

Journal of Demographic Economics

Date of delivery:**Journal and vol/article ref:** DEM 1500012**Number of pages (not including this page):** 26

This proof is sent to you on behalf of Cambridge University Press. Please print out the file and check the proofs carefully. Please ensure you answer all queries.

Please EMAIL your corrections within 2 days of receipt to:

Caela Moffet at
email: cmoffet@cambridge.org

Please clearly indicate any corrections required by page, column and line reference.

Authors are strongly advised to read these proofs thoroughly because any errors missed may appear in the final published paper. This will be your ONLY chance to correct your proof. Once published, either online or in print, no further changes can be made.

NOTE: If you have no corrections to make, please also email to authorise publication.

- The proof is sent to you for correction of typographical errors only. Revision of the substance of the text is not permitted, unless discussed with the editor of the journal. Only **one** set of corrections are permitted.
- Please answer carefully any author queries.
- Corrections which do NOT follow journal style will not be accepted.
- A new copy of a figure must be provided if correction of anything other than a typographical error introduced by the typesetter is required.

- If you have problems with the file please email

cmoffet@cambridge.org

Please note that this pdf is for proof checking purposes only. It should not be distributed to third parties and may not represent the final published version.

Important: you must return any forms included with your proof. We cannot publish your article if you have not returned your signed copyright form

Please do not reply to this email

NOTE - for further information about **Journals Production** please consult our **FAQs** at http://journals.cambridge.org/production_faqs

Author queries:

- Q1. The distinction between surnames can be ambiguous, therefore to ensure accurate tagging for indexing purposes online (eg for PubMed entries), please check that the highlighted surnames have been correctly identified, that all names are in the correct order and spelt correctly.
- Q2. References “Montalvo and Reynal-Querol (2006)”, “McNeill (1997)”, “Le Point (2003)”, “Robertson (2003)” are not present in the reference list. Please provide the same in the list with complete bibliographic details or else delete from the text.
- Q3. The name of the author in the reference citation “Bell and Gerbasch (2006)” has been changed to “Bell and Gersbach (2006)” as per the reference list. Please check for correctness.
- Q4. Reference “Fenichel et al. (2011)” is not present in the reference list. Please provide the same in the list with complete bibliographic details or else delete from the text.
- Q5. Please provide the Legend for Figure 1 and 2.
- Q6. Please provide the issue numbers in the journal references wherever it is missing.
- Q7. Please provide the complete bibliographic details of the reference “Boily (2002)”.
- Q8. Please update the reference “Dupas et al. (2014)”, if already published.
- Q9. Please provide the editors’ name and publisher’s location in the references “Kremer (2000a,b)”.
- Q10. McNeill 1977 is not cited in text.
- Q11. Please check Montalvo and Reynal-Querol 2007 is not cited in text.

Typesetter queries:

Non-printed material:

MIGRATION AND THE EQUILIBRIUM PREVALENCE OF INFECTIOUS DISEASES

ALICE MESNARD

City University London

Email: alice.mesnard.1@city.ac.uk

PAUL SEABRIGHT

Institute for Advanced Study in Toulouse and CEPR

Abstract: This paper models how migration both influences and responds to differences in disease prevalence between cities and shows how the possibility of migration away from high-prevalence areas affects long-run steady state disease prevalence. We develop a dynamic framework where migration responds to the prevalence of disease, to the costs of migration and to the costs of living. The model explores how pressure for migration in response to differing equilibrium levels of disease prevalence generates differences in city characteristics such as land rents. Competition for scarce housing in low-prevalence areas can create segregation, with disease concentrated in high-prevalence “sinks”. We show that policies affecting migration costs affect the steady-state disease prevalences across cities. In particular, migration can reduce steady-state disease incidence in low-prevalence areas while having no impact on prevalence in high-prevalence areas. This suggests that, in some circumstances, public health measures may need to avoid discouraging migration away from high-disease areas.

