabilizes. However, the subsequent growth era lasts for only about 20 generations, or 400 years. Economic growth abates because there is a renewed increase in population and a decline in the average level of investment in education after about generation 60, which does not occur in the model with only two genotypes. By generation 95, technological progress has ended and income growth is negative. The fall in per capita income continues until it has returned to the initial Malthusian level. Since technological progress is permanent, the economy, however, supports a higher population during the second Malthusian era.

Figure 3.8: Annual growth rate of technology, population and income

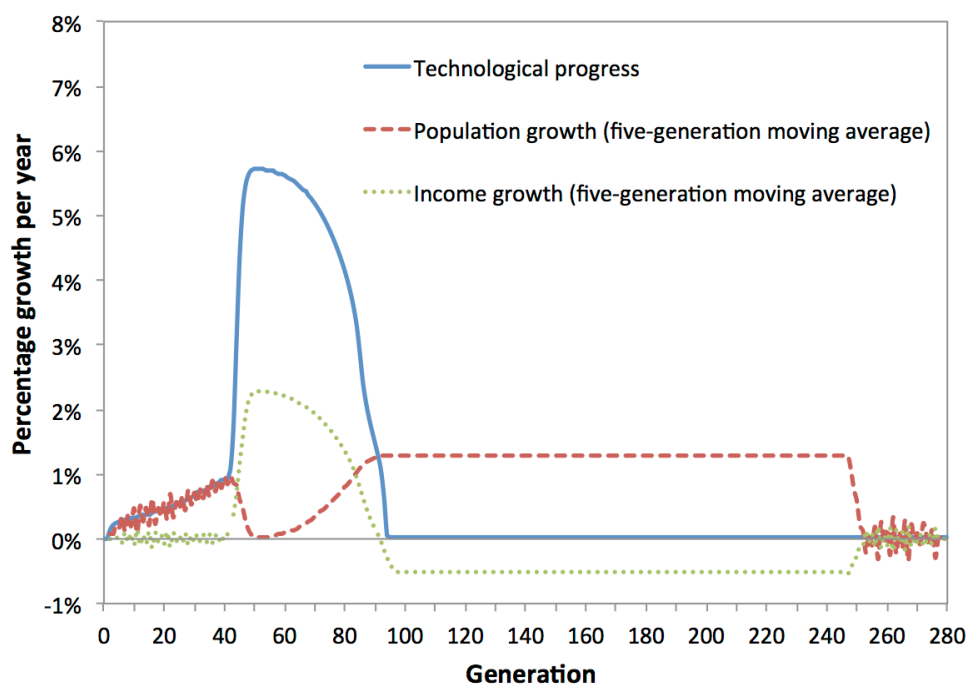
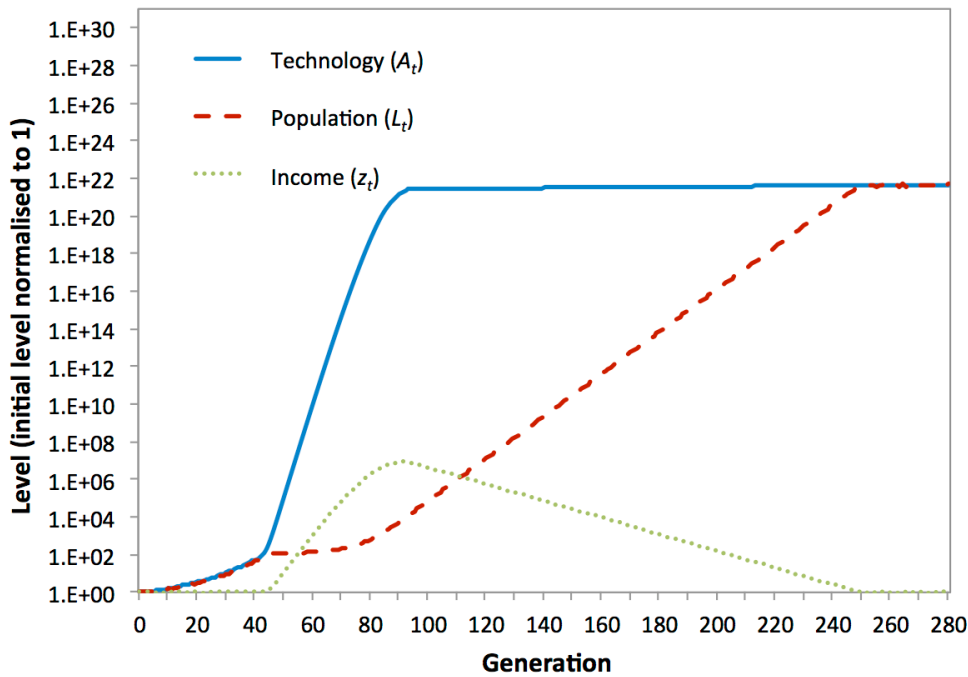


Figure 3.9: Level of technology, population and income



Figures 3.10, 3.11 and 3.12 reveal the behaviour of education, fertility and the genetic makeup of the population. In the initial Malthusian state, genotype a has the highest fitness as education increases potential income, and genotypes b and c have equal fitness as neither invests in education. Once the high growth era commences, natural selection favours genotype c because the other two genotypes overeducate their children relative to the level of education that maximises fitness. The return to the Malthusian state is caused by a decline in per capita human capital, which is driven by the higher fertility of genotype c and its increasing prevalence in the population. Since genotype c invest little in education, the average education level of the population declines and technological progress stalls. After the return to Malthusian conditions, the bulk of the population is genotype c , with a small proportion of genotype b and genotype a almost driven to extinction.

Figure 3.10: Education

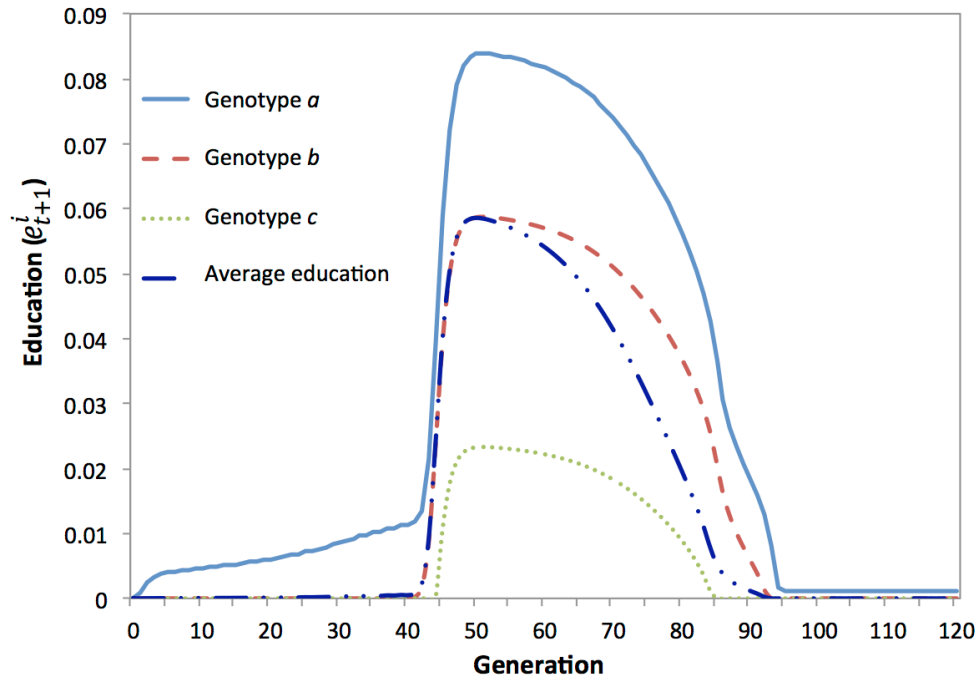


Figure 3.11: Fertility rate (five-generation moving average)

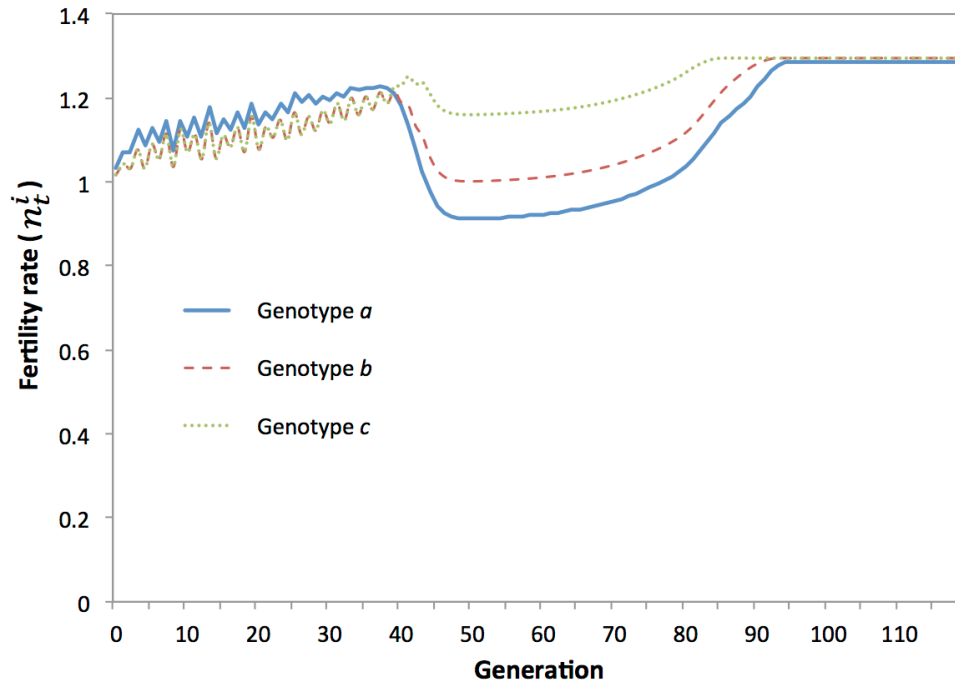
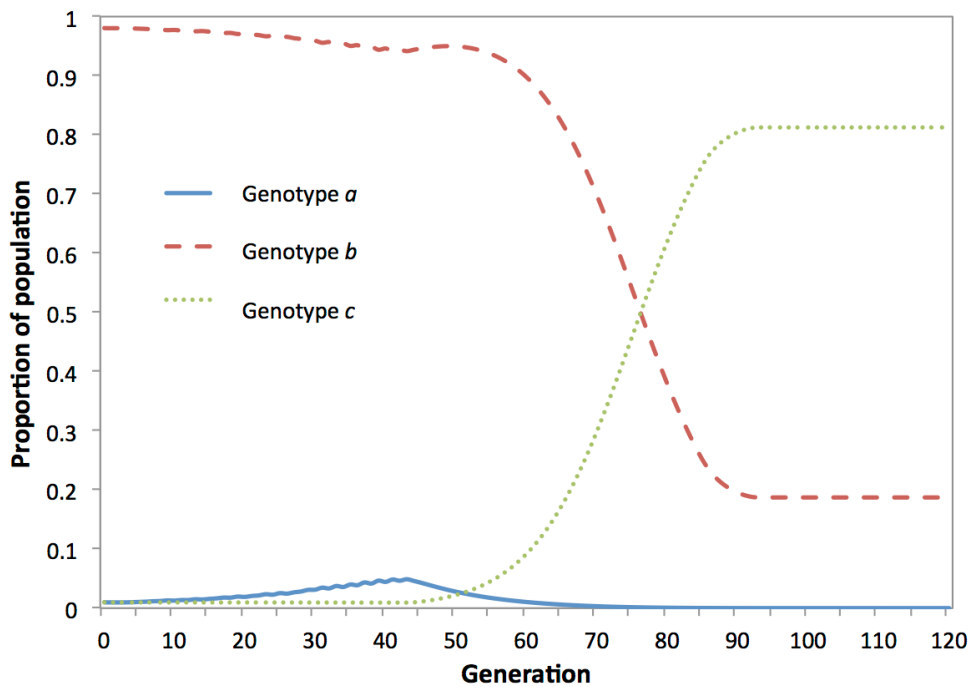


Figure 3.12: Proportion of population of each genotype



After the return to Malthusian conditions, genotype *a* at first regains the fitness advantage and again starts to increase in proportion of the population. It takes several hundred generations for genotype *a* to recover to meaningful numbers from their near extinction at the beginning of the second Malthusian state.²⁹ The renewed increase in the prevalence of genotype *a* again promotes technological progress, but it is not sufficient for another exit from Malthusian stagnation. The second Malthusian state is permanent because technological progress is matched by population growth. Thus, the situation is different from the initial Malthusian state with a small number of genotype *c* individuals. In the second Malthusian state, there is a high proportion of genotype *c* whose fertility absorbs any increase in income. After generation 565, a growth cycle repeats itself about every 25 generations without ever leading out of Malthusian stagnation. The cycles are generated by the interaction between genotype *a*, which

²⁹ No graphs are shown for the dynamics in the second Malthusian state.

drives technological progress, and genotype c , whose high fertility dilutes the average level of income in the population.

The timing of the return to Malthusian conditions is subject to the strength of the preferences of the strongly-quantity preferring type and their initial prevalence in the population. Given the other parameters used in the simulation, the economy returns to a Malthusian state for any value of $\beta^c < 0.808$, with the economy returning to the Malthusian state more quickly as β^c is reduced. For example, income growth is negative by generation 140 for $\beta^c=0.8$, by generation 95 for $\beta^c=0.75$ (as graphed above) and by generation 80 where the strongly quantity-preferring genotype does not invest in education at all (which is the case for any $\beta^c < 0.644$). However, for a value of $\beta^c = 0.809$, there is no return to a Malthusian state and per person income grows at 1.5 per cent per year in equilibrium, albeit at a slower rate than the 2.3 per cent immediately following the transition phase. Thus, the return to Malthusian conditions occurs only if the preference for quantity of genotype c is sufficiently strong.

The finding of a possible return to a Malthusian era does not alter materially if the new genotype is introduced later in the simulation period. For example, the strongly quantity-preferring genotype may emerge in a population during the period of economic growth, which creates opportunities for global migration. In this situation, the return to Malthusian conditions would be delayed to the extent that the introduction of the new genotype is delayed. A simulation was conducted with a quantity-preferring genotype with a preference parameter $\beta^c = 0.75$ comprising one percent of the population. If the new genotype is introduced after the transition phase at generation 50, income growth is negative by generation 100. A counteracting shock that introduces a strongly quality-preferring genotype into the population has, however, no long-lasting effect because the quality-preferring type will always have lower fertility in the modern growth era. The

stronger the preference for quality of children, the lower the fitness of a genotype and the quicker it will be eliminated from the population.

The return to Malthusian conditions may be prevented, however, by a scale effect of the form that features prominently in unified growth theory and the related Galor and Weil (2000) model. A scale effect would provide an additional source of technological progress, particularly following the rapid population growth at the time of the take-off into the modern growth era. The technological progress resulting from the scale effect would also provide an ongoing incentive for sufficiently quality-preferring agents to continue to invest in the human capital of their children, thereby providing a further foundation for economic growth.

3.10 Conclusion

The simulation of the Galor and Moav model produces a pattern of income and population growth that resembles the period of Malthusian stagnation before the Industrial Revolution and the take-off into a modern growth era. While the simulation demonstrated that model outcomes are sensitive to variations in the preference for quality and the fixed time cost of childrearing, a range of parameters exist for which the core features of the Industrial Revolution can be achieved. In particular, the increase in income over approximately six generations and the rapid demographic transition in response to the changing quality-quantity trade-off faced by the population agents reflect what is observed in Western Europe.

The simulations of the extended model demonstrate that, given the absence of a scale effect, the economy can regress to a Malthusian state due to an increasing prevalence of a strongly quantity-preferring genotype. If the model includes three genotypes with a wider range of preferences between quantity and quality of children, economic growth

is transitory if the third genotype is sufficiently quantity preferring. The high fitness of the quantity-preferring genotype eventually returns the economy to Malthusian conditions and in the case of the scenario simulated, the second Malthusian state is permanent.

The simulation exercise highlights other considerations relevant to a biological evolution theory of the Industrial Revolution. There may exist some degree of phenotypic plasticity, which – in the current context – is the ability of an individual with a given set of genes to change its behaviour in response to environmental conditions.³⁰ This might involve greater flexibility in the response to technological progress, which could allow quality-preferring genotypes to reduce their response to technological progress when overinvestment in education impairs their fertility. This flexibility would enhance the robustness of the modern-growth state by allowing quality-preferring genotypes to maintain a larger share of the total population. However, in the simulations presented above, a genotype that does not invest in education when income is above subsistence will always have a fitness advantage and drive the population back towards the Malthusian state.

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³⁰ The distinction between genotype and phenotype takes account of the observation that organisms with the same genetic code may look or act differently due to environmental conditions during their development.

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CHAPTER 4

POPULATION GROWTH, TECHNOLOGICAL PROGRESS AND THE EVOLUTION OF INNOVATIVE POTENTIAL

4.1 Introduction

In his seminal paper on population growth and technological change, Kremer (1993) proposed that the growth of population over most of human history is proportional to its size. As more individuals generate more ideas (Kuznets 1960; Simon 1998), larger populations generate more technological progress that can ease the Malthusian constraints on further population growth.

We propose that a complementary driver of technological progress is evolution of the human potential to innovate. We call this trait ‘innovative potential’, which might be thought of as research productivity per person. As a larger population generates more mutations (Fisher 1930), population growth will increase the rate at which new traits can emerge. If mutations that increase innovative potential raise the fitness of the bearer, these genes will spread in the population, enhance technological progress and provide an economic basis for further population growth. Larger populations would thus be expected to grow faster than smaller populations through evolution-driven technological progress.

In this paper we explore the effect of human evolution on population growth and technological progress in a model that incorporates evolutionary changes in human innovative potential.³¹ We demonstrate that the higher number of mutations in larger

³¹ The link between human evolution and economic growth proposed by Hansson and Stuart (1990) was first examined in depth by Galor and Moav (2002), who developed a unified growth model in which the preference for quantity or quality of children evolves. Galor and Michalopoulos (2012) apply a similar

populations, and hence a faster rate of evolutionary change in innovative potential, can provide an explanation for the greater than exponential population growth that Kremer documented for one million years of human history.³² Our evolutionary model of population growth is more robust to technological shocks than a model in which the innovative potential of the population is not evolving, with successively faster recovery from each shock. We also show that as the population becomes larger, population size becomes a relatively more important driver of the acceleration of technological progress than further increases in innovative potential.

In the following sections, we develop a model of population growth and technological progress in which the agents' innovative potential evolves endogenously. We examine the model under a number of specifications and test the response of the model population to technological shocks. We close our analysis with an agent-based model that allows us to endogenise factors such as the relative fitness of more innovative agents and the rate of spread of mutations through the population.

4.2 Evolutionary theory

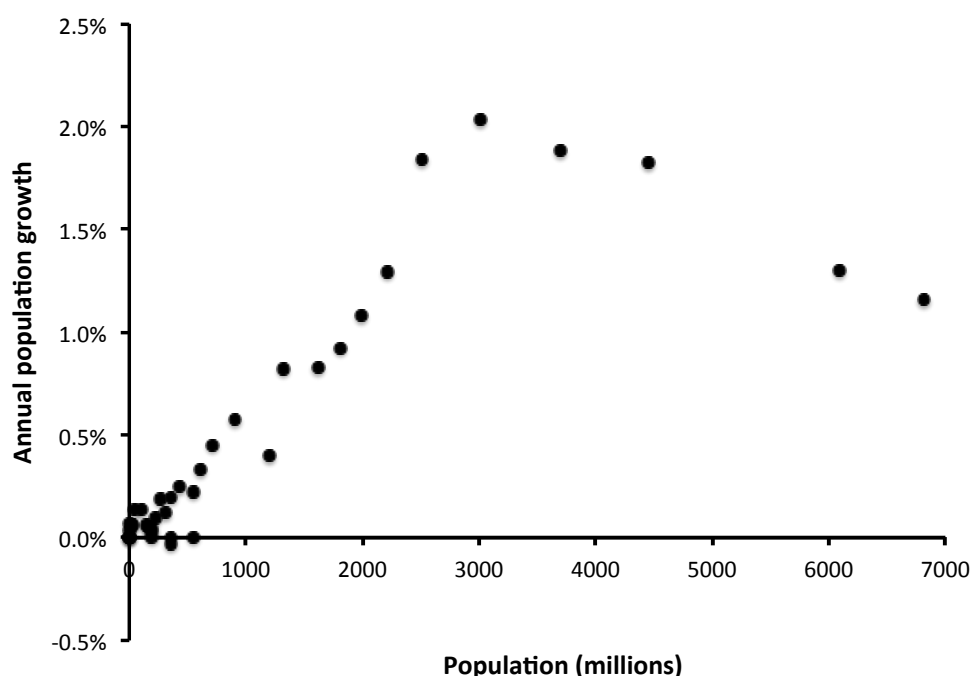
Until recently, the global annual population growth rate was positively correlated with population size, implying faster than exponential population growth. Figure 4.1 shows the relationship between global population size and population growth over one million years. The first data point indicates a global population of 125,000 and a population

framework to the evolution of entrepreneurial spirit, and Collins et al. (2014) provide a quantitative analysis of the Galor and Moav model. Clark (2007) proposed genetic change was a factor in the Industrial Revolution, while Zak and Park (2002, 2006) suggest sexual selection may affect economic growth. The growing availability of genetic data has also fostered research into the evolutionary determinants of economic development (Spolaore and Wacziarg 2009; Ashraf and Galor 2013).

³² *Homo sapiens* did not emerge as a distinct species until approximately 200,000 years ago. However, for ease of terminology, we will refer to the agents evolving over the last one million years, including various hominid precursors to modern humans, as “humans” or “people”.

growth rate of 0.0003 per cent per year, and the last data point represents the global population of close to seven billion people and a growth rate of 1.1 per cent in 2009. The population growth rate increased with population size until the global population surpassed three billion people in the mid-twentieth century, but then the positive relationship between population size and population growth broke down. The recent reduction in the population growth rate coincided with many sub-populations undergoing a demographic transition to lower fertility rates.

Figure 4.1: Population size and the annual population growth rate



Source: Until 1990, Kremer (1993), based on Deevey (1960), McEvedy and Jones (1978), the United Nations (1952, 1986) and The World Almanac (1991). The last two data points for 2000 and 2009 are from the United Nations (2006, 2011).

Kremer (1993) attributed this demographic pattern to a scale effect in innovation. A larger population leads to faster technological progress because a larger population generates more ideas. Adopting the Malthusian assumption that population size is limited by technology, the effect of population size on technological progress creates a positive feedback loop between population size and population growth. Kremer's model predicts that the growth rate of the population is proportional to its size, as is observed

in historical data until approximately 1950. Generalising the model, Kremer further suggested that there is a point where the rate of technological progress increases beyond that which population can grow, leading to an increase in per capita income. If people reduce fertility in response to increases in income, a demographic transition to lower population growth will then occur. This provides scope for the model to match the rise in the population growth rate before 1950, as well as the recent attenuation of population growth.

While Kremer characterised the driver of technological change as the total human population, humans have undergone significant evolutionary change over the one million years that he examined.³³ Evolutionary change is evident in the increase in brain size. The cranial capacity of *Homo erectus* skulls from one million years ago are typically around 900 cubic centimetres (Ruff et al. 1997; Lee and Wolpoff 2003; Rightmire 2004). A significant increase in brain size then occurred, with that increase concentrated between 600,000 and 150,000 years ago. Cranial capacity peaked at over 1,500 cubic centimetres approximately 30,000 years ago in the late upper Palaeolithic, although it has since declined to around 1,350 cubic centimetres (Henneberg 1988).

Changes in skull capacity and brain size are reflected in the emergence of behaviourally modern humans, at the earliest, 200,000 years ago (McBrearty and Brooks 2000; Henshilwood and Marean 2003). Some estimates put behavioural modernity within the last 50,000 years (Klein 2000). While there is evidence of technological progress before the appearance of behaviourally modern humans, such as increases in the quality of

³³ One million years ago, *Homo sapiens* did not exist as a species, with *Homo erectus* found in Africa, Asia and Europe (Rightmire 1998). Subsequently, a number of hominid species proposed as the ancestors of *Homo sapiens* emerged, including *Homo antecessor* (Carbonell et al. 2008) and *Homo heidelbergensis* (Rightmire 1998). Further, genomic evidence has revealed that *Homo sapiens* cross bred with *Homo neanderthalensis* in Europe (Green et al. 2010) and Denisova hominins in Asia (Rasmussen et al. 2011) within the last hundred thousand years.

hand axes and other stone tools around 600,000 years ago (Klein and Edgar 2002), technological progress was slow (and from the archaeological record, often undetectable) until behaviourally modern humans emerged.

Brain size and population size are correlated over this time. By comparing 175 hominid crania dating from between 1.9 million to 10 thousand years ago with population density, Bailey and Geary (2009) found that cranial size increased consistently with population density before declining slightly at the highest densities. They proposed that this correlation was driven by competition within large cooperative groups for control of social dynamics.

Brain size, together with any other trait that may influence innovative potential, is also affected through the link between population size and mutation rates. Fisher (1930) recognised that more mutations would occur in a larger population and proposed that larger populations should therefore evolve faster. Further, in a larger population the mutation is more likely to reoccur in subsequent generations if previously eliminated by genetic drift (Reed and Aquadro 2006), and in a growing population a mutation is less likely to be eliminated by genetic drift (Otto and Whitlock 1997).

Fisher's idea that larger populations generate more mutations has received empirical support from examinations of genomic data. Genomic evidence suggests that human evolution has accelerated over the last 40,000 years (Hawks et al. 2007).

Many studies have found heritable traits that would affect innovative potential, including brain size (Peper et al. 2007), intelligence (Johnson et al. 2010), risk preference (Cesarini et al. 2009; Le et al. 2012; Zhong et al. 2012), time preference (Bezdjian et al. 2011) and personality traits including openness, conscientiousness and

extroversion (Jang et al. 1996).³⁴ However, adaptive changes affecting innovative potential over the last one million years may have moved to fixation, effectively making heritability zero as all members of the population now have that adaptation. Further, evolutionary changes since *homo erectus*, as suggested by increases in cranial capacity, likely represent major changes in mental capacity and the basic ability to innovate. The strand of research that investigates the evolution of preferences during the transition from Malthusian conditions to modern economic growth (Galor and Moav 2002; Galor and Michalopoulos 2012) deals with a shorter time frame than the one considered by Kremer (1993) and in this study, and involves changes in preference for quality of children or risk rather than the fundamental changes in mental capacity that likely occurred over the longer timeframe contemplated in our model.

4.3 The basic model

In this section we describe a model of population growth and technological progress in the style of the base model developed by Kremer (1993). Our model incorporates an additional element in the form of the innovative potential of the population, with that potential subject to evolutionary change.

The model comprises a population of N people who live for one generation. The members of the population are of innovative potential δ , with δ genetically determined and passed from parent to child. Mutation provides a basis by which innovative potential may change between generations.

³⁴ A trait is heritable if a proportion of the variation in phenotype in a population can be explained by genetic variation.

Total output Y is:

$$Y = AN^\alpha X^{1-\alpha} \quad (4.1)$$

A is the level of technology and X is the amount of a fixed input factor, such as land or the environment, which is normalised to one. The parameter α is the elasticity of output with respect to labour input. Then, the level of per person income is:

$$y = AN^{\alpha-1} \quad (4.2)$$

In a Malthusian environment, fertility increases and mortality decreases with increases in income, keeping the population at subsistence levels. As a result, population will increase above a subsistence level of per person income, and decrease below it. We can use equation (4.2) to determine the population N that is supported by the subsistence level of income \bar{y} , as shown in equation (4.3). We assume that the population instantaneously adjusts to the Malthusian equilibrium, which is a reasonable approximation on a millennial timescale (Richerson et al. 2001).

$$N = \left(\frac{\bar{y}}{A} \right)^{\frac{1}{\alpha-1}} \quad (4.3)$$

The number and innovative potential of humans spur technological change. If research productivity of each person is independent of population size and increases in proportion with the level of technology A , the technological growth rate $g(A) \equiv (1/A)(dA/dt)$ is proportional to population size:³⁵

$$g(A) = \delta N \quad (4.4)$$

The new element in our model is the endogenously determined innovative potential of people δ . The number of beneficial mutations emerging in the population increases linearly with the size of the population (Fisher 1930). This has been shown to be the case in humans (Hawks et al. 2007). Accordingly, the growth rate in innovative potential $g(\delta) \equiv (1/\delta)(d\delta/dt)$ is:

$$g(\delta) = 2vN \quad (4.5)$$

v is the genome wide mutation rate for the emergence of beneficial mutations.³⁶ As for technological progress, we assume that the effect of new mutations builds upon previous mutations, which provides for increasing returns to mutation. We relax this assumption in the Appendix.

For traits associated with innovative potential to spread through the population, innovative individuals must have higher fitness. However, innovative potential will not lead to higher fitness if ideas are non-excludable and available to anyone regardless of their innovative potential. Therefore, to enable the spread of innovation-enhancing

³⁵ While some research casts doubt about the presence of scale effects in modern economies (Jones 1995), Kremer's (1993) findings provide evidence that scale effects are present over the longer timeframes before industrialization.

³⁶ Due to the manner in which the genome wide mutation rate is implemented in this model, v can also be interpreted as the increase in innovative potential arising due to mutations. We multiply the mutation rate by two as humans are diploid.

mutations independently of drift, we assume that innovative individuals accrue a fitness advantage. This assumption might be supported by temporary excludability of ideas in the form of trade secrets, inherently higher productivity of innovative individuals, or prestige attached to the generation of new ideas (Henrich and Gil-White 2001). A direct productivity component to innovative potential is explicitly included in the agent-based model in Section 4.5.

We can derive the growth rate of the population $g(N) \equiv (1/N)(dN/dt)$ by taking the log of equation (4.3) and differentiating with respect to time:³⁷

$$g(N) = \frac{1}{1-\alpha} g(A) \quad (4.6)$$

This equation shows that in a Malthusian environment, population growth is determined by technological progress.

Substituting equation (4.4) into equation (4.6) gives:

$$g(N) = \frac{1}{1-\alpha} \delta N \quad (4.7)$$

Equation (4.7) predicts that the growth rate of a population is proportional to the size of population and the innovative potential of its people. This leads to a prediction of stronger population growth than would be made under a model with constant innovative potential. The contribution of increasing innovative potential to population growth can be seen in Figure 4.2, which compares numerical simulations of a population with and

³⁷ Take the log of equation (4.3): $\ln N = \frac{1}{\alpha-1} (\ln \bar{y} - \ln A)$. Then $\frac{d \ln N}{dt} = \frac{1}{1-\alpha} \frac{1}{A} \frac{dA}{dt} \equiv \frac{1}{1-\alpha} g(A)$.

without the evolution of innovative potential.³⁸ Population growth accelerates faster with the evolution of innovative potential because of the positive feedback between population and innovative potential.

Figure 4.2: Population size

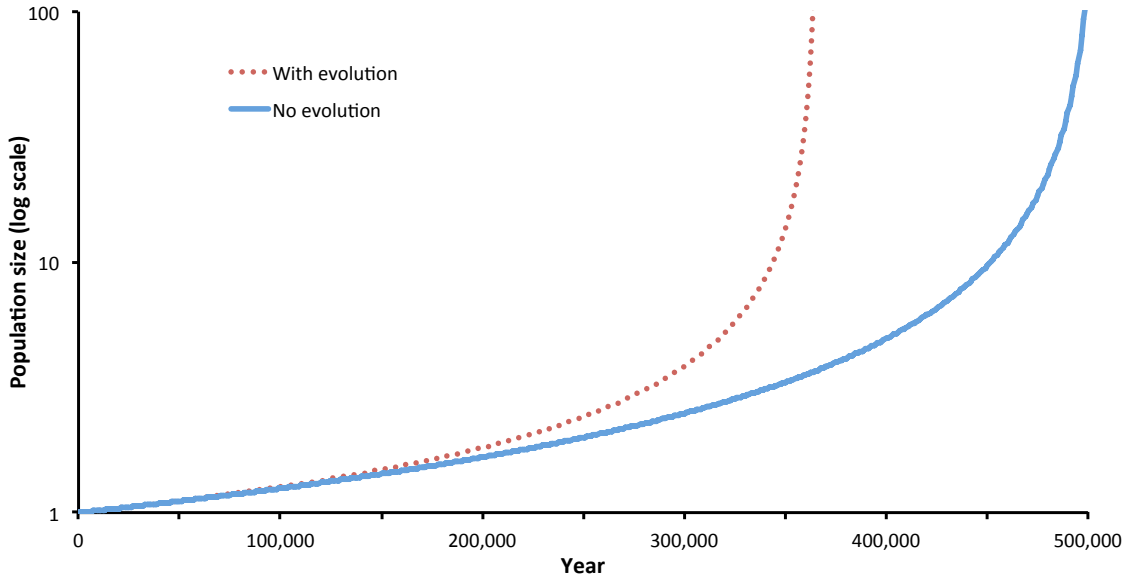
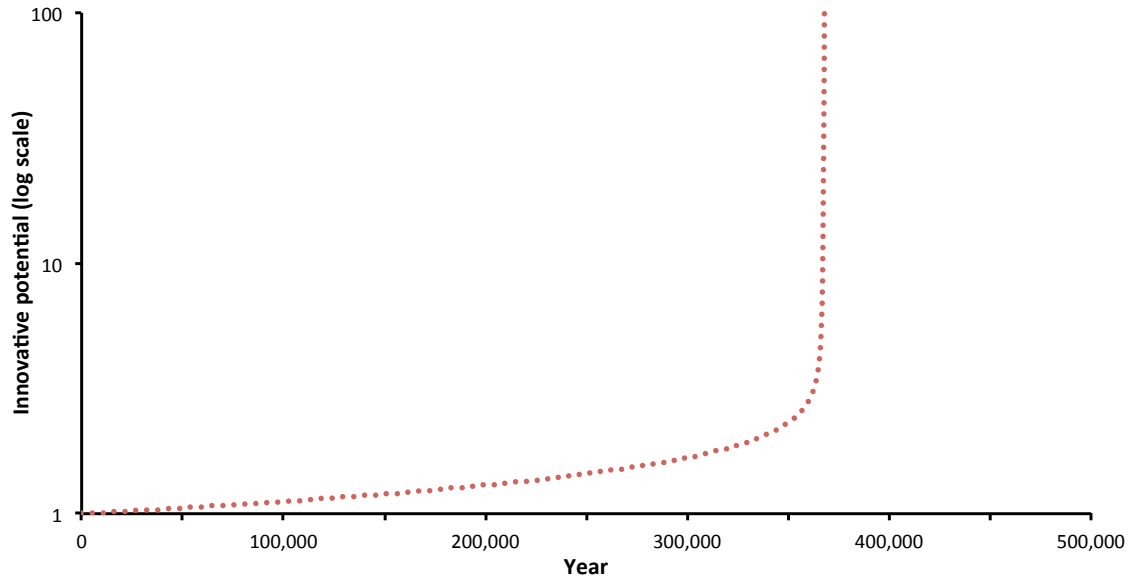


Figure 4.3 shows the evolution of innovative potential during this period. Around the time of the population explosion at approximately year 350,000 of the simulation, innovative potential growth sharply accelerates.

³⁸ The results in Figures 4.2 and 4.3 are generated by iterating equations (4.3), (4.4) and (4.5), with 1,000 years per iteration. The simulation parameters were $A_0 = 1$, $N_0 = 1$, $\delta_0 = 0.001$, $\alpha = 0.5$, $\bar{y} = 1$ and $\nu = 0.0005$.

