On market forces and human evolution

Gilles Saint-Paul*

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ABSTRACT: This paper studies how an institution such as markets affects the evolution of mankind. My key point is that the forces of natural selection are made weaker because trade allows people to specialize in those activities where they are strong, and to offset their weaknesses by purchasing adequate goods on the market. Absent trade, people must allocate their time among all the activities necessary for their fitness. A fitness advantage in any given dimension will increase survival probability, so that in the long run natural selection makes sure that population is entirely made of individuals with the best alleles at all locations. Under trade, there exist long-run equilibria where less fit individuals are able to achieve the same survival potential as the fittest, by specializing in activities where they are not at a disadvantage, and purchasing goods that are substitute for activities for which they are 'weak'.

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*Université de Toulouse I and CEPR. This paper has benefitted from discussions with Paul Seabright and Thierry Verdier.

1 Introduction

Basic Darwinian theory considers individuals competing for resources in order to achieve their goals of fitness and reproduction. Its more recent developments have considered cooperation and altruism as the outcome of selective forces at the gene level, which typically leave room for cooperation at a very small scale, within a family or tribe of genetically close individuals. Yet our species has developed institutions which allow cooperation on a much larger scale.[1] Markets, in particular, allow people to specialize according to their comparative advantage and to purchase goods needed for fitness and survival from other participants. Clearly, most of us would disappear if left alone facing the forces of natural selection; world population would shrink considerably if trade among individuals were to suddenly collapse.

This paper studies how an institution such as markets affects the evolution of mankind. My key point is that the forces of natural selection are made weaker because trade allows people to specialize in those activities where they are strong, and to offset their weaknesses by purchasing adequate goods on the market. Absent trade, people must allocate their time among all the activities necessary for their fitness. A fitness advantage in any given dimension will increase survival probability, so that in the long run natural selection makes sure that population is entirely made of individuals with the best alleles at all locations. Under trade, there exist long-run equilibria where less fit individuals are able to achieve the same survival potential as the fittest, by specializing in activities where they are not at a disadvantage, and purchasing goods that are substitute for activities for which they are 'weak'.

Hence, markets allow many genotypes that would be eliminated by natural selection to survive, thus building in greater genetic diversity in human populations. This genetic diversity may be helpful in face of environmental shocks, implying that when such shocks prevail, a population which trades

[1] Interesting surveys on interactions between the economic and biological spheres include Hirshleifer (1977), Robson (2001), and Seabright (2003), forthcoming.

will grow faster than a population which does not, eventually eliminating it in statistical terms.

The model has a variety of implications, as discussed in section 8. It explains why many human genes seem to evolve according to the "neutralist" view, i.e. as if they had no selective properties. It also sheds light, I believe, on recent controversies on intelligence and the economic importance of genes.

Note however that the model is silent about how markets themselves evolve[2]. The literature on gene/culture coevolution is mostly concerned with the genetic basis for adoptions of cultural norms such as altruism (often relying on controversial group selection hypotheses).[3] To my knowledge it has not provided a theory of how complex institutions such as markets evolve. The present paper only looks at causality in one dimension, taking institutions as given and studying their impact on the gene pool.

Another potentially relevant critique is that it is not clear whether trade has been around for long enough to significantly affect evolution. It is often argued that evolution is very slow, and that our genes are essentially determined by the hunter-gatherer societies which prevailed hundreds of thousands of years ago. However, there are two quite different aspects of evolution. The first one is that mutations do not happen frequently, which explains why it takes hundreds of thousands of years for a feature like the human brain to develop. The second one is that an *existing* allele can replace another one quite rapidly. A well-known example is that of the gene for lactose tolerance; the share of the European population with a "tolerant allele" increased from 5 % to 70 % in less than 5,000 years, due to changes in food habits. In contrast, most Asian populations are lactose intolerant because their cultures had not developed dairy farming.[4] It is this kind of evolution that I consider here: the race between existing competing alleles. The message is then that

[2]Interestingly, Adam Smith saw our propensity to trade as a genetic property of our species. See Smith (1994).

[3]See Cavalli-Sforza and Feldman (1981); Lumsden and Wilson (1981); Gintis (2002), Boyd and Richerson (1985).

[4]See e.g. Aoki (1991).

in some sense, markets "slow" evolution, by making alleles less loaded with selective pressure. The example of lactose tolerance suggests that trade can have a significant impact on our gene pool if it has been present for say 10,000 years.

2 The model: population dynamics

This section describes the basic features of the model, and of its demographic implications. It is a simplified representation of genetic evolution in a heterogeneous population. This population has a single chromosome with two loci, indexed by $\lambda = 1, 2$. At each loci there can be one of two competing alleles, indexed by $\kappa = H, L$. Consequently, there are 4 possible genotypes, denoted by HH, HL, LH and LL. Genotype $\kappa\kappa'$ has allele κ at location 1 and allele κ' at location 2.

Each of these genotypes g has a specific mortality rate $\mu(g)$, which refers to the gross outflow per unit of time in the corresponding population. $\mu(g)$ could in principle depend on time or on the total distribution of genotypes in the population. For our purposes, however, it is enough to assume it is constant.

Reproduction is sexual and takes place as follows. At any instant of time people mate randomly. Let N_t be total population at t and n_{gt} be the proportion of genotype g in the population at date t. Then the total number of matches at t is νN_t. Random matching implies that there are $(2n_{gt}n_{g't})\nu N_t$ matches between g and $g' \neq g$, and $n_{gt}^2 \nu N_t$ matches between two people of the same type g.

Each match produces one offspring and at each location the offspring inherits the corresponding gene in its mother's chromosome with probability 1/2, and that corresponding to its father with probability 1/2. The following table gives the distribution of the offspring's genotype as a function of each parent's genotype:

Match/Proportions	HH	HL	LH	LL
$HH + HH$	1	0	0	0
$HH + HL$	0.5	0.5	0	0
$HH + LH$	0.5	0	0.5	0
$HH + LL$	0.25	0.25	0.25	0.25
$HL + HL$	0	1	0	0
$HL + LH$	0.25	0.25	0.25	0.25
$HL + LL$	0	0.5	0	0.5
$LH + LH$	0	0	1	0
$LH + LL$	0	0	0.5	0.5
$LL + LL$	0	0	0	1

Table 1: proportion of offsprings of each genotype as a function of parents' genotype.

This table may then be used to compute the evolution equation of the population's composition:[5]

$$\frac{1}{N_t}\frac{d}{dt}(n_{HH}N_t) = \nu n_{HH}^2 + \nu n_{HH}n_{HL} + \nu n_{HH}n_{LH} + \frac{1}{2}\nu(n_{LH}n_{HL+}n_{HH}n_{LL}) - \mu(HH)n_{HH} \tag{1}$$

$$\frac{1}{N_t}\frac{d}{dt}(n_{HL}N_t) = \nu n_{HH}n_{HL} + \nu n_{HL}^2 + \nu n_{HL}n_{LL} + \frac{1}{2}\nu(n_{LH}n_{HL+}n_{HH}n_{LL}) - \mu(HL)n_{HL} \tag{2}$$

$$\frac{1}{N_t}\frac{d}{dt}(n_{LH}N_t) = \nu n_{LL}n_{LH} + \nu n_{LH}^2 + \nu n_{LH}n_{HH} + \frac{1}{2}\nu(n_{LH}n_{HL+}n_{HH}n_{LL}) - \mu(LH)n_{LH} \tag{3}$$

$$\frac{1}{N_t}\frac{d}{dt}(n_{LL}N_t) = \nu n_{LL}n_{LH} + \nu n_{LL}^2 + \nu n_{LL}n_{HL} + \frac{1}{2}\nu(n_{LH}n_{HL+}n_{HH}n_{LL}) - \mu(LL)n_{LL} \tag{4}$$

[5] These formulas are a simplified version of the biologists's "Hardy-Weinberg" equations.

These equations are non linear. Adding them we get the population growth rate:

$$\frac{\dot{N}}{N} = \nu - \bar{\mu}_t, \tag{5}$$

where $\bar{\mu}$ is the average mortality rate:

$$\bar{\mu}_t = \sum_g n_g \mu(g).$$

Similarly, adding the first two equations we get an evolution equation for the fraction of the population with an $H-$allele at location 1, denoted by $h_1 = n_{HH} + n_{HL}$:

$$\frac{\dot{h}_1}{h_1} = \bar{\mu}_t - \bar{\mu}_{1t}, \tag{6}$$

where

$$\bar{\mu}_{1t} = \frac{n_{HH}\mu(HH) + n_{HL}\mu(HL)}{n_{HH} + n_{HL}}$$

is the average mortality rate of this sub-population.

Thus a gene will grow if it is associated with a higher fitness, i.e. a lower mortality, than the average in the population. Similarly, for the fraction of agents with an H at location 2, we have

$$\frac{\dot{h}_2}{h_2} = \bar{\mu}_t - \bar{\mu}_{2t}.$$

As we shall see below, we assume a positive correlation between the presence of an $H-$allele and fitness. Thus the following set of assumptions must hold:

ASSUMPTIONS A1:

$$\begin{aligned} \mu(HH) &\leq \mu(HL) \\ \mu(HH) &\leq \mu(LH) \\ \mu(HL) &\leq \mu(LL) \\ \mu(LH) &\leq \mu(LL) \end{aligned}$$

Thus, we assume that fitness differences are associated with differences in mortality, while birth rates are identical. Clearly, one could have a more complex model and assume that birth rates as well depend on genotype. While mortality rates are assumed fixed for now, further below they will be the outcome of individual choice.

Most of the results we will derive will be concerned with a "long-run" equilibrium, i.e. a stationary distribution of genotypes.