Keywords: migration, infectious diseases, public health

JEL Classifications: I18, O15, O19, R23

1. INTRODUCTION

This paper studies the reciprocal causality between migration and the incidence of disease. While it is well known that migration can contribute to spreading diseases through the effect of crowding, we study its consequences for the composition of populations, which, in turn, affects the evolution of disease. If migration responds to disease incidence as well as influences it, feedback effects may either dampen

We are grateful to Jerome Adda, Emmanuelle Auriol, Jean-Paul Azam, Michael Ben-Gad, David Bardey, Clive Bell, Bastien Chabe-Ferret, Pascaline Dupas, Saqib Jafarey, Denis Gromb, Michael Kremer, Subhrendu Pattanayak, Nicola Pavoni, William Poulriot, Martin Ravallion, Colin Rowat, Patrick Rey, and participants in seminars in Toulouse, Cornell and City University and in conferences organized by the EUDN, PSE and the AFD in Paris, the AFD in Harvard, the NORFACE at UCL, the CSAE, CRETE, and the PopPov network for helpful comments. We also thank the AFD, the IRD, the AIRD, and the William, and Flora Hewlett foundation, for their financial support through the grant “Health risks and Migration”. The usual disclaimer applies.

1 or magnify initial differences in disease prevalence between locations. We show
 2 in particular how, for a large class of infectious endemic diseases, migration can
 3 magnify initial differences, since the healthy have a stronger incentive than the
 4 sick to flee unhealthy neighborhoods. This can turn some localities into “sinks”
 5 whose initial high disease prevalence attracts further sick individuals because they
 6 cannot compete with healthy individuals for scarce space in healthier localities. We
 7 show that several steady states can exist with different degrees of segregation of
 8 sick and healthy individuals. In fact, higher segregation is beneficial in the model,
 9 since it unambiguously reduces prevalence in low-prevalence locations and does
 10 not necessarily increase it in high-prevalence locations. This has implications for
 11 public policy towards migration and may provide a case for encouraging or even
 12 subsidising migration that has such results.

13 Epidemiologists have already addressed how individual migration may contribute,
 14 among other factors, to the spread of disease¹ and a few studies have
 15 assessed its relative importance in malaria’s eradication in the Early twentieth
 16 Century United States (Barreca et al., 2011) or the contribution of forced migration
 17 to the incidence of malaria in refugee-receiving countries (Montalvo and Reynal-
 18 Querol, 2006). But much less is known about how migration in turn responds to
 19 infectious diseases, although numerous historical instances have been recorded
 20 of people fleeing plague or other epidemics by migrating to distant areas (see
 21 McNeill, 1997)².

22 There is also abundant historical evidence of endemic disease as a factor in
 23 individuals’ location decisions. Historians have shown that infectious diseases
 24 causing high mortality rates among settlers were a key determinant of European
 25 colonization. Among other examples, Acemoglu et al. (2001) refer to Crosby
 26 (1986 pp. 143–144) who has shown that the Pilgrim Fathers decided to migrate to
 27 the United States rather than to Guyana because of the high mortality rates from
 28 infectious diseases in Guyana. We also know from Alexis de Tocqueville and other
 29 witnesses of that period that it took the draining of the malarial swamps in the
 30 State of Michigan in the mid nineteenth century for the interior of the state to be
 31 opened up by settlers on a scale comparable to what had already occurred further
 32 west in Illinois³.

33 Even today, there is a vast difference between different parts of the world in
 34 the incidence of infectious disease. Mortality statistics published by the World
 35 Health Organization⁴ reveal that deaths from infectious or parasitic disease make
 36 up just over 2% of all deaths in Europe, and some 3% of deaths in North America,
 37 while they make up over 52% of all deaths in Africa (9% of all deaths being due
 38 to malaria and 20% of all deaths being due to HIV/AIDS). The world average
 39 is a little under 20% of all deaths. Given the importance of infectious disease
 40 in mortality, it would be surprising if individuals did not take variations in its
 41 incidence into account in their location decisions. The location of some important
 42 cities (Nairobi in Kenya, for example, or Colombo in Sri Lanka) seems likely to
 43 have benefited from their low rates of malarial incidence compared to the rest of
 44 the country.