Figure 4.3: Innovative potential (initial level normalised to one)



As both models seek to explain the same trajectory of human history, the appropriate interpretation of Figure 4.2 is that a model incorporating evolution of innovative potential requires a lower value of δ to generate the same acceleration in population growth to a model without evolution. If there is a time where the population size for each model is the same, population growth in the evolutionary scenario will accelerate faster from that point. Accordingly, we use the simulation of the scenario without evolution of innovative potential to provide a reference point for interpreting the simulations in figures, rather than providing a direct comparison of two alternative models of population growth.

This stronger population growth rate is apparent if we look at the acceleration in the growth of the population, $a(N)$. Taking the log and first derivatives of equation (4.7) gives:

$$\begin{aligned}
 a(N) &= g(\delta) + g(N) \\
 &= g(\delta) + \frac{1}{1-\alpha} g(A) \\
 &= \left(2\nu + \frac{\delta}{1-\alpha} \right) N
 \end{aligned} \tag{4.8}$$

The acceleration in population growth is driven by the growth in innovative potential and the growth in population size. The term 2ν is the additional acceleration in growth in the evolutionary model over a model with no evolution of innovative potential. Equation (4.8) can also be thought of apportioning the acceleration of population growth between growth in innovative potential and growth in technology.

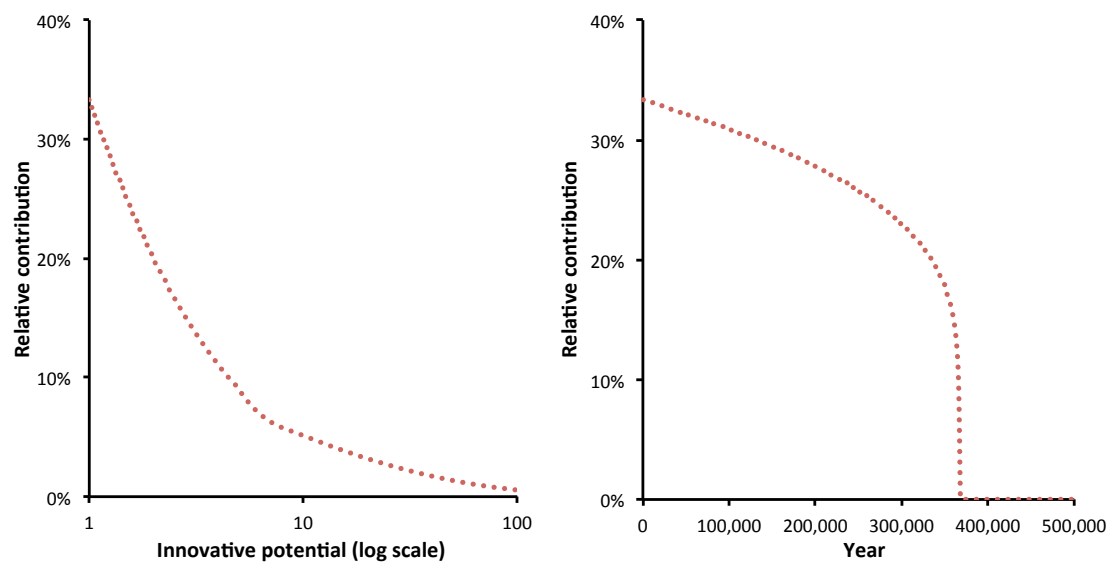
The proportion of the acceleration of population growth attributable to increasing innovative potential is obtained by dividing the first term of equation (4.8) by $a(N)$.

$$\pi(\delta) \equiv \frac{g(\delta)}{a(N)} = \frac{2\nu}{2\nu + \frac{\delta}{1-\alpha}} \tag{4.9}$$

Equation (4.9) shows that the contribution to the acceleration of population growth by increasing innovative potential grows weaker as innovative potential increases. As population members become more innovative, increasing their numbers has more effect on innovation than where innovative potential is low. Figure 4.4 shows the proportion of the acceleration in population growth that can be attributed to increasing innovative potential for the simulation shown in Figure 4.2. The panel on the left-hand side shows the negative relationship between innovative potential and its contribution to the

acceleration of population growth. The panel on the right-hand side depicts the contribution of innovative potential to the acceleration of population growth over time. Initially, improvements in innovative potential are a significant factor in accelerating population growth, with one third of the acceleration attributable to increasing innovative potential, but this effect fades as innovative potential increases. As innovative potential growth accelerates rapidly at year 370,000 of the simulation, the contribution of increasing innovative potential to accelerating population growth plunges to near zero at that time. However, the *level* of innovative potential of the population remains an important factor in the rate of technological progress.

Figure 4.4: Relative contribution of increase in innovative potential to acceleration of population growth



Adjustment of the mutation rate or initial innovative potential alters the relative contribution of innovative potential to the acceleration of population growth. However, the general pattern of declining contribution by innovative potential as innovative potential increases and the population takes off remains. The results also do not materially change if we use a more general version of equation (4.4) that allows for technological progress to vary with the level of technology, such as where there are

positive spillovers from earlier inventions to new ones, or where research productivity varies with population, such as from network effects. Similarly, a more general version of equation (4.5) that allows for spillovers between mutations does not much change the results. The effects of the more general functional forms of these equations are explored in the Appendix.

4.4 Population dynamics

The above analysis is predicated on a steadily increasing population. However, human evolutionary history comprises non-linear features, including population cycles and bottlenecks. For example, genetic evidence suggests that human populations experienced a bottleneck (or multiple bottlenecks) within the last 100,000 years that reduced the human population to around 10,000 individuals (Harpending et al. 1993).

Under a model with no human evolution, a sudden decline in population would constitute a significant setback to technological progress. The decline in population would result in a commensurate decline in idea production, and technological progress would revert to the level experienced when the population was last of that size. If the decline in population was caused by a technological shock, such as a change in environmental or climatic conditions that reduces the effective level of technology, or if the population decline reduced the level of technology available to the population through the loss of people holding ideas, the population recovery would be no faster than the rate of population growth when the population was last of that size.³⁹ Therefore, a population suffering frequent technological shocks may never escape the Malthusian state.

³⁹ If the shock does not affect the level of technology, population is able to increase back to the pre-shock level regardless of the rate of technological progress.

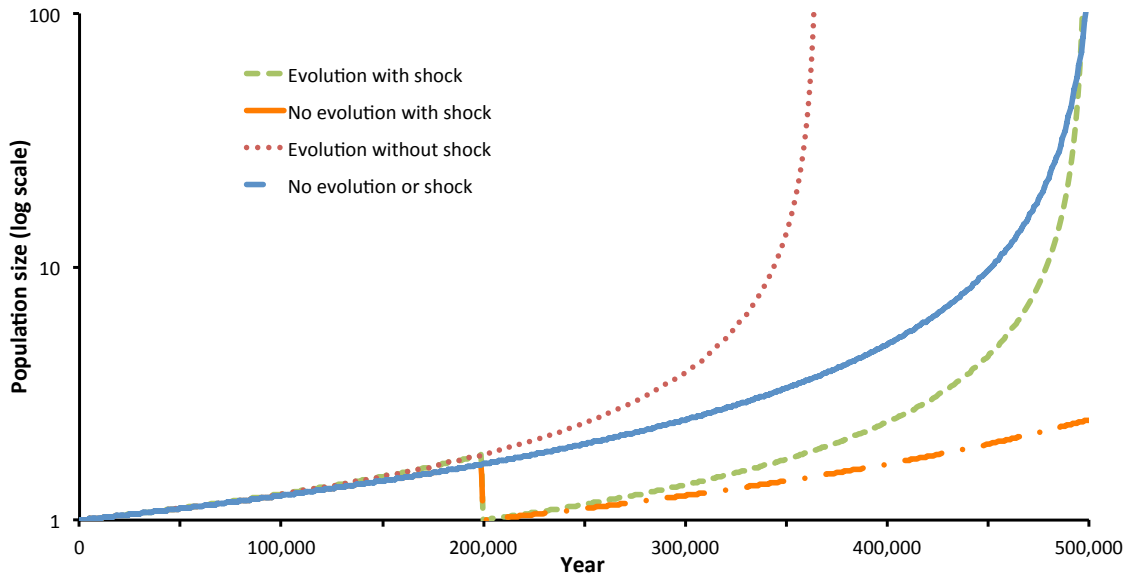
In a model incorporating the evolution of innovative potential, a population decline due to a technological shock is still a setback, but the innovative potential of people is higher after the shock than when the population was last of that size. This results in a faster recovery in population size and allows for continuing acceleration of technological progress. If an evolving population is subject to successive technological shocks, there will be successively faster recovery from each shock, as the population will have increasingly greater innovative potential.

Figure 4.5 shows a simulation of four scenarios over a period of 500,000 years: a base case for the population model without human evolution, a base case for the model incorporating human evolution, and those two scenarios being subject to technological shocks.⁴⁰ The shock, which is applied in year 200,000, reduces the level of technology to what it was at the start of the simulation ($A_{200,000} = 1$).

After the application of the technological shock, the population declines from around 1.7 in the base case without evolution and 1.8 in the base case with evolution to 1.0 in both scenarios. Without evolution, the shock effectively winds the clock back to the start of the simulation. Population growth during the next 200,000 years mirrors that for the previous 200,000 years, resulting in recovery from the shock by the year 400,000. The rebound in population following the shock is faster in the scenario with human evolution. Where innovative potential can evolve, it takes 157,000 years for the population recover to the level it was at the time of the shock. The later the shock and the higher the innovative potential of the people at the time of the shock, the faster the population will recover.

⁴⁰ The simulations in Figure 4.5 are generated by iterating equations (4.3), (4.4) and (4.5), with 1,000 years per iteration. The parameters were $A_0 = 1$, $N_0 = 1$, $\delta_0 = 0.001$, $\alpha = 0.5$, $\bar{y} = 1$ and $v = 0.0005$. At $t = 200,000$, a shock is applied such that $A_{200,000} = 1$.

Figure 4.5: Population size with environmental shocks



4.5 An agent-based model

In this section, we implement the model with heterogeneous agents. In contrast to the homogeneous agents of the above model, in this agent-based model each agent has his or her own specific level of innovative potential and interacts with the environment as an autonomous agent. This allows us to endogenise factors such as the relative fitness of more innovative agents and the rate of spread of mutations through the population, which cannot be done in closed form.

In the earlier model, we assumed that more innovative people have higher fitness. However, higher innovative potential will not lead to higher fitness and spread through the population if ideas are non-excludable. In this agent-based model we relax the assumption of higher fitness by adding an additional component to the production function so that innovative people are more productive in using ideas in addition to producing more ideas. This assumption is intuitively sensible, as those developing new technologies are likely to be better able to understand and use them. Higher productivity provides a basis for more innovative individuals to have a fitness advantage even where

no private advantage can be accrued through the production of ideas as technology is non-excludable.

Another assumption of the earlier model is that beneficial mutations spread immediately through the population once they arise. In reality, mutations take time to spread through the population, and require many generations to go to fixation. The delay in the spread of beneficial mutations may have significant effects on the model outcomes, particularly for larger populations where population growth and associated technological progress is rapid. In such cases, new mutations may initially have a weaker effect due to their low frequency in the population.

This dynamic is captured in the agent-based model. When an agent experiences a mutation, the spread of the mutation depends on the fitness of that agent relative to other agents. Their fitness advantage is the result of their level of innovative potential relative to the innovative potential of the other agents in the population. The population at any point may comprise agents of multiple levels of innovative potential, with various mutations simultaneously spreading through the population as a result of the reproductive success of those carrying the mutations.

The population in generation t comprises N_t agents who live for one generation, with each agent i ($i \in 1, 2, \dots, N_t$) having innovative potential δ_t^i . The innovative potential of each agent affects their rate of idea production and the productivity with which they utilise the available technology.

In each generation the heterogeneous agents work, producing total output Y_t .

$$Y_t = A_t \left(\sum_{i=1}^{N_t} \delta_t^i \right)^\alpha X^{1-\alpha} = A_t (\bar{\delta}_t N_t)^\alpha X^{1-\alpha} \quad (4.10)$$

A_t is the level of technology available to generation t and $\bar{\delta}_t$ is the average innovative potential of the agents in the population. X is the amount of land, which is normalised to one. The parameter α is the elasticity of output with respect to labour input. Y_t depends on innovative potential due to the effect of innovative potential on productivity. The level of per capita income is:

$$y_t = A_t \bar{\delta}_t^\alpha N_t^{\alpha-1} \quad (4.11)$$

Technological progress is a function of the number and innovative potential of people generating ideas:

$$A_{t+1} = A_t [1 + g(A)] = A_t (1 + \bar{\delta}_t N_t) \quad (4.12)$$

As we assume that there is no ownership of or return to land, the wage per unit of innovative potential is:

$$w_t = A_t (\bar{\delta}_t N_t)^{\alpha-1} \quad (4.13)$$

Therefore, the income of person i is:

$$z_t^i = w_t \delta_t^i = A_t (\bar{\delta}_t N_t)^{\alpha-1} \delta_t^i \quad (4.14)$$

Taking the level of per capita income where population is constant as \bar{y} , the population will tend towards the population size that can survive off that level of income (the Malthusian population), \bar{N}_t :

$$\bar{N}_t = \left(\frac{\bar{y}}{A_t \bar{\delta}_t^\alpha} \right)^{\frac{1}{\alpha-1}} \quad (4.15)$$

The expected number of children of agent i is proportional to agent i 's share of total income and the Malthusian population level for that level of total income. This is consistent with evidence of the higher reproductive success of men with more resources in hunter-gatherer societies and among pastoralists (Borgerhoff Mulder 1987, 1990; Cronk 1991; Hopcroft 2006). The realised number of children follows a Poisson distribution.⁴¹

$$n_t^i \sim \text{Pois} \left(\bar{N}_t \frac{z_t^i}{Y_t} \right) \quad (4.16)$$

Thus, the probability that n_t^i is equal to μ ($\mu \in 0, 1, 2, \dots$) is:

$$\Pr(n_t^i = \mu) = \frac{\left(\bar{N}_t \frac{z_t^i}{Y_t} \right)^\mu e^{-\bar{N}_t \frac{z_t^i}{Y_t}}}{\mu!} \quad (4.17)$$

The higher productivity and income of more innovative agents gives them a greater expected number of children than less innovative agents, and therefore a fitness advantage.⁴²

⁴¹ A Poisson distribution has regularly been used in the examination of the spread of beneficial mutations (Otto and Whitlock 1997). There is some evidence that a negative binomial distribution may be more appropriate for human populations as the observed variance tends to be greater than the mean (Kojima and Kelleher 1962), but that distribution may under-predict the number of childless agents (Waller et al. 1973). Regardless, the choice of distribution has limited effect on the simulation results.

⁴² An alternative specification for the number of children of each agent is based on a surplus of resources over a personal level of subsistence.

$$n_t^i \sim \text{Pois} \left(\bar{N}_t \frac{(z_t^i - \bar{c})}{(Y_t - \bar{c}N_t)} \right)$$

\bar{c} is the proportion of income that agents must allocate to their own survival before allocating any resources to the production of children. Given the rapid spread of the mutations in the agent-based simulations, this specification has limited effect on the model outcomes beyond increasing the probability that a new mutation will survive past its initial appearance.

The innovative potential of an agent j may differ from that of his or her parent i due to mutation. A child inherits their parent's level of innovative potential, plus or minus the effect of any mutations. Mutation occurs at the rate 2ν , with equal probability that the mutation is positive ($m_{t+1}^j = 1$) or negative ($m_{t+1}^j = -1$):

$$P(m_{t+1}^j = 1) = \nu \quad P(m_{t+1}^j = -1) = \nu \quad (4.18)$$

$$P(m_{t+1}^j = 0) = 1 - 2\nu$$

When an agent experiences a mutation, the mutation increases or decreases innovative potential by a factor of $1+\rho$. Thus, the innovative potential of a new agent j in period $t+1$ is:

$$\delta_{t+1}^j = \delta_t^i (1 + m_{t+1}^j \rho) \quad (4.19)$$

4.5.1 Simulation results

The agent-based simulations were developed in NetLogo (Wilensky 1999), with the agents run through the following model protocol:⁴³

- Each agent i works and generates income z_t^i [equation (4.14)].
- The agents' activity generates technological progress, which sets the level of technology available to the agents in the next generation [equation (4.12)].
- Each agent i has n_t^i children [equation (4.16)].
- The innovative potential of each child j is determined [equation (4.19)].

⁴³ The code for the agent-based model is contained in the Appendix. The full NetLogo model is available for download from <http://www.jasoncollins.org/downloads>.

- The agents from generation t die.

The parameters for the simulations are given in Table 4.2. All agents start at the same level of innovative potential. The values of δ_0 and ν are lower than used in simulations earlier in this paper, as these are rates per generation compared to rates per thousand years used previously. Initial technology is set at 10,000 to support the initial population of 1,000 agents.

Table 4.2: Agent-based model parameters

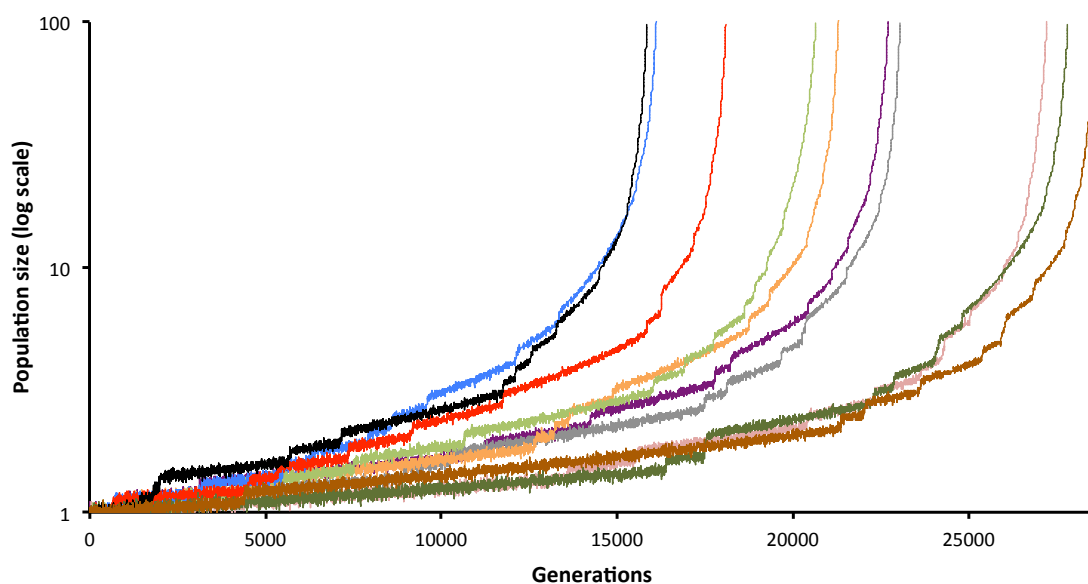
		Description	Base case value	Range explored
Parameters	α	Output elasticity of labour	0.5	
	ν	Mutation rate	10^{-6}	10^{-7} to 10^{-5}
	ρ	Mutation increment	0.1	
	\bar{y}	Subsistence income ⁴⁴	$\sqrt{10}$	
Initial values	N_0	Population	1000	
	\bar{N}_0	Malthusian population	1000	
	A_0	Level of technology	10,000	
	q_0^i	Agent innovative potential	10^{-8}	10^{-7} to 10^{-9}
	X	Land	1	

Figures 4.6 and 4.7 show the time paths of the population size and average innovative potential of the population for 10 runs of the base case model. To compare the simulation runs between the two figures, we kept the same colour code for each of the 10 runs. There is significant variation in the rate of population and innovative potential growth despite the same parameters being used in each model run because mutations arrive randomly during the simulation period. Even where mutations occur, many mutations are eliminated at low frequencies due to the number of children of an agent following a Poisson distribution. The number of children may be zero even if the mutant

⁴⁴ Set so that the population is stable at the starting population size, level of technology and innovative potential of the population.

has a higher than average expected number of children. For example, the model runs represented by the two top lines had several mutations that spread through the population within the first 5,000 generations, leading to an early increase in the level of idea production per person and population size that then drives further increases. In contrast, there were no innovation-enhancing mutations in one model run for over 15,000 generations. This reduced the positive feedback loop between population size and innovative potential relative to those model runs with early mutations, resulting in population differences greater than an order of magnitude at specific points. If we developed a version of the model that relied on individual chance inventions, rather than the steady accumulation of ideas as in the current implementation, the variation in the rate of population growth between simulation runs would be even larger.

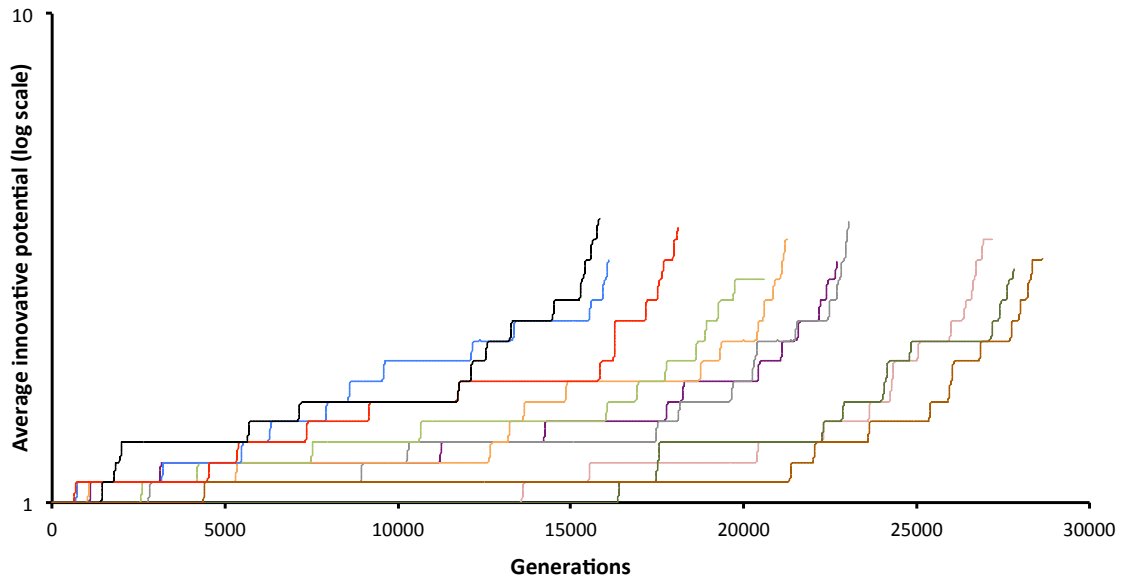
Figure 4.6: Population size in agent-based simulation (initial log level normalised to one)



The step-wise nature of innovative potential growth in Figure 4.7 is indicative of the rapid spread of fitness enhancing mutations when they arise and spread through the population. However, the delay prevents a sudden increase in innovative potential at the time of the population explosion. Innovative potential growth reflects the mutations that

arose when the population was much smaller, with the new mutations in the much larger population plentiful but at low prevalence as they have not had time to spread.

Figure 4.7: Average innovative potential in agent-based simulation (initial level normalised to one)



The parameter values for initial innovative potential (δ_0) and mutation (v) affect the rate of population growth, the growth in innovative potential of the agents, and the contribution of innovative potential growth to population growth. We explore the effect of varying these parameters in the Appendix, but we do not seek to calibrate the model as the lack of data makes this an impossible exercise at present.

4.6 Discussion

In this paper, we propose that the evolution of human innovative potential can be a complementary driver of long-term technological progress and population growth. The model provides scope for technological progress to accelerate if population size is constrained. This is particularly important if a population is subject to a setback that reduces its size. The evolution of more innovative people renders a population more resilient, with successively faster recovery from adverse shocks.

We also show that as a population increases in its ability to innovate, increases in population size become a more important driver of accelerating population growth than continued evolutionary change. Increasing population size is particularly effective where the population is already innovative, as population size has the potential to change more quickly than mutations can spread. Indeed, the global population has doubled over the last 50 years, but any genetically based human innovative potential has almost certainly not changed much during that period. However, we do not propose that our model explains population dynamics of the demographic transition and the decline in fertility of the last 200 years.

Where population size is the major driver of technological progress, there is potential for small populations to shrink and experience technological regress, such as that experienced by Tasmanian aboriginals after Tasmania was isolated from the rest of Australia by sea level rise (Diamond 1993; Kremer 1993). A similar possibility arises with the evolution of the innovative potential. In a smaller population, the low level of mutation and the increased potential for loss of beneficial mutations through genetic drift may affect the evolution of innovative potential. However, the historical presence of population bottlenecks where the human population was likely small (in the order of 10,000 individuals) suggests that human populations have often recovered from population shocks.

One feature of our model is that the evolution of innovative potential occurs along the dimension of production of ideas, and not in relation to human ability to transmit the ideas to other people in the population. The scale effect of population size on technological progress is premised on the ability of ideas to be spread and shared between population members. While some non-human species learn and communicate ideas, this is limited compared to humans (Cavalli-Sforza 1986). Further, it is suggested

that observational learning is not a by-product of intelligence and requires specific psychological mechanism (Boyd and Richerson 2005). On that basis, the slow technological change before the emergence of behaviourally modern humans may be partially attributable to the failure to transmit ideas across the population. There may have been an evolutionary change where the accumulation of ideas became possible, as humans developed the cognition and tools of cultural learning. As such, it is possible to develop a two-step evolutionary model in which the evolution of a trait allowing transmission of ideas is followed by a period where population size and increased innovative potential become the drivers of technological progress.

Data constraints make empirical tests of the model presented in this paper challenging. First, empirical tests would require some measure of human innovation potential. A proxy such as cranial capacity or brain size may serve this purpose, but would be a crude proxy at best. Another problem is the degree of resolution for population data for the last one million years. The Deevey (1960) data used in Figure 4.1 contains only three data points between one million BCE and 25,000 BCE, yet population data would be needed for each brain size data point. Higher resolution population data would likely expose the non-linear population dynamics of the human population, including population bottlenecks. These would render a simple time series regression of population and innovative potential inappropriate. Accordingly, while changes in brain size over the last one million years suggest that an evolutionary explanation of population dynamics is required over and above the explanation provided by Kremer (1993), current data constraints prevent a test between the models.

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Chapter 4 Appendix

4A.1 General function forms of innovation and mutation equations

A more general version of equation (4.4) for technological progress is given in equation (4.20) [derived from Jones (1995)]. This equation allows for technological progress to vary with the level of technology, such as where there are positive spillovers from earlier inventions to new ones, and for research productivity to vary with population, such as from network effects.

$$g(A) = \delta(qN)^\lambda A^{\phi-1} \quad (4.20)$$

Technological progress (in absolute terms) accelerates when there are positive spillovers in the creation of new ideas ($\phi > 0$) and it falls when there is a ‘fishing out’ of ideas ($\phi < 0$). Where $\lambda > 1$, the network effects are significant and groups are more productive than individuals, while $\lambda < 1$ represents a situation where there is duplication of effort as the number of researchers increases. Equation (4.4) has the implicit assumption of $\phi = 1$ and $\lambda = 1$; that is, there are positive spillovers in research.

Substituting equation (4.20) into equation (4.6) to derive equation (4.21), we can determine the growth rate of the population. The positive relationship between population growth and the size and innovative potential of the population persists using this more general functional form. The strength of the effect of innovative potential and population size on population growth is changed by the degree to which network effects are negative or positive, while less than linear returns to technology decrease the growth effect of both innovative potential and the population size in model simulations. This result reflects the findings of Kremer (1993), who also found that the more general

equation for technological progress did not substantially change the prediction of increasing population growth with population size.

$$g(N) = \frac{\delta}{1-\alpha} (qN)^\lambda A^{\phi-1} \quad (4.21)$$

Similarly, equation (4.5), which describes the evolution of the innovative potential of the population, can be generalised, allowing for spillovers between mutations. There is the possibility that the rate of “new” mutations is a function of the existing level of mutations, as one critical mutation may provide the environment for another mutation to have a fitness advantage, and there is the potential for depletion of certain mutations. For example, the ability to digest lactose past childhood evolved in at least three locations through different mutations (Tishkoff et al. 2006; Ingram et al. 2008). Once the ability to digest lactose had evolved, new mutations that cause this trait may not have a selective advantage relative to the existing allele.

A more general form of equation (4.5) where the mutation rate varies with the existing level of innovative potential is:

$$g(q) = 2vq^{\theta-1}N \quad (4.22)$$

There are positive returns (in absolute terms) to the existing number of fitness-enhancing mutations when $\theta > 0$ and decreasing when $\theta < 0$. This contrasts with the implicit assumption in equation (4.5) that there are positive returns to mutations ($\theta = 1$). Changing the functional form for mutations does not change equation (4.21).

Values of ϕ that vary from one are of interest as time series evidence from industrialised economies suggests that the value of ϕ is less than one (Jones 1995). While there is no

equivalent evidence that bears directly on the value of θ , it is plausible that it may also be less than one.

To determine the acceleration in population growth, we substitute equation (4.22) into equation (4.8).

$$\begin{aligned} a(N) &= \lambda g(q) + \lambda g(N) - (1-\phi)g(A) \\ &= \lambda \left(2vq^{\theta-1}N + \frac{\delta}{1-\alpha}(qN)^{\lambda} A^{\phi-1} \right) - (1-\phi)\delta(qN)^{\lambda} A^{\phi-1} \end{aligned} \quad (4.23)$$

The acceleration in population growth is now mitigated or enhanced depending on whether there are negative or positive network effects to innovation, and whether there is depletion of mutations. Diminishing returns to innovation lower the acceleration in population growth.

We can apportion the source of the acceleration of population growth between innovative potential growth and population growth. Dividing the first term of equation (4.23) by the full equation, the proportion of the acceleration in growth attributable to increasing innovative potential is:

$$\pi(q) = \frac{2v\lambda q^{\theta-1}N}{\lambda \left[2vq^{\theta-1}N + \frac{\delta}{1-\alpha}(qN)^{\lambda} A^{\phi-1} \right] - (1-\phi)\delta(qN)^{\lambda} A^{\phi-1}} \quad (4.24)$$

$$\frac{d\pi(q)}{dq} = \frac{(\theta-1-\lambda) \left[\frac{\lambda}{1-\alpha} - (1-\phi) \right] 2v\delta\lambda q^{\theta+\lambda-2} N^{\lambda+1} A^{\phi-1}}{\left[\lambda \left(2vq^{\theta-1}N + \frac{\delta}{1-\alpha}(qN)^{\lambda} A^{\phi-1} \right) - (1-\phi)\delta(qN)^{\lambda} A^{\phi-1} \right]^2} \quad (4.25)$$

$$\frac{d\pi(q)}{dN} = \frac{(1-\lambda) \left[\frac{\lambda}{1-\alpha} - (1-\phi) \right] 2\nu\delta\lambda q^{\lambda+\theta-1} N^\lambda A^{\phi-1}}{\left[\lambda \left(2\nu q^{\theta-1} N + \frac{\delta}{1-\alpha} (qN)^\lambda A^{\phi-1} \right) - (1-\phi) \delta (qN)^\lambda A^{\phi-1} \right]^2} \quad (4.26)$$

From equation (4.25), the relative contribution to the acceleration of population growth by increasing innovative potential declines with innovative potential unless $\lambda < (1-\phi)(1-\alpha)$, which is unlikely to be the case. Equation (4.26) shows that the relative contribution of increasing innovative potential to the acceleration of population growth declines with population size under the same condition.

4A.2 Sensitivity testing of agent-based model

In Tables 4.3 and 4.4 we show the results of sensitivity testing in which we increase and decrease these parameters by a factor of 10 in each direction from the base case. This represents exploration of a compound relative change by a factor of 10,000 ($\delta_0 = 10^{-9}$ and $\nu = 10^{-5}$ through to $\delta_0 = 10^{-7}$ and $\nu = 10^{-7}$). For each pair of parameters, we report the average result of 10 model runs. Table 4.3 reports the number of generations it takes for the population to increase by a factor of 100, from 1,000 to 100,000, while Table 4.4 reports the change in innovative potential of the agents at that endpoint.

Table 4.3: Sensitivity testing - Generations to 100,000 population.

		Initial innovative potential (δ_0)				
		10^{-9}	5×10^{-9}	10^{-8}	5×10^{-8}	10^{-7}
Mutation rate (ν)	10^{-7}	204,409 (35,859)	74,039 (13,214)	43,844 (3,633)	9,407 (631)	4,885 (182)
	5×10^{-7}	90,674 (17,616)	44,632 (7,010)	29,729 (3,056)	8,575 (681)	4,573 (243)
	10^{-6}	47,518 (12,261)	33,702 (7,172)	21,457 (3,656)	7,879 (750)	4,555 (341)
	5×10^{-6}	13,418 (1,821)	10,872 (2,619)	9,767 (1,781)	4,402 (502)	3,268 (317)
	10^{-5}	7,689 (1,478)	6,799 (716)	5,992 (1,088)	3,600 (451)	2,563 (288)

Note: Mean and standard deviation (in brackets) of 10 model runs.

Table 4.4: Sensitivity testing - Average innovative potential at 100,000 population (Initial value normalised to one)

		Initial innovative potential (δ_0)				
		10^{-9}	5×10^{-9}	10^{-8}	5×10^{-8}	10^{-7}
Mutation rate (ν)	10^{-7}	3.83 (0.59)	1.70 (0.27)	1.32 (0.12)	1.09 (0.08)	1.02 (0.04)
	5×10^{-7}	8.90 (0.85)	3.58 (0.64)	2.36 (0.23)	1.29 (0.10)	1.19 (0.10)
	10^{-6}	12.75 (1.01)	4.51 (0.54)	3.17 (0.40)	1.58 (0.19)	1.26 (0.10)
	5×10^{-6}	23.00 (0.85)	9.46 (0.79)	6.58 (0.50)	2.60 (0.30)	1.85 (0.12)
	10^{-5}	26.45 (1.33)	12.32 (0.83)	8.03 (0.25)	3.36 (0.27)	2.24 (0.18)

Note: Mean and standard deviation (in brackets) of 10 model runs.

The adjustment of these parameters strongly affects the rate of population growth and the contribution of innovative potential to that growth. Taking a generation to be approximately 20 years, the parameters explored can generate a 100-fold increase in population size in 50,000 years (lower-right cell of Table 4.3) or four million years (upper-left cell). Innovative potential may play almost no part in population growth (upper-right cell of Table 4.4), or may increase by 26-fold (lower-left cell) being the major driver of population growth.

CHAPTER 5

SEXUAL SELECTION, CONSPICUOUS CONSUMPTION AND ECONOMIC GROWTH

5.1 Introduction

In the majority of species, females invest more into offspring than males. Females produce costly eggs instead of cheap sperm, invest substantial amounts of resources into offspring during pregnancy or provide extensive brood care of young. Consequently, females are choosy and prefer males who give them or their offspring fitness enhancing benefits (Trivers 1972). This forces males to compete for access to females, often resulting in large differences in reproductive success between males relative to that between females (Bateman 1948; Wade 1979). The resulting sexual selection can generate rapid genetic and phenotypic change in a population (Maynard Smith 1978; Andersson 1994).

Males have evolved a range of traits that are advantageous when competing with rival males for access to females. This includes extravagant traits that are costly for the bearer, such as the plumage of peacocks, the bright coloration of butterflies or ornamental morphological structures such as the antlers of deer (Zahavi 1975). By imposing a cost or handicap on the male that cannot be borne by males with limited abilities or resources, these secondary sexual characteristics can provide an honest signal of underlying quality to the female (Grafen 1990a, 1990b). As such signals are honest, females benefit if they prefer males who give signals, while the increase in mating opportunities compensates the males for the cost of the signal.

Sexual selection has been an important force in human evolution, as emphasized by Darwin (1871). The higher variance in reproductive success for men than for women (Fisher 1930; Brown, Laland & Borgerhoff Mulder 2009) is suggestive of the struggle between males for mating opportunities with females. Using estimates of genetic diversity from a range of studies, Wade and Shuster (2004) estimated that sexual selection accounts for about half of total selection in *Homo sapiens*. Accordingly, men have evolved secondary sexual characteristics to signal their quality, which include behavioural traits such as the propensity to engage in conspicuous consumption. Through its cost, conspicuous consumption can provide an honest signal of male quality and give those men who engage in conspicuous consumption greater reproductive success (Frank 1999; Miller 1999, 2001; Saad 2007).