This section's central result is then the following lemma:

LEMMA 1 – Assume (A1) holds. Then, in any stationary equilibrium such that $\dot{n}_g = 0, \forall g$, *and* $h_i > 0, \forall i$, *one must have*

(i) $n_{HH} = 1$, $n_{HL} = n_{LL} = n_{LH} = 0$ *if* $\mu(HH) < \mu(HL)$ *and* $\mu(HH) < \mu(LH)$.

(ii) $n_{LL} = n_{HL} n_{LH} = 0$ *if* $\mu(LL) > \mu(HH)$

(iii) all surviving genotypes must have the same mortality rate, which is the minimum across all genotypes.

Proof of (i)–Assuming without loss of generality that $\mu(LH) \geq \mu(HL)$, we get that $\frac{\dot{h}_1}{h_1} = \bar{\mu}_t - \bar{\mu}_{1t}$ is positive and bounded away from zero unless $n_{LH} = n_{LL} = 0$. Since h_1 cannot exceed 1, then the latter equalities must hold. Substituting into (1), we see that in steady state we must have $\bar{\mu}_t = \bar{\mu}_{1t} = \mu(HH)$, which only holds if $n_{HL} = 0$.

Proof of (ii)–If $\min(\mu(HL), \mu(LH)) > \mu(HH)$, then (i) applies. Assume then $\min(\mu(HL), \mu(LH)) = \mu(HH)$. Under the same innocuous assumption that $\mu(LH) \geq \mu(HL)$, we have that $\frac{\dot{h}_1}{h_1} = \bar{\mu}_t - \bar{\mu}_{1t}$ is positive and bounded away from zero unless $n_{LL} = 0$ and either $n_{LH} = 0$ or $\mu(LH) = \mu(HL) = \mu(HH)$. In the first case, the proof is complete. In the second case, substituting a common mortality rate and $n_{LL} = 0$ into (1) yields $n_{LH} n_{HL} = 0$.

Proof of (iii)–This derives from the proofs of (i) and (ii). All surviving genotypes have the same mortality, and it is lower than that of nonsurviving

genotypes.

This lemma tells us that, as long as the H-alleles are present in the population, only the fittest types survive. Furthermore, as (ii) implies, HL and LH cannot simultaneously survive–even though they may be as fit as HH– if LL has a strictly lower fitness than HH. This is because they occasionally mate together, thus yielding some LL types which have a lower survival probability. This process tends to drive L alleles out of the gene pool until they have disappeared at at least one location, thus preventing any LL–individuals from arising.

3 Fitness and survival

We now describe how mortality rates are determined. People have a total time endowment (in flow terms) equal to 1. They allocate time between two activities, referred by "f" (fight) and "d" (defence). Furthermore, they have different productivities in each activity, and these productivities are genetically determined. Productivity at the f-acticity is determined by the gene at location 1 on the chromosome, and productivity in d is determined by location 2. More specifically, if the individual has allele H (resp. L) at location 1, his productivity at f is f_H (resp. f_L). Similarly, productivity at activity d is d_H (resp. d_L) for people with allele H (resp. L) at location 2.

Consequently, an individual with genotype $g = \kappa\kappa'$ chooses his fight and defence levels f and d subject to the following time allocation constraint:

$$\frac{f}{f_\kappa} + \frac{d}{d_{\kappa'}} = 1 \tag{7}$$

Mortality is then given by the inverse of "fitness", where fitness is increasing in the total amount of f and d activities:

$$\mu = \frac{1}{\varphi(f,d)}; \varphi_1' > 0, \varphi_2' > 0$$

We shall assume that having an H–allele increases fitness, that is:

$$f_H > f_L$$

$$d_H > d_L$$

Finally, we assume that people set f and d in order to maximize their fitness.[6] Thus they maximize $\varphi(f,d)$ subject to the time allocation constraint (7). In order to avoid analytical problems we shall assume that φ is concave, and satisfies the following conditions:

ASSUMPTIONS A2 –

$$\begin{aligned} \lim_{f\to 0} \frac{\varphi_1'(f,d_0)}{\varphi_2'(f,d_0)} &= +\infty, \quad d_0 > 0 \\ \lim_{d\to 0} \frac{\varphi_2'(f_0,d)}{\varphi_1'(f_0,d)} &= +\infty, \quad f_0 > 0. \end{aligned}$$

To get analytical solutions we shall often use a Leontief fitness function:

$$\varphi(f,d) \equiv \min(f,d)$$

4 Autarky

The preceding section describes an economy without trade. To get the long-run composition of the population is quite simple: The HH type has a more favorable time allocation contraint. Therefore, it is able to achieve a greater fitness. By virtue of Lemma 1, it must be the only remaining type in the long run.

[6] By bluntly making this assumption, we depart from biology and enter economics. A biologist would ask why people should behave like that, and would probably assume the existence of a gene for such behavior, and try to show that it drives out genes for alternative behaviors. This "as if" argument is out of the scope of this paper, and we directly assume maximization of fitness. See Hirschleifer (1977) and Robson (2001) for discussions.

PROPOSITION 1 – The solution to people's maximization problem satisfies (A1) with strict inequalities. Consequently, in any long-run equilibrium:

$$n_{HH} = 1; n_{HL} = n_{LH} = n_{LL} = 0$$

Proof – Straightforward by application of Lemma 1.

In the long run, the fittest gene is "fixed" at both locations. The less fit genes have disappeared. That is in conformity with basic principles of natural selection.

5 Trade

We now introduce the possibility of trade among people and derive its implications for the long-run composition of the population. We now assume that f and d, instead of being activities, are tradeable goods. At any date their price is denoted by p_f and p_d, and it is convenient to normalize this price vector so that

$$p_f + p_d = 1.$$

An equilibrium is then determined the standard way. Each individual of genotype $g = \kappa\kappa'$ determines his allocation of time by maximizing income:

$$\max \; p_f f^S + (1 - p_f) d^S$$

Subject to his time allocation constraint:

$$\frac{f^S}{f_\kappa} + \frac{d^S}{d_{\kappa'}} = 1$$

This determines his supply to the market of goods f and d, $f^S(g, p_f)$ and $d^S(g, p_f)$, as well as his total income $R(g, p_f) = p_f f^S + (1 - p_f) d^S$.

People purchase quantities f^D and d^D of each good on the market, by maximizing $\varphi(f^D, d^D)$ subject to

$$p_f f^D + (1 - p_f) d^D = R(g, p_f). \tag{8}$$

This determines the individual demand functions $f^D(g, p_f)$, and $d^D(g, p_f)$, and the resulting level of mortality $\mu(g, p_f) = \varphi(\ f^D(g, p_f), d^D(g, p_f))^{-1}$. An equilibrium is an allocation and a price vector which are solution to these optimization problems and such that markets clear, i.e.

$$\sum_g n_g f^D(g, p_f) = \sum_g n_g f^S(g, p_f). \tag{9}$$

By Walras' law, if this holds, then the market for d is also in equilibrium. Standard results tell us that, given the current distribution of genotypes $\{n_g\}$, an equilibrium exists and is Pareto optimal, in that an agent's fitness can't be increased without reducing another agent's fitness.

We are now interested in how economic forces affect the long-run genetic composition of the population. For this we introduce the concept of a "Long run equilibrium", which is a situation where the economy is in Walrasian equilibrium and the distribution of genotypes in the population is stationary. The two are interrelated because the economic equilibrium determines the level of fitness of each genotype, which in turn affects its population dynamics.

DEFINITION – A Long-Run Equilibrium (LRE) is an allocation $\{(f_g^S, d_g^S, f_g^D, d_g^D)$, *a price vector* p_f, *and a genotypic distribution* $\{n_g\}$ *such that*

(i) Markets clear, i.e. $f_g^S = f^S(g, p_f)$, $f_g^D = f^D(g, p_f)$, $d_g^S = d^S(g, p_f), d_g^D = d^D(g, p_f)$, *and (9) holds.*

(ii) The genotypic distribution is stationary, i.e. the following equations (SP) *hold:*

$$0 = \nu n_{HH}(n_{HH} - 1 + \bar{\mu} - \mu(HH, p_f)) + \nu n_{HH} n_{HL} + \nu n_{HH} n_{LH} + \frac{1}{2}\nu(n_{LH} n_{HL} + n_{HH} n_{LL})$$

$$0 = \nu n_{HH} n_{HL} + \nu n_{HL}(n_{HL} - 1 + \bar{\mu} - \mu(HL, p_f)) + \nu n_{HL} n_{LL} + \frac{1}{2}\nu(n_{LH} n_{HL+} n_{HH} n_{LL})$$

$$0 = \nu n_{LL} n_{LH} + \nu n_{LH}(n_{LH} - 1 + \bar{\mu} - \mu(LH, p_f)) + \nu n_{LH} n_{HH} + \frac{1}{2}\nu(n_{LH} n_{HL+} n_{HH} n_{LL})$$

$$0 = \nu n_{LL} n_{LH} + \nu n_{LL}(n_{LL} - 1 + \bar{\mu} - \mu(LL, p_f)) + \nu n_{LL} n_{HL} + \frac{1}{2}\nu(n_{LH} n_{HL+} n_{HH} n_{LL}),$$

where

$$\bar{\mu} = \sum_g n_g \mu(g, p_f).$$

We now turn to the central result of this section, which characterizes the properties of an LRE. We first state it formally and prove it, and then discuss it.

PROPOSITION 2 – In any LRE

(i) The price of f must be equal to

$$p_f = \frac{d_H}{d_H + f_H} \tag{10}$$

(ii) The L-allele has disappeared at at least one location.