1 Obviously, there are correlations between high rates of disease prevalence and
 2 a high incidence of poverty, and the complex linkages between poverty and in-
 3 fectious disease make their interaction an interesting area of study. Economists
 4 have begun to study the channels through which health outcomes interact with
 5 economic factors (Bell and Gersbach, 2006, Bell et al. 2006, Duncan et al. 2002,
 6 Hurd, et al. 2003, Marmot, 2002). And there is strong evidence that epidemic
 7 outbreaks cause important economic losses⁵. Furthermore, asset markets may also
 8 be affected, as was observed on the housing market in Hong Kong after the SARS
 9 outbreak (Wong, 2008). However, among these linkages, migration has attracted
 10 rather little attention and yet has been under increasing focus since the SARS
 11 outbreak in China.

12 This paper models how the decisions of individuals to live in different areas are
 13 determined both by the health environment, captured in our model by different
 14 prevalence rates of diseases, and by economic factors such as costs of living. To
 15 capture the costs of living or of any fixed asset or amenity attached to a given area,
 16 the price of which increases with more people willing to settle in the area, we
 17 assume that there is a constant stock of land in each city and that rents will vary
 18 to clear the market for land.⁶ This requires of course a dynamic set-up where the
 19 economic and health environment are affected by migration, and in turn determine
 20 individual decisions such as migration.

21 At the beginning of each period, individuals find themselves in one of two
 22 cities, which differ in a number of characteristics including the prevalence of
 23 disease. The two cities could also be interpreted as countries or regions, or even
 24 in some circumstances as different sectors of the economy.⁷ Individuals have
 25 characteristics of their own, and in the model we focus on their health status
 26 (wealth, which normally differs among individuals, is here assumed to be the
 27 same for all individuals in one city so as to focus attention on differences in
 28 health). These individuals must make decisions about whether to stay in their city
 29 of origin or to migrate to the other city, which determines their consumption levels
 30 and their risk of being infected in the future : the benefits of risk reduction must
 31 be balanced against the costs, which here comprise not just migration costs but
 32 also any difference in the cost of living in the two cities. Time is infinite and
 33 the significance of the future is summarized in terms of a value function whose
 34 parameters are the health status of the individual and the characteristics of the
 35 city where she lives, both of these considered at the start of the following period.
 36 Each individual's decisions therefore involve balancing the impact of varying the
 37 migration choice on her current utility and her future discounted value function.

38 We assume that healthy individuals in any one city are *ex ante* identical in
 39 terms of risk of infection. In this respect, we differ from Mesnard and Seabright
 40 (2009), where individuals differ in an individual risk parameter. As individuals
 41 may have more information than the health authorities about their probability of
 42 being infected, we showed that quarantine measures may have unexpected effects
 43 on the spread of diseases, as they may give too high or too low incentives to
 44 migrate. This comes from the fact that migrants exert an externality on other

1 individuals living in the destination and origin areas, which depends on their own
 2 risk of having caught the disease. The type of externality outlined in that two
 3 period model affects the spread of disease in the short run – just after the outbreak
 4 of an epidemic disease. In contrast, in the present paper, we show that there is
 5 a qualitative difference between short run and long run externalities. When the
 6 costs of migration are prohibitively high, an individual living in a city with zero
 7 prevalence imposes a very large externality in the long run if she acquires an
 8 infectious disease, because the people she infects directly and indirectly lead to
 9 the disease eventually reaching a positive steady state prevalence. However, if
 10 the same individual can migrate to a high prevalence city, the long run externality
 11 he/she imposes is zero. The fact that migration may lead to a sorting of sick/healthy
 12 individuals to high/low infected areas, has thus important policy implications that
 13 are studied in the present paper.