Since Veblen's (1899) identification of the preference for conspicuous consumption, conspicuous consumption has been recognized as a social phenomenon relevant to economic analysis. However, economic models typically ascribe no evolutionary foundation for consumption. From an evolutionary perspective, high consumption will only persist if it increases the fitness of the agent relative to those who consume less. Thus, an assumption that people seek to maximise consumption can only hold if maximising consumption enhances fitness. De Fraja (2009) addressed this problem by providing an evolutionary foundation to the economic hypothesis that humans seek to maximise consumption. Using a modified version of Grafen's (1990a, 1990b) models on biological signals as handicaps, he demonstrated that conspicuous consumption could be explained as an honest signal of male quality.

This paper extends previous analyses of the evolutionary foundations of conspicuous consumption by examining conspicuous consumption in a dynamic evolutionary framework. Our goal is to extend the work of De Fraja (2009) by moving from a steady

state population equilibrium analysis to an examination of the dynamic evolution of the trait and its economic effects. This makes it necessary to extend the analysis to include the interaction between reproductive success, conspicuous consumption and economic activity.

In our model some males carry a gene that predisposes them to signal their quality through engaging in conspicuous consumption, while others do not. Conspicuous consumption might involve autonomous activities such as developing art or other objects of beauty in traditional societies, or participating in the labour force to earn income in modern times. The definition of conspicuous consumption should be considered to be broad, and to include any consumption activities beyond those required for survival. The female's receptivity to male conspicuous consumption results in males being under strong selection to express the trait, which increases the prevalence of the gene underlying the behaviour and the level of conspicuous consumption in the population.

To fund conspicuous consumption, a male must participate in activities to obtain the resources to consume, which reduces the time available for subsistence and therefore the probability of survival. Males will only signal through conspicuous consumption if the fitness benefits through increased mating opportunity outweigh the handicap of lower survival probability. We show that a separating equilibrium exists in which signalling males increase in prevalence, with the female preference for high-quality males who signal through conspicuous consumption compensating for the survival cost of the signal.

We propose that this increase in conspicuous consumption increases economic growth through two avenues: increased labour engaged in productive uses to acquire resources

to conspicuously consume, and a scale effect (Romer 1990; Kremer 1993) whereby the level of human capital engaged in production drives technological progress. As an illustration, handaxes have been observed at archaeological sites at frequencies and levels of symmetry far above that required for purely for survival. Kohn and Mithen (1999) proposed that these handaxes were the products of sexual selection, with the ability to manufacture highly symmetrical handaxes a reliable indicator of the quality of the maker. As such, progress in stone tools was partly driven by competition for mates. As a modern example, a male must engage in high-value activities in the labour force to acquire sufficient wealth to purchase discretionary consumer items, which might range from fashionable clothes to a sports car. Their labour effort and any innovation they exercise to acquire these goods contribute to economic activity. Further, the production of many of these goods for conspicuous consumption involves high levels of innovation, such as through precision engineering.

As female mating choice increases male conspicuous consumption and the level of economic activities to fund it, we propose that sexual selection was a contributing factor to the emergence of modern levels of economic growth. As such, the model provides a basis for the observation that males engage in work effort and consumption at levels above that required for survival (or at the cost of survival) and proposes that these behaviours have substantial economic effects.

In Section 5.3 we develop and simulate a model of human evolution and economic growth in which only male behaviour is subject to selective pressure, and in Section 5.4 the model is extended to include male-female co-evolution.

5.2 Related literature

Over the last 40 years, a range of economic preferences of humans has been examined in an evolutionary context. This includes the evolution of altruism (Becker 1976; Hirshleifer 1977), risk preferences (Rubin & Paul II 1979), and time preference (Hansson & Stuart 1990; Rogers 1994; Robson & Samuelson 2007; Robson & Szentes 2008; Netzer 2009; Robson & Samuelson 2009).

More recently, a growing literature deals with the link between the evolutionary dynamics of these traits and economic growth. In a seminal paper, Galor and Moav (2002) proposed that changes in prevalence of a genetically based preference for quality or quantity of children were a trigger for the Industrial Revolution. Galor and Michalopoulos (2012) argued that selection for a genetically determined entrepreneurial spirit (proxied by risk aversion) could be the cause of modern levels of economic growth. In these papers, selection is ecological, being based on survival due to availability of resources above a subsistence level and allocation of those resources to children.

Zak and Park (2002) incorporated sexual selection into a model of economic growth as part of a broader analysis of gene-environment interactions and their economic effects. In their agent-based model, female choice affects the evolution of cognitive ability, as females prefer smarter males. Sexual selection may explain the observation of Clark (2007) that fertility was higher among wealthy men in the lead-up to the Industrial Revolution. He proposed that fecundity of well-off families increased the proportion of the population with the preferences and habits conducive to economic progress. Clark's findings match other evidence of higher reproductive success of men with more resources, particularly in hunter-gatherer societies and among pastoralists (Mulder

1987; Borgerhoff Mulder 1990; Cronk 1991; Hopcroft 2006). Although the link between resources and reproductive success may have weakened in recent times, positive sexual selection on male income has been observed in contemporary Sweden, Britain and the United States (Nettle & Pollet 2008).

Conspicuous consumption is likely to have deep evolutionary roots. Evidence of conspicuous consumption dates back to the development of stone axes, with many axes constructed to a degree of specification beyond that required for practical use, or at a cost to practical use (Kohn & Mithen 1999). Trade in shell beads has been dated to over 80,000 years ago (Bouzouggar et al. 2007), and the prevalence of monument building across cultures is also suggestive of a propensity to conspicuously consume (for example, Neiman 1997). Recent research in evolutionary psychology has linked conspicuous consumption with mating displays. Griskevicius et al. (2007) found that men who are shown photos of women or who read a romantic scenario were more willing to spend on luxuries than men who were exposed to neutral images. Sundie et al. (2011) showed that men looking for short-term partners wished to spend more on conspicuous consumption when primed with mating scenarios. Women asked to rate two otherwise identical men preferred the owner of a luxury car as a short-term partner. Yet, men showed no response to female conspicuous consumption.

5.3 Model with evolution of male preference

This section describes an evolutionary model in which males evolve a genetic propensity to signal their quality through conspicuous consumption. All females observe male signals and use this information to assess male quality.

5.3.1 The agents

The model comprises a population of male and female agents who live for one mating season. The number of males and females at the start of generation t , $M(t)$ and $F(t)$, are normalised to a constant level such that $M(t) = M(t+1)$ and $F(t) = F(t+1)$. For ease of notation, the indicator t relating to the generation is omitted except where a distinction is required to be made between two generations.

Males vary in inherent quality ($0 < h^k \leq 1$), which is allocated randomly at birth. We assume that males can be of high ($k = H$) or low ($k = L$) quality with probability p and $1 - p$. The assumption of random allocation of quality allows the evolution of conspicuous consumption to be analysed without conflating the analysis with inherited changes in the agents' qualitative traits.

The agents are haploid: that is, a single gene codes for each trait. Each male has one genetic locus, with the allele (variant of the gene) at that locus expressing for signalling behaviour. There are two alleles that determine the agents' set of actions, signalling (S) and not signalling (N), which are transmitted directly from father to son. The frequency of each male phenotype in the population is denoted by π^{ik} ($i \in S, N; k \in H, L$). For example, π^{SH} indicates the frequency of high-quality signalling males in the population. $\pi^i = \pi^{iH} + \pi^{iL}$ denotes the prevalence of males of type i of either level of quality.

As humans are diploid, this haploid arrangement can be thought of as representing the inheritance of a phenotypic characteristic between generations, with the alleles standing for the different phenotypes. Abstraction of this nature is common in modelling the evolution of social behaviour in humans as it avoids complications presented by diploid reproduction, genetic interactions and multi-gene traits (Grafen 1991). Where the genetics underlying the trait of interest are not understood and the area of interest is the

selective force on the expressed trait, the assumption of haploidy provides a convenient way of modelling the evolution of phenotypic traits. This arrangement could also be thought of as vertical transmission of a cultural trait, where the son learns the signalling behaviour from his father.

The utility of a male is defined as the number of children he fathers. The male utility function can only be defined in terms of the particular model details, so is given below in equations (5.20) and (5.21) after the model is further specified. The male utility function is only a correlate of fitness, as fitness is maximised if the number of viable offspring in the long-term is maximised. The single-generation form of the utility function is adopted through this paper for mathematical tractability. However, in this version of the model, a male's decision to maximise offspring numbers in the next generation is equivalent to long-term fitness maximisation, as the male cannot affect the genotype or quality of his children.

Female agents are identical and are passive, except for their mating decision. Females prefer males of higher quality, as the number of surviving children, n , is an increasing function of the quality of the male with whom she mates.

$$n = n(h^k) \quad n_h(h^k) > 0 \quad (5.1)$$

The utility of a female depends on the number of surviving children.

$$u^F = n(h^k) \quad (5.2)$$

As for the male, the female utility function is not a direct statement of the female's long-term fitness. She cares about the genotype of the male with whom she mates, as the genotype of her child will affect that child's reproductive success and that of future

generations. However, in a separating equilibrium, the single-generation form of the female utility function is still fitness maximising, with the choice of utility function affecting only the condition under which a separating equilibrium occurs. The change to this condition is discussed in further detail below.

Females are assumed to have an innate preference for observing male signals and, as they cannot directly observe male quality, use male conspicuous consumption as a proxy in the mating decision. The assumption that females cannot directly observe a dimension of quality is supported by the ubiquity of costly signalling behaviour in humans (Miller 2001). The innate preference of females reflects a situation where male evolution is shaped by a pre-existing female sensory bias (Basolo 1990; Ryan 1990, 1998; Miller 2001). Rather than male and female behaviour co-evolving, which is explored in the second model in this paper, here the female preference is a fixed trait that does not evolve.

5.3.2 The economy

The economy consists of two sectors: the subsistence sector and the luxury sector. The subsistence sector comprises activities that increase the probability of agent survival, such as hunting, gathering and resting. The luxury sector comprises labour market activities to access a surplus with which to engage in conspicuous consumption. In early evolutionary times before a modern division of labour, luxury sector activities might have involved conspicuous leisure (Veblen 1899), production of art or ornaments, body ornamentation or other costly displays of underlying quality (Miller 2001). When the development of agriculture allowed greater specialisation, time engaged in the luxury sector expanded to include specialised production activities and ultimately participation in the modern labour force.

Production in each sector uses inputs of labour and a scarce environmental factor, such as land, whose quantity we fix and normalise to one. Males have one unit of time that they allocate between the subsistence and luxury sectors. The proportion of time that a male is engaged in subsistence activities is s^{ik} , with the remaining time, $1 - s^{ik}$, spent in the luxury sector. Only males who carry the signalling allele S allocate time to the luxury sector and engage in conspicuous consumption. Thus, non-signalling males spend all their time in the subsistence sector ($s^{Nk} = 1$).

Each agent has an equal share of the fixed factor, giving each agent $1/M$ of the fixed factor, where M is the number of males. Agents allocate their use of the fixed factor between sectors in the same proportions as they do their time.

Agents receive the product of their own subsistence sector and luxury sector labour. Effective labour input in the luxury sector is a function of both the time allocated to labour activity in that sector and the quality of that agent. Accordingly, each agent receives income in the subsistence sector, z^{ik} , and conspicuous consumption sector, c^{ik} , as follows:

$$z^{ik} = A^S (s^{ik})^\rho \left(\frac{s^{ik}}{M} \right)^{1-\rho} = A^S s^{ik} M^{\rho-1} \quad \rho \in (0,1) \quad (5.3)$$

$$c^{ik} = A^L (1 - s^{ik})^\alpha h^{ik} \left(\frac{1 - s^{ik}}{M} \right)^{1-\alpha} = A^L (1 - s^{ik}) h^{ik} M^{\alpha-1} \quad \alpha \in (0,1) \quad (5.4)$$

The parameters ρ and α are the elasticity of output with respect to labour input in each sector. The shift factors A^S and A^L are the level of technology in the subsistence sector and luxury sector.

Aggregate output, Y , is given by the sum of output in the subsistence sector and the luxury sector.

$$Y = \sum_{i=S,N} \sum_{k=H,L} \pi^{ik} \left(A^S s^{ik} M^\rho + A^L (1-s^{ik}) h^{ik} M^\alpha \right) \quad (5.5)$$

Under equations (5.3) and (5.4), the wage per unit of labour in the subsistence sector, r^{ik} , and the wage per efficiency unit of labour in the luxury sector, w^{ik} , are:

$$r = A^S M^{\rho-1} \quad (5.6)$$

$$w = A^L M^{\alpha-1} \quad (5.7)$$

In a given sector, all agents receive the same wage per unit or efficiency unit of labour because the production technology exhibits constant returns to scale and they invest the same proportion of the fixed factor in each sector, as they do their labour.

As the wage in the luxury sector is per efficiency unit, low-quality males will receive a lower wage per unit of time engaged in the luxury sector than high-quality males. Therefore, low-quality males face a higher effective cost for conspicuous consumption, raising their cost of signalling. Signalling males are subject to the following budget constraint.

$$c^{ik} \leq w h^k (1-s^{ik}) \quad (5.8)$$

We assume that the subsistence sector has zero technological progress and set A^S to one. In a Malthusian economy, technological progress would be matched with population growth, effectively constraining income growth. As the population is normalised to a fixed level for each generation, the assumption of zero technological progress in the

subsistence sector allows for maintenance of a Malthusian environment without introducing population growth into the model.

The level of technology in the luxury sector is determined endogenously in the model. It is assumed that technological progress, g , is an increasing and concave function of the number of efficiency units of labour engaged in the luxury sector, L . This is similar to the scale effect as a driver of technological progress in Romer (1990), in which technological progress is a function of the human capital engaged in research, or Kremer (1993), who assumed that technological progress is a function of population size. The number of efficiency units of labour engaged in the luxury sector is given in equation (5.9), with the function for technological progress given in equation (5.10).

$$L = M \sum_{i=S,N} \sum_{k=H,L} \pi^{ik} (1 - s^{ik}) h^k \quad (5.9)$$

$$g = \frac{A(t+1) - A(t)}{A(t)} = g(L, A^L) \quad (5.10)$$

$$g_L > 0 \quad g_{LL} \leq 0 \quad g(0, A^L) = 0$$

5.3.3 The mating season

Each generation lives for one mating season, which comprises three stages denoted by A, B and C. The mating season can be thought of as one of a series of short-term partnerships, as a sequence of “serial monogamy” during a lifetime, or (in the case of some agents) as a complete monogamous life. In stage A males work and in stages B and C mating takes place. In this section we describe how males and females move from stage A to B to C in the mating season.

Equal numbers of males and females are born and enter stage A ($M_A = F_A$). Males suffer from pre-breeding mortality in stage A. Male survival probability, δ^{ik} , is a function of their subsistence income relative to a minimum subsistence level of income, \bar{z} .

$$\begin{aligned} \delta^{ik} &= \delta(z^{ik}, \bar{z}) & \delta_z(z^{ik}, \bar{z}) &> 0 & \delta_{zz}(z^{ik}, \bar{z}) &\leq 0 \\ \delta(0, \bar{z}) &= 0 & \delta(r, \bar{z}) &\leq 1 \end{aligned} \quad (5.11)$$

The number of surviving males who are available to mate in stage B is:

$$M_B = \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik} \delta^{ik} M_A \quad (5.12)$$

As male mortality is not uniform, the prevalence of males of each type in stage B varies from that in stage A:

$$\pi_B^{ik} = \pi_A^{ik} \delta^{ik} \frac{M_A}{M_B} \quad (5.13)$$

The number of females does not change from stage A to B as there is no female mortality ($F_B = F_A$).

In stages B and C, males and females are randomly paired and the female chooses whether to mate with the male. As males are polygynous and make no investment in the offspring, they can mate in both periods. Females can mate only once, as they must make a maternal investment in their children. While this paper has two mating periods, the results can be generalised to more than two mating periods.

The probability of a male or female being matched depends on the number of males and females in the mating pool. In stage B, the probability of being matched is one for a male [$q_B^M = q(M_B, F_B) = 1$], as male mortality ensures that there are fewer males than females. The probability of being matched for a female is:

$$q_B^F = q(M_B, F_B) = \frac{M_B}{F_B} = \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik} \delta^{ik} \quad (5.14)$$

When a female is paired in stage B, she decides whether she will mate with the male. If she does, the female exits the mating pool and gives birth to that male's children. A male always agrees to mate with the female he is matched with, as there is no cost to mating for a male.

There is no further mortality of males after stage A. The number of males and the ratio of types of males do not change between stages B and C ($M_C = M_B$ and $\pi_C^{ik} = \pi_B^{ik}$). The number of females available for mating in stage C comprises the females who did not mate in stage B.

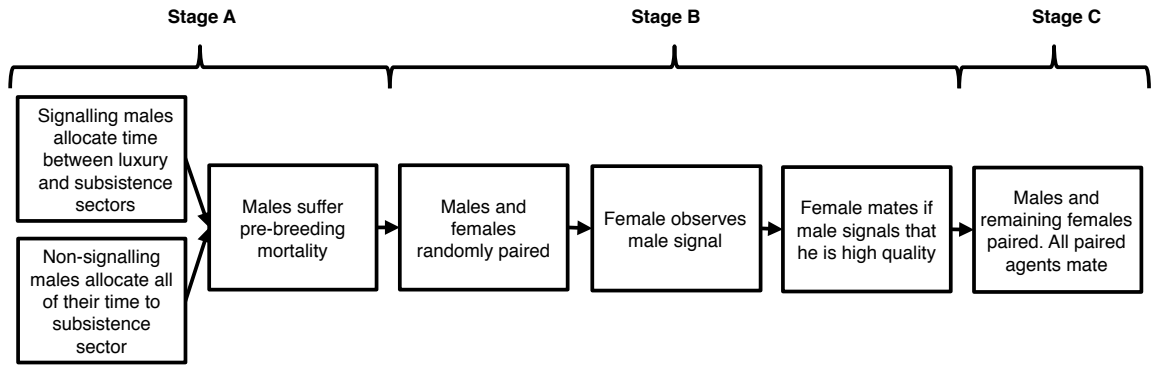
Depending on male mortality in stage A and the frequency of mating acceptance by females in stage B, it is possible for males and females to be unmatched until stage C, which is the final breeding period. A female's probability of being matched in stage C, q_C^F , will be greater than the corresponding probability in stage B as some females mate and exit the mating pool in stage B, whereas the number of available males remains constant.

$$q_C^M = q(M_C, F_C) = \min \left\{ 1, \frac{F_C}{M_C} \right\} \quad (5.15)$$

$$q_C^F = q(M_C, F_C) = \min \left\{ 1, \frac{M_C}{F_C} \right\} \quad (5.16)$$

In stage C, both females and males will mate with whomever they are matched with as females will have no further opportunities to mate and mating for males does not involve a cost. Offspring from the mating in stages B and C are then born and form the next generation. The mating season is summarised in Figure 5.1. Females observe male signals after they are randomly paired with males in stage B. There is no male signalling in stage C as females will accept any male they are matched with.

Figure 5.1: The mating season



5.3.4 Female optimisation

The female decision whether to agree to mate with a given male is a binary decision: yes or no. A female will mate in stage B if the benefit is greater than the benefit she expects to receive from mating in stage C. As the female is unable to distinguish between a high-quality non-signalling male and a low-quality male, she must weight the expected quality of the male with whom she is paired in stage B against expected future quality in stage C. She will reject a non-signalling male in stage B if:

$$E[n(h^k)] \leq q_C^F \sum_{i=S,N} \sum_{k=H,L} \pi_C^{ik} n(h^k) \quad (5.17)$$

If all remaining females are paired in stage C, this condition will always be met as $q_C^F = 1$ and $n(h^L) < n(h^H)$. Equation (5.17) is always satisfied unless there is a low probability of a female being paired with a male in stage C. This might occur if male mortality rates were high in stage A and few females mate in stage B.

If the female utility function were an exact representation of fitness, females would also weight the probability that the male is of the signalling genotype. The absence of a signal in stage B, in addition to signifying that the male is potentially of low quality, could mean that the male is of a non-signalling genotype. As signalling males have higher fitness in a separating equilibrium, this increases the incentive of the female to delay mating until stage C. This makes a separating equilibrium more likely. Once this separating equilibrium occurs, the mating decision of the female is identical regardless of whether the utility function is strictly a statement of long-term fitness. Females mate only with high-quality males who signal in stage B.

As a female observes conspicuous consumption rather than male quality, the mating decision of the female depends on whether the level of conspicuous consumption is sufficient, with the threshold level denoted by \bar{c} .

$$\theta(c^{ik}) = \begin{cases} 0 & \text{if } c^{ik} \leq \bar{c} \\ 1 & \text{if } c^{ik} > \bar{c} \end{cases} \quad (5.18)$$

If equation (5.17) is not satisfied, a female will set \bar{c} at a level that will only be achieved by high-quality males. In that case, we can state the number of females available to mate in stage C as:

$$F_C = (1 - q_B^F \pi_B^{SH}) F_B \quad (5.19)$$

In this specification of the model, a female who delays her mating decision incurs no cost to the delay beyond the small probability of not being paired in stage C. The model could incorporate costs to delay such as a probability of death before the second mating period [as was included in the model by De Fraja (2009)] or by recognising the increased relative fertility inherent with a shorter time between generations.

5.3.5 Male optimisation

The male's utility function can now be stated. The number of children fathered by a male is a function of his survival probability, whether a female accepts him as a mating partner, and the male's quality. Survival probability and mating success are a function of the level of conspicuous consumption. If females only mate with high-quality signalling males in stage B, the signalling and non-signalling males vary in the manner in which they optimise the number of children. Their respective utility functions are:

$$u^{Sk} = \delta^{Sk} (\theta q_B^M + q_C^M) n(h^k) \quad (5.20)$$

$$u^{Nk} = \delta^{Nk} q_C^M n(h^k) \quad (5.21)$$

Substituting the budget equation (5.8) into equations (5.20) and (5.21), a male of each type faces the following optimisation problem:

$$\{s^{Sk}\} = \operatorname{argmax} \left\{ \delta^{Sk} (rs^{Sk}, \bar{z}) \left[\theta (wh^k (1 - s^{Sk})) q_B^M + q_C^M \right] n(h^k) \right\} \quad (5.22)$$

$$\{s^{Nk}\} = \operatorname{argmax} \left\{ \delta^{Nk} (rs^{Nk}, \bar{z}) q_C^M n(h^k) \right\} \quad (5.23)$$

If females will not mate with non-signalling males in stage B [i.e. equation (5.17) is satisfied], high-quality signalling males will maximise utility by signalling if the gain from the additional mating opportunity in stage B [the left-hand side of equation (5.24)]

exceeds the decreased mating opportunity due to the increased probability of death (the right hand side).

$$\delta^{SH} q_B^M \geq (\delta^{NH} - \delta^{SH}) q_C^M \quad (5.24)$$

If there were more than two mating periods, the required decrease in survival probability before the high-quality signalling males would have lower fitness would be even greater. This condition is easier to satisfy if $M_C > F_C$, as the reduced probability of being paired in stage C makes the opportunity to pair in stage B relatively more important.

Finally, the non-signalling and low-quality males spend all of their time on survival activities ($s^{SL} = s^{Nk} = 1$).

5.3.6 Offspring

Offspring are born and raised at the end of the mating season. Assuming the females accept the high-quality males who signal, the offspring born from mating in stage B, who all inherit the signalling allele, are:

$$n_B^S = \pi_A^{SH} \delta^{SH} n(h^H) M_A \quad (5.25)$$

The mating during stage C results in offspring of both genotypes:

$$n_C^i = \sum_{k=H,L} \pi_C^{ik} n(h^k) \min\{M_C, F_C\} \quad (5.26)$$

The number of offspring born in each stage determines the prevalence of each genotype in the next generation. The prevalence of the signalling genotype in generation $t + 1$ is:

$$\begin{aligned}\pi_A^S(t+1) &= \frac{n_B^S + n_C^S}{n_B^S + n_C^S + n_C^N} \\ &= \pi(p, \pi_A^S, h^L, h^H)\end{aligned}\tag{5.27}$$

As the population is normalised to a constant level at the start of the next generation, equation (5.27) gives the relative proportion of each genotype in total population $M(t)$.

The prevalence of the signalling type in the population increases if $\pi_A^S(t+1) > \pi_A^S(t)$. Using this condition, equation (5.27) can be used to derive equation (5.24), which determines whether high-quality males signal and mate in stage B. This shows that the signalling allele increases in prevalence if condition (5.24) is met.

5.3.7 Signalling equilibrium

As shown by Grafen (1990a, 1990b), the core condition for the emergence of a separating equilibrium on the basis of a signalling handicap is that the signallers of different quality experience different costs (or benefits) to their signalling behaviour.⁴⁵ The low-quality male must experience greater costs (or lower benefits) for the same size signal as that produced by a high-quality signaller.

⁴⁵ Spence (1973) observed the requirement of differential cost for an honest signal in his analysis of job signaling markets.

In our model, the cost of signalling to a male is the reduction in offspring due to the lower probability of survival associated with a signal of size c . The cost can be derived from equations (5.8) and (5.11), and is weighted by the probability of being paired in stage C.

$$Cost = q_C^M \left[\delta(r, \bar{z}) - \delta\left(r \left[1 - \frac{c}{wh^k}\right], \bar{z}\right) \right] \quad (5.28)$$

The cost of conspicuous consumption of level c is larger for the low-quality male as they receive a lower wage for their labour and must sacrifice a greater quantity of subsistence activity to match a high-quality male's signal.

The benefit of conspicuous consumption is the expected increase in children due to the signal. The benefit of signalling is equal to the probability of surviving to stage B, being paired and the female allowing the male to mate with her. The benefit can be derived from equations (5.11) and (5.18). The benefit depends negatively on signal strength c as the increasing probability of death reduces the weighted benefit of the additional mating opportunity.

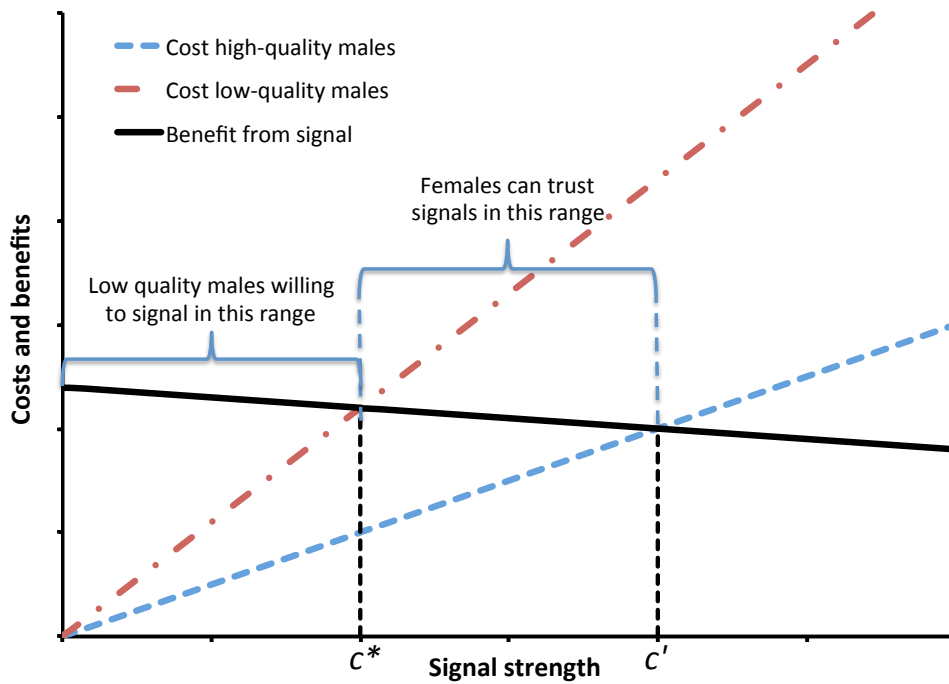
$$Benefit = q_B^M \theta \delta\left(r \left[1 - \frac{c}{wh^k}\right], \bar{z}\right) \quad (5.29)$$

Adding the costs and benefits together in equations (5.28) and (5.29) gives us the condition in which males are willing to signal in equation (5.24).

Figure 5.2 shows the costs and benefits of signalling for the male. The costs for high and low-quality males are as calculated in equation (5.28). The benefits are derived in equation (5.29). Figure 5.2 shows that there is a range of signal strength that a high-quality male is willing to make that a low-quality male will not match as the cost is

above the mating benefit. A low-quality male will set conspicuous consumption at or below c^* , while a high-quality male is prepared to signal up to a level of c' . In the interval from c^* to c' , conspicuous consumption gives an honest signal of male fitness because only high-quality males signal. Accordingly, females set the threshold level \bar{c} for the signal above the level c^* at which a low quality male is willing to signal.

Figure 5.2: Costs and benefits of signalling



Combining the male and female strategies, it can be observed that females will mate with males who signal in the interval $(c^*, c']$. High-quality males do not wish to signal more than necessary and they have first mover advantage as they set the signal before the female decides whether to mate. Therefore, high-quality males will signal at the level (or an infinitesimal amount above) that low-quality males are indifferent about. In other words, high-quality males will set the signal just above c^* .

In a separating equilibrium, no-one has an incentive to deviate. If high-quality males signal at a higher level, they reduce their survival for no mating gain, while a lower signal size that would be copied by low quality males will result in no mating benefit as

females cannot trust the signal. Low-quality males will not copy the signal, as its cost exceeds its benefit to them. Females will not raise their threshold level of acceptance as they would then miss the opportunity to mate with high-quality males, while a reduction in threshold would make signalling attractive to low-quality males.

Given $\bar{c} = c^*$, we can use equation (5.24) to determine the proportion of time that a high-quality male will allocate to subsistence activities. If we denote the level of subsistence activity of a low-quality male at which he is indifferent between signalling or not as \hat{s} , we can derive equation (5.30), which implicitly defines \hat{s} as a function of r , q_B^M and q_C^M .

$$\delta(r\hat{s}, \bar{z}) = \frac{\delta(r, \bar{z})q_C^M}{q_B^M + q_C^M} \quad (5.30)$$

Equation (5.8) describes the trade-off between subsistence activity and conspicuous consumption. Using that equation to determine the relationship between subsistence activities of the high-quality and low-quality types, the high-quality signalling type will set s^{SH} as in equation (5.31) to distinguish themselves from low-quality males:

$$s^{SH} = 1 - (1 - \hat{s}) \frac{h^L}{h^H} \quad (5.31)$$

To determine the value for q_C^M in equation (5.30), we must consider whether there will be more males or females in stage C. Using equations (5.12) and (5.19), $q_C^M = 1$ if:

$$\delta(rs^{SH}, \bar{z}) > \frac{1 - (1 - \pi_A^{SH})\delta(r, \bar{z})}{2\pi_A^{SH}} \quad (5.32)$$

Equation (5.32) is solved in iteration with equations (5.30) and (5.31) to see whether there are more males or females in stage C. Specification of a function for δ will allow the values of \hat{s} and s^{SH} to be derived.

5.3.8 Model dynamics

When equations (5.17) and (5.24) are satisfied, there will be a separating equilibrium where high-quality males signal and females consent to mate in stage B only if paired with one of those high-quality signalling males. In a separating equilibrium, the prevalence of the signalling allele in the population will increase because the reproductive success of the signalling males will be higher than that for non-signalling high-quality males. This will in turn increase labour force participation in the luxury sector and the rate of technological progress, driving an increase in income and economic growth.

The rate at which the signalling allele spreads in the population is affected by a number of variables. The per generation percentage point change in the prevalence of the signalling allele is:

$$\Delta\pi^S = \frac{p\pi_A^S(1-\pi_A^S)\left[(1+q_C^M)\delta^{SH} - q_C^M\delta^{NH}\right]n(h^H)}{p\pi_A^S\delta^{SH}n(h^H) + q_C^M \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik}\delta^{ik}n(h^k)} \quad (5.33)$$

The term $\pi_A^S(1-\pi_A^S)$ in the numerator shows the frequency dependent nature of the change in the prevalence of the signalling allele. The term equals zero at $\pi_A^S = 0$ and at $\pi_A^S = 1$, and it reaches a maximum at $\pi_A^S = 0.5$. At low frequencies of the signalling allele, there are few signallers who experience reproductive success, and at high frequencies the rate slows as the prevalence approaches the upper bound of 100 per cent.

The rate of change, $\Delta\pi^S$, increases with the proportion of the population that is high quality, p , as an increased prevalence of high-quality males results in more males indicating the presence of the signalling allele through signalling. The rate of change also increases with the difference in reproductive success between high and low-quality males, $n(h^H)$ and $n(h^L)$, as the high-quality signalling males obtain a relatively greater benefit from their mating success.

According to equation (5.24), which gives the condition for an increase in the signalling allele, the term $(1 + q_C^M)\delta^{SH} - q_C^M\delta^{NH}$ in the numerator of equation (5.33) is positive. An increase in this term increases the selection rate through the greater relative benefit of signalling. $1 + q_C^M$ depends on the number of mating periods in a season, with high-quality signalling males having $1 + q_C^M$ expected mating opportunities, compared to the q_C^M opportunities of the other types. If there were more than two mating periods in a season, the signalling allele would spread more quickly as signalling males would have even more opportunities to attract a mate. The survival rates of the signallers and non-signallers, δ^{ik} , are also relevant, with more costly signalling slowing the spread of the signalling allele. The size of the required signal is, however, likely to fall as the survival cost increases, reducing the effect of changes in survival probability on the spread of the allele.

5.3.9 Simulation

The evolutionary process is driven by the difference equation (5.27), which describes the change in prevalence of signalling males in the population. The following simulation illustrates the dynamics of the model.

We set equation (5.1) as $n = h^k$. The growth rate in technology, given in equation (5.10), takes the form $g = \psi L^\beta$ [$\beta \in (0,1)$]. ψ is the research productivity per person.

Equation (5.11) is specified as $\delta^{ik} = rs^{ik}/\bar{z}$. The parameter values and initial values are given in Table 5.2. The initial prevalence of the signalling allele is minute, amounting to 0.001 per cent of the population.