Proof of (i) – This price makes the HH type indifferent between supplying f and supplying d. Suppose, say, $p_f > \frac{d_H}{d_H+f_H}$. Then HH only supplies f, and so does HL. As for LH, his maximum income does not exceed $\max(p_f f_L, (1-p_f)d_H) < \max(p_f f_L, \frac{d_H f_H}{d_H+f_H}) < p_f f_H$. Consequently, $\mu(LH) > \mu(HH)$. Next, lemma 1, (iii), implies that both LL and LH must have disappeared in equilibrium. But, then total supply of good d is zero, which can't be true because of conditions (A2). A similar line of reasoning holds if $p_f < \frac{d_H}{d_H+f_H}$.

Proof of (ii) – The LL type has a strictly lower income than HH, therefore $\mu(LL) > \mu(HH)$. Applying (ii) in lemma 2 does the rest.

Part (i) of Proposition 2 is striking. It tells us that in an LRE, the price vector is entirely pinned down by the productivity levels of the H alleles, regardless of the shape of the fitness function φ and of the composition of the population. This is a property of any LRE but not of a situation which would just be a Walrasian equilibrium. In other words, p_f may transitorily differ from $\frac{d_H}{d_H+f_H}$ but in the long-run it has to be equal to it.

The intuition is as follows: if the HH type were not indifferent between the two goods, it would specialize in one of them, and any type which supplies the other would be strictly worse-off, i.e. less fit, than HH, since its productivity at doing it is at most as high as that of HH, which strictly prefers not supplying it. Then, all suppliers of this good gradually disappear relative to the rest of the population, and this cannot be in equilibrium.

Part (ii) tells us that one of the two L–alleles has to disappear, because of the LL type acting as a genetic well. What is important, however, is that only one low fitness gene has to disappear, whereas all of them were eventually eliminated under autarky. Trade allows less fit people to specialize in the activity where they can match the best, thus making their genetic deficiencies irrelevant for their survival. Consequently, these inferior genes are passed to the next generation with the same frequency as superior ones, and are no longer eliminated in the long run. The LL-type, on the other hand, has an absolute disadvantage in all activities and as long as mating

between people with an L-gene produces some $LL's$, the L genes gradually disappear in relative terms. But this process stops when H is fixed at one of the two locations, since no new LL is then produced.

It is easy to construct equilibria where the L-allele survives at one location. To do this let us simply take the Leontief fitness function $\varphi(f,d) \equiv \min(f,d)$. Let us construct an LRE where $n_{HH} > 0$ and $n_{HL} > 0$. Since HH is indifferent between the two activities, HL must specialize in f. Thus its supply of f is $n_{HL}f_H$, and its income is $R(HL, \frac{d_H}{d_H+f_H}) = \frac{d_H f_H}{d_H+f_H}$. Its demand for f is then obtained by plugging $f = d$ in its budget constraint (8). We get

$$f^D(HL) = d^D(HL) = \frac{d_H f_H}{d_H + f_H}$$

The income of HH is also equal to $\frac{d_H f_H}{d_H+f_H}$ and its demand is therefore the same:

$$f^D(HH) = d^D(HH) = \frac{d_H f_H}{d_H + f_H}$$

To get an equilibrium it must be that the total demand for f exceeds what HL is supplying. The difference is then supplied by HH. Thus it must be that:

$$\frac{d_H f_H}{d_H + f_H}(n_{HH} + n_{HL}) \geq n_{HL}f_H,$$

or equivalently

$$n_{HL} < \frac{d_H}{d_H + f_H}. \tag{11}$$

This condition is necessary and sufficient for an economic equilibrium. Then noting that HL and HH have the same mortality rate, one can trivially check that the stationarity conditions (SP) are satisfied. In fact any initial distribution satisfying $n_{LH} = n_{LL} = 0$ and (11) will indefinitely reproduce itself, without any transitional dynamics.

Thus, as long as the proportion of HL in the population is not too high, the economy can be in an LRE with stationary proportions of each type.

In such an equilibrium, fitness is simply equal to $f = d = R = \frac{d_H f_H}{d_H + f_H}$. Thus mortality is equal to

$$\mu(HH) = \mu(HL) = \bar{\mu} = \frac{d_H f_H}{d_H + f_H}. \tag{12}$$

Note also that the RHS of (11) is greater, the greater the maximum productivity level in the $d-$activity relative to the $f-$activity. When d_H/f_H is large, only a few people are needed to produce society's demand for the $d-$good (because this demand is inelastic due to the complementarity between the two activities in producing fitness). Since all of these people must be of genotype HH in equilibrium, equilibrium exists if n_{HH} is large enough relative to the required number of people who must produce d. This is more likely to be the case, the smaller this number, i.e. the greater d_H/f_H.

Similarly for an LRE with HH and LH in the population, a necessary and sufficient condition is

$$n_{LH} < \frac{f_H}{d_H + f_H}.$$

While Proposition 2 has characterized the equilibrium in terms of prices and genotypic composition, the following one compares trade and autarky in terms of total population.

PROPOSITION 3 – (i) The long-run population growth rate is the same under trade and autarky

(ii) For given initial conditions, population at any subsequent date is larger under trade than autarky.

Proof – (i) follows from the observation that average mortality is the same under both cases. To see this, note that the income of any type under trade

must be the same as that of HH, i.e. $d_H f_H/(d_H + f_H)$. Consequently, the budget constraint under trade, i.e.

$$\frac{d_H f^D}{d_H + f_H} + \frac{f_H d^D}{d_H + f_H} = \frac{d_H f_H}{d_H + f_H},$$

is equivalent to the HH type's time allocation constraint under autarky, i.e.

$$\frac{f}{f_H} + \frac{d}{d_H} = 1.$$

Thus all types choose the same fitness as the HH type under autarky, yielding the same long-run population growth rate. To prove (ii), just note that for any given type, fitness is always higher under trade than under autarky.

Proposition 3 clearly ignores phenomena such as the demographic transition, when increased fitness may mean lower population growth (See Galor and Moav (2000) for an analysis). Such effects could be reintroduced by endogenizing birth rates.

6 A generalization

This section generalizes the preceding results by analyzing the case of a chromosome with more than 2 loci. To do so, we must first introduce some notations, and establish some basic results regarding the structure of genotypic distribution in a steady state, independently of how fitness is influenced by markets.

6.1 Notations and genetic properties of stationary populations

There are q loci and at each locus 2 possible alleles, denoted by H and L. A genotype g is a sequence $(\kappa_1, ..., \kappa_q)$ of genes with $\kappa_i \in \{H, L\}$. Given g, we denote by $g[i]$ its allele at locus i.

A key task in extending the previous section's results is to put enough structure on the ordering of different genotypes with respect to fitness. Thus,

we define the genetic downgrading operator at locus i as associating to each genotype g a genotype $T_i g$ such that $T_i g[j] = g[j]$, $j \neq i$, $T_i g[i] = L$. Similarly one can downgrade over any subset S of loci, by using the operator T_S defined by

$$T_S g = \bigcirc_{i \in S} T_i g$$

Similarly the genetic upgrading operator U_i is defined by $U_i g[j] = g[j], j \neq i$, $U_i g[i] = H$. One has $U_i T_i = U_i$ and $T_i U_i = T_i$.

As previously, we assume that an H-allele favors fitness. That is, each genotype g has a specific mortality rate $\mu(g)$ and the following inequality holds:

$$\mu(T_i g) \geq \mu(g). \forall g$$

Or, equivalently[7]

$$\mu(U_i g) \leq \mu(g). \forall g \tag{13}$$

We denote by $g_{\max} = \{H, ...H\}$ the fittest possible genotype, and by $g_{\min} = \{L, ..., L\}$ the least fit.

Consider now two genotypes, g and g'. We denote by $g \cdot g'$ the number of loci where alleles differ between g and g'. Then, $g \otimes g'$ is defined as the set of possible offsprings of g and g'. It is obtained by considering all the possible combinations of alleles at the locations where they differ in the two genotypes. That is:

$$g \otimes g' = \{g'' \text{ s.t. } g''[i] = g[i] \vee g''[i] = g'[i], \forall i\} \tag{14}$$

[7] To see that these two conditions are equivalent, assume that the first one holds and that the second does not. Then for some $g, \mu(U_i g) > \mu(g)$. Therefore, $U_i g \neq g$, implying $g[i] = L$. But, then $T_i U_i g = g$, and $\mu(g) = \mu(T_i U_i g) \geq \mu(U_i g)$, which is clearly a contradiction. Symmetrical reasoning proves that if the second condition holds, the first must also hold.

There are $2^{g \cdot g'}$ such combinations, and each of them comes out with probability $1/2^{g \cdot g'}$.

Let g'', and g be an arbitrary genotype. We denote by $g'' * g$ the set of mates of g which may possibly yield g'' as an offspring. That is:

$$g'' * g = \{g' \text{ s.t. } g''[i] = g[i] \vee g''[i] = g'[i], \forall i\}.$$

The "star" and "upgrade" operators are linked by the following useful property:

PROPERTY P1 – If $g[i] = L$ and $g'[i] = H$, then

$$g * T_i g' = U_i g * g'.$$

PROOF – This is equivalent to proving the following:

$$g \in g'' \otimes T_i g' \Longleftrightarrow U_i g \in g'' \otimes g',$$

for any g''. To see this, just note that for $j \neq i$, $T_i g'[j] = g'[j]$ and $U_i g[j] = g[j]$, therefore $(g''[j] = g[j] \vee T_i g'[j] = g[j]) \Leftrightarrow (g''[j] = U_i g[j] \vee U_i g[j] = g'[j])$. Finally, note that $g[i] = L$ and $g'[i] = H$, so that both $(g''[i] = g[i] \vee T_i g'[i] = g[i])$ and $(g''[i] = U_i g[i] \vee U_i g[i] = g'[i])$ are always true. Q.E.D.