14 We first present the model and then solve for a steady state in prevalence in
 15 circumstances where migration costs are sufficiently high to discourage migration
 16 both in and out of the steady state. As expected, we show that cities with a healthier
 17 environment will have lower disease prevalence rates.

18 Next, we examine the properties of the steady states when migration costs are
 19 sufficiently low that healthy individuals in the high prevalence city will wish to
 20 migrate to the low-prevalence city, thereby bidding up rents in that city and en-
 21 couraging sick individuals to migrate in the opposite direction. Historically, there
 22 is good reason to think that segregation of neighborhoods by disease incidence
 23 has an important influence on the spatial composition of cities and countries,
 24 and that relative housing costs play an important part in this process. The East
 25 End of London was considered a sink of disease (as well as of other unsavory
 26 characteristics) and prosperous citizens paid considerable sums to live in the West
 27 End, which thanks to the prevailing (westerly) winds could escape the noxious
 28 odors emanating from the east more easily than the east could avoid those from
 29 the west (the winds were thought to transmit disease – through “miasma” – as well
 30 as foul odors). Thus, although we do not claim that the phenomena, we model are
 31 the only or even the main influences of infectious diseases on migration, it seems
 32 likely that they help explain some important characteristics of the geography of
 33 development, past, and present.

34 We show that, if all sick individuals do thus migrate, the prevalence rate in the
 35 low-prevalence city declines to zero so that there are no more infected individuals
 36 and migration in the steady state no longer occurs. However, the *possibility* of mi-
 37 gration is important because it removes potentially infectious individuals from the
 38 locality where they can infect the most other people. The possibility of migration
 39 strictly lowers steady-state prevalence levels, and, it is unambiguously desirable
 40 to reduce migration costs to make this possible.

41 Our prediction that migration may under certain conditions induce sorting by
 42 health status has obvious echoes of the literature on segregation by individuals
 43 induced by their demand for local public goods such as education. The idea
 44 that individuals might sort between locations according to their preferences for

1 local public goods goes back to Tiebout (1956), and there is a substantial more
 2 recent literature exploring the conditions under which that sorting would result
 3 in segregation of households by income (see Ellickson, 1971, for a pioneering
 4 contribution and Epple, 2003, for an overview). As documented by Timmins
 5 (2005), there is much controversy about the strength of such effects and about how
 6 to estimate them econometrically. Particular applications have included sorting by
 7 preference for education provision (see Bénabou, 1996a, b, and Fernandez and
 8 Rogerson, 1996). There is a sense in which our model here documents broadly
 9 similar effects, since the prevalence rate of a disease has the relevant characteristics
 10 of a public good (or more precisely a public bad).

11 A distinctive feature of our model is that cities are likely to be in different
 12 steady states depending on the level of migration costs, in some of which there is
 13 sorting by migration, and with endogenous differences in health status even in the
 14 equilibria without sorting. It is also true in our model, unlike in many public-good
 15 models, that some of these equilibria can be Pareto-ranked, and in particular that
 16 there exist equilibria with the possibility of sorting outside the steady state that
 17 dominate equilibria without that possibility. These features in our view cast useful
 18 light on the possible role of migration policies in selecting one equilibria.

19 The paper proceeds as follows. Section 2 presents the links to the literature
 20 on epidemiology. Section 3 presents the model, Section 4 explains how indi-
 21 viduals make their decisions, Section 5 studies the steady-state equilibria and
 22 shows that the existence of one or the other equilibrium depends importantly
 23 on the level of migration costs. Section 6 discusses the policy implications and
 24 concludes.