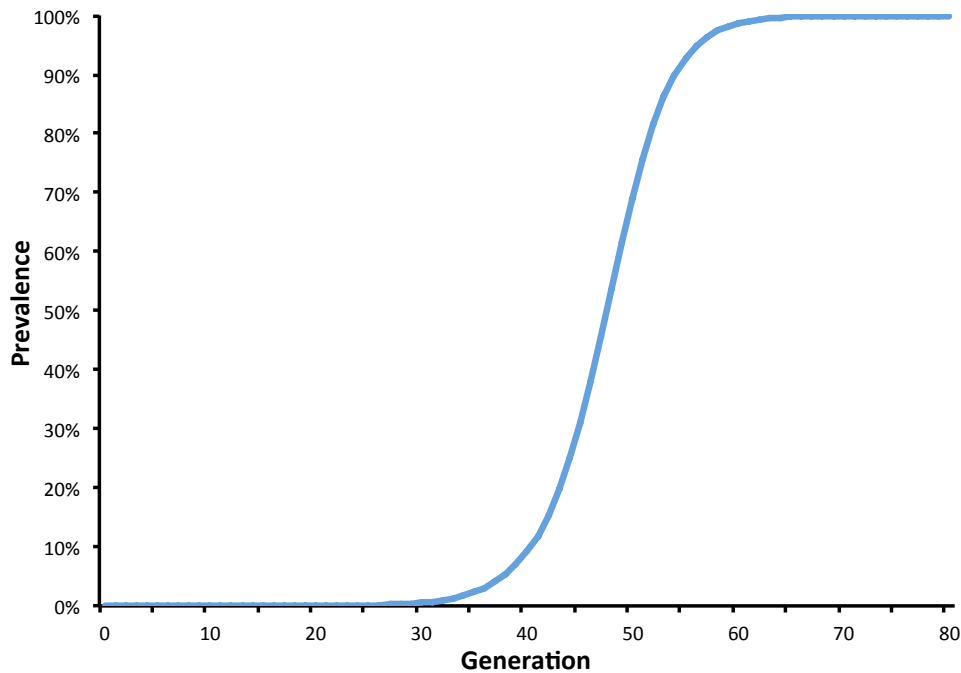
Table 5.2: Model parameters

		Description	Value
Parameters	α	Output elasticity of labour in luxury sector	0.5
	ρ	Output elasticity of labour in subsistence sector	0.5
	h^L	Quality of low-quality males	0.5
	h^H	Quality of high-quality males	1
	p	Proportion of high-quality males at birth	0.5
	Ψ	Research productivity per person	1
	\bar{z}	Subsistence income	1
	β	Returns to innovation	0.5
Initial values	π^S	Initial prevalence of signalling allele	0.00001
	A^L	Level of technology in the luxury sector	1
	g	Rate of technological progress in the luxury sector	0

As shown in Figure 5.3, at low prevalence the signalling allele spreads slowly as few males signal. It takes 33 generations (approximately 700 years) until signalling males account for one per cent of the male population. Afterwards, the signalling allele spreads rapidly, with another 28 generations (approximately 600 years) to move from one per cent prevalence to over 99 per cent. The point of inflection occurs close to a prevalence of 50 per cent, as $\pi_A^S(1 - \pi_A^S)$ in the numerator of equation (5.33) is highest at $\pi_A^S = 0.5$.⁴⁶ Eventually the spread of the signalling allele slows as it reaches fixation at 100 per cent prevalence.

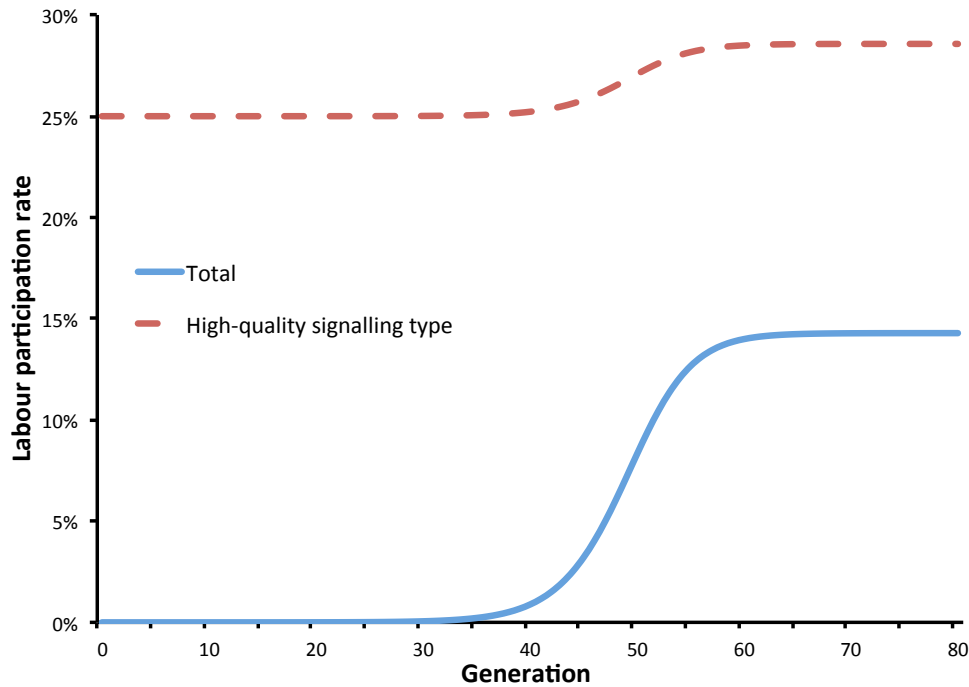
⁴⁶ The point of inflection occurs at $\pi_A^S < 0.5$ because π_A^S also appears in the denominator in equation (5.33).

Figure 5.3: Prevalence of signalling allele



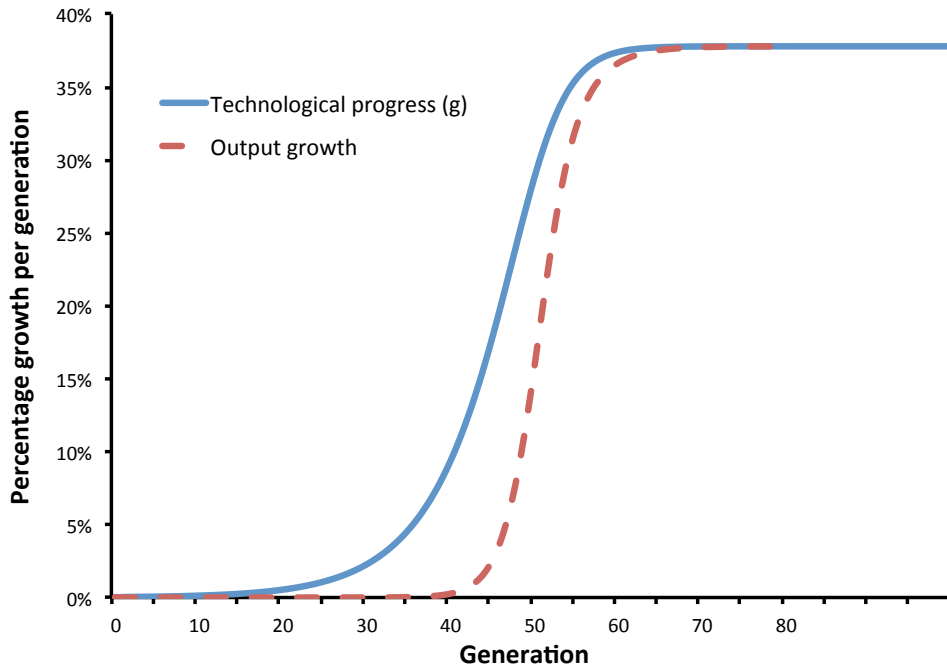
The prevalence of signalling males influences economic growth in two ways. First, the increase in prevalence increases participation in the luxury sector. Figure 5.4 shows that high-quality signalling males initially allocate 25 per cent of their time to the luxury sector. Their work effort increases to about 29 per cent in response to an increase in the benefit of mating in stage B, as fewer females remain in the breeding pool for stage C. This is equivalent to an upward shift in the benefits to mating line in Figure 5.2. Between generations 40 and 70, as the prevalence of the signalling allele increases, the overall labour force participation [equation (5.9)] rapidly increases. This increase in work effort is reflected in the historical record. Reviewing a number of studies, Voth (2000) notes that the average hours worked per day increased from an average of 4.9 hours in hunter-gatherer communities, to 7.4 in mixed communities and to 10.9 hours per day in advanced sedentary agricultural societies.

Figure 5.4: Labour force participation



The second way that signalling males contribute to economic growth is through technological progress. As technological progress is a function of the population engaged in the labour force, technological progress increases with the increase in the prevalence of signalling males. Figure 5.5 shows the growth paths in technology and output, both of which show significant increases with the increased prevalence of signalling behaviour. Initially, technological progress is not matched by output growth due to the low proportion of the population that is engaged in the luxury sector. It is only when there is significant use of the growing technological base by a greater proportion of the population that output growth takes off. It takes about 300 years (15 generations) from the start of the growth period until economic growth settles at the rate of technological progress. Once the signalling allele moves to fixation in the population, technological progress becomes the sole source of output growth as there are no new entrants into the labour force.

Figure 5.5: Growth rate of technology and output



The combination of technological progress in the luxury sector and an increase in labour participation by the high-quality signalling type cause a material increase in the size of their signal (as measured in units of output). Once the signalling allele has moved to fixation, the growth in conspicuous consumption mirrors technological progress due to the wage in the luxury sector being a function of technology [as in equation (5.7)].

The take-off in economic activity is robust to changes in parameter values, and is largely a function of the two mating periods per mating season. More than two mating periods in a season would increase the rate of evolutionary change because it would give signalling males more opportunities to mate. Sensitivity testing of parameters relating to the relative fertility of low and high quality types and the prevalence of high-quality males is contained in the Appendix. Adjustment of other parameters makes it possible to achieve any rate of technological progress and output growth per generation in equilibrium. As we do not propose that the evolution of the propensity to engage in

conspicuous consumption is the sole factor behind increasing economic growth, we did not calibrate the rate of technological progress and income growth.

5.4 Model with coevolution of male and female preferences

This section describes a model in which both male and female behaviour is determined genetically. Depending on their type, female agents observe the male signal and use that information to infer quality, or they ignore the signal. As a result, male and female signalling and observing behaviour must co-evolve, rather than male signalling behaviour exploiting an existing female sensory bias as occurred in the previous model version. Except where noted, the new model retains the features of the first model.

5.4.1 The agents

Each haploid agent has one genetic locus. The allele at the locus expresses signalling and observing strategies for the male and female respectively. The signalling allele is carried and expressed phenotypically in the male only, and the observing allele is present in the female only. Children inherit the allele from their parent of the same sex, such as through a sex chromosome.

The choice of haploid sex-linked genetic loci improves the tractability of the model relative to both sexes having loci for signalling and observing alleles, but ignores potential linkage disequilibrium effects whereby reproductive success associated with one type of allele affects the frequency of another. The decision to use this framework has negligible effects on the model results beyond doubling the rate of evolutionary change compared to a model in which males and females both carry the signalling and observing alleles. This doubling occurs because the alleles are always expressed in one of the sexes, rather than being carried but not under selection in the other sex.

There are two alleles of each gene: signalling and not signalling ($i \in S, N$), and observing and not observing ($j \in S, N$). Males differ in quality as well as signalling behaviour, whereas the observing allele is the only difference among females. Observing females use the male signal to infer quality. The frequency of each male phenotype by quality is denoted by π^{ik} ($i \in S, N$, $k \in H, L$), while for females each genotype is denoted by ϕ^j . π^i denotes the prevalence of males of genotype i of either level of quality.

The utility function of a male is the number of children he fathers. Similarly, the utility function of a female is the number of children she bears, which is equal to the quality of the male with whom she mates, as in equations (5.1) and (5.2).

As before, expressing the utility function as the number of viable children is not an exact statement of the male or female's fitness maximising function. In this model, this distinction is more important, however, as a male cares about the genotype of his children and accordingly, the genotype of the female with whom he mates. While the male mates with any female with whom he is paired and he has no opportunity to control the pairing process, the benefits of mating with an observing female may affect the size of the signal the male is willing to make. This is discussed further below.

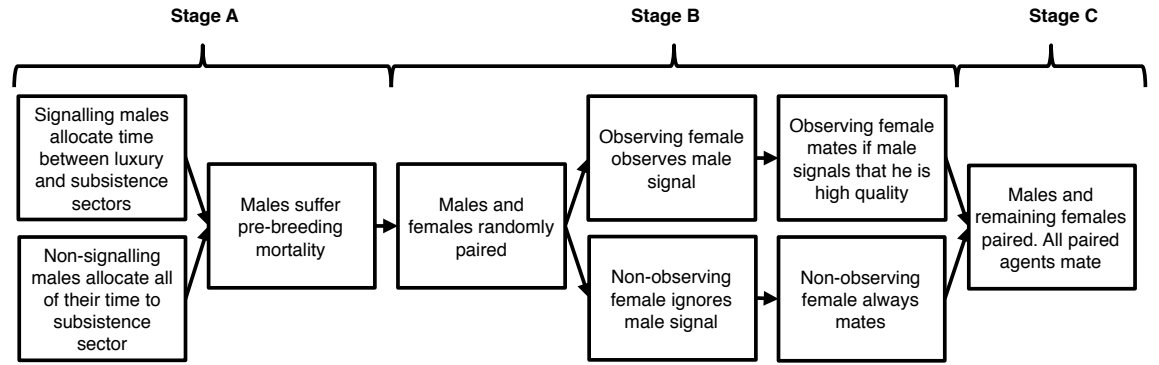
The consequence of this form of the utility function for females is as for the previous model. Female actions in a separating equilibrium are not changed, but the conditions for a separating equilibrium are affected.

5.4.2 The mating season

Figure 5.6 summarises the mating season. The difference to the preceding model arises in stage B where males and females are randomly paired. If a female is paired in stage

B, a female of the observing type uses the male signal to determine whether she will mate with the male. If she does choose to mate, the female exits the mating pool. If she is of the non-observing type, she will mate with the male regardless of his signal or quality. Accordingly, non-observing females are only present in stage C if they are not paired in stage B.

Figure 5.6: The mating season



The number of males and the ratio of types of males do not change between stages B and C ($M_C = M_B$ and $\pi_C^{ik} = \pi_B^{ik}$). The number of females available for mating in stage C decreases by the number who mate in stage B. Females exit the breeding pool if they are an observing type and are matched with a high-quality signalling male, or, if they are not an observing type and are matched at all.

$$F_C = \left[\phi_B^S (1 - q_B^F \pi_B^{SH}) + (1 - \phi_B^S) (1 - q_B^F) \right] F_B \quad (5.34)$$

The proportion of females of each type present in stage C is:

$$\phi_C^S = \phi_B^S (1 - q_B^F \pi_B^{SH}) \frac{F_B}{F_C} \quad (5.35)$$

$$\phi_C^N = (1 - \phi_B^S) (1 - q_B^F) \frac{F_A}{F_C} \quad (5.36)$$

It is possible for males and females to be unmatched in stage C [as in equations (5.15) and (5.16)]. In stage C, males and females will mate with whomever they are matched, as there are no further opportunities to mate. Offspring from the mating in stages B and C form the next generation.

5.4.3 Female optimisation

A female carrying the observing allele will mate in stage B if the benefit of mating exceeds the expected benefit of waiting. The future benefit is a function of the probability that she will be matched to a male in stage C and the expected quality of that male. She will not mate with a non-signalling male she is paired with in stage B if equation (5.17) is satisfied. Equation (5.17) is more likely to be satisfied in this model than that presented in Section 5.3 because paired non-observing females mate and exit the breeding pool in stage B, making it unlikely that there will be excess females in stage C. As in the earlier model, the mating decision of the female is based on the threshold level of conspicuous consumption \bar{c} that is given in equation (5.18).

5.4.4 Male optimisation

The utility functions of signalling and non-signalling males are:

$$u^{Sk} = \delta^{Sk} \left[\left(\theta(c^{ik}) \phi_B^S + \phi_B^N \right) q_B^M + q_C^M \right] n(h^k) \quad (5.37)$$

$$u^{Nk} = \delta^{Sk} \left(\phi_B^N q_B^M + q_C^M \right) n(h^k) \quad (5.38)$$

Substituting equation (5.8) into equations (5.37) and (5.38), males of each genotype face the following optimisation problem:

$$\{s^{Sk}\} = \operatorname{argmax} \left\{ \delta^{Sk} \left[\left(\theta \left(wh^k (1 - s^{Sk}) \right) \right) \phi_B^S + \phi_B^N \right] q_B^M + q_C^M \right] n(h^k) \right\} \quad (5.39)$$

$$\{s^{Nk}\} = \operatorname{argmax} \left\{ \delta^{Nk} q_C^M n(h^k) \right\} \quad (5.40)$$

If observing females discriminate between signalling and non-signalling males in stage B, high-quality signalling males will signal where the following condition holds:

$$(q_B^M + q_C^M) \delta^{SH} > \left[(1 - \phi_A^S) q_B^M + q_C^M \right] \delta^{NH} \quad (5.41)$$

This condition implies that high-quality signalling males always mate in stage B (as $q_B^M = 1$) and if paired in stage C, while all other male phenotypes mate in stage B if they are paired with a non-choosy female, and if paired in stage C. If most females are not of the observing genotype, a male will be willing to sacrifice only a small probability of survival to signal to females.

If $M_C > F_C$, the condition in equation (5.41) is easier to satisfy than if $M_C < F_C$. This is because a reduced probability of being paired in stage C makes the opportunity to pair in stage B relatively more important. The probability that the condition would be met would also be affected if males pursued true fitness maximising behaviour through maximising children in the long-term. In that case, attracting an observing female has the benefit of both a mating opportunity and the higher fitness associated with the observing allele, making the increased mating opportunity relatively more attractive. As such, males would be willing to give a stronger signal to attract the female, resulting in a larger signal in a separating equilibrium. The larger signal would increase signalling

male mortality, decreasing the likelihood that the condition in equation (5.41) would be met.

The non-signalling and low-quality phenotypes spend all of their time on survival activities (i.e. $s^{SL} = s^{Nk} = 1$).

5.4.5 Offspring

Offspring are born and raised at the end of the mating season. Assuming the signal-observing females accept only the high-quality males who signal, the male and female offspring of each genotype, $n(\pi^i)$ and $n(\phi^j)$, born from mating in stage B are:

$$n(\pi_B^S) = \left[\left(\pi_B^{SH} \phi_B^S n(h^H) \right) + \sum_{k=H,L} \pi_B^{Sk} \phi_B^N n(h^k) \right] M_B \quad (5.42)$$

$$n(\pi_B^N) = \sum_{k=H,L} \pi_B^{Nk} \phi_B^N n(h^k) M_B \quad (5.43)$$

$$n(\phi_B^S) = \pi_B^{SH} \phi_B^S n(h^H) M_B \quad (5.44)$$

$$n(\phi_B^N) = \sum_{i=S,N} \sum_{k=H,L} \pi_B^{ik} \phi_B^N n(h^k) M_B \quad (5.45)$$

The offspring born because of mating in stage C are:

$$n(\pi_C^i) = \sum_{k=H,L} \pi_B^{ik} n(h^k) \min\{M_C, F_C\} \quad (5.46)$$

$$n(\phi_C^j) = \sum_{i=S,N} \sum_{k=H,L} \pi_B^{ik} \phi_C^j n(h^k) \min\{M_C, F_C\} \quad (5.47)$$

The prevalence of the signalling allele in generation $t+1$ is:

$$\begin{aligned}\pi^S(t+1) &= \frac{n(\pi_B^S) + n(\pi_C^S)}{\sum_{i=S,N} [n(\pi_B^i) + n(\pi_C^i)]} \\ &= \pi(p, \pi_A^i(t), \phi_A^j(t), h^L, h^H)\end{aligned}\quad (5.48)$$

The prevalence of the signalling type in the population increases if $\pi_A^S(t+1) > \pi_A^S(t)$.

Using this condition, equation (5.48) can be used to derive the condition for the male deciding to signal, which was shown in equation (5.41). The prevalence of the signalling allele increases in the population if that condition is met.

The prevalence of the observing allele in generation $t+1$ is:

$$\begin{aligned}\phi^S(t+1) &= \frac{n(\phi_B^S) + n(\phi_C^S)}{\sum_{j=S,N} (n(\phi_B^j) + n(\phi_C^j))} \\ &= \phi(p, \pi_A^S(t), \phi_A^S(t), h^k, h^H)\end{aligned}\quad (5.49)$$

The prevalence of the observing type in the population increases if $\phi_A^S(t+1) > \phi_A^S(t)$.

Accordingly, the prevalence of the observing allele increases if the condition given in equation (5.50), derived from equation (5.49), is satisfied.

$$\frac{p(1-\pi_A^S)n(h^H) + (1-p)n(h^L)\delta^{NL}}{1-\pi_B^{SH}} < q_C^F \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik} \delta^{ik} n(h^k) \quad (5.50)$$

This condition, which is the same as that in equation (5.17), shows that the frequency of the observing allele will increase if a female, who postpones mating until stage C, bears more children than if she mates in stage B without paying attention to male signals. If $M_C > F_C$, this condition is always satisfied as there is no cost to females for rejecting

males in stage B. If $M_C < F_C$, the additional probability of mating with a high-quality male must outweigh the cost of not being paired in stage C.

The signalling equilibrium is similar to that in the first model. As before, the optimal level of conspicuous consumption by a high-quality male is the level that a low-quality male is indifferent about making. However, as many of the females in the population do not respond to signals, the benefits to signalling for a male are initially lower, given the high probability that he will be paired with a female who does not respond to his signal. Accordingly, high-quality signalling males tend to signal through a lower level of conspicuous consumption until the prevalence of observing females increases.

We can use equation (5.41) to determine the proportion of time a high-quality male will allocate to subsistence activities. With \hat{s} the level of subsistence that a low-quality male is indifferent between signalling or not, we can derive the following equation:

$$\delta(r\hat{s}) = \frac{(1 - \phi_A^S + q_C^M)\delta(r)}{1 + q_C^M} \quad (5.51)$$

Again, the level of signalling by the high-quality signalling male, s^{SH} , can be derived from equation (5.51) using equation (5.31). Specification of a function for δ will allow for the relationship between \hat{s} and s^{SH} to be solved.

To determine the probability that a male is being paired in stage C, q_C^M , we must consider whether there will be more males or females. As such, we will have to solve equation (5.52) in iteration with equation (5.51). $q_C^M < 1$ if:

$$(2 - \phi_B^S) \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik} \delta^{ik} > 1 - \phi_B^S \pi_A^{SH} \delta^{SH} \quad (5.52)$$

As noted before, the size of the signal may be larger if the male maximised fitness rather than his genetic contribution to the next generation only. However, as the male is willing to mate with any female with whom he is paired and he has no opportunity to control the pairing process, the male's actions after setting the signal are not affected.

5.4.6 Model dynamics

When equations (5.17) and (5.41) are satisfied, there will be a separating equilibrium where high-quality males signal in stage B and receiving females consent only if paired with one of those high-quality signalling males. In a separating equilibrium, the percentage point change in frequency of the signalling allele per generation is:

$$\Delta\pi^S = \frac{p\pi_A^S(1-\pi_A^S)\left[(1+q_C^M)\delta^{SH} - (\phi_A^N + q_C^M)\delta^{NH}\right]n(h^H)}{\pi_A^{SH}\delta^{SH}\phi_A^S n(h^H) + (\phi_A^N + q_C^M) \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik}\delta^{ik} n(h^k)} \quad (5.53)$$

The effect of the variables on the rate of change in prevalence of the signalling allele is similar to that in the first model. The major difference is the effect of the frequency of the observing allele in the population. If the observing allele is at 100 per cent frequency ($\phi_A^S = 1$, $\phi_A^N = 0$), equation (5.53) equals equation (5.33) in the first model. At lower frequencies of the observing allele, non-signallers have greater reproductive success in stage B, and selection on the signalling allele is therefore weaker.

The percentage point change in frequency of the observing allele per generation is:

$$\Delta\phi^S = \frac{\phi_A^S(1-\phi_A^S) \left[q_C^F \left(1 - p\pi_A^S \delta^{SH} \frac{M_A}{M_B} \right) \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik}\delta^{ik} n(h^k) - \left(p(1-\pi_A^S)n(h^H) + (1-p)n(h^L) \right) \delta^{NL} \right]}{\pi_A^{SH}\delta^{SH}\phi_A^S n(h^H) + (\phi_A^N + q_C^M) \sum_{i=S,N} \sum_{k=H,L} \pi_A^{ik}\delta^{ik} n(h^k)} \quad (5.54)$$

The rate of change of the prevalence of the observing allele is greatest at intermediate frequencies due to the term $\phi_A^S(1 - \phi_A^S)$. The rate of change also increases with the prevalence of the signalling allele and the proportion of males who pair in the final period. An increase in the prevalence of high-quality males in the population reduces the spread of the observing allele as it increases the expected quality of males without signals in stage B by more than the expected quality of all males in stage C. The term in brackets in the numerator is equivalent to the condition for the increase in the observing allele shown in equation (5.50).

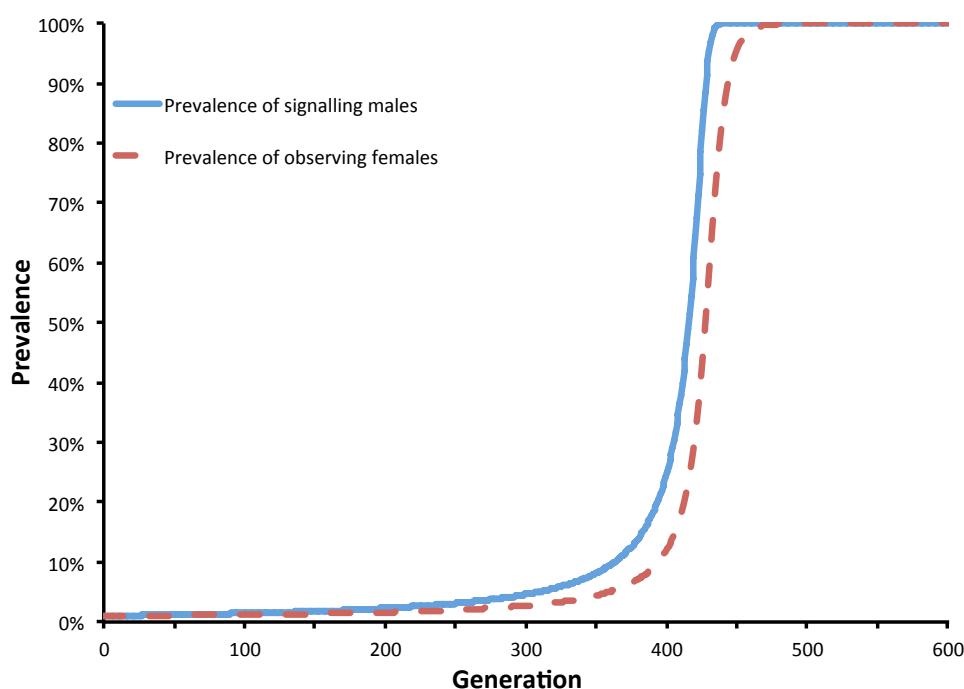
5.4.7 Simulation

We simulate the model using the same functional forms and parameter values as we used in the first model, except for the initial prevalence of the signalling and observing alleles in the population, which is one per cent. This is more than for the signalling allele in the first model as the signalling and observing types have a fitness advantage only when they are paired. At lower prevalence, it takes thousands of additional generations to reach material frequencies. In an evolutionary scenario, the relatively higher initial prevalence of each type may be achieved by collocation of mutations in a geographically or socially isolated population.

Figure 5.7 shows the evolution of the signalling and observing alleles in the population. The increase in prevalence of the signalling allele is significantly slower than that in the first model in which all females have a genetic predisposition to observe male conspicuous consumption. It takes approximately 170 generations for the prevalence of the signalling allele and almost 250 generations for the prevalence of the observing allele to double to two per cent. Over 350 generations have passed before the prevalence of either reaches 10 per cent, but then the spread of the alleles increases markedly and

over the next 120 generations, each allele increases to a prevalence of over 99 per cent. The prevalence of observing females increases with the proportion of signalling males. This makes the signalling or observing behaviour more rewarding, meaning that the strength of selection on each allele increases as the prevalence of the other increases.

Figure 5.7: Prevalence of signalling and observing alleles



The prevalence of signalling males affects the economy through increased labour force participation (Figure 5.8) and increased technological progress through the scale effect (Figure 5.9). Labour force participation, even for high-quality, signalling males, is initially low due to the low benefits to signalling as there are few observing females. It is only when the prevalence of observing females increases that significant time is allocated to labour in the luxury sector. Total labour force participation increases due to both increasing participation by signalling males and their increasing prevalence.

Figure 5.8: Labour force participation

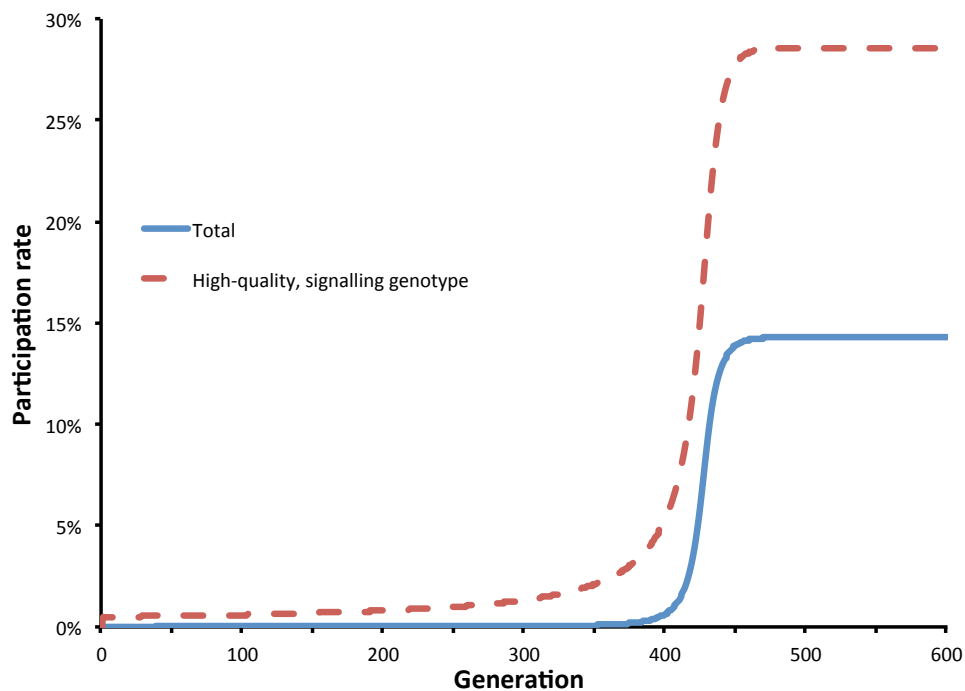
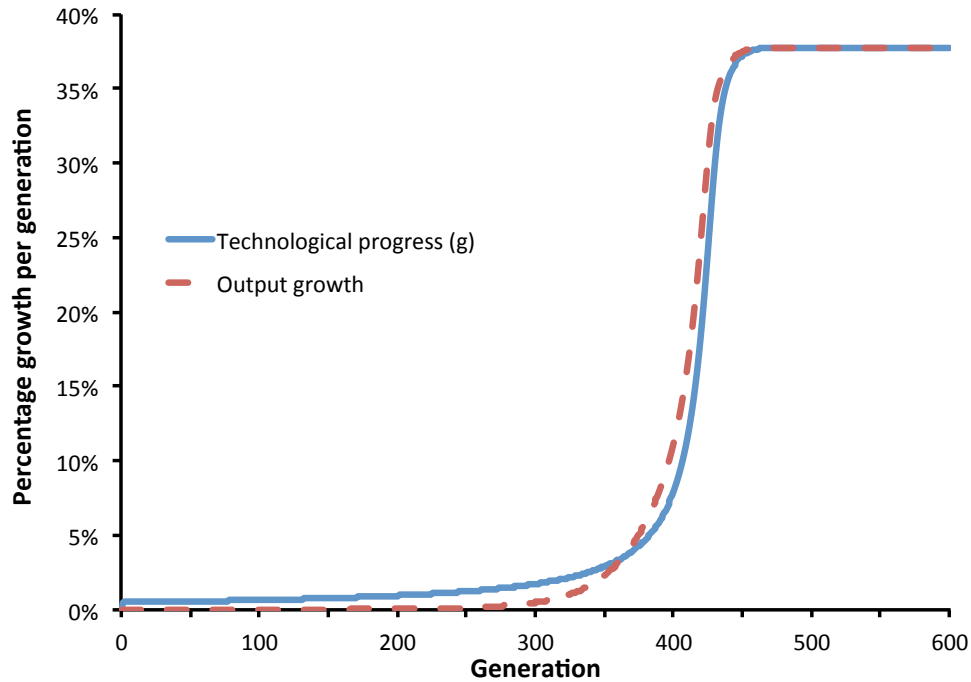


Figure 5.9 shows the growth paths in technology and output. Initially, the rate of technological progress exceeds output growth due to the low proportion of the population that is engaged in the luxury sector. It is only when there is significant use of the growing technological base by a greater proportion of the population that output growth takes off. It takes about 150 generations from the start of the growth period until economic growth settles at the rate of technological progress. Once the signalling allele moves to fixation in the population, technological progress becomes the sole source of income growth, as there are no new entrants into the labour force.

Figure 5.9: Growth rate of technology and output



Conspicuous consumption by high-quality signalling males remains low until the prevalence of the observing allele reaches material levels and then increases markedly. Once the signalling allele moves to fixation and there is no further increase in labour force participation, conspicuous consumption grows at the rate of technological progress.

Sensitivity testing of parameters relating to the relative fertility of low and high quality types and the prevalence of high-quality males is contained in the Appendix. As before, adjustment of other parameters makes it possible to set any level of output and technological growth per generation in equilibrium.

5.5 Discussion

The two models presented in this paper provide a basis for the hypothesis that sexual selection was a contributing factor to the emergence of modern levels of economic growth. As females prefer males who conspicuously consume, males are under selection

to increase their allocation of time to innovation, labour and other productive activities in order to engage in conspicuous consumption. These activities contribute to technological progress and economic growth.

The two models differ in the speed at which the conspicuous consumption behaviour spreads through the population. The first model produces significant changes in economic behaviour within one to two hundred years. The second model gives rise to longer-term trend in economic growth, which, when combined with population growth, would not result in observable per capita income changes for long periods. Positive responses to modern consumer goods, to which females have had no exposure in our evolutionary history, suggest significant flexibility over short timeframes in female perception of what is a reliable signal. Conversely, anthropological evidence of the ubiquity of conspicuous consumption in human society suggest a deep evolutionary basis to this trait, which may have evolved over significant time (Sundie et al. 2011).

The core condition for an honest signal such as conspicuous consumption is different costs (or benefits) of signalling between high and low-quality signallers. In our model, the difference in costs to signalling arises from the difference in wages that each high and low-quality male can earn in the luxury sector of the economy for purchasing conspicuous consumption goods. Even if there was no such wage difference, the necessary condition for existence of a handicap as an honest signal could have been met through alternative means. The models could be reframed so that high and low-quality males face different costs of decreased investment into subsistence activities, with the survival probability for low-quality males declining faster. The increased survival cost faced by a low-quality male would allow a separating equilibrium to exist. If we assumed that the “quality” trait affected multiple outcomes, including survival

probability and labour efficiency, the condition for the handicap would be met in multiple dimensions.

In both models, sexual selection does not affect the quality of the agents. Quality is allocated randomly at birth, which made the model tractable for an analysis of the handicap principle. If quality were heritable, selection of high-quality individuals would tend to drive the genes associated with high quality to fixation, at which point female choice would become obsolete. A more realistic but complicated scenario would be to introduce multiple genes associated with quality and allow selection and mutation of these genes to occur. This would allow female choice to remain important, while allowing qualitative population changes to occur. We consider that this scenario would be more representative of the human evolutionary history, with the propensity for conspicuous consumption and qualitative traits both being selected for in the population.

In addition to being a signal of quality, conspicuous consumption may be a signal of accumulated resources, which are likely to be of value to a female. Female interest in resource accumulation is likely to play a significant role in the evolution of a preference for conspicuous consumption. In the models in this paper, agents do not accumulate resources as there is no capital and there is no transmission of resources from males to females. The ability to accumulate resources may change the inherent trade-offs between quality and signalling ability, particularly if resources can be transmitted to children.

If capital accumulation were incorporated into the model, the effect of conspicuous consumption on capital accumulation may create an ambiguous effect on growth, particularly in modern economies. The lower savings rate implied by higher

conspicuous consumption may reduce long-term growth as there is less accumulated capital for productive uses. However, capital investment is itself likely to be driven by the desire for conspicuous consumption, which is increased over the long-term through the return on that capital. The preference for conspicuous consumption provides an incentive to invest in resources that will income in excess of that required for survival.