With this notational apparatus, we are now ready to extend the population analysis of section 2. The distribution of genotypes evolves according to the following equation:

$$\frac{1}{N_t}\frac{d}{dt}(n_g N_t) = \nu \sum_{g'} \sum_{g'' \in g * g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} - \mu(g) n_g \tag{15}$$

Summing over all types, we get that the population growth rate still obeys (5):

$$\begin{aligned}
\frac{1}{N_t}\frac{d}{dt}N_t &= \nu \sum_{g} \sum_{g'} \sum_{g'' \in g * g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} - \sum_{g} \mu(g) n_g \\
&= \nu \sum_{g''} \sum_{g'} \sum_{g \in g' \otimes g''} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} - \sum_{g} \mu(g) n_g \\
&= \nu \sum_{g''} n_{g''} \sum_{g'} n_{g'} - \sum_{g} \mu(g) n_g \\
&= \nu - \bar{\mu}_t,
\end{aligned}$$

where $\bar{\mu}_t$ is again the average mortality rate in the population:

$$\bar{\mu}_t = \sum_g \mu(g) n_g.$$

Equations such as (6) still hold. Let $h_i = \sum_{g,g[i]=H} n_g$ the share of the population with the H-allele at locus i. Let

$$\bar{\mu}_i = \frac{\sum_{g,g[i]=H} \mu(g) n_g}{h_i}.$$

Then, summing (15) over all types such that $g[i] = H$, we get:

$$\begin{aligned}
\dot{h}_i + (\nu - \bar{\mu}_t) h_i &= \nu \sum_{g,g[i]=H} \sum_{g'} \sum_{g'' \in g * g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} - \sum_{g,g[i]=H} \mu(g) n_g \\
&= \nu \left[\begin{array}{c} \sum_{g',g'[i]=L} \sum_{g'',g''[i]=H} \frac{1}{2} \sum_{g \in g' \otimes g''} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ + \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L} \frac{1}{2} \sum_{g \in g' \otimes g''} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ + \sum_{g',g'[i]=H} \sum_{g'',g''[i]=H} \sum_{g \in g' \otimes g''} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \end{array} \right] - \sum_{g,g[i]=H} \mu(g) n_g \\
&= \nu \left[\begin{array}{c} \frac{1}{2} \sum_{g',g'[i]=L} n_{g'} \sum_{g'',g''[i]=H} n_{g''} \\ + \frac{1}{2} \sum_{g',g'[i]=H} n_{g'} \sum_{g'',g''[i]=L} n_{g''} \\ + \sum_{g',g'[i]=H} n_{g'} \sum_{g'',g''[i]=H} n_{g''} \end{array} \right] - \sum_{g,g[i]=H} \mu(g) n_g \\
&= \nu \left[h_i (1 - h_i) + h_i^2 \right] - \bar{\mu}_i h_i = (\nu - \bar{\mu}_i) h_i,
\end{aligned}$$

where the 1/2 coefficients capture the fact that 50 % of the offpsrings of the mating between a type with H at i and a type with L at i have an H at i, and $\bar{\mu}_i$ is the average mortality rate of people with an H at i. Hence we again get the equivalent of (6):

$$\frac{\dot{h}_i}{h_i} = \bar{\mu}_t - \bar{\mu}_{it} \tag{16}$$

We are now in a position to extend Lemma 1, which played a key role in characterizing long-run equilibria.

LEMMA 2 – In any stationary equilibrium such that $\dot{n}_g = 0, \forall g$,

(i) All types such that $n_g > 0$ must have the same mortality rate

(ii) $\forall g, g'; n_g > 0, n_{g'} > 0, g'' \in g \otimes g' \Rightarrow n_{g''} > 0$.

(iii) The distribution of genotypes can be deduced from the distribution of genes by the following formula:

$$n_g = \prod_{i,g[i]=L} (1 - h_i) \prod_{i,g\{i]=H} h_i$$

PROOF – See Appendix.

Lemma 2 tells us that in steady state, all existing types must have the same mortality rate, which is typically that of the fittest type $\mu(g_{\max})$ if no $L-$allele is fixed. This is because high mortality types typically have more L-alleles because of the fitness ranking property (13), so that as long as they exist this tends to diminish the proportion of at least some L-alleles in the population. Finally, part (iii) tells us that in the long-run, sexual reproduction and random matching achieve the maximum "mixing" of a given distribution of genes in the population, in that the probability of getting a given allele at a given locus is given by the proportion of that allele in the population, and therefore independent of whatever other alleles are present at other loci. Note however that the distribution of genes $\{h_i\}$ must be the equilibrium one, i.e. it is endogenous, unless all existing types in the *initial* distribution of genotypes have the same mortality and satisfy (iii)–in which case the distribution of genes is preserved and in the long-run is equal to the initial one.

Another way to view this mixing property is to use the concept of entropy. Let us define *genetic entropy* as

$$S = \sum_g n_g \ln n_g$$

The properties of entropy as a measure of disorder, i.e. an inverse measure of information, are well known (see Shannon(1948)). Now, the following lemma tells us that in the long-run, genes must be distributed in the population so as to maximize genetic entropy.

LEMMA 3 – In any stationary equilibrium such that $\dot{n}_g = 0, \forall g$, the distribution of genotypes must maximize genetic entropy subject to the following set of constraints:

$$\sum_{g,g[i]=H} n_g = h_i$$

PROOF – See Appendix

6.2 Reintroducing markets

We now describe how an individual's genotype affects his productivity at various activities.

The allele present at a given locus i determines the individual's productivity at a corresponding activity denoted by the same index i. This productivity is equal to z_i, which is z_{Hi} if the allele is H and z_{Li} if it is L. We shall assume $z_{Li} < z_{Hi}$. The time allocation constraint of an individual with genotype g is therefore given by

$$\sum_{i=1}^{q} \frac{x_i}{z_i} \leq 1, \tag{17}$$

where x_i is the individual's output in activity i, and $z_i = z_{Hi}$ (resp. z_{Li}), iff $g[i] = H$ (resp. $g[i] = L$)

Finally the individual's fitness is

$$\frac{1}{\mu(g)} = \varphi(y_1, ..., y_q),$$

where y_i is the individual's consumption of activity i, and φ satisfies the same properties as previously. Under autarky, we have $y_i = x_i$, and the following result holds:

PROPOSITION 3 – Under autarky, $\mu(T_i g) > \mu(g)$ if $g[i] = H$. Consequently, in any LRE such that $h_i > 0, \forall i$, all individuals are of genotype $g_{\max}$, i.e. the H-allele is fixed at all locations.

Proof – Type $T_i g$ has a more unfavorable time budget constraint than type g. Therefore, it achieves a lower fitness. The rest follows from Lemma 2. Q.E.D.

Let us now look at the trade case. As in the preceding analysis, the price vector (p_i) satisfies the following normalization

$$\sum_{i=1}^{q} p_i = 1 \tag{18}$$

People allocate their time between various activities so as to maximize their income $R(g) = \sum_{i=1}^{q} p_i x_i$, subject to the time allocation constraint (17). Their demand vector is the one which maximizes φ subject to their budget constraint:

$$\sum_{i=1}^{q} p_i y_i = R(g)$$

Types with lower incomes must achieve lower fitness and therefore disappear in the long-run. The following proposition generalizes the results derived for the two loci case.

PROPOSITION 4 – (i) In any LRE such that $h_i > 0, \forall i$, *a given type only supplies goods corresponding to H–alleles in its genotype:* $x_i(g) > 0 \Longrightarrow g[i] = H$

(ii) In any LRE such that $h_i > 0$, *the price vector is* $p^* = (p_1^*, ..., p_q^*)$ *such that*

$$p_i^* = \frac{1/z_{Hi}}{\sum_j \frac{1}{z_{Hj}}} \tag{19}$$

(iii) In any LRE, there exists a locus j *such that* $h_j = 1$, *i.e. allele H is fixed at locus* j.

Proof of (i) – If $h_i > 0$ for all i, $n_{g_{\max}} > 0$ (Lemma 2, (iii)). Assume there exists a genotype g such that $x_l(g) > 0$ for l such that $g[l] = L$. Clearly,

the plan $(x_1(g), ..., x_l(g)\frac{z_{Hl}}{z_{Ll}}, ..., x_q(g))$ achieves a strictly higher income level and is feasible (i.e. satisfies (17)) for $g_{\max}$. Consequently, $R(g_{\max}) > R(g)$, implying $\mu(g_{\max}) < \mu(g)$, which can't hold in LRE since it is precluded by Lemma 2. Consequently, any type g only supplies goods where it has an H-allele.

Proof of (ii) – The price vector defined by (19) is the one which makes type $g_{\max}$ indifferent between all activities. Assume there exists an LRE with a different price vector. Then there exists a pair of goods (j, k) such that

$$\frac{p_j}{p_k} < \frac{z_{Hk}}{z_{Hj}}, \tag{20}$$

and $x_j(g_{\max}) = 0$ since more income is yielded for type $g_{\max}$ by offering good k than good j.

Since φ satisfies the Inada conditions, the demand for good j is strictly positive; since $g_{\max}$ does not supply good j, there exists $g \neq g_{\max}$ such that $n_g > 0$ and $x_j(g) > 0$. By virtue of (i), $g[j] = H$. The income of type g is $R(g) = \sum_{i=1}^{q} p_i x_i(g) = \sum_{i \neq j} p_i x_i(g) + p_j x_j(g)$. The supply vector $(x_i(g))$ is feasible for type $g_{\max}$ since $g_{\max}$ is more productive than any other type at all activities. The supply vector (x'_i) defined by $x'_i = x_i(g)$, $i \neq j, k, x'_j = 0, x'_k = x_k(g) + x_j(g)\frac{z_{Hk}}{z_{Hj}}$ also satisfies (17). Therefore,

$$\begin{aligned} R(g_{\max}) &\geq \sum_{i=1}^{q} p_i x'_i \\ &= R(g) - p_j x_j(g) + p_k \frac{x_j(g) z_{Hk}}{z_{Hj}} \\ &> R(g), \end{aligned}$$

where the last inequality comes from (20). But, this cannot hold since it again implies $\mu(g_{\max}) < \mu(g)$, which contradicts lemma 2. This proves (ii). Q.E.D.