25 2. LINKS TO THE LITERATURE ON EPIDEMIOLOGY

26 Related work on infectious diseases can be classified in three main strands. A first
 27 strand of the literature uses dynamic models of epidemic diseases to understand
 28 the effects of different policies on the evolution of infectious diseases. For exam-
 29 ple, Sethi (1978) studies optimal quarantine programs, which are modeled as an
 30 exogenous decrease in the infectivity parameter characterizing a specific disease.
 31 One shortcoming of these papers is that they assume away any potential behavioral
 32 response to the policies/changes considered. However, there is growing evidence
 33 pointing out that individual behavior is key to explain the evolution of aggregate
 34 disease (see Auld 2003, Reluga 2010 or Fenichel et al., 2011, and Fenichel,
 35 2013).

36 Two further strands of the literature capture this key role of individual decisions.

37 Models of decisions where individuals are rational have mostly focused on
 38 preventive behaviors such as vaccine or safe sex adoption (Geoffard and Philip-
 39 son 1996, Kremer 2000a, 2000b, Philipson, 2000); or partner choice decision
 40 (Philipson, 2000, Dupas et al. 2014). These studies obtain the common result that
 41 centralized measures may be ineffective for a number of reasons overviewed by
 42 Chen and Toxvaerd (2014). For example, Geoffard and Philipson (1996) show

1 that if demand for prevention treatments such as vaccines is prevalence elastic
 2 initially successful public health efforts typically run into diminishing returns,
 3 not simply for technical reasons but because the decline of a disease discourages
 4 prevention. Similar considerations apply to the factors determining the adoption
 5 of means of contraception as barrier methods for Sexually Transmissible Dis-
 6 eases, and a growing literature now focuses on the microeconomic determinants
 7 of such individual decisions, in order to reach a better understanding of epi-
 8 demiological patterns (See Gersovitz and Hammer (2003, 2004) and Pattanayak
 9 et al. (2007) for surveys of the evidence on the prevalence elasticity of preventive
 10 behavior).

11 The third strand of literature, which can be characterized as Behavioral Epidemi-
 12 ology, explores the consequences of individuals' exposure to risk using models
 13 of bounded rationality or studies how information about disease or the value of
 14 treatments spreads via word-of-mouth learning (Medlock et al., 2009; d'Onofrio
 15 et al., 2013; Bauch et al., 2013; Fenichel and Wang, 2013).

16 Although most of these studies appear to focus on a single aspect of epidemi-
 17 ology, namely preventive behaviors, they explore a wide range of behavior types
 18 with very different policy implications. Depending on whether individual deci-
 19 sions are complements such as for adoption of safe sex or substitutes such as for
 20 vaccination, interventions may have very different effects on the overall disease
 21 prevalence. In particular, when prevention leads to complementarities between
 22 individual utilities, multiple equilibria are possible. Our model will show that the
 23 decision to migrate displays similar properties, which leads to multiple equilibria
 24 and an interesting policy coordination issue.

25 To our knowledge, migration as a preventive behavior has not been studied
 26 before Mesnard–Seabright (2009) and the effects of migration restrictions such as
 27 quarantine measures have been overlooked by the literature using decentralized
 28 decision making frameworks. Yet, as was observed in the aftermath of the Ebola
 29 crisis, individuals may respond to strict quarantine measures by moving to dif-
 30 ferent areas, which may have unexpected effects on the evolution of diseases. In
 31 such circumstances, Mesnard–Seabright (2009) highlighted potential unexpected
 32 effects of too strong quarantine measures pushing still asymptomatic but sick
 33 individuals to escape from the epicenter of an epidemic disease: individuals who
 34 are more accurately informed than the authorities about their previous exposure
 35 to infection, may choose to migrate “strategically” just after the outbreak of an
 36 epidemic disease into a low prevalence area while still asymptomatic, which entails
 37 negative externalities.

38 The present paper highlights another channel through which migration restric-
 39 tions for controlling spread of infectious diseases may have undesirable effects.
 40 Even without asymmetric informational issues, we show that too strict migration
 41 restrictions may lead in the long run to an equilibrium with a higher overall
 42 prevalence rate of disease than without restrictions. Indeed, strong migration re-
 43 strictions may distort the sorting of sick/healthy individuals to high/low infected
 44 areas.