One omission from our models is the positive effect on survival of the activities undertaken to support conspicuous consumption. The labour and innovation of previous centuries has not only improved the methods to acquire resources for conspicuous consumption, but has also affected basic survival probability. In advanced economies, survival to adulthood is likely with probability above 99 per cent (Department of Economic and Social Affairs 2011). Once conspicuous consumption imposes no cost on the individual, it loses its reliability as a signal. Accordingly, we might expect the nature of conspicuous consumption to change in modern contexts, or for alternative methods of signalling to emerge. However, if the preference for conspicuous consumption is a heritable trait, females may still prefer conspicuous consumption despite its diminished usefulness as a signal. This trait would persist in the population until the preference for signals that are more accurate spread.

This is not to say, however, that conspicuous consumption can have no survival cost today. Conspicuous consumption also occurs in poor societies, often at significant cost to the signallers. Moav and Neeman (2012) theorised that conspicuous consumption is more prevalent in poor societies than in societies with higher income, as in advanced economics people can signal through career, qualifications or other costly methods of demonstrating quality.

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Chapter 5 Appendix

5A.1 Sensitivity testing: Model with evolution of male preference

As noted in the discussion of equation (5.33), the spread of the signalling allele is accelerated where there is a greater difference between the fertility of the high and low-quality types and where the prevalence of the high-quality males is greater. Providing a sensitivity analysis, Table 5.3 displays the number of generations for the prevalence of the signalling allele to increase from one per cent to 99 per cent for different fertility ratios and prevalence of high-quality males. It is only with low differences in fertility between low and high-quality males and a low prevalence of high-quality males that the spread of the signalling allele is materially slowed.

Table 5.3: Sensitivity testing (generations for spread from 1% to 99% prevalence)

		Low-quality to high-quality fertility ratio					
		0.1	0.2	0.4	0.6	0.8	0.9
Proportion males of high quality	0.1	23	36	73	149	378	837
	0.2	18	24	42	80	194	424
	0.4	14	17	27	45	102	217
	0.6	13	15	21	33	71	148
	0.8	11	13	18	27	56	113
	0.9	11	12	17	25	51	102

5A.2 Sensitivity testing: Model with coevolution of male and female preference

The take-off in economic activity is robust to changes in parameter values. Table 5.4 shows the number of generations for the prevalence of the signalling and observing allele to increase from one per cent to 99 per cent prevalence for different fertility ratios between low and high-quality males and prevalence of high-quality males. The first number in each cell is for the signalling allele, the second for the observing allele. Increasing the fertility ratio speeds up the spread of both alleles, reducing the number of

generations until they dominate the population. Unlike the first model, the spread is fastest for intermediate numbers of high-quality males, which is a result of the opposing effects of the prevalence of high-quality males on selection for the signalling and observing alleles and the interaction between those two alleles.

Table 5.4: Sensitivity testing (generations for spread from 1% to 99% prevalence)

		Low-quality to high-quality fertility ratio					
		0.1	0.2	0.4	0.6	0.8	0.9
Proportion males of high quality	0.1	235 / 243	385 / 399	837 / 868	1,712 / 1,801	4,234 / 4,522	8,873 / 9,618
	0.2	185 / 192	264 / 275	502 / 525	968 / 1,020	2,306 / 2,466	4,769 / 5,175
	0.4	175 / 183	220 / 231	357 / 376	624 / 662	1,394 / 1,499	2,815 / 3,070
	0.6	194 / 207	231 / 246	340 / 364	555 / 597	1,174 / 1,279	2,320 / 2,560
	0.8	247 / 275	284 / 316	393 / 439	609 / 681	1,233 / 1,393	2,393 / 2,743
	0.9	314 / 376	356 / 426	482 / 578	731 / 879	1,454 / 1,764	2,808 / 3,455

5A.3 Simulation notes

5A.3.1 Model with evolution of male preference

To determine the level of signalling by the high-quality male, solve equation (5.30).

Setting $\delta(z^{ik}, \bar{z}) = z^{ik} / \bar{z}$, $\bar{z} = 1$ and the initial population $M(t)=1$ such that that $z^{ik} < 1$ when an agent invests in the luxury sector, when $M_C < F_C$ equation (5.30) becomes:

$$\hat{s} = \frac{1}{2} \quad (5.55)$$

Substitute equation (5.55) into equation (5.31):

$$s^{SH} = 1 - \frac{1}{2} \frac{h^L}{h^H} \quad (5.56)$$

If $M_C > F_C$, equation (5.30) becomes:

$$\hat{s} = \frac{1 - \pi_A^{SH} s^{SH}}{2 - \pi_A^{SH}} \quad (5.57)$$

Substitute equation (5.31) into equation (5.57):

$$\hat{s} = \frac{1 - \pi_A^{SH} \left(1 - \frac{h^L}{h^H}\right)}{2 - \pi_A^{SH} \left(1 - \frac{h^L}{h^H}\right)} \quad (5.58)$$

Using equations (5.31) and (5.58):

$$s^{SH} = \frac{2 - \pi_A^{SH} \left(1 - \frac{h^L}{h^H}\right) - \frac{h^L}{h^H}}{2 - \pi_A^{SH} \left(1 - \frac{h^L}{h^H}\right)} \quad (5.59)$$

To determine whether M_C is greater than F_C , the agent will need to check whether equation (5.32) holds. $M_C < F_C$ where $s^{SH} < 1/2$.

With $\delta(z^{ik}, \bar{z}) = z^{ik} / \bar{z}$ and $\bar{z} = 1$, the signalling level determined from equation (5.56) is always such that \hat{s} equals one half and s^{SH} is higher, meaning that M_C will always exceed F_C . Hence, equation (5.56) must be used to determine the level of signalling by the high-quality, signalling male.

5A.3.2 Model with coevolution of male and female preference

When $M_C < F_C$, using equation (5.51) and setting $\delta(z^{ik}, \bar{z}) = z^{ik}/\bar{z}$, $\bar{z} = 1$ and the initial population $M(t)=1$, gives:

$$\hat{s} = \frac{\phi_A^N + 1}{2} \quad (5.60)$$

To solve for s^{SH} , substitute equation (5.60) into equation (5.31):

$$s^{SH} = 1 - \left(\frac{\phi_A^S}{2} \right) \frac{h^L}{h^H} \quad (5.61)$$

When $M_C > F_C$, from equation (5.51) and $\delta(rs) = s$:

$$\begin{aligned} \hat{s} &= \frac{\phi_A^N + q_C^M}{1 + q_C^M} \\ &= \frac{1 - \phi_A^S \pi_A^{SH} s^{SH}}{1 + \phi_A^S (\pi_A^{SL} + \pi_A^{NH} + \pi_A^{NL})} \end{aligned} \quad (5.62)$$

Substitute equation (5.31) into equation (5.62):

$$\hat{s} = \frac{1 - \phi_A^S \pi_A^{SH} \left(1 - \frac{h^L}{h^H} \right)}{1 + \phi_A^S \pi_A^{SH} \left(1 + \frac{h^L}{h^H} \right)} \quad (5.63)$$

Using equations (5.31) and (5.63):

$$s^{SH} = \frac{1 + \phi_A^S (1 - \pi_A^{SH}) \left(1 - \frac{h^L}{h^H} \right)}{1 + \phi_A^S \left[1 - \pi_A^{SH} \left(1 - \frac{h^L}{h^H} \right) \right]} \quad (5.64)$$

To determine whether M_C is greater than F_C , we check whether equation (5.52) holds and solve in iteration with equations (5.61) and (5.64). $M_C < F_C$ where:

$$s^{SH} < \frac{\pi_A^{SH} - \phi_A^N (1 - \pi_A^{SH})}{2\pi_A^{SH}} \quad (5.65)$$

CHAPTER 6

EVOLUTION, FERTILITY AND THE AGEING POPULATION

6.1 Introduction

Demographic change is projected to present fiscal and economic challenges to governments in forthcoming decades, particularly in developed countries. Population ageing caused by reduced fertility and increasing life expectancy has been predicted to increase the demands placed on pension systems and aged-care services at a time when the relative tax base is forecast to be declining [for example, Lee & Mason (2011)]. This twin assault on government finances has led developed world governments to expect deterioration in their fiscal positions.

While a range of assumptions underpins these projections, the focus of this paper is on the fertility rate. Fertility has been declining in most developed countries since the demographic transition in the late nineteenth century. After the intervening post-war baby boom in the 1950s and early 1960s, fertility resumed its decline before rebounding slightly in recent years. The total fertility rate in developed countries dropped from 2.81 in 1950-55 to 1.56 in 1995-2000, before recovering to 1.66 in 2005-10 (United Nations 2013). While some of this recovery from recent fertility lows may be attributable to the postponement to childbearing, fertility rate measures adjusted for the timing of births have also increased in many countries (Goldstein et al. 2009). Further, the recent increase in fertility in developed countries does not depend on immigration. It is present in countries with few immigrants, such as Japan (United Nations 2013), and is typically exhibited amongst native born populations once immigration is excluded (Goldstein et al. 2009; Tromans et al. 2009).

In view of the recent recovery in fertility, many population forecasts have been revised but few project a return of fertility to the replacement level of about 2.1 children per woman. For example, the United Nations (2013) predicts that the fertility rate in developed countries will increase to 1.85 by 2045-50 and 1.93 by 2095-2100 in its medium fertility scenario.

Evolutionary theory suggests that fertility will bounce back after an environmental shock. Since the beginning of the modern growth era, shocks such as changes in income, contraception and female roles have changed the composition of fertility-related traits under selection and temporarily raised the proportion of variability in fertility in the population that can be attributed to genetic variation. That is, the shocks have increased the heritability of fertility. Following this increase, the prevalence of fertility-related genes is subject to evolution by natural selection. Those genes associated with higher fertility have a selective advantage and thereby increase their frequency, which increases the fertility rate over time. As a result, heritable traits and behaviours that lead individuals to have few or no children eventually disappear from the population.

Adopting an evolutionary perspective, we propose that the recent rise in the fertility rate may be the beginning of a sustained increase in developed world fertility towards above-replacement levels. Using quantitative and population genetic models, we model the evolution of the fertility rate, population size and population structure following an environmental shock that reduces fertility. We show that even with relatively low levels of genetically based variation in fertility, there is a return to a high-fertility state, with recovery to above replacement levels usually occurring within a few generations.

Our hypothesis is relevant for the design of pension systems, aged care and health services. The postulated increase in fertility would reduce the fiscal burden to the extent that it reduces the proportion of elderly in the population. However, a rise in the fertility rate will also increase population growth and the proportion of dependent young, presenting new fiscal and economic policy challenges.

6.2 Heritability of fertility

Selection for high fertility requires variation in fertility in the population and a genetic basis for the intergenerational transmission of this variation. Fisher's (1930) fundamental theorem of natural selection states that the "rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time."⁴⁷ Fisher's theorem implies that variation in traits in a population can only persist where they have a weak relationship with fitness. If there were a strong relationship with fitness, those traits associated with higher fitness would rapidly outcompete traits with lower fitness. This implication has often led researchers to assume that the heritability of fertility, a primary factor affecting fitness, would effectively be zero. Accordingly, any variation in fertility would be due to environment. Consistent with this, Murphy (1999) has shown that the relationship between the fertility of parents and children was close to zero in pre-twentieth century populations that had not undergone a demographic transition.

The trade-off between quantity and quality of children is a driver of the low heritability of fertility in Malthusian environments. Since the analysis of Lack (1947), it has been recognised that maximisation of offspring numbers is generally not a fitness maximising strategy, as resources used in the production of more offspring cannot be used to

⁴⁷ Fitness is defined as the average contribution of a genotype to the gene pool in the next generation.

provision existing offspring. Examination of pre-industrial societies using a life-history framework has confirmed that humans face a quantity-quality trade-off for children (Hill and Kaplan 1999; Borgerhoff Mulder 2000; Walker et al. 2008). Hence, genetically based variation from this optimal number and quality of offspring would reduce fitness and eliminate that variation from the population.

However, as Fisher (1930) recognised, genetically based variation in traits that affect fitness may persist in the population for extended periods if there are large and continuous perturbations in the environment. Environmental change can alter the selective pressure upon a genotype. Neutral genes that were not under selection (or already in their steady-state equilibrium) may suddenly become decisive factors as to whether an organism survives or reproduces in the new environment. For example, an ancestral gene relating to salt retention had an allele (variant of a gene) that did not increase salt retention relative to the common allele that did. When humans moved out of Africa into cooler latitudes where salt retention was costly, the previously rare allele spread rapidly (Thompson et al. 2004; Cochran and Harpending 2009).

In a fertility context, any shock that changes the nature of the quantity-quality trade-off of children would be expected to change the fitness associated with fertility decisions. Life history analysis generally concludes that modern fertility behaviour deviates from fitness maximisation (Kaplan et al. 1995; Kaplan 1996; Kaplan and Lancaster 2000). This provides a basis for higher fitness for those whose fertility behaviour is fitness maximising and accordingly, for fertility behaviour to be heritable.

A range of environmental shocks may have affected the heritability of fertility: increased entry of women into the workforce (Becker 1960; Mincer 1963; Galor and Weil 1996); preference for decreased quantity and increased quality of children in

response to increased income (Becker and Lewis 1974); the development of capital markets to provide alternative sources of old-age security (Neher 1971); substitution from quantity to quality of children in response to increased technological progress and changing returns to human capital (Galor and Weil 2000; Galor and Moav 2002); decreased child mortality (Preston 1978); and a series of contraceptive devices of increasing effectiveness and ease of use (Becker 1960). In this paper, we deal with a generic shock to fertility as for the purposes of our hypothesis it is sufficient that that variation in fertility is heritable after the decline.

There is substantial empirical evidence that the heritability of fertility has increased after the demographic transition. Fisher (1930) analysed the number of children by women as a function of the number of children born to their mothers. He found that a woman could expect 0.21 additional children for each additional child that her mother had and 0.11 additional children for each additional child that her grandmother had. Since the second-generation effect was one half of the first-generation effect, Fisher suggested that the correlation between mother and child must be almost exclusively due to “organic” inheritance. Using those estimates, he concluded that heritability of fertility at that time was 0.4 (40 per cent of the variation in fertility is explained by genetic factors).

Recent studies support the view that fertility has become heritable and is transmitted between generations. Murphy (1999) found that while there was almost no relationship between the fertility of parents and children before the demographic transition, the heritability of fertility averaged around 0.2 in post-demographic transition societies, with the estimates increasing in recent periods. Kohler et al. (1999) examined data

obtained for Danish twins for the periods 1870 to 1910 and 1953 to 1964.⁴⁸ The heritability of fertility varied from close to zero in the pre-transition period to approximately 0.4 to 0.5 during the demographic transition, and it remained strong for the 1953 to 1964 cohort. The continuing high heritability of fertility is suggestive of multiple shocks, or of shocks hitting different groups within the population at different times.

Rodgers et al. (2001b), who analysed data for Danish twins from the 1950s, attribute one quarter of the variation in fertility to genetic factors. In a review of recent literature, Rodgers et al. (2001a) concluded “Fertility differentials are genetically influenced, and at least part of the influence derives from behavioural precursors that are under volitional control, which are themselves genetically mediated.” Using another Danish database, Murphy and Knudsen (2002) found strong intergenerational correlation between fertility of parents and children. They observed “Those from larger families not only provide a disproportionate contribution to the next generation, they also have an even greater impact on numbers of more distant kin”.

Despite growing research into and evidence of the heritability of fertility, there has been limited research into its consequences. Fisher (1930) noted that a higher fertility genotype would eventually dominate the population if fertility had a heritability greater than zero. Based on a fertility ratio of two to one between the more and less fertile genotypes (which was in line with the data Fisher examined), he demonstrated a

⁴⁸ Twin studies are used to provide an estimate of heritability, and suggest that the reported correlations between parent and child are not solely due to vertical cultural transmission or the transmission of resources.

population consisting of 97 per cent of the less fertile genotype and three per cent of the more fertile one would have equal prevalence of each within five generations.⁴⁹

Foster (2000) proposed that the inherited “need to nurture” is sufficiently strong that the majority of women will want to bear at least one child. She concluded fertility has likely approached the limits of its decline. Murphy and Wang (2002) developed a model of population dynamics based on England and Wales between 1850 and 2050. Even with low intergenerational inheritance of fertility, the simulations reveal the 2050 population would be 2.5 times larger than where there is no inheritance. The authors suggest the heritability of fertility has played an important role in supporting developed world fertility rates, low as they are. Rowthorn (2011) showed if religiosity is heritable [for which there is substantial evidence (Koenig et al. 2005)], the higher fertility of more religious individuals will cause the religiosity allele to spread in the population, thereby boosting fertility and population levels.

6.3 Three models of fertility

This section sets out three models of fertility: a diploid model, a model with varying heritability, and an age-structured haploid model. Humans are diploid organisms with two sets of chromosomes, one from each parent. In contrast, a haploid organism

⁴⁹ There is considerable evidence for rapid evolutionary change in humans. For example, agriculture resulted in the spread of alleles related to lactose and gluten tolerance and malaria resistance (Voight et al. 2006). Genomic evidence also suggests that human evolution has accelerated (Hawks et al. 2007).

Stephens et al. (1998) noted the increase in prevalence of the CCR5-Δ32 AIDS resistance gene from one in 10,000 of the population to one in 10 within 700 years required a selection coefficient – a measure the proportional amount that a phenotype is less fit than the phenotype with the highest fitness - of 28 per cent (dominance) or 37 per cent (additive). They hypothesised the gene was associated with resistance to the medieval plague. Similarly, Mead et al. (2009) found rapid spread during the twentieth century of an allele that provided immunity to kuru in Papua New Guinea, with selection coefficients around 30 per cent and as high as 64 per cent in some communities. Relating to fertility, Milot et al. (2011) observed that in a frontier French-Canadian population, the average age of first reproduction dropped from 26 to 22 over 140 years. As the age of first reproduction is heritable, the reduction was evidence of selection acting on an existing trait.

possesses only one set of chromosomes. As a diploid approach is unwieldy, we take a haploid approach in the age-structured model. This abstraction is common in the study of the evolution of social behaviour in humans, with behaviour usually based on observable phenotypes (Grafen 1991). This approach avoids complications in genetic inheritance, such as diploid reproduction, multi-gene traits, interactions between genes and phenotypic expression. Thus, the allele for high or low fertility in the age-structured model is effectively a phenotypic character transmitted from parent to child. The second model, which features varying heritability, does not distinguish between diploid and haploid organisms.

6.3.1 A diploid model of human fertility

The population consists of male and female agents who live for one period in which they pair and have children. The children, who inherit their genes from their parents, form the population in the next generation. There are equal numbers of males and females born in each generation.

As agents are diploid, males and females carry two genes whose alleles represent high and low fertility ($i = H, L$). This results in three potential genotypes in the population: HH , HL and LL . Each child receives one allele from each parent, with the particular allele from each parent contributed with a 50 per cent probability. Female fertility is determined by her genotype. The genotype of the male does not influence the fertility of his female partner, but influences the genotype of his children.

A single environmental fertility shock takes place at time t_0 . Before t_0 , all females have the same fertility, with n^{ii} being the number of children born by females of genotype ii . As fertility is perfectly correlated with fitness in this model, each genotype has the same

fitness before t_0 and any change in prevalence of the fertility alleles would be the result of random sampling between generations.

$$n_t^{HH} = n_t^{HL} = n_t^{LL} \quad t < t_0 \quad (6.1)$$

Following the fertility shock, the fertility of genotype LL decreases, while the fertility of genotype HH , who is immune to the shock, is unchanged. The fertility of the heterozygote genotype HL is dependent on which allele is dominant. The fertility of each genotype following the shock is:

$$\begin{aligned} n_t^{LL} &= (1-s)n_t^{HH} & 0 \leq s \leq 1 \\ n_t^{HL} &= (1-hs)n_t^{HH} & h \in 0, 1 \end{aligned} \quad t \geq t_0 \quad (6.2)$$

The selection coefficient s indicates the proportional reduction in fertility (and fitness) for genotype LL that is caused by the shock. The dominance coefficient h determines the relative fertility of the female heterozygote. The high-fertility H allele is dominant and the low-fertility L allele recessive when $h = 0$, and vice versa when $h = 1$. Thus, the heterozygote genotype is unaffected by the fertility shock if the high fertility allele is dominant. Equation (6.2) gives the following fertility relation, where at least one of the inequalities is a strict inequality:

$$n_t^{HH} \geq n_t^{HL} \geq n_t^{LL} \quad t \geq t_0 \quad (6.3)$$

The frequency of each allele is represented by π_t^i . As each agent has two alleles, the proportion of each of the three genotypes in the population, π_t^{ii} , is a function of the prevalence of the allele.⁵⁰ The proportion of each genotype in generation t is:

$$\pi_t^{ii} = \frac{X_t^{ii}}{X_t} \quad (6.4)$$

X_t is the total population and X_t^{ii} is the population of each genotype.

If there is random mating, the number of each genotype in generation $t + 1$ is:

$$X_{t+1}^{HH} = \frac{1}{2} \left((\pi_t^{HH})^2 n^{HH} + \pi_t^{HH} \pi_t^{HL} \left(\frac{n^{HH} + n^{HL}}{2} \right) + \frac{1}{4} (\pi_t^{HL})^2 n^{HL} \right) X_t \quad (6.5)$$

$$\begin{aligned} X_{t+1}^{HL} = \frac{1}{2} \left(\pi_t^{HH} \pi_t^{HL} \left(\frac{n^{HH} + n^{HL}}{2} \right) + \pi_t^{HL} \pi_t^{LL} \left(\frac{n^{HH} + n^{LL}}{2} \right) \right. \\ \left. + \frac{1}{2} (\pi_t^{HL})^2 n^{HL} + 2 \pi_t^{HH} \pi_t^{LL} \left(\frac{n^{HH} + n^{LL}}{2} \right) \right) X_t \end{aligned} \quad (6.6)$$

$$X_{t+1}^{LL} = \frac{1}{2} \left((\pi_t^{LL})^2 n^{LL} + \pi_t^{HL} \pi_t^{LL} \left(\frac{n^{HL} + n^{LL}}{2} \right) + \frac{1}{4} (\pi_t^{HL})^2 n^{HL} \right) X_t \quad (6.7)$$

For any selection coefficient greater than zero, the frequency of the high-fertility allele and high-fertility phenotypes will increase. This is the case both when the high-fertility allele is recessive and dominant. Further detail on the rate of increase is provided in the Appendix.

⁵⁰ As there is selection occurring, these are not the Hardy-Weinberg frequencies, although they are a close approximation. The Hardy-Weinberg equilibrium involves constant allele and genotype frequencies:

$$\pi^{HH} = (\pi^H)^2 \quad \pi^{HL} = \pi^H (1 - \pi^H) \quad \pi^{LL} = (1 - \pi^H)^2$$

To illustrate the speed of the return to a high-fertility state, we simulated a population with an initial fertility rate of 3.6, falling to 1.8 after a fertility shock. These fertility rates approximate the baby boom peaks and fertility rate lows experienced in Australia and the United States before the recent rebound in fertility (United Nations 2013), although given that the populations in these countries have been experiencing fertility shocks for 200 years, the use of these fertility rates is illustrative only. The fertility rate of the high-fertility phenotype is unaffected by the shock, whereas the fertility rate of the low-fertility phenotype is determined by the selection coefficient s . The selection coefficient is chosen so that total fertility rate equals 1.8 immediately after the shock. Simulations were conducted for different initial values for the prevalence of the high-fertility phenotype of between one and 40 per cent of the population (see Table 6.1 in the Appendix for the implied fertility of the low-fertility genotype).

When the high-fertility allele is dominant, the high-fertility phenotype (genotypes HH and HL) quickly dominates the population and the total fertility rate rapidly returns to pre-shock levels (Figure 6.1). The long-run fertility rate does not depend on the size of the fertility shock because the low-fertility allele is always selected out of the population. The speed of the return to the pre-shock fertility rate, however, depends on the initial prevalence of the high-fertility phenotype. If the high-fertility phenotype forms only one per cent of the population, fertility increases to above replacement within seven generations and it takes more than 20 generations until fertility returns to previous levels. If it comprises 40 per cent of the population, the first generation following the shock has fertility above replacement and fertility rate approaches previous levels within 10 generations.

Figure 6.1: Fertility rate with dominant high-fertility allele

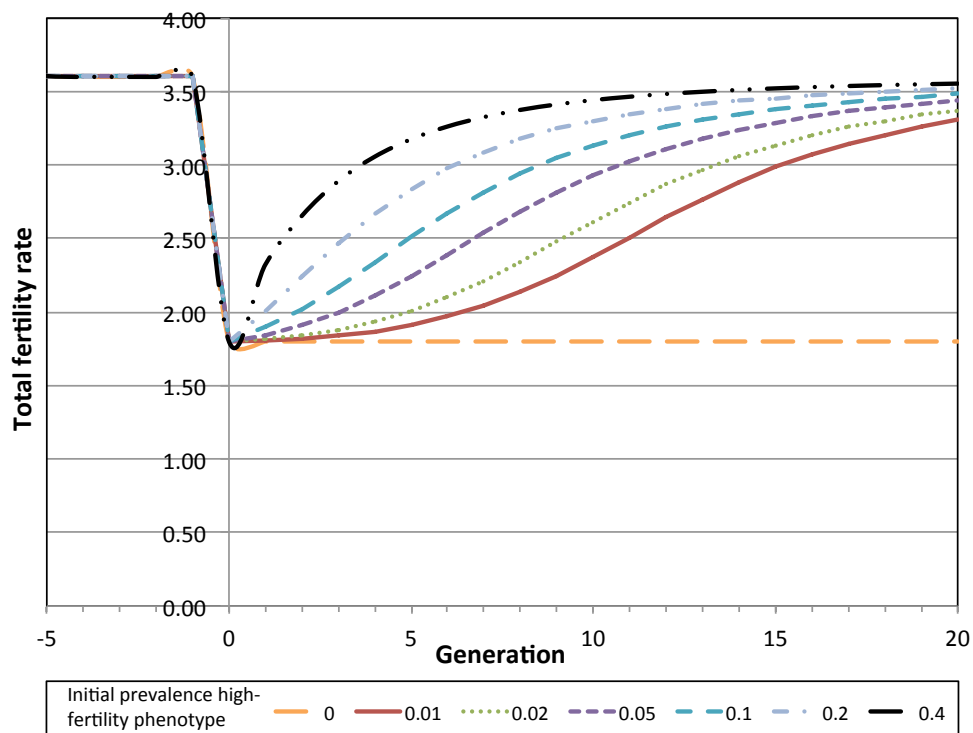
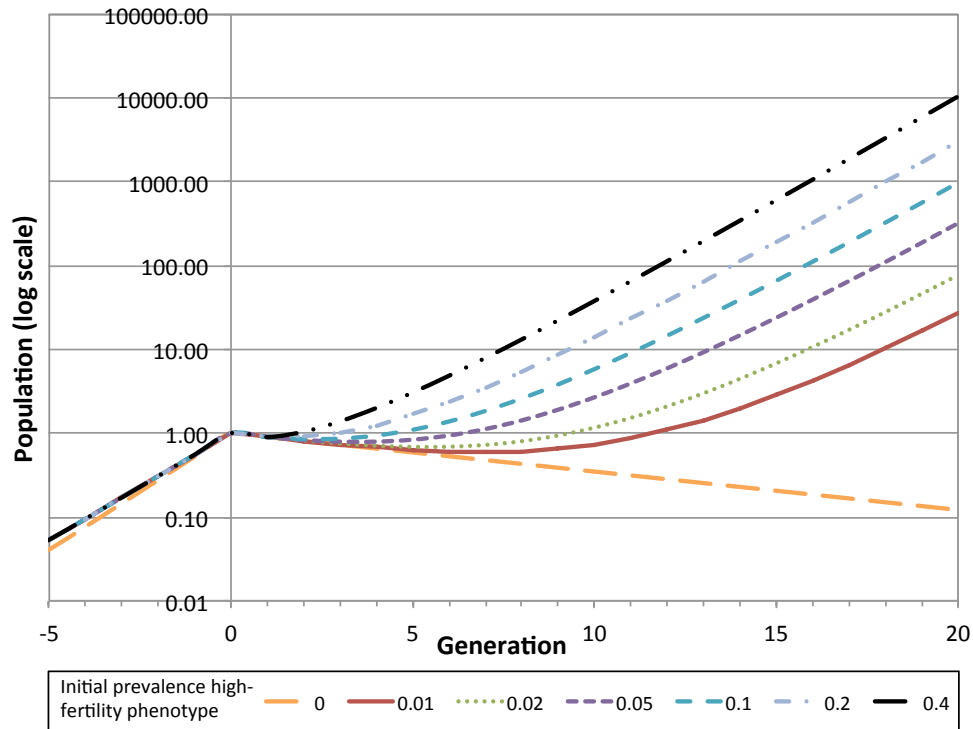


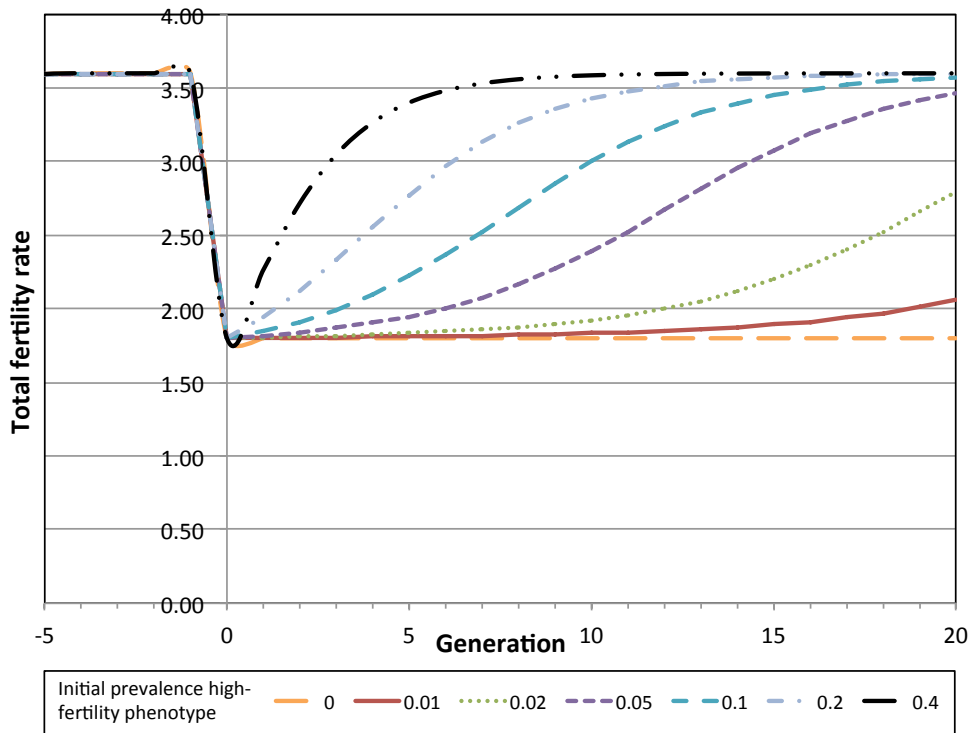
Figure 6.2 shows that natural selection for the high-fertility phenotype is important for population projections. Whatever the initial prevalence of the high-fertility phenotype, the population after 20 generations is orders of magnitude higher than if the total fertility rate remained at 1.8. In the case of an initial one per cent prevalence of the high-fertility phenotype, the population is over double that of the constant fertility case after 10 generations, while for higher initial prevalence the population is an order of magnitude or more higher.

Figure 6.2: Population with dominant high-fertility allele



Where the high-fertility allele is recessive, the high fertility phenotype does not include the heterozygote HL . In this situation, there is greater variation in the rate of increase in the fertility rate after the shock than in the dominant case (Figure 6.3). If the high-fertility phenotype (genotype HH) forms only one per cent of the population, it takes 19 generations until the fertility rate increases above replacement and many more generations until the old fertility rate is restored. If the high-fertility phenotype comprises 40 per cent of the population, the first generation following the shock has fertility above replacement and the total fertility rate approaches previous levels within six generations.

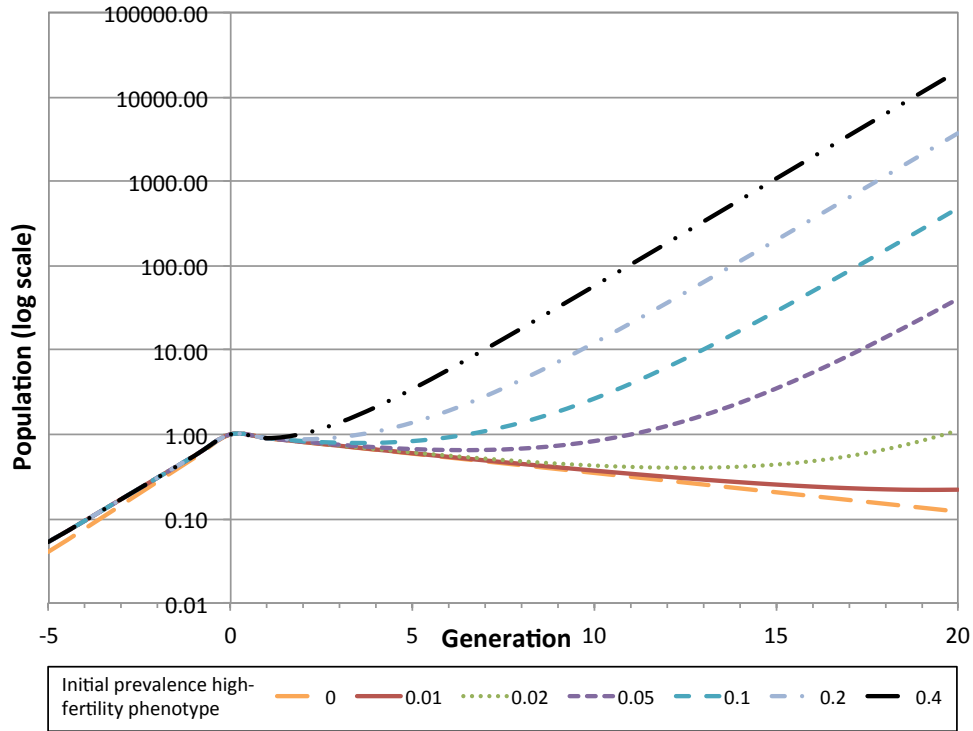
Figure 6.3: Fertility rate with recessive high-fertility allele



The greater range of change in fertility in the case of the recessive allele is due to the greater range of frequency of the high-fertility allele implied by the initial phenotype frequencies. In the dominant case, the frequency of the high-fertility allele ranges from 0.5 per cent to 22 per cent as the high-fertility phenotype changes from one per cent to 40 per cent of the population. When the high-fertility allele is recessive, the frequency of the high-fertility allele ranges from 10 per cent to 63 per cent for similar high-fertility phenotype prevalence. Further detail on the rates of change for the recessive and dominant cases is provided in the Appendix.

The population growth in each scenario also varies more when the high-fertility allele is recessive than when it is dominant (Figure 6.4). If less than five per cent of the starting population is the high-fertility phenotype, it takes 12 or more generations before the population passes its initial level, although by this time the population is over four times that of a case with no variation in fertility.

Figure 6.4: Population with recessive high-fertility allele



Finally, it is unlikely that the high populations shown in some scenarios in Figures 6.2 and 6.4 would be realised in full without interruption as hitting Malthusian limits would be expected to restrict fertility and total population growth. That said, the current world population is many times larger than would have been thought possible in Malthus's day. This observation is also relevant to the models described below.

6.3.2 A model of heritability of fertility

In the previous model children fully inherit fertility from their parents. This contrasts with the observation that the heritability of fertility is below one and is often measured at around 0.2 to 0.4 in modern populations. In this section, we present a model of family size in which we vary the heritability of fertility.