Proof of (iii) – If not, then $n_{g_{\min}} > 0$ (Lemma 2, iii). But then (i) would be violated.

The preceding proposition tells us what properties an LRE must necessarily have, but does not tell us whether an LRE exists and whether, as in the preceding analysis, one can construct equilibria with a positive level of some L-alleles. We now establish a result which generalizes condition (11), which tells us that an LRE exists with a strictly positive proportion of L-alleles, provided these alleles are not too frequent.

To do so, for any subset S of $\{1, ...q\}$ we define $\tilde{S}$ as $\tilde{S} = \{g, g[i] = H \Rightarrow i \in S\}$. $\tilde{S}$ is the set of all genotypes such that the loci of their H-alleles are all in S. By extension we set $\tilde{\emptyset} = \{g_{\min}\}$.

PROPOSITION 5 – Let $Rf_i(\mathbf{p})$ be the inverse demand function for the fitness maximization problem of an individual with income R facing price vector $\mathbf{p}$. Let

$$D_i = \frac{1}{\sum_j \frac{1}{z_{Hj}}} f_i(\mathbf{p^*})$$

Then there exists an LRE with a distribution $\{n_g\}$ of genotypes if and only if this distribution satisfies the following property:

$$\forall S \subset \{1, ...q\}, \sum_{i \in S} \frac{D_i}{z_{Hi}} \geq \sum_{g \in \tilde{S}} n_g. \tag{21}$$

Proof – See Appendix.

Clearly, conditions (21) are pretty stringent, so that it is not straightforward to construct an equilibrium. However for $n_{g_{\max}}$ close enough to 1, i.e. n_g small enough when $g \neq g_{\max}$, they are clearly satisfied, since $n_{g_{\max}}$ appears on the RHS only for $S = \{1, ...q\}$, in which case (21) is always satisfied with equality, due to Walras' law: $\sum_{i=1}^{q} \frac{D_i}{z_{Hi}} = \sum_{i=1}^{q} p_i^* f_i(\mathbf{p}^*) = 1 = \sum_g n_g$. Therefore there always exist equilibria with a strictly positive fraction of genotypes with L-alleles, provided this fraction is small enough.

7 The impact of trade on collective fitness: the role of environmental shocks

One question of interest is: does a population which trades do better than one which does not, by enough to drive out the latter from existence in the long-run? As we have seen above, for the same initial distribution of genotypes a trading society will have a larger population in the long-run than a non trading one, but these two populations will grow at the same rate. Hence, the former will be larger than the latter but will not eliminate it. If, on the other hand, it were the case that trade increased population growth, then a trading population would eventually become infinitely large relative to a non trading one, so that the latter has been eliminated in relative terms.

It turns out that trade achieves a greater population growth rate if one allows for shocks to the environment such that the relative survival value of H vs. L-alleles can be inverted. A typical example of such an inversion in nature is skin color. Bears in warm climates are dark, while bears in cold climate are white. Thus an allele favouring a white skin would be considered as "H" in a cold climate, but would have to be re-classified as "L" if there is climate change.

If such environmental changes can occur, then the persistence of "L" genes under trade is an asset for the population as a whole. It allows it to diversify its genetic composition so as to better cope with environmental change.

Going back to the simple model of section 2, and assuming a Leontief fitness function, consider a population without trade. We know that in steady state only the fittest type survives, so that $n_{HH} = 1$. This population grows at rate $\nu - \mu_0$, where μ_0 is the minimum autarkic mortality of the HH type, i.e.

$$\mu_0 = \frac{d_H + f_H}{d_H f_H}$$

Now, assume that this population is subject to an environmental shock such that the productivity of HH at activity d is now d_L, rather than d_H,

while that of type HL is now d_H. Population now grows at the rate corresponding to a population entirely made of HL individuals, i.e. $\nu - \mu_1$, where μ_1 is given by

$$\mu_1 = \frac{d_L + f_H}{d_L f_H} > \mu_0.$$

This suggests that on average, when there are shocks to the environment, population will grow at a rate strictly lower than $\nu - \mu_0$. Now, this reasoning is not quite correct, since when there are repeated environmental shocks, there is no reason why population should entirely be made of HH types. Rather, as long as environmental shocks do not affect productivity of the f-activity, it will be made of both HH and HL types, with the former tending to outnumber the latter when the environment favours them, and tending to disappear when it hurts them. In such a case, mortality will always be a weighted average of μ_0 and μ_1, with strictly positive weights, and population growth will be smaller than $\nu - \mu_0$.

Assume for example that the environment changes between two states, $\hat{H}$ and $\hat{L}$, such that the productivity of HH (resp. HL) at the d-activity is d_H (resp. d_L) in the $\hat{H}$ state and d_L (resp. d_H) in the $\hat{L}$ state. Assume transitions between two states follow a Poisson process, with a flow transition probability equal to λ_L from $\hat{H}$ to $\hat{L}$ and equal to λ_H from $\hat{L}$ to $\hat{H}$. Then one can show (see Appendix) that the unconditional expectation of mortality is equal to

$$E(\bar{\mu}) = \frac{\mu_0(\mu_1 - \mu_0)}{\lambda_H + \lambda_L + \mu_1 - \mu_0} + \frac{\mu_0(\lambda_H^2 + \lambda_L^2) + 2\mu_1\lambda_H\lambda_L}{(\lambda_H + \lambda_L + \mu_1 - \mu_0)(\lambda_H + \lambda_L)},$$

which, as the reader can trivially check, is strictly above μ_0.

On the other hand, consider a trading economy with a proportion n_{HH} of HH types, and $1 - n_{HL}$ of HL types, such that

$$\max\ (n_{HH}, 1 - n_{HH}) < \frac{d_H}{d_H + f_H}.$$

According to the results at the end of section 2, this distribution is an equilibrium in both the $\hat{H}$ and $\hat{L}$ states. In the $\hat{H}$ state, the "weak" type HL achieves the lowest mortality rate by specializing in the f good, while in the $\hat{L}$ state, the weak type is HH and it is the one which specializes in f. Therefore, in both states mortality is the same for both types and given by (12), i.e. it is equal to μ_0. Consequently, population grows at rate $\nu - \mu_0$.

From there one may argue that if populations with different institutions compete with each other for land and natural resources, trading populations will eventually eliminate non trading ones because of their faster population growth rate. This would not be true in the absence of environmental shocks. Hence we get the interesting prediction that environmental volatility speeds up institutional evolution.

8 Applications

In this section, I discuss some real-world applications of the theory outlined above.

First, it may shed light on a puzzle regarding *heritability* of economic performance. Here, to simplify, there is a conflict between social scientists, who, looking at *income,* tend to favor the environment,[8] and psychometrists, who, by looking at *test scores,* insist on the genetic determinants of intelligence.[9] For example, Becker and Tomes (1986) find that regression to the mean in terms of income takes place quite quickly, which is somewhat at variance with the view that economic performance is heritable (although randomness in mating and complexity in the gene combinations that might determine ability implies that these findings are not incompatible with the importance of genes). On the other hand, recent evidence on brain structure, as well as studies of heritability of test scores, suggest that intelligence has substantial

[8] See Ashenfelter and Krueger (1994).

[9] This debate is somewhat captured by the heated "Bell curve" debate in the 1990s. (Herrnstein and Murray, 1995; Devlin et al. 1997; Cawley et al. 1996; Ashenfelter and Rouse (1998))

genetic determinants.[10] Some psychometricians also insist on the existence of a *general* intelligence factor g, meaning that people who perform well in some test will also tend to perform well in other tests.

The above model suggests that specialization according to comparative advantage loosens the link between ability and income. Under our assumptions, people who have a greater number of $L-$alleles will tend to perform poorly on a general ability test. However this is of no consequence for their income as long as the economy is in an equilibrium where they can achieve the same income level by specializing in activities associates with their H-alleles.

Second, the above model is related to the *neutralist* hypothesis promoted by a school of biologists such as Kimura (1983). This approach contends that most of the observed genetic variation (not only in humans but in all organisms) is due to random drift of selectively neutral alleles. While the truth is probably that some alleles are neutral and others not, our analysis suggests that neutrality itself is not independent of social organization. Trade and specialization increase the number of genes that are neutral because exchange offsets the fitness deficit of inferior alleles. Formally, this is captured by the following property of the above model: Under autarky, the long-run composition of the population is entirely made of $H-$alleles (more fundamentally, it is determined by the environment). Under trade, however, there exists a continuum of steady state distribution of genotypes compatible with steady state equilibrium, and which one is reached depends on initial conditions. As long as there are random shocks which change this distribution while maintaining the economy in the zone where the long-run equilibrium implies the same fitness for all genotypes (i.e. in the zone where the equivalent of (11) is satisfied), evolution is driven by random drift rather than selective pressure.[11]

[10] See Bouchard and McGue (1981), and recent studies by Tang et al. (1999), Tsien (2000)). and, Thompson et al. (2001).

[11] Note however that such evolution cannot fix an L-allele at any locus, because market forces would push up the relative price for the corresponding good, eventually violating (11) and giving a fitness advantage to genotypes with an H-allele at that locus.

Third, the model has implications for differences in the distribution of specific genes across different human populations. In principle, one should be able to empirically test the model by comparing human populations with different degrees of economic advancement, and looking at differences in the frequency of genes across these populations. One should expect to see genes that make people fitter for a given activity to be more frequent in the less advanced populations, to the extent that it implies less frequent trade, and less specialisation, among individuals.