3. THE MODEL

Consider a discrete time, infinite horizon model with two equally-sized cities in terms of population, indexed by $i \in \{1, 2\}$.⁸ Since our purpose here is to capture externalities due to pure compositional effects, we focus on the fact that migration incentives in one direction create pressures for migration in the opposite direction because of resource constraints and adopt the simplifying assumption that populations are constant, and normalized to unity⁹.

Y denotes the constant per-period exogenous income in both cities (income is an endowment, and agents are assumed identical in income).¹⁰ Out of this income, individuals must pay a rent r_{it} in the city in which they choose to live. To simplify the calculations, we assume that land is not scarce at the margin in the city with low rent, so that $r_{it} = 0$ whenever $r_{it} < r_{jt}$. This means we can write $r_t \equiv r_{jt}$ for the city with high rent. This rent will be endogenously determined by a land market that clears when the net demand of individuals for migration to the city with high rent is zero.

We assume that one city has a more disease-prone environment, which favors the spread of disease vectors (think for example as low altitude or a high degree of humidity, which may favor airborne or insect borne diseases). We will capture the degree of disease-proneness of city i 's environment by a parameter α_i and assume, without loss of generality, that $\alpha_1, \alpha_2 < 1$, which ensures interior solutions by ruling out theoretically possible but empirically uninteresting cases of diseases which affect the entire population.

Each individual can be in two states of health, denoted by θ : sick (S) or healthy (H).¹¹ At time t , a proportion p_{it} of city i 's population are sick, the proportion of healthy inhabitants being therefore $(1 - p_{it})$. We call p_{it} the "prevalence" of the disease in city i at time t . We assume that the parameters α_i are such as to ensure that

$$p_{1t} + p_{2t} < 1 \tag{1}$$

to capture the fact that the sick are a minority of the total population.

There is an exogenous utility cost m of migrating from one city to the other. This represents any kind of costs, which, like psychological costs, may be non-monetary and directly affect individuals' utility. Utility is separable in this cost and in a term $U(c_t, \theta_t)$ that is increasing and weakly concave in consumption c_t at time t .

The assumption that utility is separable in the migration cost greatly simplifies the calculations because the derivatives of utility with respect to rents and disease prevalence do not depend on whether an individual has migrated in any given period, but it is unlikely that the qualitative findings of the model turn importantly on this restriction.

At the beginning of each period, individuals observe their current health status. They also have perfect foresight of the values of all parameters and city-level endogenous variables (namely Y, m, p , and r). They choose whether or not to

1 migrate to the other city and all individuals receive the incomes and pay the rents
2 in the city they have chosen to live in and consume the residual.

3 Accordingly, individuals living in city i face a per period budget constraint:

$$Y - r_{it} = c_t. \quad (2)$$

4 The health status of individuals evolves as follows:

5 Healthy individuals' likelihood of becoming infected increases with the local
6 prevalence, p_{it} , and the degree of disease-proneness of their environment:

$$P [\theta_{t+1} = S/\theta_t = H] = \alpha_i p_{it}. \quad (3)$$

7 Sick individuals recover from the disease naturally with exogenous probability
8 π .¹²

$$P [\theta_{t+1} = H/\theta_t = S] = \pi. \quad (4)$$

9 We assume $\pi < \alpha_2 < \alpha_1$ in order to focus on interior solutions (diseases with
10 higher recovery rates never become established as endemic in the population).

11 The expected present value of current and future utility of individuals of type
12 θ_t in city i at time t is

$$W_{it} = \sum_{\tau=t}^{\infty} \gamma^{\tau-t} [U(c_t, \theta_t) - mI_t], \quad (5)$$

13 where I_t is an indicator function taking the value 1 if they migrate in period t ,
14 otherwise 0.