The population is composed of male and female agents who are paired into families. In each generation, the female gives birth to zero, one or more children. The number of children in generation t is determined by a binomial distribution, with conditional mean

μ_t and variance σ_t^2 . The maximum number of children (n) is set at 10. This approximates Hutterite fertility, which is considered a good example of an unrestricted fertility rate (Clark 2007). The frequency of families with i children in generation t , f_t^i , is given by:

$$f_t^i = \frac{n!}{i!(n-i)!} (p_t)^i (1-p_t)^{n-i} \quad (6.8)$$

$$\mu_t = np_t \quad \sigma_t^2 = np_t(1-p_t)$$

Given the condition mean μ_t , these equations determine the probability p_t , the variance σ_t^2 and the frequency f_t^i .

The mean number of children that each child has in their family is greater than the mean number of children in each family, as there are more children in the larger families. The proportion of children who are in a family with i children, π_t^i , is:

$$\pi_t^i = \frac{if_t^i}{\mu_t} \quad (6.9)$$

The mean number of children each child has in its family is:

$$c_t = \sum_{i=0}^n i\pi_t^i \quad (6.10)$$

Equation (6.10) determines family size if fertility is completely heritable. If each child born in generation t had the same number of children as their parents, the population in generation $t + 1$ would have a mean of c_t children in each family. This would represent an increase in the mean number of children born to each family as those from larger families come to form a larger part of the population.

If fertility is not fully heritable, the increase in family size can be modelled using the breeder's equation, which provides an estimate of the evolutionary change in a phenotypic trait under selection (Lush 1937). Under the breeder's equation, the change in the mean family size from one generation to the next is proportional to the selection differential and the narrow sense heritability of fertility, h^2 .⁵¹ The selection differential is the difference between the mean number of children that each child has in their family, c_t , and the mean fertility of the parents, μ_t .

$$\Delta\mu_t = h^2(c_t - \mu_t) \quad (6.11)$$

$$\mu_{t+1} = \mu_t + h^2(c_t - \mu_t) \quad (6.12)$$

Using these equations, we can calculate the effects of different heritability levels on the rate of change in population fertility, where the mean number of children per family is equal to the total fertility rate. Iterating forward, the new mean number of children as calculated by equation (6.12) is input into equation (6.8) to give the family structure of the new generation.

Figure 6.5 shows the results of a simulation with initial mean family size of 1.8 children (which is effectively the total fertility rate). Even at relatively low heritability, the increase in fertility is rapid. For a heritability of 0.2, the fertility rate is above the replacement level of two children within two generations. As fertility is constrained by an upper bound of 10 children per family, fertility and population projections are higher than for the diploid model presented earlier in this paper where fertility is effectively constrained at 3.6. Population growth is also rapid in all scenarios of heritability

⁵¹ Broad-sense heritability is the proportion of the variation in a phenotypic trait that is explained by all genetic factors. Narrow-sense heritability is the limited to the additive genetic variation, excluding dominance, epistasis and other non-additive effects.

(Figure 6.6). This second model demonstrates that increases in fertility may still be relatively rapid, even where heritability of fertility is below one.

Figure 6.5: Fertility rate with varying heritability

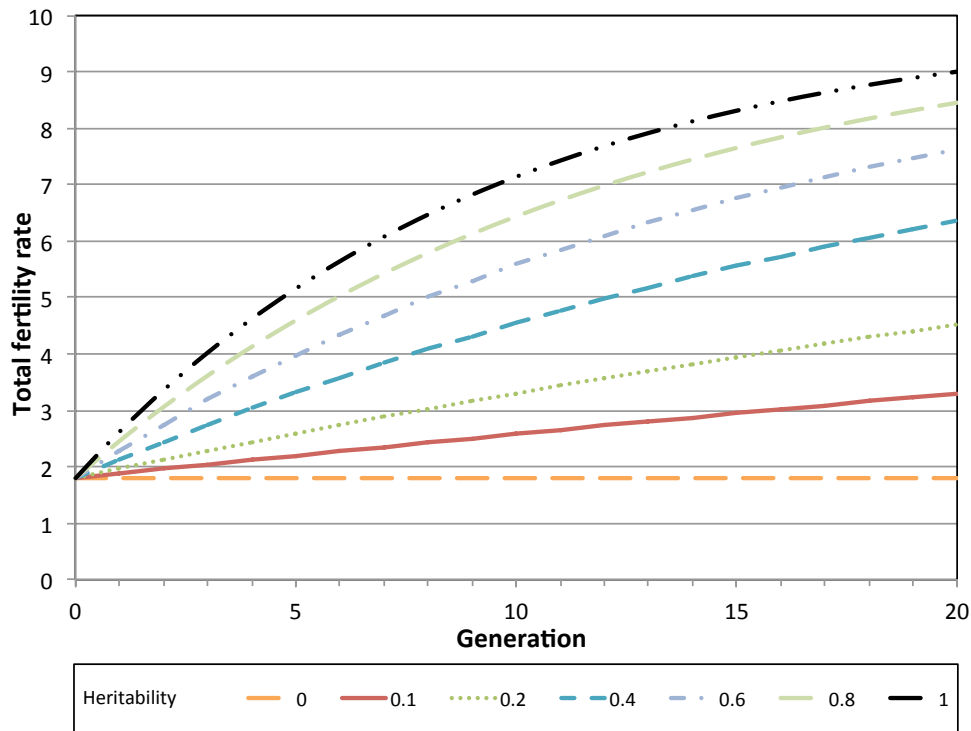
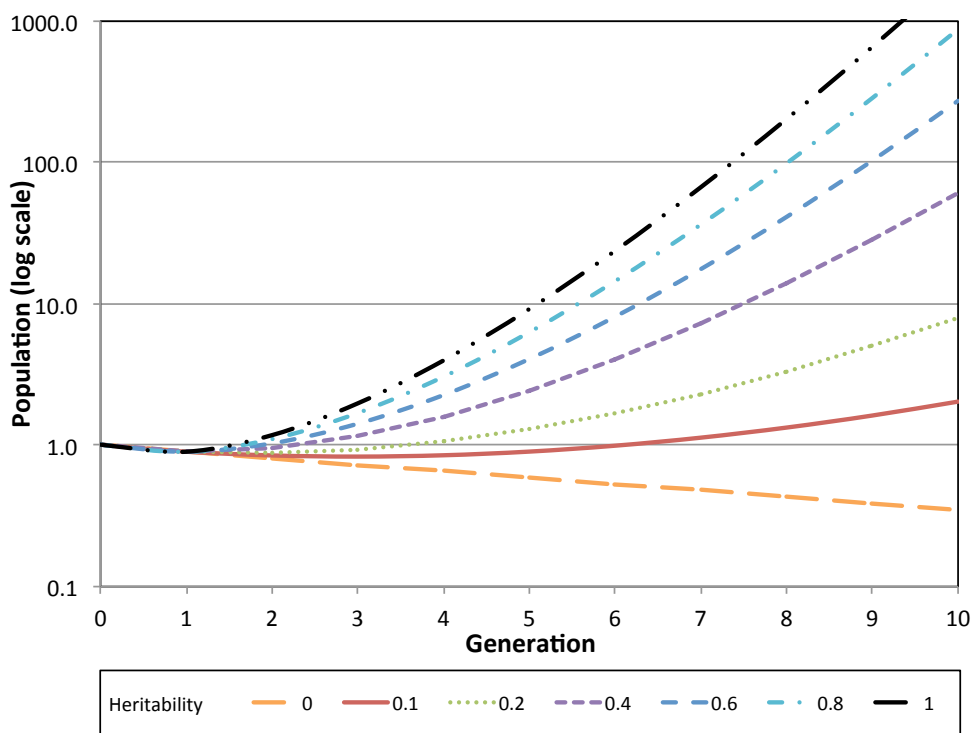


Figure 6.6: Population with varying heritability



6.3.3 A haploid age-structured model

Many of the economic challenges arising from low fertility rates pertain to the age structure of the population, rather than the fertility rate in itself. In this section, we present an age-structured model that shows how the age structure affects the short-run population dynamics.

The population is composed of haploid male and female agents, who are grouped into five-year age cohorts. In each five-year period, females bear children, with equal numbers of male and female children. The children born in one five-year period become the population of 0 to 4 year olds in the next five-year period and then move through the age cohorts in additional five-year steps. Agents also have an age-specific probability of death, which does not vary between males and females and remains constant through time. The fertility rate for each age cohort in period t_0 and the death rates approximate those in a modern, industrialised economy. The initial population structure in period t_0 is similar to that in Australia and the United States in 1960 following the post-war baby boom, giving a starting point that now lies 50 years in the past. Given that these populations have been experiencing fertility shocks for 200 years, the use of these particular data points should be considered illustrative only. The fertility rates, death rates and initial population structure are shown in Table 6.2 in the Appendix.

Using a haploid approach, two alleles of a single gene determine the fertility of a female, with these alleles representing high and low fertility ($i = H, L$). Males and females carry a high or low fertility allele and the allele inherited by the child may come from either parent, with a 50 per cent probability for each. As fertility is completely determined by the genotype of the mother, heritability is one.

An environmental fertility shock occurs at time t_0 . Each genotype responds differently to the shock, with $n^{i\tau}$ being the number of children born by females of genotype i for age cohort τ . The selection coefficient s , which is the same for each age cohort, indicates the relative response of the different genotypes to the fertility shock.

$$n_t^{L\tau} = (1-s)n_t^{H\tau} \quad 0 \leq s \leq 1 \quad t \geq t_0 \quad (6.13)$$

The population proportion of genotype i belonging to age cohort τ is:

$$\pi_t^{i\tau} = \frac{X_t^{i\tau}}{X_t} \quad (6.14)$$

X_t is the total population and $X_t^{i\tau}$ is the population of genotype i in age cohort τ . The number of children of each genotype in cohort $t+5$ is:

$$X_{t+5}^{i(0-4)} = \frac{1}{2} X_t \sum_{\tau=0-4}^{100+} \pi_t^{i\tau} n^{i\tau} \quad (6.15)$$

The total fertility rate at time t is:

$$n_t = \sum_{\tau=0-4}^{100+} \frac{X_t^{H\tau} n^{H\tau} + X_t^{L\tau} n^{L\tau}}{X_t^{H\tau} + X_t^{L\tau}} \quad (6.16)$$

As a numerical example, we ran a simulation where the initial total fertility rate is 1.8, the high-fertility genotype initially comprises 20 per cent of the population and the ratio of fertility between the high and low-fertility genotypes is 3.0 (i.e. $s = 0.66$). This implies fertility rates of approximately 1.3 and 3.9 for the low and high-fertility genotypes after the shock.

Figure 6.7 shows the age structure of the population over a 250-year period, moving first 25 years from one panel to the next and then 50 years. The bars drawn on the right

in each panel show the simulated age structure of the population, while the bars on the left represent a scenario where there is no heritable variation in fertility. The top-left panel shows the initial age structure with few young adults and many children due to the post-war baby boom. After 50 years, the cohort of children is substantially larger if fertility is heritable and after 75 years there is a larger workforce. The cohort of children continues to increase, approaching a maximum proportion of the population by year 150. By year 100, any resemblance between the population structure with heritable fertility and the scenario in which fertility is not heritable has disappeared.

Figure 6.7: Population age structure with fertility ratio of 3.0

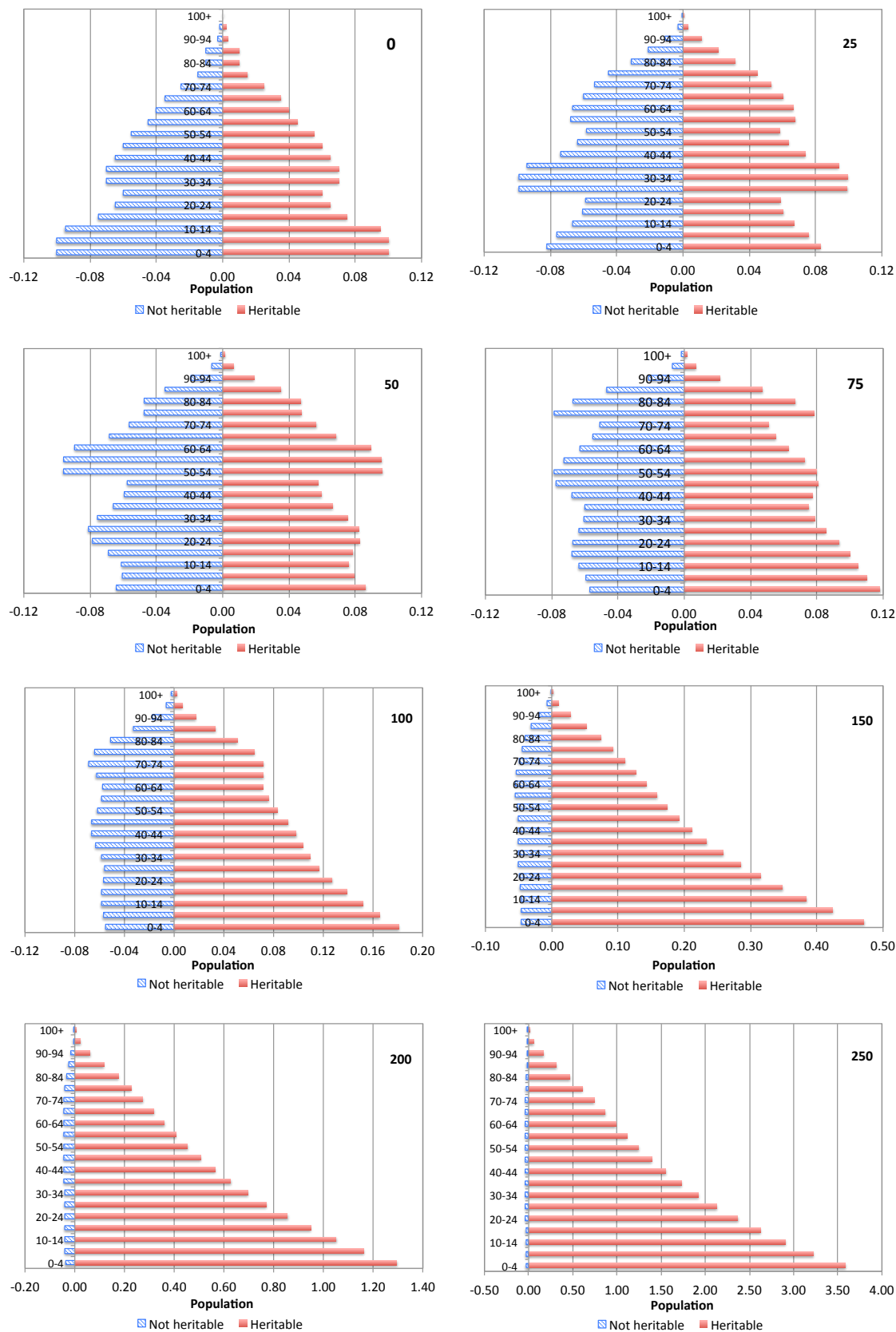


Figure 6.8 shows the dependency ratio for the simulated population. The dependency ratio for children aged 0 to 14 initially falls, remains low until year 45 and then increases to 32 per cent, while the dependency ratio of those aged over 65 increases for the first 65 years to a peak of 26 per cent and then falls to 11 per cent. The net result of these two effects is a relatively stable but high total dependency ratio of 43 per cent. This contrasts with a long-run dependency ratio of 15 per cent for the young and 28 per cent for the elderly if fertility is not heritable, with a similar total dependency ratio of 43 per cent (Figure 6.9). This pattern holds for other initial ratios between the high and low-fertility types, with the total dependency ratio approaching 43 per cent, but the proportion of young increasing as the fertility ratio increases. This finding of invariance of the total dependency ratio to changes in the fertility rate is consistent with other analyses [for example, Lattimore & Pobke (2008)].

Figure 6.8: Dependency ratio with fertility ratio of 3.0

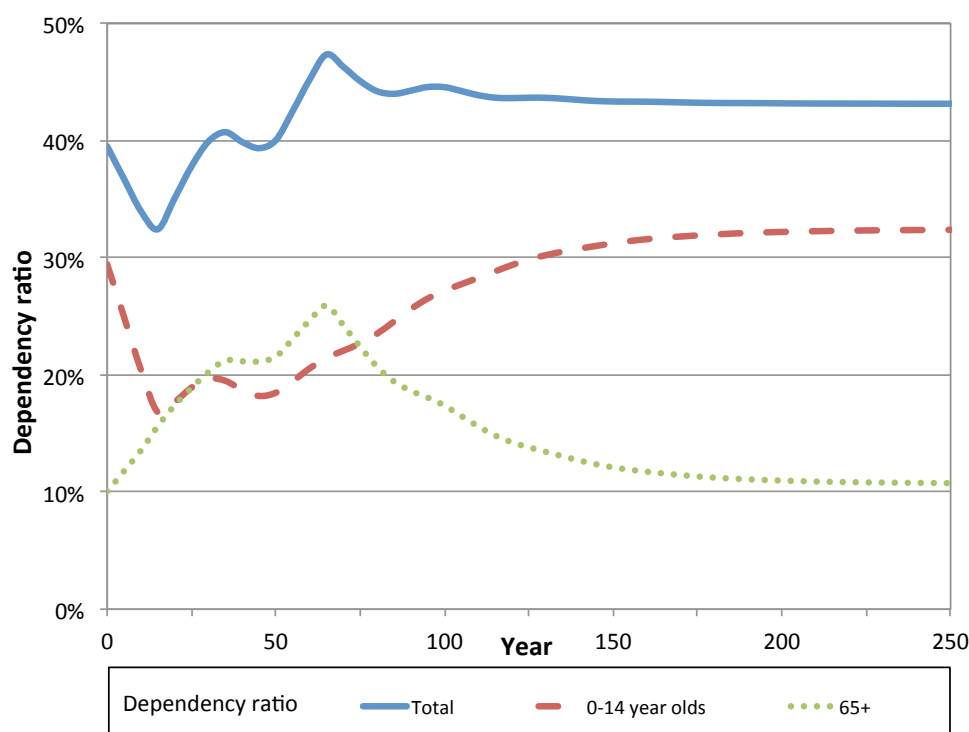


Figure 6.9: Dependency ratio with no heritability of fertility

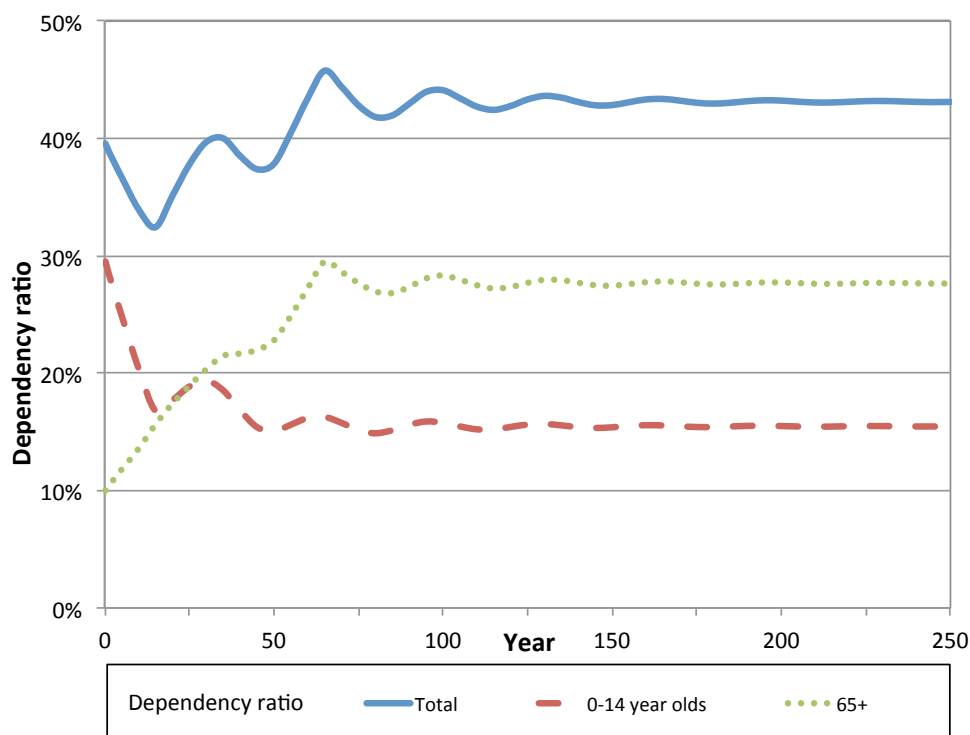


Figure 6.10 shows the total fertility rate over 250 years from the start of the simulation. The fertility rate increases above the replacement level within 30 years if the initial fertility ratio between high and low-fertility types is 3.0. The evolution of the fertility rate for three other simulations with a post-shock fertility ratio of 1.5, 2.0 and 4.0 is also shown. The fertility rate rises above the replacement within 100 years if the fertility ratio is 1.5. The total fertility rate does not change over time if fertility is not heritable.

Figure 6.10: Total fertility rate

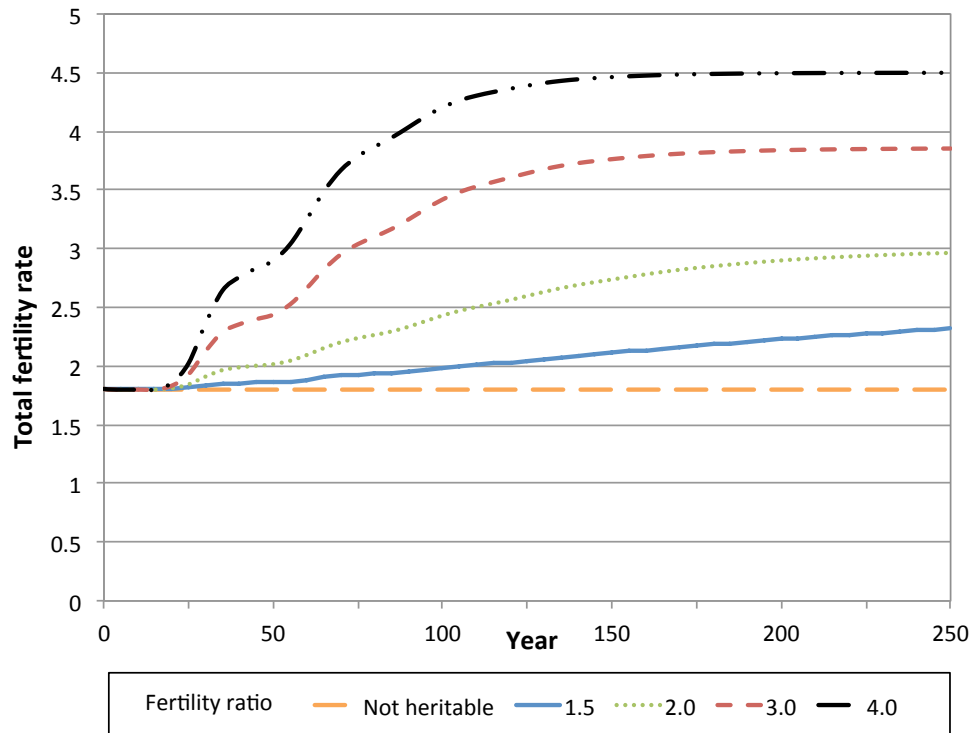
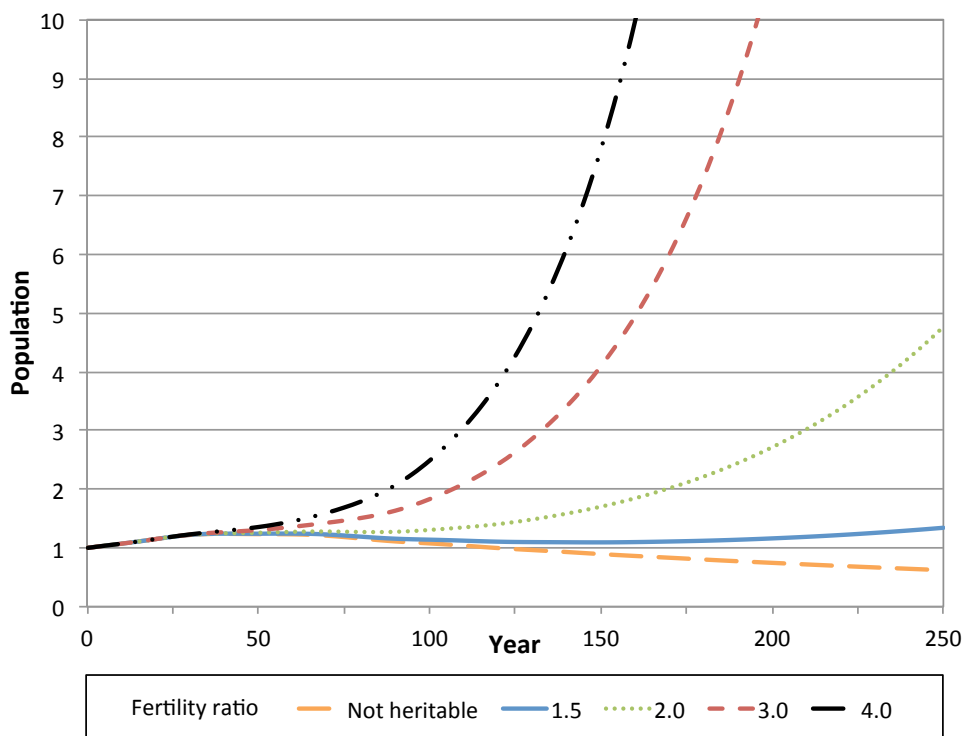


Figure 6.11 shows that the population response is similarly rapid, increasing monotonically and doubling within 110 years with a 3.0 fertility ratio, rather than declining steadily from year 30 when fertility is not heritable. The population for three other simulations with a fertility ratio of 1.5, 2.0 and 4.0 is also shown.

Figure 6.11: Population



In these age-structured model simulations with heritable fertility, there is a lag before there is a noticeable change in the age structure of the population. At best, it takes two generations before the high-fertility genotype has materially boosted the working population. Whether there will be any amelioration of the economic challenges created by an ageing population over the next 50 years depends on the biological parameters and whether there will be more shocks to fertility in the future. The use of haploid agents is likely to increase the rate of spread of the high-fertility allele relative to a diploid model, and the presence of perfect heritability will result in a faster fertility recovery faster than if the heritability were less than one. However, other assumptions lower the rate of change, particularly the restricted variation in family size.

6.4 The Galor and Moav model

In this section, we examine our hypothesis in the context of the unified growth model of Galor and Moav (2002). Unified growth theory (Galor and Weil 2000; Galor 2011)

seeks to capture within a single analytical framework the pattern of income, technology and population during the periods of Malthusian stagnation, the transition to modern growth and the modern growth state. Using a unified growth framework, Galor and Moav proposed that the evolution of the population's preference for quality or quantity of children was a factor in the transition from Malthusian stagnation to modern levels of economic growth.

The population in the Galor and Moav model consists of two types of agents ($i = a, b$) with different preferences for quantity and quality of children. Type a agents place a higher value on the quality of their children, whereas type b agents place greater value on the quantity of children. The utility functions of type a and b agents are expressed in equation (6.17).

$$u_t^i = (1 - \gamma) \ln c_t^i + \gamma (\ln n_t^i + \beta^i \ln h_{t+1}^i) \quad (6.17)$$

$$\gamma \in (0, 1); \quad \beta^i \in (0, 1]; \quad i \in a, b \quad \beta^a > \beta^b$$

c_t^i is the consumption of an individual of type i in period t , n_t^i is the number of children, and h_{t+1}^i is the level of human capital of each child. The parameter γ , which is the same for both agents, determines the relative weights of children and consumption. The parameter β^i measures the preference for quantity and quality of children for an agent of type i . Subsequent generations inherit without change the parameter β^i .

A parent incurs a fixed time cost τ for each child and a variable time cost to educate the child to the level of education e_{t+1}^i . Therefore, the total cost of raising a family with n children is $n_t^i(\tau + e_{t+1}^i)$. The human capital of a child, h_{t+1}^i , depends on the child's education, e_{t+1}^i , and on the level of technology g_{t+1} , as expressed in equation (6.18).

$$\begin{aligned}
h_{t+1}^i &= h(e_{t+1}^i, g_{t+1}) & h_e(e_{t+1}^i, g_{t+1}) &> 0 & h_{ee}(e_{t+1}^i, g_{t+1}) &< 0 \\
h_g(e_{t+1}^i, g_{t+1}) &< 0 & h_{gg}(e_{t+1}^i, g_{t+1}) &> 0 & h_{eg}(e_{t+1}^i, g_{t+1}) &> 0 \\
h(0, 0) &= 1 & \lim_{g \rightarrow \infty} h(0, g_{t+1}) &= 0
\end{aligned} \tag{6.18}$$

Education increases human capital but human capital is rendered obsolete by technological progress. The positive cross-derivative h_{eg} indicates that an increase in technology increases the return to education in terms of accumulation of human capital (and vice versa by Young's theorem).

Technological progress is a function of the average level of education of the population, as shown in equation (6.19).

$$g_{t+1} = \varphi(e_t) \tag{6.19}$$

$$\varphi_e > 0; \quad \varphi_{ee} < 0; \quad \varphi(0) = 0$$

As the return to education increases with technological progress ($h_{eg} > 0$), there is a positive feedback loop between the two. More education increases the rate of technological progress in the next generation. This increases the return to education, inducing the next generation to invest more in the education of their children.

In the Malthusian state, the quality-preferring types have higher fitness as their children have higher human capital and income that can be spent on the children of future

generations. As a result, the quality-preferring type a increases in prevalence. This causes the rate of technological progress to undergo a slow but steady increase. Eventually, the rate of technological progress increases to an extent that even the quantity-preferring type b is induced to educate its children, yielding a sudden increase in technological progress and triggering a transition to a modern growth state. As the quantity-preferring types now allocate resources to quality as well as quantity of children, fertility rates fall and the population undergoes a demographic transition.

In the modern growth state, the fitness advantage between the quantity and quality-preferring type reverses. The quality-preferring type loses the fitness advantage because technological progress degrades human capital ($h_g < 0$), making it costly to maintain a high level of human capital when the rate of technological progress is high. As the quality-preferring type overinvests in education of children, it declines in prevalence and ultimately only the quantity-preferring type remains.

Considering the Galor and Moav model in the context of this paper, the population experiences a fertility shock through the increase in technological progress. Technological progress affects the agents' allocation of resources between the quality and quantity of children, with increases in technological progress causing an increase in the investment in quality and a decrease in the investment in quantity. Fertility then declines.

In a simulation of the Galor and Moav model, Collins et al. (2014; Chapter 3) demonstrated quantitatively the evolution of fertility patterns in the population. Figure 6.12 shows the results of the simulation with two types of agents ($\beta^a = 1$; $\beta^b = 0.9$). The model parameters were calibrated such that the quantity-preferring type b had

fertility of one in the modern growth state. As reproduction is asexual and there is no mortality before reproduction, a fertility of one is the replacement level.

From the beginning of the simulation, fertility slowly increases as accelerating technological progress eases the Malthusian constraint. At generation 40, the fertility peaks at around 1.2 children per agent, before the technological shock unfolds over the course of several generations. Rapid technological progress commences when the quantity-preferring type *b* starts to educate its children. As both types start to spend on quality at the cost of quantity, the population undergoes a demographic transition in which the fertility rate declines to one. After the transition, the quality-preferring type, which comprises five per cent of the population, has a below replacement fertility level of 0.91. The quality-preferring type disappears due to its low fertility in the modern growth state, leaving only the quantity-preferring type with a fertility of one in the population (Figure 6.13). Accordingly, the fertility rate and population size is constant after the technological shock.

Figure 6.12: Fertility rate– two types

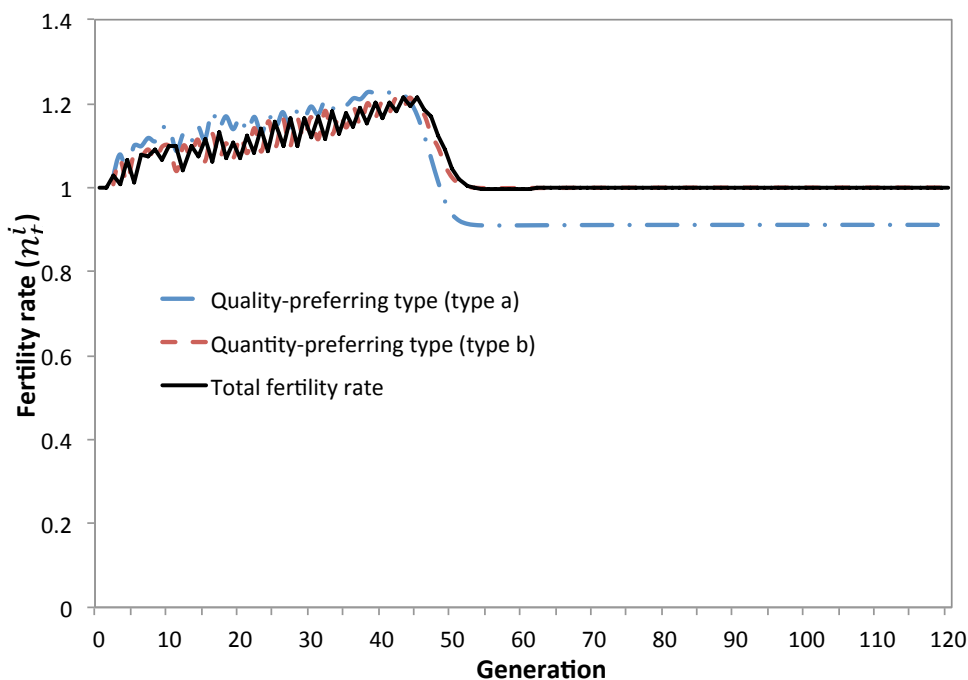
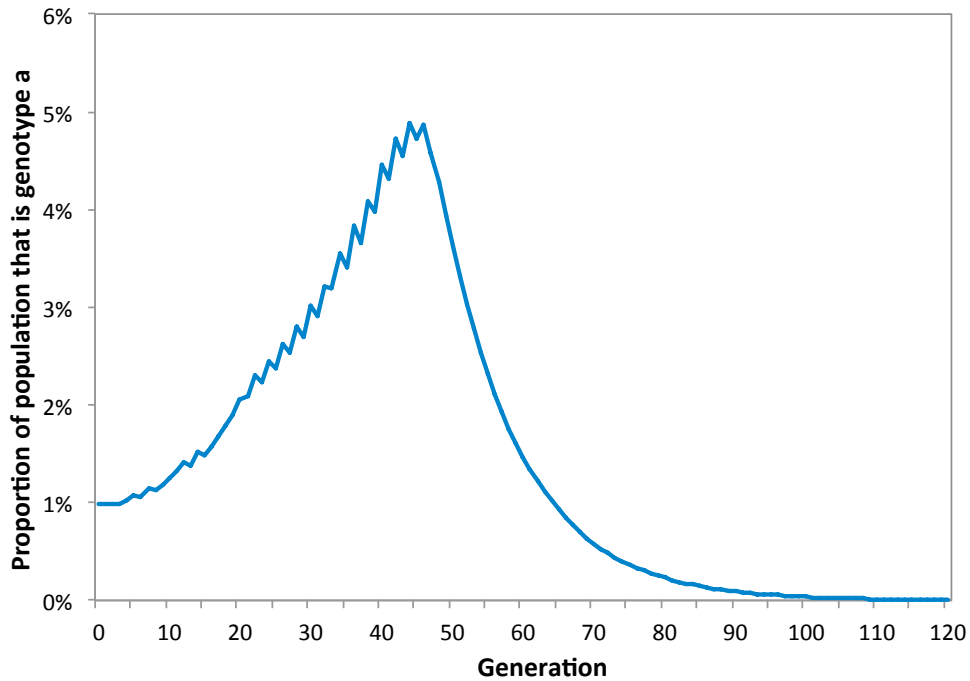


Figure 6.13: Population composition – two types



In this model specification, there is no fertility recovery following the shock, as there is effectively no genetic variation in fertility in the population. The low-fertility quality-preferring types are eliminated by selection, and the quantity-preferring types are calibrated to have fertility at the replacement level. There is therefore no scope for fertility increase and population growth after the technological shock.