Unfortunately, this is more easily said than done. First, one should identify differences in economic advancement that are persistent over long periods by historical standards, since biological evolution is slow. This is not easy. While in the XXI century the most developed countries are located in North America, Europe and East Asia, things were quite different just one thousand years ago. Scandinavian people, for example, who now are among the richest, were then a primitive tribe compared to the Muslim world. The ancient civilizations of China, Egypt and America were flourishing while Europe was stagnating at prehistoric levels. As a first pass, however, one may speculate that populations coming from Asia, the middle East, or Europe have had sophisticated institutions for thousands of years, while this is not the case for more primitive populations in Australasia or sub-saharan Africa. It is reasonable to assume that exchanges have been more intense in the former than in the latter societies.But one should expect a lot of heterogeneity within these broadly defined groups.[12]

Second, one should identify alleles that give a clear adaptive advantage. But measuring the selective value of specific alleles is far from obvious. In particular, an allele can be good for a given population in a given environment, and bad for another population in another environment (recall the

[12] In Africa, for example, co-exist descendents of ancient civilizations such as Egypt as well as primitive tribes.

On the other hand, this level of aggregation allows to use casual knowledge about world history, rather than detailed data on comparative economic developments at the population level, which may be difficult to gather and/or construct.

above example of skin color in bears); for example, an allele can increase resistance to tuberculosis, but reduce resistance to malaria. So if we find that it is more frequent in one population than in another, it may be due to differences in the environments faced by these populations rather than differences in economic development. Furthermore, for some genes a fitness advantage may be in favor of heterozygotes, a phenomenon ignored in our 1-chromosome model. Finally, most documented selective consequences of alleles are associated with rather serious diseases, whose consequences can hardly be offset by trade and specialization, at least until recently.

With these caveats in mind, let us illustrate how the model's prediction could in principle be tested by looking at the world distribution of some alleles. One of the most studied gene is the one for blood group (ABO), which comes in three main alleles, A, B, and O. According to Cavalli-Sforza et al. (1994, p.129), it has been found that the $O-$allele confers a selective advantage with respect to several diseases: syphilis, plague, cholera, diarrhea, smallpox, tuberculosis, malaria, diabetes, anemia, thrombosis, liver cirrhosis, and so on. With such overwhelming properties, we should expect A and B to disappear. Indeed, this is what has happened among native south Americans. However, O is not uniformly advantageous. Rhuematoid arthritis, and ulcers, are more likely to occur in O individuals. Furthermore, many puzzles remain regarding its selective properties.

While specialization is unlikely to offset all the disadvantages associated with greater exposure to the diseases just mentioned, there is at least one property of the ABO gene which is useful for our purpose, namely that the incidence of myopia is 2.5 times greater in A and B individuals than in O individuals.[13] Clearly, under autarky myopia confers a substantial disadvantage, even when corrected with glasses[14]. Hunting, gathering, escaping

[13] The source for all this discussion is Cavalli-Sforza et al. (1994).

[14] Glasses are believed to have been invented in the 13th century, but those which correct for short sightedness were developed only in the 16th century. See Cipolla (1994). While trade and specialization have been around for say several thousand years, which is arguably enough to have influenced evolution, this is much less likely for a period of 500 years.

predators, and many other activities are hampered by myopia. But in a well developed labor market, one may think of many activities where short-sightedness does not harm productivity. Therefore, regardless of whether A and B would disappear in the very long run, we expect a lower frequency of O in more advanced societies.

The following table shows the frequency of the O alleles in populations that originate from various regions of the world. Typically, historically more advanced societies (Europe and Asia) have a lower frequency of the O allele. This evidence is very mild. New Guinea, for example, has a lower frequency than Africa and America, although one would tend to think of it as less advanced. Perhaps this reflects some specific factors.

Area	Frequency
Asia	0.596
Europe	0.650
New Guinea	0.654
Pacific Oceania	0.668
Africa	0.694
Australia	0.758
America	0.896

Table 2: Frequency of blood group O-allele. Source: Cavalli-Sforza et al. (1994).

As already mentioned, the selective properties of even a deeply studied human gene such as ABO are not very well known. Ideally, one would like to design a test which would not rely on specific assumptions about the selective properties of a given gene. One way to do so is to compare the actual distribution of alleles to the one which would prevail under "neutralism". As argued above, evolution should be more "neutral" under trade than under autarky. Fortunately, Cavalli-Sforza et al. provide such a test by looking at the correlation between genetic distance and geographical distance in each continent, and compare it to the predictions of a simulated model based on the neutralist hypothesis. Their results (Figure 2.9.2, p.123) imply substantial deviations in Africa and Australasia, and no significant deviation

in Europe and Asia. This confirms our coarse test that more economically advanced societies are closer to the neutral hypothesis. Again this is mild evidence, many other environmental explanations can be proposed, but at least it is consistent with the theory outlined in this paper.

9 Conclusion

Understanding how genes and culture coevolve is an important step in integrating biology with the social sciences. One important cultural institution of our species is the market. Leaving aside the question of what genetic characteristics of our species led to such a development, this paper has considered the reverse influence: how do markets in turn affect human genetic evolution? The answer it that it makes evolution more selectively neutral by allowing individuals to offset their genetic disadvantages by specializing. A potential interesting route for further reseach would be to study how this could be further enhanced by the use of technology.

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APPENDIX

Proof of Lemma 2

The proof of (ii) is obvious: the proportion of an offspring of g and g' cannot be zero as long as g and g' are in positive proportions in the population, since they mate with positive probability.

Proof of (i) – Ignore all loci where genes are fixed. Consider a locus i. Let

$$\rho_i = \min_{g, g[i]=L} \frac{n_{U_i g}}{n_g}$$

Let $B(g) = \sum_{g'} \sum_{g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''}$ be the proportion of offsprings with genotype g. Then, for g such that $g[i] = L$, $B(g)$ can be written as

$$\begin{aligned} B(g) &= \sum_{g',g'[i]=L} \sum_{g'',g''[i]=H, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} + \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ &\quad + \sum_{g',g'[i]=L} \sum_{g'',g''[i]=L, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ &= 2 \sum_{g',g'[i]=L} \sum_{g'',g''[i]=H, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ &\quad + \sum_{g',g'[i]=L} \sum_{g'',g''[i]=L, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''}, \end{aligned} \tag{22}$$

where the equality comes from the fact that

$$\begin{aligned} \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} &= \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g \in g' \otimes g''} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ &= \sum_{g'',g''[i]=L} \sum_{g',g'[i]=H, g \in g'' \otimes g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \\ &= \sum_{g',g'[i]=L} \sum_{g'',g''[i]=H, g'' \in g*g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} \end{aligned}$$

Similarly,

$$B(U_i g) = 2 \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g'' \in U_i g * g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''} + \sum_{g',g'[i]=H} \sum_{g'',g''[i]=H, g'' \in U_i g * g'} \frac{1}{2^{g' \cdot g''}} n_{g'} n_{g''}. \quad (23)$$

Now, (22) may be rewritten as

$$B(g) = 2 \sum_{g',g'[i]=H} \sum_{g'',g''[i]=H, g'' \in g * T_i g'} \frac{1}{2^{g' \cdot g'' + 1}} n_{T_i g'} n_{g''} + \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g'' \in g * T_i g'} \frac{1}{2^{g' \cdot g'' - 1}} n_{T_i g'} n_{g''}.$$

That is, the g' such that $g'[i] = L$ are constructed by taking all the g' such that $g'[i] = H$, and downgrading them. One has then $T_i g' \cdot g'' = g' \cdot g'' + 1$ if $g''[i] = H$, and $T_i g' \cdot g'' = g' \cdot g'' - 1$ if $g''[i] = L$.

This may be rewritten as

$$B(g) = \sum_{g',g'[i]=H} \sum_{g'',g''[i]=H, g'' \in U_i g * g'} \frac{1}{2^{g' \cdot g''}} n_{T_i g'} n_{g''} + 2 \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g'' \in U_i g * g'} \frac{1}{2^{g' \cdot g''}} n_{T_i g'} n_{g''},$$

where the changes below the sum signs come from property (P1), i.e. the fact that $U_i g * g' = g * T_i g'$, for $g[i] = L$ and $g'[i] = H$.

Next, (23) implies that

$$\begin{aligned} B(U_i g) &\geq \rho_i (2 \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g'' \in U_i g * g'} \frac{1}{2^{g' \cdot g''}} n_{T_i g'} n_{g''} \\ &\quad + \sum_{g',g'[i]=H} \sum_{g'',g''[i]=H, g'' \in U_i g * g'} \frac{1}{2^{g' \cdot g''}} n_{T_i g'} n_{g''}) \\ &= \rho_i B(g). \end{aligned}$$

In steady state, we have that

$$n_g = \frac{\nu B(g)}{\nu + \mu(g) - \mu}.$$

Assume now that there exists a genotype $\tilde{g}$ and a locus i such that $n_{\tilde{g}} > 0$ and $\mu(\tilde{g}) > \mu(U_i\tilde{g})$. Then, the preceding equation implies that $n_{U_i\tilde{g}} > \rho_i n_{\tilde{g}}$. Next, consider $\hat{g} = \arg\min_{g,g[i]=L} \frac{n_{U_i g}}{n_g}$, implying $n_{U_i\hat{g}} = \rho_i n_{\hat{g}}$. Using again (23), we have that

$$\begin{aligned}
B(U_i\hat{g}) &= 2 \sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g''\in U_i\hat{g}*g'} \frac{1}{2^{g'\cdot g''}} n_{g'} n_{g''} + \\
&\quad \sum_{g',g'[i]=H,,g'\neq U_i\tilde{g}} \sum_{g'',g''[i]=H, g''\in U_i\hat{g}*g'} \frac{1}{2^{g'\cdot g''}} n_{g'} n_{g''} \\
&\quad + n_{U_i\tilde{g}} \sum_{g'',g''[i]=H, g''\in U_i\hat{g}*U_i\tilde{g}, g''\neq U_i\hat{g}} \frac{1}{2^{U_i\tilde{g}\cdot g''}} n_{g''} + \frac{n_{U_i\tilde{g}} n_{U_i\hat{g}}}{2^{U_i\tilde{g}\cdot U_i\hat{g}}} \\
&> \rho_i \left[\begin{array}{c} 2\sum_{g',g'[i]=H} \sum_{g'',g''[i]=L, g''\in U_i\hat{g}*g'} \frac{1}{2^{g'\cdot g''}} n_{T_i g'} n_{g''} \\ + \sum_{g',g'[i]=H,,g'\neq U_i\tilde{g}} \sum_{g'',g''[i]=H, g''\in U_i\hat{g}*g'} \frac{1}{2^{g'\cdot g''}} n_{T_i g'} n_{g''} \\ + n_{\tilde{g}} \sum_{g'',g''[i]=H, g''\in U_i\hat{g}*U_i\tilde{g}, g''\neq U_i\hat{g}} \frac{1}{2^{U_i\tilde{g}\cdot g''}} n_{g''} + \frac{n_{\tilde{g}} n_{U_i\hat{g}}}{2^{U_i\tilde{g}\cdot U_i\tilde{g}}} \end{array} \right] \\
&= \rho_i B(\hat{g}),
\end{aligned}$$

where the strict inequality comes from the fact that $n_{U_i\tilde{g}} > \rho_i n_{\tilde{g}} > 0$ and $n_{U_i\hat{g}} > 0$. That is, the last term in the preceding equation captures the fact that a strictly positive number of offsprings of matches between $U_i\hat{g}$ and $U_i\tilde{g}$ will be of type $U_i\hat{g}$, and that exactly half the same proportion among the (twice as numerous) offsprings of matches between $\hat{g}$ and $U_i\tilde{g}$ will be of type $\hat{g}$. Therefore, $B(U_i\hat{g}) > \rho_i B(\hat{g})$, implying

$$n_{U_i\hat{g}} = \frac{\nu B(U_i\hat{g})}{\nu + \mu(U_i\hat{g}) - \mu} > \frac{\nu \rho_i B(\hat{g})}{\nu + \mu(U_i\hat{g}) - \mu} \geq \frac{\nu \rho_i B(\hat{g})}{\nu + \mu(\hat{g}) - \mu} = \rho_i n_{\hat{g}},$$

which is a contradiction. Consequently, $\tilde{g}$ cannot exist and $\mu(U_i g) = \mu(g)$ for all i and g. Similarly, $\mu(T_i g) = \mu(g)$ for $n_g > 0$.

Consider now two arbitrary types g and g', such that $n_g > 0$ and $n_{g'} > 0$. Let $G_H = \{i, g[i] = H \text{ and } g'[i] = L\}, G_L = \{i, g[i] = L \text{ and } g'[i] = H\}, m =$

$|G_H| + |G_L|$. Then,

$$g' = \bigcirc_{i \epsilon G_H} T_i(\bigcirc_{j \in G_L} U_j g).$$

That is, we can move from g to g' by a succession of genetic downgradings and upgradings at the loci where alleles differ between g and g'. Let $(\tilde{g}_k)_{k=0}^{k=m}$ denote the sequence of genotypes obtained in this process. We have $\tilde{g}_0 = g$, and $\tilde{g}_m = g'$.

Now, (14) implies that we have the following property:

$$T_i(g \otimes g') \subseteq g \otimes g'; U_i(g \otimes g') \subseteq g \otimes g', \forall i \in G_H \cup G_L$$

Consequently, since $g \in g \otimes g'$, $\tilde{g}_k \in g \otimes g'$. That is, the intermediate genotypes between g and g' all exist as offsprings of g and g'. Furthermore, (ii) implies that $n_{g''} > 0$ for all $g'' \in g \otimes g'$. Thus, $n_{\tilde{g}_k} > 0$. Since $\tilde{g}_{k+1} = T_i \tilde{g}_k$ or $\tilde{g}_{k+1} = U_j \tilde{g}_k$, then $\mu(\tilde{g}_{k+1}) = \mu(\tilde{g}_k)$. By iteration, it follows that $\mu(g) = \mu(g')$. Q.E.D.

Proof of (iii) – Repeating the steps of the proof of (ii) proves that there cannot be any $\tilde{g}$ such that $\tilde{g}[i] = L$ and $n_{U_i \tilde{g}} > \rho_i n_{\tilde{g}}$. Consequently, $n_{U_i g} = \rho_i n_g$ for all g such that $g[i] = L$ and $n_g > 0$. Summing over all g's such that $g[i] = L$ implies that $\rho_i = h_i/(1 - h_i)$. This implies that

$$n_g = n_{g_{\max}} \prod_{i, g[i]=L} \frac{1 - h_i}{h_i}$$

Finally, the only value of $g_{\max}$ such that the preceding equation holds and such that $\sum_g n_g = 1$ is $g_{\max} = \prod_{i=1}^{q} h_i$. Consequently:

$$n_g = \prod_{i, g[i]=L} (1 - h_i) \prod_{i, g\{i]=H} h_i \tag{24}$$

Q.E.D.

Proof of Lemma 3

Let λ_i be the Lagrange multiplier of the corresponding constraint. Then

the FOC is

$$1 + \ln n_g = \sum_{i, g[i]=H} \lambda_i.$$

Consequently,

$$\frac{n_{U_i g}}{n_g} = e^{\lambda_i}.$$

Following the same steps as the proof of (iii) in Lemma 2, if follows that $e^{\lambda_i} = h_i/(1-h_i)$, so that (24) holds. Q.E.D.

Proof of Proposition 5

Proof – We first prove that this condition is necessary. The RHS of (21) is the total time supplied by genotypes in $\tilde{S}$. Proposition 4, (i) implies that it must be allocated among goods i such that $g[i] = H$, i.e. among goods in S. The LHS of (21) is the total time input needed to produce all the goods in S. It must be greater than or equal to its RHS, since genotypes in $\tilde{S}$ cannot produce any other good. Otherwise, supply would exceed demand. Note that (21) applied to $S = \emptyset$ implies $n_{g_{\min}} = 0$, and therefore that 1 H-gene is fixed. Also, (21) applied to $S = \{1, ...q\}$ boils down to Walras' law, since it is equivalent to $\sum_{i=1}^{q} p_i^* f_i(p^*) \geq 1$, and by Walras law $\sum_{i=1}^{q} p_i^* f_i(p^*) = 1$.

Let us now prove sufficiency. In order to do so, we construct a set of functions $m_i(g)$, representing the share of time of genotype g devoted to activity i, such that:

$$m_i(g) > 0 \Longrightarrow g[i] = H \tag{25}$$

$$\sum_{g, g[i]=H} m_i(g) n_g = \frac{D_i}{z_{Hi}}. \tag{26}$$

$$\sum_i m_i(g) = 1 \tag{27}$$

If we are able to construct such functions, then this is indeed an equilibrium, since supply equals demand for all goods, and since the price vector in (19) implies that a genotype is indifferent between supplying all the good in which it has an H-allele.

To construct the $m_i(g)$, we use the following algorithm. We start from any arbitrary allocation $m_i^{(0)}(g)$ satisfying (25) and (27). This defines the initial stage.[15] Then we move from stage (k) to stage $(k+1)$ as follows. At the beginning of stage (k), the set $\{1, ...q\}$ can be partioned into three subsets:

$$\begin{aligned} H_0^{(k)} &= \{i, \sum_{g, g[i]=H} m_i^{(k)}(g) n_g = \frac{D_i}{z_{Hi}}\} \\ H_+^{(k)} &= \{i, \sum_{g, g[i]=H} m_i^{(k)}(g) n_g < \frac{D_i}{z_{Hi}}\} \\ H_-^{(k)} &= \{i, \sum_{g, g[i]=H} m_i^{(k)}(g) n_g > \frac{D_i}{z_{Hi}}\}. \end{aligned}$$

That is, those goods for which supply equals demand, those for which there is excess demand, and those for which there is excess supply. Note that since $\sum_i D_i/z_{Hi} = 1$, $H_+^{(k)}$ is empty if and only if $H_-^{(k)}$ is empty. If $H_+^{(k)} = H_-^{(k)} = \emptyset$, then we have an equilibrium, and the algorithm stops. Otherwise, we can show that:

$$\exists g, i, j, \text{s.t. } g[i] = H, g[j] = H, m_i^{(k)}(g) > 0, i \in H_-^{(k)}, j \in H_+^{(k)} \tag{28}$$

To prove this, assume (28) does not hold. Let

$$G(i) = \{g, g[i] = H, m_i^{(k)}(g) > 0\}.$$

Then for any $i \in H_-^{(k)}$ and $g \in G(i)$, it must be that $g[j] = L$ for all $j \in H_+^{(k)}$–otherwise, (28) would hold. Consequently, $G(i) \subseteq \tilde{H}_{-=}^{(k)}$, where

[15] One can trivially check that such an allocation exists, since $g_{\min}$ is the only genotype which has no H-alleles, and $n_{g_{\min}} = 0$.