However, Collins et al. (2014; Chapter 3) extended Galor and Moav's model by allowing the existence a third type of agent in the population with a very low preference for quality (i.e. $\beta^a > \beta^b \gg \beta^c$). This extension illustrates the potential for fertility recovery after a shock.⁵² In the modern growth state, the strongly quantity-preferring type c has a lower response to the technological shock and allocates more resources to the quantity of children than the other two types. Accordingly, type c has higher fertility. As there are no resource constraints in the modern growth state, this quantity-

⁵² This situation differs somewhat from the scenario presented earlier in this paper where there is no genetic variation in fertility before the fertility shock, but this does not materially affect the interpretation of the model.

focused strategy engenders higher fitness, the strongly quantity-preferring types increase in prevalence and they drive an increase in the total fertility rate.

The recovery in fertility is shown in Figures 6.14 and 6.15, which display the results of the simulation presented in Collins et al. (2014; Chapter 3), where a third type with a strong preference for quantity of children is present in the population ($\beta^c = 0.75$). As in the first simulation, the quality-preferring type a has higher fitness before the demographic transition as neither the quantity-preferring type b or strongly quantity-preferring type c invest in the quality of their children. After the technological shock, the strongly quantity-preferring type forms less than one per cent of the population but has a fertility of 1.16, which exceeds the fertility of one of the majority of the population. Type c has higher fertility because of their lower (but non-zero) investment in education, freeing resources for increasing the number of children. Accordingly, type c increases in prevalence, reaching 10 per cent of the population around generation 60, 30 per cent of the population at generation 70 and over 80 per cent by generation 90. Their increasing prevalence in the population causes a decline in technological progress through their lower investment in education, and by generation 90, the reduced rate of technological progress, which decreases the return to education, has caused the quantity-preferring type b to cease educating their children also. As a result, between generations 50 and 90, the fertility rate increases from 1.00 to 1.29.

Figure 6.14: Fertility rate – three types

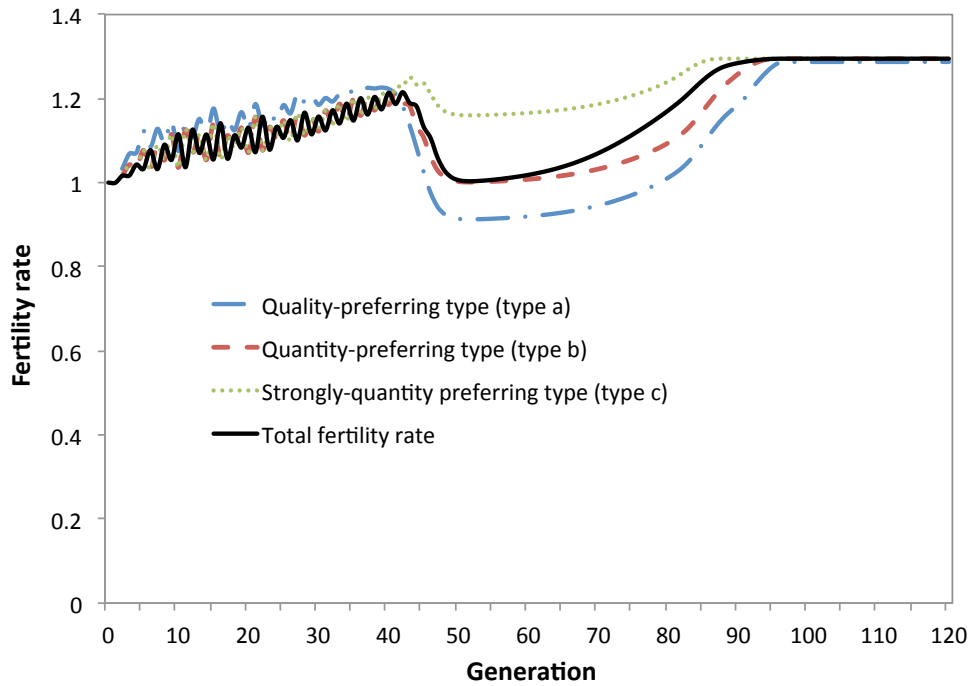
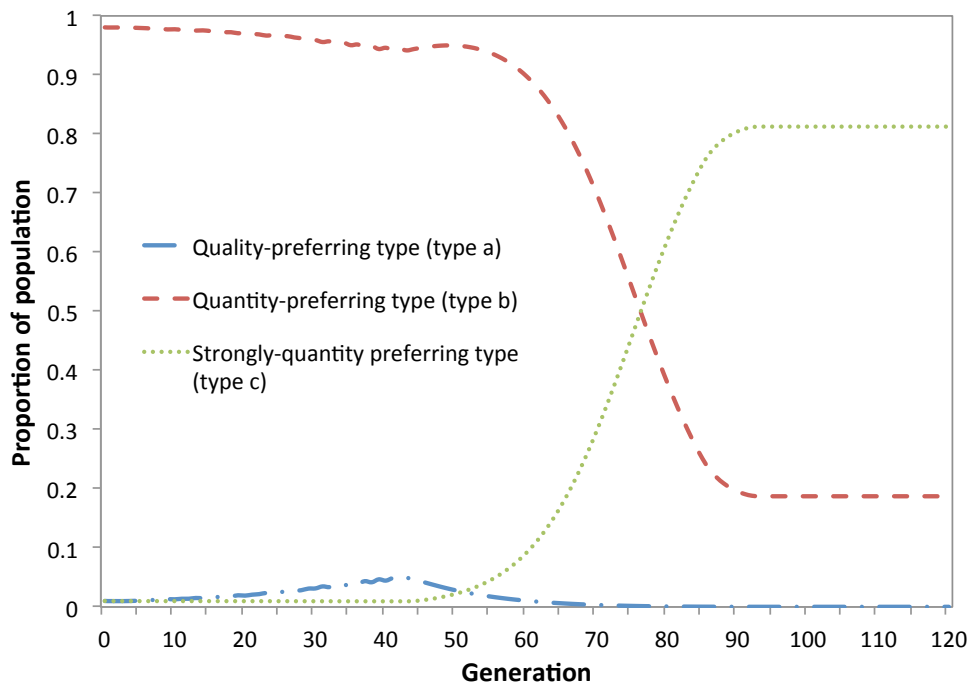


Figure 6.15: Population composition – three types



For economic policy-making, the increase in the fertility rate tens of generations after the entry into the modern growth state may be considered to be outside policy horizons. However, the slow response in the fertility rate is largely due to type *c* still having some

preference for quality in the modern growth state when technological progress is high and their initial prevalence of only one per cent of the population. The effective fertility rate cap of 1.29 due to the parameterisation of the agent's preference between consumption and children (γ) and the fixed time cost of children (τ) also constrain the fertility recovery.

In Figures 6.16 and 6.17 we show the time path of fertility and the prevalence of each type for a simulation where we relax the first two of these assumptions. The preference of the strongly quantity-preferring type c is set to zero, so that they prefer not to educate their children no matter what the rate of technological progress (i.e. $\beta^c = 0$), and type c comprises 10 per cent of the population at the start of the simulation. The first 45 generations resemble the previous simulation. At the time of the shock, the total fertility rate reduces to around 1.08, with type c keeping fertility above the replacement level. From that point, there is strong recovery in fertility, with fertility above the pre-shock level within eight generations, even though the fertility of type c is effectively capped by the maximum fertility rate of 1.29. The recovery would be faster to the extent that there was an even higher prevalence of type c at the beginning of the simulation. Further increases in their prevalence would, however, reduce the size of the technological shock and the potential for the technological take-off, as the lack of education of their children prevents the positive feedback loop between education and technological progress that characterises the Galor and Moav model.

Figure 6.16: Fertility rate – no education by third type

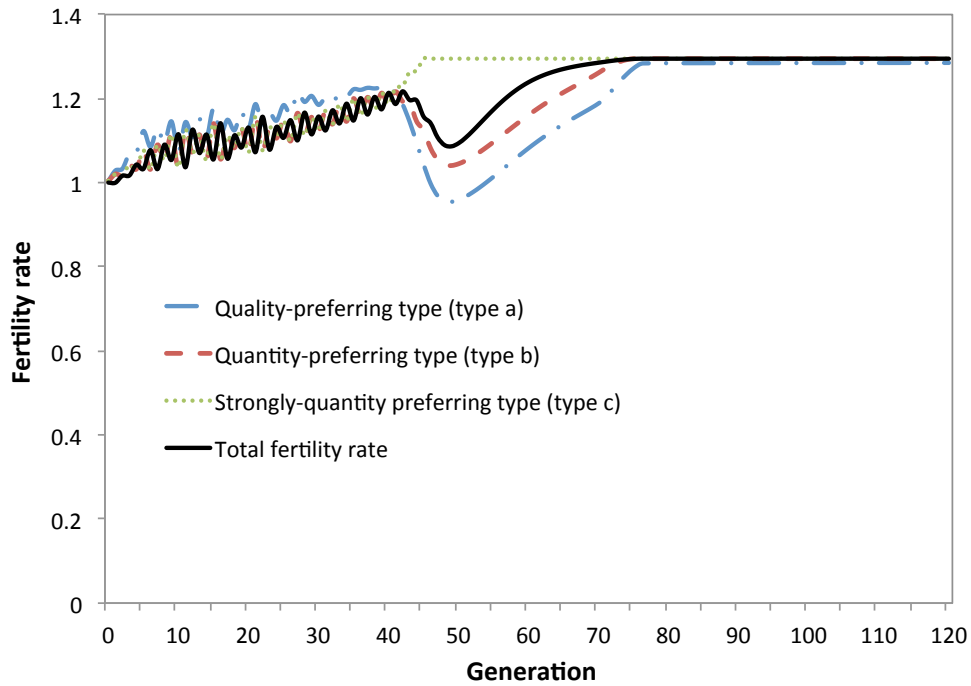
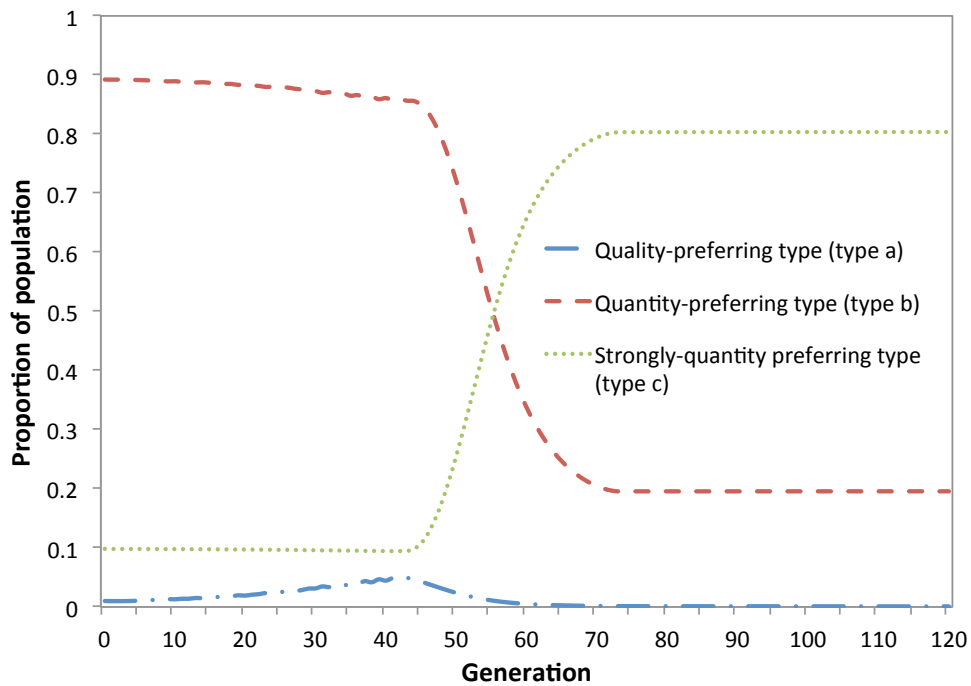


Figure 6.17: Population composition – no education by third type



Another source of fertility variation that could drive a fertility recovery would be if some agents had a heritable and lower preference for consumption in the modern growth state (i.e. higher γ). In that case, the dynamics in the Malthusian state would be

unchanged, as the subsistence constraint would still bind. However, after the entry into the modern growth state, those types that prefer to allocate resources to their children instead of consumption would have higher fertility, increase in prevalence in the population and drive an increase in the total fertility rate. Still another plausible basis for fertility variation would be children that have a lower minimum time-cost to raise, or declining time-cost of additional children, which would ease the effective fertility rate cap of 1.29.

6.5 Discussion

Most current population projections assume that the fertility rate is at, or close to, a steady state and fertility shocks are essentially modelled as permanent shocks [for example, see Lee and Tuljapurkar's (2001) analysis of United States Census Bureau forecasts]. For some genotypes this may be accurate, since they may have experienced permanent fertility reductions following the recent changes in the human environment. However, heritable variation in fertility-related traits favours those genotypes with the greatest immunity to the shock, giving rise to an increase in fertility after a fertility shock. This result holds in all three models presented in this paper; only the timing of the recovery in fertility is in serious question.

One factor that affects the speed of the recovery in fertility is the nature of the environmental shock. The models in this paper are premised on a one-off negative fertility shock, whereas the historical pattern of decline in fertility is more consistent with multiple shocks, striking different population subgroups at different times. For example, the demographic transition in England occurred first among high-income families, before spreading to the broader population (Clark and Cummins 2015). The persistence of fertility heritability and the failure of the fertility rate to recover is

evidence that multiple negative shocks have delayed a return to steady state in the past. The projected recovery in fertility does not, however, require an absence of further environmental change. Even with more environmental shocks, fertility will recover if the population response is small, which may be the case if humans are becoming better adapted to the types of environmental shocks generated by modern living.

Myrskylä et al. (2009) related the recent increase in fertility to the human development index (HDI), published by the United Nations Development Programme. They proposed that development above a certain level reverses the negative relationship between development and fertility that prevailed in the twentieth century and noted that the recent recovery in fertility could ameliorate projected socioeconomic challenges. The hypothesis presented in this paper is not inconsistent with their observation, as the HDI tends to exceed the threshold level in those countries that have been developed for longer and experienced the associated fertility shocks earlier. The earlier that a country experiences a fertility shock, the sooner those immune to the shock will increase in prevalence in the population and drive increases in the total fertility rate.

Other proposed explanations of recent fertility increases, including government policies [for example, Milligan (2005), Lalive and Zweimüller (2009) and Goldstein et al. (2009)], may also be consistent with our models. Government policies may accelerate or mitigate the selection of high fertility genotypes because different genotypes may respond differently to government incentives. In that sense, policy changes are a further type of environmental shock.

Another factor influencing fertility is the effect of immigration and immigration policy. Migrants tend to increase the total fertility rate in the short-run through their higher fertility levels. However, while their fertility may initially be higher, their fertility may

be lower than that of the host population in the long-run if they are less adapted to the new environment, which would make them more susceptible to the environmental fertility shocks of the host country.

For these reasons, it is possible that the return to a replacement-level fertility rate will not occur rapidly. However, our models provide a theoretical framework for thinking about fertility projections and their policy implications. Focusing on the heritability of fertility, our models suggest a biological mechanism by which fertility will recover after a shock. By ignoring the evolutionary underpinnings of fertility, demographers and policy makers risk underestimating future fertility rates and population growth. As a result, governments may prepare for challenges different from those that their populations will actually face.

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Chapter 6 Appendix

6A.1 Model parameters

In Table 6.1 we show the implied fertility of the low-fertility phenotype for different initial frequencies of the high-fertility phenotype in the diploid model.

Table 6.1: Diploid model phenotype fertility

Initial frequency high-fertility phenotype	Implied fertility low-fertility phenotype
0	1.80
0.01	1.78
0.02	1.76
0.05	1.71
0.10	1.60
0.20	1.35
0.40	0.60

In Table 6.2 we show the population parameters for the age-structured model.

Table 6.2: Age-structured model population parameters

Age cohort (τ)	Total fertility rate at t_0	Death rate	Population at t_0
0-4	0	0.005	0.10
5-9	0	0.0005	0.10
10-14	0	0.0005	0.095
15-19	0.1	0.002	0.075
20-24	0.3	0.002	0.065
25-29	0.6	0.003	0.060
30-34	0.5	0.004	0.070
35-39	0.2	0.005	0.070
40-44	0.1	0.006	0.065
45-49	0	0.01	0.060
50-54	0	0.01	0.055
55-59	0	0.02	0.045
60-64	0	0.03	0.040
65-69	0	0.05	0.035
70-74	0	0.1	0.025
75-79	0	0.15	0.015
80-84	0	0.25	0.010
85-89	0	0.4	0.010
90-94	0	0.6	0.003
95-99	0	0.8	0.002
100+	0	1	0
Total	1.8		1

6A.2 Selection pressure in the diploid model

The rate of change in frequency of the high-fertility allele depends on the selection coefficient and the fertility of the heterozygote phenotype (HL). If the ratio of fertility between the high-fertility homozygote (HH), heterozygote (HL) and low-fertility homozygote (LL) is $1:(1-hs):(1-s)$, the rate of change of the proportion of the high-fertility allele in the population is:

$$\Delta\pi_t^H = \frac{s\pi_t^H(1-\pi_t^H)(1-\pi_t^H + h(2\pi_t^H - 1))}{1-s(1-\pi_t^H) + s\pi_t^H(1-2h)(1-\pi_t^H)} \quad (6.20)$$

s is the selection coefficient and h is the degree of dominance of the high-fertility allele.

Where the high-fertility allele is dominant, the heterozygote has the same fertility as the high-fertility homozygote. Equation (6.20) simplifies to:

$$\Delta\pi_t^H = \frac{s\pi_t^H(1-\pi_t^H)^2}{1-s(1-\pi_t^H)^2} \quad (6.21)$$

Where the high-fertility allele is recessive, the heterozygote has the same fertility as the low-fertility homozygote. Equation (6.20) simplifies to:

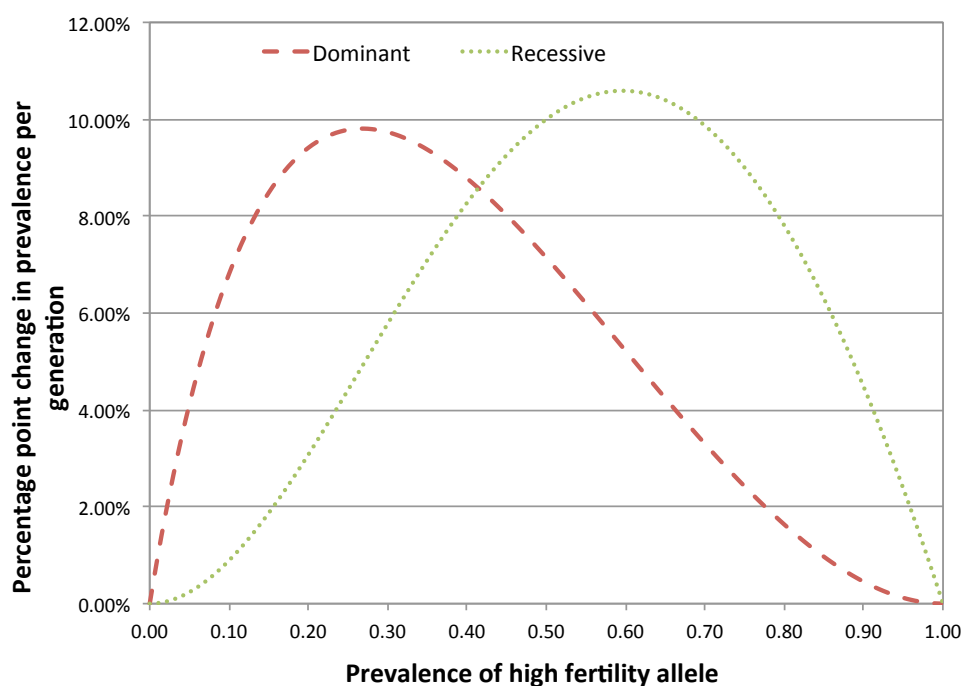
$$\Delta\pi_t^H = \frac{s(\pi_t^H)^2(1-\pi_t^H)}{1-s[1-(\pi_t^H)^2]} \quad (6.22)$$

Equations (6.21) and (6.22) show that the high-fertility allele spreads fastest when it is present in proportions at less than 50 per cent when it is dominant and at more than

50 per cent when it is recessive. In each case, it is likely to reach fixation in the population at a similar time.

Figure 6.18 illustrates the spread of the high fertility allele at different levels of prevalence for a selection coefficient of 0.5 (such as for high-fertility phenotype fertility of 3.6 and a low-fertility phenotype fertility of 1.8). The upper axis shows the selection pressure (the percentage point change in allele frequency per generation) and the lower axis indicates the prevalence of the high-fertility allele in the population. If the high-fertility allele is dominant, the selection pressure peaks at lower prevalence than if it is recessive. In the dominant case, at low frequencies most high-fertility alleles are in the homozygote (HH) and are selected for, which speeds the initial spread. At higher frequencies, the success of the heterozygote (HL) prevents elimination of the low-fertility allele. In the recessive case, most high-fertility alleles are eliminated in the heterozygote at low prevalence, while at higher prevalence, selection against the heterozygote assists in the elimination of the now rare low-fertility allele.

Figure 6.18: Selection pressure



APPENDIX: COMPUTER CODE

Chapter 3 computer code

The simulations in Chapter 3 were conducted using R (R Development Core Team 2010), a free language and environment for statistical computing. The software is available from <http://www.r-project.org>

3C.1 Base case simulation

```
#load BB package - used for solving nonlinear equations
library(BB)

#initial conditions
A<-1
g<-0
ea<-0
eb<-0
e<-0
La<-0.007
Lb<-0.7
L<-La+Lb
qa<-La/L
qb<-Lb/L
za<-1.25
zb<-1.25
na<-1
nb<-1

#parameters
Ba<-1
Bb<-0.9
alpha<-0.4
Tau<-0.2
rho<-0.99
a<-rho*Tau
m<-2
gamma<-0.259
k<-8.885139596
r<-0.108150721
X<-1
sc<-1
zsc<-sc/(1-gamma)
```

```

time<-200 #number of generations

#Build data frame which will be used to store results
Growth<-data.frame(time=0, A, g, ea, eb, La, Lb, na, nb, za, zb)

#establish a loop
for (t in 1:time) {

# population
La<-na*La
Lb<-nb*Lb
L<-La+Lb
qa<-La/L
qb<-Lb/L

# technology in this period (based on education given to
  children in last period)
e<-qa*ea+qb*eb
g<-k*e^0.5
A<-(1+g)*A

# human capital
ha<-(m*ea+a)/(ea+r*g+a)
hb<-(m*eb+a)/(eb+r*g+a)

#level of education of each genotype
ea<-max(0, (1/(2*m))*((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)+(((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)^2+4*m*(Ba*m*r*g*Tau+Ba*m*a*Tau-Ba*a*Tau-a*r*g-a^2))^0.5)))
eb<-max(0, (1/(2*m))*((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)+(((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)^2+4*m*(Bb*m*r*g*Tau+Bb*m*a*Tau-Bb*a*Tau-a*r*g-a^2))^0.5)))

#calculate possibilities for income for genotype a (and given
  zb=za*hb/ha)
#if subsistence constraint binding for neither
zaa1b1<-(((A*X)/(L*((1-gamma)*(qa*ha+(1-qa)*hb))))^alpha)*ha
zba1b1<-zaa1b1*hb/ha
if(zaa1b1>=zsc & zba1b1>=zsc) za<-zaa1b1

#if subsistence constraint binding for type b
fzaa1b2<-function(zaa1b2){
y<-(((A*X)/(L*(((1-gamma)*qa*ha)+(1-
qa)*((sc*ha)/zaa1b2))))^alpha)*ha-zaa1b2
y
}
ansfzaa1b2<-multiStart(c(0.2,1,10,100),fzaa1b2,
  control=list(M=200), quiet=TRUE)

```



```

zaa1b2<-max(ansfzaa1b2$par[,1], ansfzaa1b2$par[,2],
  ansfzaa1b2$par[,3], ansfzaa1b2$par[,4])
zba1b2<-zaa1b2*hb/ha
if(zaa1b2>=zsc & zba1b2>=sc & zba1b2<zsc) za<-zaa1b2

#if subsistence constraint binding for both types
fzaa2b2<-function(zaa2b2){
y<-((((A*X)/(L*(((sc/zaa2b2)*qa*ha)+(1-
qa)*((sc*ha)/zaa2b2))))^alpha)*ha-zaa2b2
y
}
ansfzaa2b2<-multiStart(c(0.2,1,10,100),fzaa2b2,
  control=list(M=200), quiet=TRUE)
zaa2b2<-max(ansfzaa2b2$par[,1], ansfzaa2b2$par[,2],
  ansfzaa2b2$par[,3], ansfzaa2b2$par[,4])
zba2b2<-zaa2b2*hb/ha
if(zaa2b2>=sc & zaa2b2<zsc & zba2b2>=sc & zba2b2<zsc) za<-zaa2b2
if(zaa2b2<sc) za<-0

#given income for genotype a, calculate income for genotype b
zb<-za*hb/ha

#population growth
if(za>=zsc) na<-gamma/(Tau+ea)
if(za<zsc & za>sc) na<-(1-(sc/za))/(Tau+ea)
if(za<=sc) na<-0
if(zb>=zsc) nb<-gamma/(Tau+eb)
if(zb<zsc & za>sc) nb<-(1-(sc/zb))/(Tau+eb)
if(zb<=sc) nb<-0

Growth<-rbind(Growth, c(t, A, g, ea, eb, La, Lb, na, nb, za,
  zb))

}

Growth

```

3C.2 Simulation with three genotypes

```

#load BB package - used for solving nonlinear equations
library(BB)

#initial conditions
A<-1
g<-0
ea<-0

```

```

eb<-0
ec<-0
e<-0
La<-0.007
Lb<-0.7
Lc<-0.007
L<-La+Lb+Lc
qa<-La/L
qb<-Lb/L
qc<-Lc/L
za<-1.25
zb<-1.25
zc<-1.25
na<-1
nb<-1
nc<-1

#parameters
Ba<-1
Bb<-0.9
Bc<-0.75
alpha<-0.4
Tau<-0.2
rho<-0.99
a<-rho*Tau
m<-2
gamma<-0.259
k<-8.885139596
r<-0.108150721
X<-1
sc<-1
zsc<-sc/(1-gamma)
time<-2000 #number of generations

#Build data frame which will be used to store results
Growth<-data.frame(time=0, A, g, ea, eb, ec, La, Lb, Lc, na, nb,
nc, za, zb, zc)

#establish a loop
for (t in 1:time) {

# population
La<-na*La
Lb<-nb*Lb
Lc<-nc*Lc
L<-La+Lb+Lc
qa<-La/L
qb<-Lb/L

```

```

qc<-Lc/L

# technology in this period (based on education given to
  children in last period)
e<-qa*ea+qb*eb+qc*ec
g<-k*e^0.5
A<-(1+g)*A

# human capital
ha<-(m*ea+a)/(ea+r*g+a)
hb<-(m*eb+a)/(eb+r*g+a)
hc<-(m*ec+a)/(ec+r*g+a)

#level of eduction of each genotype
ea<-max(0, (1/(2*m))*((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)+(((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)^2+4*m*(Ba*m*r*g*Tau+Ba*m*a*Tau-Ba*a*Tau-a*r*g-a^2))^0.5)))
eb<-max(0, (1/(2*m))*((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)+(((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)^2+4*m*(Bb*m*r*g*Tau+Bb*m*a*Tau-Bb*a*Tau-a*r*g-a^2))^0.5)))
ec<-max(0, (1/(2*m))*((Bc*m*r*g+Bc*m*a-Bc*a-m*r*g-a*m-
a)+(((Bc*m*r*g+Bc*m*a-Bc*a-m*r*g-a*m-
a)^2+4*m*(Bc*m*r*g*Tau+Bc*m*a*Tau-Bc*a*Tau-a*r*g-a^2))^0.5)))

#calculate possibilities for income for genotype a
#if subsistence constraint binding for noone
zaa1b1c1<-(((A*X)/(L*(1-gamma)*(qa*ha+qb*hb+qc*hc)))^alpha)*ha
zba1b1c1<-zaa1b1c1*hb/ha
zca1b1c1<-zaa1b1c1*hc/ha
if(zaa1b1c1>=zsc & zba1b1c1>=zsc & zca1b1c1>=zsc) za<-zaa1b1c1

#if subsistence constraint binding for type c only
fzaa1b1c2<-function(zaa1b1c2){
y<-(((A*X)/(L*(((1-
gamma)*(qa*ha+qb*hb))+qc*((sc*ha)/zaa1b1c2))))^alpha)*ha-
zaa1b1c2
y
}
ansfzaa1b1c2<-multiStart(c(0.2,1,10,100),fzaa1b1c2,
  control=list(M=200), quiet=TRUE)
zaa1b1c2<-max(ansfzaa1b1c2$par[,1], ansfzaa1b1c2$par[,2],
  ansfzaa1b1c2$par[,3], ansfzaa1b1c2$par[,4])
zba1b1c2<-zaa1b1c2*hb/ha
zca1b1c2<-zaa1b1c2*hc/ha
if(zaa1b1c2>=zsc & zba1b1c2>=zsc & zca1b1c2>=sc & zca1b1c2<zsc)
  za<-zaa1b1c2

```

```

#if subsistence constraint binding for type b and c
fzaa1b2c2<-function(zaa1b2c2){
y<-((((A*X)/(L*(((1-
gamma)*qa*ha)+qb*((sc*ha)/zaa1b2c2)+qc*((sc*ha)/zaa1b2c2))))^alpha)*ha-zaa1b2c2
y
}
ansfzaa1b2c2<-multiStart(c(0.2,1,10,100),fzaa1b2c2,
control=list(M=200), quiet=TRUE)
zaa1b2c2<-max(ansfzaa1b2c2$par[,1], ansfzaa1b2c2$par[,2],
ansfzaa1b2c2$par[,3], ansfzaa1b2c2$par[,4])
zba1b2c2<-zaa1b2c2*hb/ha
zca1b2c2<-zaa1b2c2*hc/ha
if(zaa1b2c2>=zsc & zba1b2c2>=sc & zba1b2c2<zsc & zca1b2c2>=sc &
zca1b2c2<zsc) za<-zaa1b2c2

#if subsistence constraint binding for all types
fzaa2b2c2<-function(zaa2b2c2){
y<-((((A*X)/(L*(((sc/zaa2b2c2)*qa*ha)+(1-
qa)*((sc*ha)/zaa2b2c2))))^alpha)*ha-zaa2b2c2
y
}
ansfzaa2b2c2<-multiStart(c(0.2,1,10,100),fzaa2b2c2,
control=list(M=200), quiet=TRUE)
zaa2b2c2<-max(ansfzaa2b2c2$par[,1], ansfzaa2b2c2$par[,2],
ansfzaa2b2c2$par[,3], ansfzaa2b2c2$par[,4])
zba2b2c2<-zaa2b2c2*hb/ha
zca2b2c2<-zaa2b2c2*hc/ha
if(zaa2b2c2>=sc & zaa2b2c2<zsc & zba2b2c2>=sc & zba2b2c2<zsc)
za<-zaa2b2c2
if(zaa2b2c2<zsc) za<-0

#given income for genotype a, calculate income for genotype b
zb<-za*hb/ha
zc<-za*hc/ha

#population growth
if(za>=zsc) na<-gamma/(Tau+ea)
if(za<zsc & za>sc) na<-(1-(sc/za))/(Tau+ea)
if(za<=sc) na<-0
if(zb>=zsc) nb<-gamma/(Tau+eb)
if(zb<zsc & zb>sc) nb<-(1-(sc/zb))/(Tau+eb)
if(zb<=sc) nb<-0
if(zc>=zsc) nc<-gamma/(Tau+ec)
if(zc<zsc & zc>sc) nc<-(1-(sc/zc))/(Tau+ec)
if(zc<=sc) nc<-0

```

```
Growth<-rbind(Growth, c(t, A, g, ea, eb, ec, La, Lb, Lc, na, nb,  
nc, za, zb, zc))
```

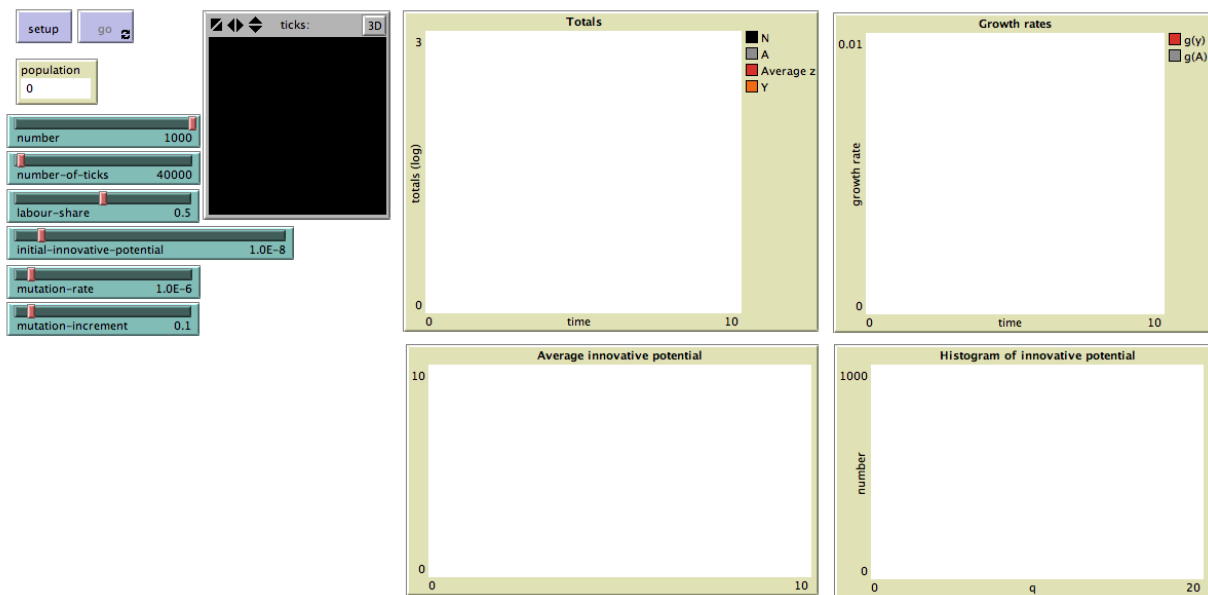
```
}
```

```
Growth
```

Chapter 4 computer code

The simulations in Chapter 4 were conducted using NetLogo (Wilensky 1999), a multi-agent programmable modelling environment. The full NetLogo model is available for download from <http://www.jasoncollins.org/downloads>. Figure 1 shows a screenshot of the NetLogo interface.