$H_{-=}^{(k)} = H_{-}^{(k)} \cup H_{0}^{(k)}$.Therefore:

$$\sum_{g \in G(i)} n_g \leq \sum_{g \in \tilde{H}_{-=}^{(k)}} n_g. \tag{29}$$

At the same time, we have that for any $g \in G(i)$:

$$\sum_{i \in H_{-=}^{(k)}} m_i^{(k)}(g) = 1,$$

since $m_j(G) = 0$ for all $j \in H_{+}^{(k)}$. Consequently:

$$\begin{aligned} \sum_{g \in G(i)} n_g &= \sum_{g \in G(i)} n_g \sum_{i \in H_{-=}^{(k)}} m_i^{(k)}(g) \\ &= \sum_{i \in H_{-=}^{(k)}} \sum_{g \in G(i)} m_i^{(k)}(g) n_g \\ &= \sum_{i \in H_{-=}^{(k)}} \sum_{g, g[i]=H} m_i^{(k)}(g) n_g \\ &> \sum_{i \in H_{-=}^{(k)}} \frac{D_i}{z_{Hi}}, \end{aligned} \tag{30}$$

where the last inequality comes from the fact that $H_{-}^{(k)}$ is non empty. Now, confronting (29) with (30) implies $\sum_{g \in \tilde{H}_{-=}^{(k)}} n_g > \sum_{i \in H_{-=}^{(k)}} \frac{D_i}{z_{Hi}}$, which violates (21) for $S = H_{-=}^{(k)}$. Consequently, (28) must hold.

Let us now define

$$L^{(k)} = \{g \text{ s.t. } \exists i \in H_{-}^{(k)}, \exists j \in H_{+}^{(k)}, g[i] = H, g[j] = H, m_i^{(k)}(g) > 0\}.$$

The above reasoning implies that unless $\{m_i^{(k)}(g)\}$ is an equilibrium allocation, i.e. $H_{+} = H_{-} = \emptyset$, then $L^{(k)}$ is non empty. To move to iteration $(k+1)$, take $g^* \in L^{(k)}$ and some i and j such that g^*, i, j satisfy (28).

1. If

$$m_i^{(k)}(g^*) n_{g^*} < \min \left(\sum_{g, g[i]=H} m_i^{(k)}(g) n_g - \frac{D_i}{z_{H_i}}, \frac{D_j}{z_{Hj}} - \sum_{g, g[j]=H} m_j^{(k)}(g) n_g \right),$$

then set $m_i^{(k+1)}(g^*) = 0, m_j^{(k+1)}(g^*) = m_j^{(k)}(g^*) + m_i^{(k)}(g^*)$, and $m_r^{(k+1)}(g') = m_r^{(k)}(g')$ for $g' \neq g^*$ or $r \neq i, j$. Clearly, in such a case, $g^* \notin L^{(k+1)} \subset L^{(k)}$. Thus$\left|L^{(k+1)}\right| < \left|L^{(k)}\right|$.

2. If

$$\begin{aligned} m_i^{(k)}(g^*)n_{g^*} &\geq \frac{D_j}{z_{Hj}} - \sum_{g,g[j]=H} m_j^{(k)}(g)n_g, \text{ and} \\ \sum_{g,g[i]=H} m_i^{(k)}(g)n_g - \frac{D_i}{z_{H_i}} &\geq \frac{D_j}{z_{Hj}} - \sum_{g,g[j]=H} m_j^{(k)}(g)n_g \end{aligned}$$

then set $m_i^{(k+1)}(g^*) = m_i^{(k)}(g^*) - \left(\frac{D_j}{z_{Hj}} - \sum_{g,g[j]=H} m_j^{(k)}(g)n_g\right)/n_{g^*}, m_j^{(k+1)}(g^*) = m_j^{(k)}(g^*) + \left(\frac{D_j}{z_{Hj}} - \sum_{g,g[j]=H} m_j^{(k)}(g)n_g\right)/n_{g^*}$, and $m_r^{(k+1)}(g') = m_r^{(k)}(g')$ for $g' \neq g^*$ or $r \neq i, j$. In such a case, $j \notin H_+^{(k+1)} \subset H_+^{(k)}$, while $H_-^{(k+1)} \subseteq H_-^{(k)}$ and $j \in H_0^{(k+1)} \supset H_0^{(k)}$.

3. If

$$\begin{aligned} m_i^{(k)}(g^*)n_{g^*} &\geq \sum_{g,g[i]=H} m_i^{(k)}(g)n_g - \frac{D_i}{z_{H_i}}, \text{ and} \\ \frac{D_j}{z_{Hj}} - \sum_{g,g[j]=H} m_j^{(k)}(g)n_g &\geq \sum_{g,g[i]=H} m_i^{(k)}(g)n_g - \frac{D_i}{z_{H_i}} \end{aligned}$$

then set $m_i^{(k+1)}(g^*) = m_i^{(k)}(g^*) - \left(\sum_{g,g[i]=H} m_i^{(k)}(g)n_g - \frac{D_i}{z_{H_i}}\right)/n_g, m_j^{(k+1)}(g^*) = m_j^{(k)}(g^*) + \left(\sum_{g,g[i]=H} m_i^{(k)}(g)n_g - \frac{D_i}{z_{H_i}}\right)/n_g$,and $m_r^{(k+1)}(g') = m_r^{(k)}(g')$ for $g' \neq g^*$ or $r \neq i, j$. In such a case, $i \notin H_-^{(k+1)} \subset H_-^{(k)}$, while $H_+^{(k+1)} \subseteq H_+^{(k)}$ and $i \in H_0^{(k+1)} \supset H_0^{(k)}$.

Consequently, we always have

$$\left|L^{(k+1)}\right| + \left|H_+^{(k+1)}\right| + \left|H_-^{(k+1)}\right| < \left|L^{(k)}\right| + \left|H_+^{(k)}\right| + \left|H_-^{(k)}\right|$$

After a finite number of iterations, this quantity must be equal to zero, which can only happen if one is at an equilibrium. This proves sufficiency. Q.E.D.

A Poisson process for environmental shocks

Assume H is fixed at locus 1. To compute average mortality, first note that in the H state n_{HH} evolves according to

$$\dot{n}_{HH} = (1 - n_{HH})(\mu_1 - \mu_0).$$

The solution to this differential equation is

$$n_{HH}(t) = n_{HH}(0)e^{-(\mu_1-\mu_0)t} + 1 - e^{-(\mu_1-\mu_0)t},$$

where $n_{HH}(0 \mid H)$ is the initial value of n_{HH}, i.e. prevailing at the date when this spell of H state started. Similarly, in the L state we have

$$n_{HH}(t) = n_{HH}(0)e^{-(\mu_1-\mu_0)t}.$$

Mortality in the H-state is given by

$$\mu_H = n_{HH}\mu_0 + (1 - n_{HH})\mu_1.$$

Similarly, mortality in the L-state is

$$\mu_H = n_{HH}\mu_1 + (1 - n_{HH})\mu_0.$$

Let $g_H(t)$ be the stationary probability density of having been in the H state for duration t, and $g_L(t)$ the corresponding density for the L state. The Poisson process implies that

$$g_H(t) = \frac{\lambda_H\lambda_L}{\lambda_H + \lambda_L}e^{-\lambda_L t}$$

$$g_L(t) = \frac{\lambda_H\lambda_L}{\lambda_H + \lambda_L}e^{-\lambda_H t}.$$

The unconditional probability of being in the H state is thus

$$P_H = \frac{\lambda_H}{\lambda_H + \lambda_L}$$

The unconditional expectation of mortality is

$$E\bar{\mu} = P_H E(\mu_H) + (1 - P_H)E(\mu_L) \tag{31}$$

Using the above formula for mortality, we have

$$\begin{aligned} E(\mu_H) &= E(n_{HH} \mid H)\mu_0 + (1 - E(n_{HH} \mid H))\mu_1 \\ &= E(n_{HH} \mid H)(\mu_0 - \mu_1) + \mu_1 \end{aligned} \tag{32}$$

Similarly

$$E(\mu_L) = -E(n_{HH} \mid L)(\mu_0 - \mu_1) + \mu_0 \tag{33}$$

The conditional expectations $E(n_{HH} \mid H)$ is computed by summing over all durations in the H state. We get

$$\begin{aligned} E(n_{HH} \mid H) &= \frac{\int_0^{+\infty} g_H(t) E(n_{HH}(t) \mid H) dt}{P_H} \\ &= \int_0^{+\infty} \lambda_L e^{-\lambda_L t} \left[E(n_{HH}(0) \mid H) e^{-(\mu_1 - \mu_0)t} + 1 - e^{-(\mu_1 - \mu_0)t} \right] dt \\ &= 1 - \frac{\lambda_L}{\lambda_L + \mu_1 - \mu_0} \left[1 - E(n_{HH}(0) \mid H) \right]. \end{aligned} \tag{34}$$

$E(n_{HH}(0) \mid H)$ is the expected proportion of the population being HH conditional on just having shifted to the H state. Since one was in the L state one instant before we have

$$E(n_{HH}(0) \mid H) = E(n_{HH} \mid L). \tag{35}$$

Similarly, the expected number of firms conditional on being in the L-state can be computed as

$$E(n_{HH} \mid L) = \frac{\lambda_H}{\lambda_H + \mu_1 - \mu_0} E(n_{HH}(0) \mid L), \tag{36}$$

and we gain have

$$E(n_{HH}(0) \mid L) = E(n_{HH} \mid H). \tag{37}$$

These four equations allow to compute $E(n_{HH} \mid H)$ and $E(n_{HH} \mid L)$. We get:

$$E(n_{HH} \mid H) = \frac{\lambda_H + \mu_1 - \mu_0}{\lambda_L + \lambda_H + \mu_1 - \mu_0}$$

$$E(n_{HH} \mid L) = \frac{\lambda_L}{\lambda_L + \lambda_H + \mu_1 - \mu_0}.$$

Substituting into (32), (33) and then (31) we finally get

$$E(\bar{\mu}) = \frac{\mu_0(\mu_1 - \mu_0)}{\lambda_H + \lambda_L + \mu_1 - \mu_0} + \frac{\mu_0(\lambda_H^2 + \lambda_L^2) + 2\mu_1\lambda_H\lambda_L}{(\lambda_H + \lambda_L + \mu_1 - \mu_0)(\lambda_H + \lambda_L)},$$

which is the expression in the text.