Figure 1: NetLogo interface



Program code for the agent-based simulation:

```
globals [
  A                ;; technology
  A-old            ;; technology in the previous period
  A-growth         ;; technological progress
  N                ;; population
  average-delta    ;; average innovative potential of the population
  average-delta-old ;; innovative potential in the previous period
  average-delta-growth ;; innovative potential growth rate
  w                ;; wage
  Y                ;; total income
  Y-old            ;; total income in the previous period
  Y-growth         ;; total income growth rate
  average-z        ;; average income
  malthusian-limit ;; population at equilibrium for a given level of technology
  subsistence      ;; subsistence income at the Malthusian limit
  alpha            ;; labour share
```

```

v                ;; mutation rate - set by slider mutation-rate
m                ;; mutation increment - set by slider mutation-increment
]

turtles-own [
  delta          ;; innovative potential
  z              ;; turtle income
  children        ;; expected number of children for a turtle
  age            ;; use for mechanism to kill off turtles after one generation
]

to setup
  clear-all
  setup-turtles
  set alpha labour-share
  set v mutation-rate
  set m mutation-increment
  set A 1          ;; set technology to 1
  set average-delta initial-innovative-potential ;; to give non-zero initial value for
plot
  set average-z A * number ^ (alpha - 1) * average-delta ^ alpha ;; to give non-zero
initial value for plot of logarithms equal to subsistence
  set Y average-z * number
  set subsistence A * (number ^ (alpha - 1)) * (average-delta ^ alpha) ;; to set
subsistence such that users can select any initial number of turtles
  reset-ticks
end

to setup-turtles
  create-turtles number
  ask turtles [
    setxy random-xcor random-ycor
    set delta initial-innovative-potential
  ]
end

to go
  if N >= 100000 [
    stop          ;; stop population getting too large and hanging simulation
  ]
  if ticks >= number-of-ticks [
    stop          ;; stop after a certain number of ticks
  ]
  work
  innovate
  reproduce
  tick
end

```

```

to work
  set average-delta-old average-delta
  set average-delta mean [delta] of turtles
  set average-delta-growth average-delta / average-delta-old - 1
  set N count turtles
  set w  $A * ((\text{average-delta} * N) ^ (\alpha - 1))$ 
  set malthusian-limit  $(\text{subsistence} / (A * \text{average-delta} ^ \alpha)) ^ (1 / (\alpha - 1))$ 
  ask turtles [
    set z delta * w
    set age 1 ;; age turtles so can kill off at end of generation
  ]
  set Y-old Y
  set Y sum [z] of turtles
  set Y-growth Y / Y-old - 1
  set average-z Y / N
end

to innovate
  set A-old A
  set A  $A + A * (\text{average-delta} * N)$ 
  set A-growth A / A-old - 1
end

to reproduce
  ask turtles [
    set children malthusian-limit * z / Y
    hatch random-poisson children [ ;; number of children of each turtle is given
    by a Poisson distribution
      set age 0
      setxy random-xcor random-ycor
      if  $(2 * v * 1000000000) > \text{random } 10000000000$  [ ;; set innovative potential of
turtles of next generation - mutation rate of 2v
        ifelse 1 = random 2 ;; one in two chance that mutation is either
positive or negative
          [set delta delta + (m * delta)] ;; positive mutation if random 2 = 1
          [set delta delta - (m * delta)] ;; negative mutation if random 2 = 0
        ]
      ]
      if age = 1 [die] ;; kills off turtles from the last generation
      if delta <= 0 [die] ;; kills of turtles where innovative potential
has mutated to zero or below
    ]
  ]
end

```


Chapter 5 computer code

The simulations in Chapter 5 were conducted using R, a free language and environment for statistical computing. The software is available from <http://www.r-project.org> (R Development Core Team 2010)

5C.1 Model with evolution of male preference

```
#parameters
hH=1                #quality of high-quality males
hL=0.5              #quality of low-quality males
piH=0.5             #proportion of males of high quality
piL=1-piH           #proportion of males of low quality
piS=0.000001        #initial prevalence signalling allele
piN=1-piS           #initial prevalence non-sngialling allele
zbar=1              #subsistence income
rho=0.5             #output elasticity of labour in subsistence
                    #sector
alpha=0.5           #output elasticity of labour in luxury
                    #sector
N=1                 #initial population of males and females
A=1                 #initial technology in the luxury sector
psi=1               #research productivity per person
B=0.5               #returns to innovation
time<-100           #number of generations

#Build data frame which will be used to store results
Population<-data.frame(time=0, piS, piN, sSH=0.75, sSL=1, sNH=1,
  sNL=1, r=1, gammaSH=1, gammaSL=1, gammaNH=1, gammaNL=1, nSB=0,
  nSC=0, nNC=0, nS=0, nN=0, pLSH=0.25, pL=0, A, g=0, Y=1,
  cSH=0.25)

#establish a loop
for (t in 1:time) {

#population composition
piSH=piS*piH
piSL=piS*piL
piNH=piN*piH
piNL=piN*piL
```

```

#number of males
MA=N
MASH=piSH*MA
MASL=piSL*MA
MANH=piNH*MA
MANL=piNL*MA

#number of females
FA=N

# signalling
sSH=max(0, (2-piSH*(1-hL/hH)-hL/hH)/(2-piSH*(1-hL/hH)))
sSL=1
sNH=1
sNL=1

#economic outputs
r=MA^(rho-1)                                #subsistence sector wage
pLSH=1-sSH                                  #labour force partipation rate of
                                              #high-quality signalling males
pL=piSH*(1-sSH)                             #labour force participation rate for
                                              #population
L=MA*piSH*(1-sSH)*hH                       #luxury sector efficiency units of
                                              #labour
w=A*MA^(alpha-1)                            #luxury sector wage
cSH=w*hH*(1-sSH)                           #conspicuous consumption of high-
                                              #quality signalling male
Y=((1-piSH)+piSH*sSH)*r+piSH*cSH           #output
g=psi*L^B                                   #Technological progress
A=(1+g)*A                                   #Technology available to next
                                              #generation

#survival
gammaSH=sSH*r
gammaSL=sSL*r
gammaNH=sNH*r
gammaNL=sNL*r

#surviving males
MBSH=MASH*gammaSH
MBSL=MASL*gammaSL
MBNH=MANH*gammaNH
MBNL=MANL*gammaNL
MB=MBSH+MBSL+MBNH+MBNL

```

```

#proportion surviving by type
piSH=MBSH/MB
piSL=MBSL/MB
piNH=MBNH/MB
piNL=MBNL/MB

#number of children in Stage B
nSB=MBSH*hH

#number of females available in Stage C
FC=max(0, FA-MBSH)

#number of children in Stage C
#note - always more males than females in Stage C under
#parameters chosen
nSC=(piSH*hH+piSL*hL)*FC
nNC=(piNH*hH+piNL*hL)*FC

#total number of children of each type
nS=nSB+nSC
nN=nNC
n=nS+nN

#NEXT GENERATION
#proportion of each type in next generation
piS=nS/n
piN=nN/n

#population in next generation
MA=N
FA=N

#Bind the new generation of results to the data frame
Population<-rbind(Population, c(t, piS, piN, sSH, sSL, sNH, sNL,
r, gammaSH, gammaSL, gammaNH, gammaNL, nSB, nSC, nNC, nS, nN,
pLSH, pL, A, g, Y, cSH))

#close loop
}

Population

```

5C.2 Model with coevolution of male and female preferences

```
#parameters
hH=1          #quality of high-quality males
hL=0.5        #quality of low-quality males
piH=0.5       #proportion of males of high quality
piL=1-piH     #proportion of males of low quality
piS=0.01      #initial prevalence signalling allele
piN=1-piS     #initial prevalence non-signalling allele
phiS=0.01     #initial prevalence receiving allele
phiN=1-phiS   #initial prevalence non-receiving allele
zbar=1        #subsistence income
rho=0.5       #output elasticity of labour in subsistence
               sector
alpha=0.5     #output elasticity of labour in luxury
               sector
N=1           #initial population of males and females
A=1           #initial technology in the luxury sector
psi=1         #research productivity per person
B=0.5         #returns to innovation
time<-1000    #number of generations

#Build data frame to store results
Population<-data.frame(time=0, piS, piN, phiS, phiN, sSH=1,
  sSL=1, sNH=1, sNL=1, r=1, gammaSH=1, gammaSL=1, gammaNH=1,
  gammaNL=1, npis=0, npin=0, nphis=0, nphin=0, pLSH=0, pL=0, A,
  g=0, Y=1, cSH=0)

#establish a loop
for (t in 1:time) {

#population composition
piSH=piS*piH
piSL=piS*piL
piNH=piN*piH
piNL=piN*piL

#number of males
MA=N
MASH=piSH*MA
MASL=piSL*MA
MANH=piNH*MA
MANL=piNL*MA

#number of females
FA=N
FAS=phiS*FA
```

```

FAN=phiN*FA
#signalling - determine signalling for male shortage in Stage C,
check male/female ratio and amend if required
sSHtest1=1-(phiS/2)*(hL/hH)
sSHtest2=(1+phiS*(1-piSH)*(1-hL/hH))/(1+phiS*(1-piSH*(1-hL/hH)))
sSH=max(0, if(sSHtest1<piSH-phiN*(1-piSH)/(2*piSH)) sSHtest1
else sSHtest2)
sSL=1
sNH=1
sNL=1

#economic outputs
r=MA^(rho-1) #subsistence sector wage
pLSH=1-sSH #labour force partipation rate of
high-quality signalling males
pL=piSH*(1-sSH) #labour force participation rate for
population
L=MA*piSH*(1-sSH)*hH #labour force efficiency units of
labour
w=A*MA^(alpha-1) #luxury sector wage
cSH=w*hH*(1-sSH) #conspicuous consumption of high-
quality signalling male
Y=((1-piSH)+piSH*sSH)*r+piSH*cSH
#output
g=psi*L^B #Technological progress
A=(1+g)*A #Technology available to next
generation

#survival
gammaSH=sSH*r
gammaSL=sSL*r
gammaNH=sNH*r
gammaNL=sNL*r

#surviving males
MBSH=MASH*gammaSH
MBSL=MASL*gammaSL
MBNH=MANH*gammaNH
MBNL=MANL*gammaNL
MB=MBSH+MBSL+MBNH+MBNL

#proportion surviving by type
piSH=MBSH/MB
piSL=MBSL/MB
piNH=MBNH/MB
piNL=MBNL/MB

```

```

#proportion of males to females in Stage B
qFB=MB/FA

#number of children in Stage B
npiSB=(piSH*phiS*hH+piSH*phiN*hH+piSL*phiN*hL)*MB
npiNB=(piNH*hH+piNL*hL)*phiN*MB
nphiSB=(piSH*phiS*hH)*MB
nphiNB=phiN*(piSH*hH+piSL*hL+piNH*hH+piNL*hL)*MB

#number of females available in Stage C
FC=max(0,FA*(phiS*(1-qFB*piSH)+phiN)*(1-qFB))
MC=MB

#number of females of each type:
FCS=FAS*(1-qFB*piSH)
FCN=FAN*(1-qFB)

#proportion of females of each type
phiS=FCS/FC
phiN=FCN/FC

#number of children in Stage C
npiSC=(piSH*hH+piSL*hL)*min(MC,FC)
npiNC=(piNH*hH+piNL*hL)*min(MC,FC)
nphiSC=phiS*((piSH+piNH)*hH+(piSL+piNL)*hL)*min(MC,FC)
nphiNC=phiN*((piSH+piNH)*hH+(piSL+piNL)*hL)*min(MC,FC)

#total number of children of each type
npiS=npiSB+npiSC
npiN=npiNB+npiNC
nphiS=nphiSB+nphiSC
nphiN=nphiNB+nphiNC

#NEXT GENERATION
#proportion of each type in next generation
piS=npiS/(npiS+npiN)
piN=npiN/(npiS+npiN)
phiS=nphiS/(nphiS+nphiN)
phiN=nphiN/(nphiS+nphiN)

#population in next generation
MA=N
FA=N

#Bind the new generation of results to the data frame
Population<-rbind(Population, c(t, piS, piN, phiS, phiN, sSH,
sSL, sNH, sNL, r, gammaSH, gammaSL, gammaNH, gammaNL, npiS,
npiN, nphiS, nphiN, pLSH, pL, A, g, Y, cSH))

```

```
#close loop  
}  
Population
```

Chapter 6 computer code

The simulations in Chapter 6 were conducted using R (R Development Core Team 2010), a free language and environment for statistical computing. The software is available from <http://www.r-project.org>

6C.1 A diploid model of human fertility

```
#Conditions at t0 to be set by user
h<-0          #Set as 0 if the high fertility allele is
              #dominant, 1 if the allele is recessive
piHigh<-0.4   #Proportion of high fertility phenotypes at t=0
N0<-1         #Population at t=0
FRB<-3.6      #Fertility rate before the shock
FR0<-1.8      #Fertility rate at time of the shock (t=0)

#Other conditions at t0 derived by user set conditions
#Frequency of alleles and genotypes
if(h==0) {piL0<-(1-piHigh)^0.5}
if(h==1) {piL0<-1-piHigh^0.5}
piH0<-1-piL0
piHH0<-piH0^2
piHL0<-2*piH0*piL0
piLL0<-piL0^2
#Number of each genotype
NHH0<-(piH0^2)*N0
NHL0<-2*piH0*piL0*N0
NLL0<-(piL0^2)*N0
#Fertility of each genotype
nHH<-FRB
if (h==0) nLL<-(FR0*N0-nHH*(NHL0+NHH0))/NLL0
if (h==1) nLL<-(FR0*N0-nHH*NHH0)/(NLL0+NHL0)
if (h==0) nHL<-nHH
if (h==1) nHL<-nLL

#Generate data for generations before shock
#Data for generation t=-5
NHH<-NHH0*(FRB/2)^(-5)
NHL<-NHL0*(FRB/2)^(-5)
NLL<-NLL0*(FRB/2)^(-5)
N<-NHH+NHL+NLL
piHH<-NHH/N
piHL<-NHL/N
```



```

piLL<-NLL/N

#Build data frame which will be used to store results
time<-(-1)          #last generation for which we want
data in this loop
Growth<-data.frame(time=(-5), piHH, piHL, piLL, NHH, NHL, NLL,
N, FR=FR0)

#Establish a loop
for (t in -4:time) {

#Change in population each generation
NHH<-NHH*(FRB/2)
NHL<-NHL*(FRB/2)
NLL<-NLL*(FRB/2)
N<-NHH+NHL+NLL

#Frequency of each type
piHH<-NHH/N
piHL<-NHL/N
piLL<-NLL/N

#Fertility rate of population
FR<-FRB

Growth<-rbind(Growth, c(t, piHH, piHL, piLL, NHH, NHL, NLL, N,
FR))

}

#Simulate period after the shock
#Parameters at t=0 (need to refresh)
NHH<-NHH0
NHL<-NHL0
NLL<-NLL0
N<-N0
piHH<-piHH0
piHL<-piHL0
piLL<-piLL0
FR<-FR0

t<-0
Growth<-rbind(Growth, c(t, piHH, piHL, piLL, NHH, NHL, NLL, N,
FR))

time<-20 #number of generations after the fertility shock

```

```

#Establish a loop
for (t in 1:time) {

# number of children by each type
NHH<-
0.5*((piHH^2)*nHH+piHH*piHL*(nHH+nHL)/2+0.25*(piHL^2)*nHL)*N
NHL<-
0.5*(piHH*piHL*(nHH+nHL)/2+piHL*piLL*(nHL+nLL)/2+0.5*(piHL^2)*n
HL+2*piHH*piLL*(nHH+nLL)/2)*N
NLL<-
0.5*((piLL^2)*nLL+piHL*piLL*(nHL+nLL)/2+0.25*(piHL^2)*nHL)*N
N<-NHH+NHL+NLL

#Frequency of each type
piHH<-NHH/N
piHL<-NHL/N
piLL<-NLL/N

#Fertility rate of population
FR<-nHH*piHH+nHL*piHL+nLL*piLL

Growth<-rbind(Growth, c(t, piHH, piHL, piLL, NHH, NHL, NLL, N,
FR))

}

Growth

```

6C.2 A model of heritability of fertility

```

#Conditions at t0 to be set by user
h2<-0.2 #Heritability
n<-10 #Maximum family size (note - need to
add additional code below if want to change this from n<-10)
u<-1.8 #Mean family size at t=0
N<-1 #Population at t=0

#Other conditions at t0 derived by user set conditions
#Derive probability and variance for binomial distribution from
initial conditions
p<-u/n
var<-n*p*(1-p)

#The frequency of families with i children in generation t=0
f0<-dbinom(0, size=n, prob=p)
f1<-dbinom(1, size=n, prob=p)

```

```

f2<-dbinom(2, size=n, prob=p)
f3<-dbinom(3, size=n, prob=p)
f4<-dbinom(4, size=n, prob=p)
f5<-dbinom(5, size=n, prob=p)
f6<-dbinom(6, size=n, prob=p)
f7<-dbinom(7, size=n, prob=p)
f8<-dbinom(8, size=n, prob=p)
f9<-dbinom(9, size=n, prob=p)
f10<-dbinom(10, size=n, prob=p)

#Proportion of children who are in a family with i children
pi0<-0
pi1<-1*f1/u
pi2<-2*f2/u
pi3<-3*f3/u
pi4<-4*f4/u
pi5<-5*f5/u
pi6<-6*f6/u
pi7<-7*f7/u
pi8<-8*f8/u
pi9<-9*f9/u
pi10<-10*f10/u

#Mean number of children each child has in its family
i<-c(0,1,2,3,4,5,6,7,8,9,10)
pi<-c(pi0,pi1,pi2,pi3,pi4,pi5,pi6,pi7,pi8,pi9,pi10)
c<-sum(i*pi)

#Build data frame which will be used to store results
time<-20                                #Number of generations following the
fertility shock
Growth<-data.frame(time=0, u, p, var, N, f0, f1, f2, f3, f4, f5,
f6, f7, f8, f9, f10, pi0, pi1, pi2, pi3, pi4, pi5, pi6, pi7,
pi8, pi9, pi10, c)

#Establish a loop
for (t in 1:time) {

#Update parameters based on last generation
N<-N*u
u<-u+h2*(c-u)
p<-u/n
var<-n*p*(1-p)

#The frequency of families with i children
f0<-dbinom(0, size=n, prob=p)
f1<-dbinom(1, size=n, prob=p)
f2<-dbinom(2, size=n, prob=p)

```

```

f3<-dbinom(3, size=n, prob=p)
f4<-dbinom(4, size=n, prob=p)
f5<-dbinom(5, size=n, prob=p)
f6<-dbinom(6, size=n, prob=p)
f7<-dbinom(7, size=n, prob=p)
f8<-dbinom(8, size=n, prob=p)
f9<-dbinom(9, size=n, prob=p)
f10<-dbinom(10, size=n, prob=p)

#Proportion of children who are in a family with i children
pi0<-0
pi1<-1*f1/u
pi2<-2*f2/u
pi3<-3*f3/u
pi4<-4*f4/u
pi5<-5*f5/u
pi6<-6*f6/u
pi7<-7*f7/u
pi8<-8*f8/u
pi9<-9*f9/u
pi10<-10*f10/u

#Mean number of children each child has in its family
i<-c(0,1,2,3,4,5,6,7,8,9,10)
pi<-c(pi0,pi1,pi2,pi3,pi4,pi5,pi6,pi7,pi8,pi9,pi10)
c<-sum(i*pi)

Growth<-rbind(Growth, c(t, u, p, var, N, f0, f1, f2, f3, f4, f5,
f6, f7, f8, f9, f10, pi0, pi1, pi2, pi3, pi4, pi5, pi6, pi7,
pi8, pi9, pi10, c))

}

Growth

```

6C.3 A haploid age-structured model

```

#Conditions at t0 to be set by user
#Fertility rate of each age cohort
n0<-0      #number children born by females age cohort 0-4
n5<-0      #number children born by females age cohort 5-9
n10<-0     #number children born by females age cohort 10-14
n15<-0.1   #number children born by females age cohort 15-19
n20<-0.3   #number children born by females age cohort 20-24
n25<-0.6   #number children born by females age cohort 25-29
n30<-0.5   #number children born by females age cohort 30-34

```

```

n35<-0.2      #number children born by females age cohort 35-39
n40<-0.1      #number children born by females age cohort 40-44
n45<-0        #number children born by females age cohort 45-49
n50<-0        #number children born by females age cohort 50-54
n55<-0        #number children born by females age cohort 55-59
n60<-0        #number children born by females age cohort 60-64
n65<-0        #number children born by females age cohort 65-69
n70<-0        #number children born by females age cohort 70-74
n75<-0        #number children born by females age cohort 75-79
n80<-0        #number children born by females age cohort 80-84
n85<-0        #number children born by females age cohort 85-89
n90<-0        #number children born by females age cohort 90-94
n95<-0        #number children born by females age cohort 95-99
n100<-0       #number children born by females age cohort 100+
n<-
c(n0,n5,n10,n15,n20,n25,n30,n35,n40,n45,n50,n55,n60,n65,n70,n75
,n80,n85,n90,n95,n100)  #Vector of fertility
r<-3.0           #Ratio of fertility between high and
low types
TFR<-sum(n)      #Total fertility rate

#High and low fertility types
H<-0.2           #Initial proportion high fertility types
L<-1-H           #Initial proportion low fertility types
R<-3             #Ratio of high to low fertility
s<-1/(1-R)       #Selection coefficient
LTFR<-1/(H*R+L)  #FR of low fertility type relative to
initial TFR
HTFR<-LTFR*R     #FR of high fertility type relative to
initial TFR
nL<-LTFR*n       #Vector of FR of low fertility types
nH<-HTFR*n       #Vector of FR of high fertility types
FRL<-sum(nL)     #FR of low fertility types
FRH<-sum(nH)     #FR of high fertility types

#Death schedule
d0<-0.005        #death rate for age cohort 0-4
d5<-0.0005       #death rate for age cohort 5-9
d10<-0.0005      #death rate for age cohort 10-14
d15<-0.002       #death rate for age cohort 15-19
d20<-0.002       #death rate for age cohort 20-24
d25<-0.003       #death rate for age cohort 25-29
d30<-0.004       #death rate for age cohort 30-34
d35<-0.005       #death rate for age cohort 35-39
d40<-0.006       #death rate for age cohort 40-44
d45<-0.01        #death rate for age cohort 45-49
d50<-0.01        #death rate for age cohort 50-54
d55<-0.02        #death rate for age cohort 55-59

```

```

d60<-0.03          #death rate for age cohort 60-64
d65<-0.05          #death rate for age cohort 65-69
d70<-0.09          #death rate for age cohort 70-74
d75<-0.15          #death rate for age cohort 75-79
d80<-0.26          #death rate for age cohort 80-84
d85<-0.42          #death rate for age cohort 85-89
d90<-0.62          #death rate for age cohort 90-94
d95<-0.77          #death rate for age cohort 95-99
d100<-1            #death rate for age cohort 100+
d<-
c(d0,d5,d10,d15,d20,d25,d30,d35,d40,d45,d50,d55,d60,d65,d70,d75
,d80,d85,d90,d95,d100) #Vector of death

#Starting population
N0<-0.100          #number of age cohort 0-4
N5<-0.100          #number of age cohort 5-9
N10<-0.095         #number of age cohort 10-14
N15<-0.075         #number of age cohort 15-19
N20<-0.065         #number of age cohort 20-24
N25<-0.060         #number of age cohort 25-29
N30<-0.070         #number of age cohort 30-34
N35<-0.070         #number of age cohort 35-39
N40<-0.065         #number of age cohort 40-44
N45<-0.060         #number of age cohort 45-49
N50<-0.055         #number of age cohort 50-54
N55<-0.045         #number of age cohort 55-59
N60<-0.040         #number of age cohort 60-64
N65<-0.035         #number of age cohort 65-69
N70<-0.025         #number of age cohort 70-74
N75<-0.015         #number of age cohort 75-79
N80<-0.010         #number of age cohort 80-84
N85<-0.010         #number of age cohort 85-89
N90<-0.003         #number of age cohort 90-94
N95<-0.002         #number of age cohort 95-99
N100<-0            #number of age cohort 100+
N<-
c(N0,N5,N10,N15,N20,N25,N30,N35,N40,N45,N50,N55,N60,N65,N70,N75
,N80,N85,N90,N95,N100) #Vector of population
Ntot<-sum(N)
NL<-N*L
NH<-N*H
NLtot<-sum(NL)
NHtot<-sum(NH)

#Build data frame which will be used to store results
time<-50           #Number of five-year periods following
the fertility shock

```

```

Growth<-
  data.frame(time=0,TFR,N0,N5,N10,N15,N20,N25,N30,N35,N40,N45,N50
    ,N55,N60,N65,N70,N75,N80,N85,N90,N95,N100,Ntot)

#Establish a loop
for (t in 1:time) {

#Births in last generation
XL<-0.5*NL*nL
XH<-0.5*NH*nH
XLtot<-0.5*sum(NL*nL)
XHtot<-0.5*sum(NH*nH)

#Deaths in last generation
dL<-NL*d
dH<-NH*d

#Population after deaths
NL<-NL-dL
NH<-NH-dH

#Age population 5 years and add births to population vector
NL<-NL[-21]
NL<-c(XLtot,NL)
NH<-NH[-21]
NH<-c(XHtot,NH)
N<-NL+NH
Ntot<-sum(NH)+sum(NL)
L<-sum(NL)/Ntot
H<-sum(NH)/Ntot

#TFR of this new population
TFR<-sum((NL*nL+NH*nH)/N)

Growth<-rbind(Growth, c(t, TFR,N,Ntot))

}

Growth

```

6C.4 Galor and Moav model - base case simulation

```
#load BB package - used for solving nonlinear equations
library(BB)

#initial conditions
A<-1
g<-0
ea<-0
eb<-0
e<-0
La<-0.007
Lb<-0.7
L<-La+Lb
qa<-La/L
qb<-Lb/L
za<-1.25
zb<-1.25
na<-1
nb<-1

#parameters
Ba<-1
Bb<-0.9
alpha<-0.4
Tau<-0.2
rho<-0.99
a<-rho*Tau
m<-2
gamma<-0.259
k<-8.885139596
r<-0.108150721
X<-1
sc<-1
zsc<-sc/(1-gamma)
time<-200 #number of generations

#Build data frame which will be used to store results
Growth<-data.frame(time=0, A, g, ea, eb, La, Lb, na, nb, za, zb)

#establish a loop
for (t in 1:time) {
```



```

# population
La<-na*La
Lb<-nb*Lb
L<-La+Lb
qa<-La/L
qb<-Lb/L

# technology in this period (based on education given to
  children in last period)
e<-qa*ea+qb*eb
g<-k*e^0.5
A<-(1+g)*A

# human capital
ha<-(m*ea+a)/(ea+r*g+a)
hb<-(m*eb+a)/(eb+r*g+a)

#level of eduction of each genotype
ea<-max(0, (1/(2*m))*((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)+(((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)^2+4*m*(Ba*m*r*g*Tau+Ba*m*a*Tau-Ba*a*Tau-a*r*g-a^2))^0.5)))
eb<-max(0, (1/(2*m))*((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)+(((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)^2+4*m*(Bb*m*r*g*Tau+Bb*m*a*Tau-Bb*a*Tau-a*r*g-a^2))^0.5)))

#calculate possibilities for income for genotype a (and given
  zb=za*hb/ha)
#if subsistence constraint binding for neither
zaa1b1<-(((A*X)/(L*((1-gamma)*(qa*ha+(1-qa)*hb))))^alpha)*ha
zba1b1<-zaa1b1*hb/ha
if(zaa1b1>=zsc & zba1b1>=zsc) za<-zaa1b1

#if subsistence constraint binding for type b
fzaa1b2<-function(zaa1b2){
y<-(((A*X)/(L*(((1-gamma)*qa*ha)+(1-
qa)*((sc*ha)/zaa1b2))))^alpha)*ha-zaa1b2
y
}
ansfzaa1b2<-multiStart(c(0.2,1,10,100),fzaa1b2,
  control=list(M=200), quiet=TRUE)
zaa1b2<-max(ansfzaa1b2$par[,1], ansfzaa1b2$par[,2],
  ansfzaa1b2$par[,3], ansfzaa1b2$par[,4])
zba1b2<-zaa1b2*hb/ha
if(zaa1b2>=zsc & zba1b2>=sc & zba1b2<zsc) za<-zaa1b2

```

```

#if subsistence constraint binding for both types
fzaa2b2<-function(zaa2b2){
y<-((((A*X)/(L*(((sc/zaa2b2)*qa*ha)+(1-
qa)*((sc*ha)/zaa2b2))))^alpha)*ha-zaa2b2
y
}
ansfzaa2b2<-multiStart(c(0.2,1,10,100),fzaa2b2,
  control=list(M=200), quiet=TRUE)
zaa2b2<-max(ansfzaa2b2$par[,1],               ansfzaa2b2$par[,2],
  ansfzaa2b2$par[,3], ansfzaa2b2$par[,4])
zba2b2<-zaa2b2*hb/ha
if(zaa2b2>=sc & zaa2b2<zsc & zba2b2>=sc & zba2b2<zsc) za<-zaa2b2
if(zaa2b2<sc) za<-0

#given income for genotype a, calculate income for genotype b
zb<-za*hb/ha

#population growth
if(za>=zsc) na<-gamma/(Tau+ea)
if(za<zsc & za>sc) na<-(1-(sc/za))/(Tau+ea)
if(za<=sc) na<-0
if(zb>=zsc) nb<-gamma/(Tau+eb)
if(zb<zsc & za>sc) nb<-(1-(sc/zb))/(Tau+eb)
if(zb<=sc) nb<-0

Growth<-rbind(Growth, c(t, A, g, ea, eb, La, Lb, na, nb, za,
zb))

}

Growth

```

6C.5 Galor and Moav model - simulation with three genotypes

```

#load BB package - used for solving nonlinear equations
library(BB)

#initial conditions
A<-1
g<-0
ea<-0
eb<-0
ec<-0
e<-0
La<-0.007
Lb<-0.7

```

```

Lc<-0.007
L<-La+Lb+Lc
qa<-La/L
qb<-Lb/L
qc<-Lc/L
za<-1.25
zb<-1.25
zc<-1.25
na<-1
nb<-1
nc<-1

#parameters
Ba<-1
Bb<-0.9
Bc<-0.75
alpha<-0.4
Tau<-0.2
rho<-0.99
a<-rho*Tau
m<-2
gamma<-0.259
k<-8.885139596
r<-0.108150721
X<-1
sc<-1
zsc<-sc/(1-gamma)
time<-2000 #number of generations

#Build data frame which will be used to store results
Growth<-data.frame(time=0, A, g, ea, eb, ec, La, Lb, Lc, na, nb,
  nc, za, zb, zc)

#establish a loop
for (t in 1:time) {

# population
La<-na*La
Lb<-nb*Lb
Lc<-nc*Lc
L<-La+Lb+Lc
qa<-La/L
qb<-Lb/L
qc<-Lc/L

# technology in this period (based on education given to
  children in last period)
e<-qa*ea+qb*eb+qc*ec

```

```

g<-k*e^0.5
A<-(1+g)*A

# human capital
ha<-(m*ea+a)/(ea+r*g+a)
hb<-(m*eb+a)/(eb+r*g+a)
hc<-(m*ec+a)/(ec+r*g+a)

#level of education of each genotype
ea<-max(0, (1/(2*m))*((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)+(((Ba*m*r*g+Ba*m*a-Ba*a-m*r*g-a*m-
a)^2+4*m*(Ba*m*r*g*Tau+Ba*m*a*Tau-Ba*a*Tau-a*r*g-a^2))^0.5)))
eb<-max(0, (1/(2*m))*((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)+(((Bb*m*r*g+Bb*m*a-Bb*a-m*r*g-a*m-
a)^2+4*m*(Bb*m*r*g*Tau+Bb*m*a*Tau-Bb*a*Tau-a*r*g-a^2))^0.5)))
ec<-max(0, (1/(2*m))*((Bc*m*r*g+Bc*m*a-Bc*a-m*r*g-a*m-
a)+(((Bc*m*r*g+Bc*m*a-Bc*a-m*r*g-a*m-
a)^2+4*m*(Bc*m*r*g*Tau+Bc*m*a*Tau-Bc*a*Tau-a*r*g-a^2))^0.5)))

#calculate possibilities for income for genotype a
#if subsistence constraint binding for noone
zaa1b1c1<-(((A*X)/(L*(1-gamma)*(qa*ha+qb*hb+qc*hc)))^alpha)*ha
zba1b1c1<-zaa1b1c1*hb/ha
zca1b1c1<-zaa1b1c1*hc/ha
if(zaa1b1c1>=zsc & zba1b1c1>=zsc & zca1b1c1>=zsc) za<-zaa1b1c1

#if subsistence constraint binding for type c only
fzaa1b1c2<-function(zaa1b1c2){
y<-(((A*X)/(L*(((1-
gamma)*(qa*ha+qb*hb))+qc*((sc*ha)/zaa1b1c2))))^alpha)*ha-
zaa1b1c2
y
}
ansfzaa1b1c2<-multiStart(c(0.2,1,10,100),fzaa1b1c2,
control=list(M=200), quiet=TRUE)
zaa1b1c2<-max(ansfzaa1b1c2$par[,1], ansfzaa1b1c2$par[,2],
ansfzaa1b1c2$par[,3], ansfzaa1b1c2$par[,4])
zba1b1c2<-zaa1b1c2*hb/ha
zca1b1c2<-zaa1b1c2*hc/ha
if(zaa1b1c2>=zsc & zba1b1c2>=zsc & zca1b1c2>=sc & zca1b1c2<zsc)
za<-zaa1b1c2

#if subsistence constraint binding for type b and c
fzaa1b2c2<-function(zaa1b2c2){
y<-(((A*X)/(L*(((1-
gamma)*qa*ha)+qb*((sc*ha)/zaa1b2c2)+qc*((sc*ha)/zaa1b2c2))))^alpha
pha)*ha-zaa1b2c2
y

```

```

}
ansfzaa1b2c2<-multiStart(c(0.2,1,10,100),fzaa1b2c2,
  control=list(M=200), quiet=TRUE)
zaa1b2c2<-max(ansfzaa1b2c2$par[,1],          ansfzaa1b2c2$par[,2],
  ansfzaa1b2c2$par[,3], ansfzaa1b2c2$par[,4])
zba1b2c2<-zaa1b2c2*hb/ha
zca1b2c2<-zaa1b2c2*hc/ha
if(zaa1b2c2>=zsc & zba1b2c2>=sc & zba1b2c2<zsc & zca1b2c2>=sc &
  zca1b2c2<zsc) za<-zaa1b2c2

#if subsistence constraint binding for all types
fzaa2b2c2<-function(zaa2b2c2){
y<-(((A*X)/(L*(((sc/zaa2b2c2)*qa*ha)+(1-
  qa)*((sc*ha)/zaa2b2c2))))^alpha)*ha-zaa2b2c2
y
}
ansfzaa2b2c2<-multiStart(c(0.2,1,10,100),fzaa2b2c2,
  control=list(M=200), quiet=TRUE)
zaa2b2c2<-max(ansfzaa2b2c2$par[,1],          ansfzaa2b2c2$par[,2],
  ansfzaa2b2c2$par[,3], ansfzaa2b2c2$par[,4])
zba2b2c2<-zaa2b2c2*hb/ha
zca2b2c2<-zaa2b2c2*hc/ha
if(zaa2b2c2>=sc & zaa2b2c2<zsc & zba2b2c2>=sc & zba2b2c2<zsc)
  za<-zaa2b2c2
if(zaa2b2c2<sc) za<-0

#given income for genotype a, calculate income for genotype b
zb<-za*hb/ha
zc<-za*hc/ha

#population growth
if(za>=zsc) na<-gamma/(Tau+ea)
if(za<zsc & za>sc) na<-(1-(sc/za))/(Tau+ea)
if(za<=sc) na<-0
if(zb>=zsc) nb<-gamma/(Tau+eb)
if(zb<zsc & zb>sc) nb<-(1-(sc/zb))/(Tau+eb)
if(zb<=sc) nb<-0
if(zc>=zsc) nc<-gamma/(Tau+ec)
if(zc<zsc & zc>sc) nc<-(1-(sc/zc))/(Tau+ec)
if(zc<=sc) nc<-0

Growth<-rbind(Growth, c(t, A, g, ea, eb, ec, La, Lb, Lc, na, nb,
  nc, za, zb, zc))

}

```

Growth