15 We make the following assumptions about the effect of sickness on individual
16 utilities:

17 Sickness lowers current welfare

$$U(c_t, S) \leq U(c_t, H). \quad (6)$$

18 The marginal utility of consumption is independent of health status

$$U'(c_t, H) = U'(c_t, S) \text{ for all } c_t. \quad (7)$$

19 This assumption, it should be noted, implies (by integration of the function over
20 a finite interval) that the utility cost of any given reduction in consumption de-
21 pends only on the amount of the reduction in consumption and not on the health
22 status of the individual experiencing it. Noting that utility is strictly monotonic in
23 consumption, this assumption implies that, for any θ_i and θ_j ,

$$U(Y, \theta_i) - U(Y - r_i, \theta_i) > U(Y, \theta_j) - U(Y - r_j, \theta_j) \text{ if and only if } r_i > r_j. \quad (8)$$

24 In the context of our model, this has the consequence, as will be seen, that the
25 healthy are willing to pay more than the sick to migrate to live near to other
26 healthy people.¹³ Our model should hence be seen as exploring the consequences

1 in equilibrium of an empirically interesting phenomenon – namely the greater
 2 willingness of the healthy than the sick to migrate to live near other healthy
 3 people.

4 Note that we do not allow individuals to smooth consumption across time.
 5 Allowing for savings in our model would make each individual's decisions in any
 6 period dependent on the entire history of their consumption decisions as well as
 7 on their entire medical history, which would greatly complexify the model with
 8 no extra gains for the understanding of our main results. As it is, individuals'
 9 decisions are fully determined by their current health status and their city of
 10 residence, which gives us four distinct cases to study. We therefore write the
 11 objective function explicitly as a function of current health status as $W_{it}(\theta_t)$.

12 4. INDIVIDUAL DECISIONS

13 We first note that the objective function can be rewritten as follows, where i is
 14 the individual's city at the beginning of the period and k is the city in which she
 15 chooses to live:

$$W_{it}(\theta_t) = U(Y - r_{kt}, \theta_t) - mI_t + \gamma \sum_{\theta_{t+1}} W_{kt+1}(\theta_{t+1}) P(\theta_{t+1}|\theta_t),$$

16 where $I_t = 1$ if $i \neq k$ and 0 otherwise.

17 From this, it follows that $W_{it}(\theta_t)$ is strictly increasing in Y and strictly decreasing
 18 in r_{kt} , and weakly decreasing in m .

19 Next, for each value of the current health status, we compare the utility of each
 20 individual in case she chooses not to migrate to the utility in case she migrates.

21 4.1. Individual Migration Choices

22 A healthy individual who chooses not to migrate and for whom therefore $k = i$
 23 will obtain utility $V_{it}^N(H)$, which is equal to

$$V_{it}^N(H) = U(Y - r_{it}, H) + \gamma \alpha_i p_{it} W_{it+1}(S) + \gamma(1 - \alpha_i p_{it}) W_{it+1}(H). \quad (9)$$

24 A healthy individual who chooses instead to migrate will obtain utility $V_{it}^M(H)$,
 25 which is equal to

$$V_{it}^M(H) = U(Y - r_{jt}, H) - m + \gamma \alpha_j p_{jt} W_{jt+1}(S) + \gamma(1 - \alpha_j p_{jt}) W_{jt+1}(H). \quad (10)$$

26 The agent who is currently sick and chooses not to migrate will receive utility
 27 $V_{it}^N(S)$, which is equal to

$$V_{it}^N(S) = U(Y - r_{it}, S) + \gamma \pi W_{it+1}(H) + \gamma(1 - \pi) W_{it+1}(S). \quad (11)$$

28 A sick individual who chooses instead to migrate from city i to city j will obtain
 29 utility

1 $V_{it}^M(S)$, which is equal to

$$V_{it}^M(S) = U(Y - r_{jt}, S) - m + \gamma\pi W_{jt+1}(H) + \gamma(1 - \pi)W_{jt+1}(S). \quad (12)$$

2 Consequently, the condition for healthy individuals to migrate rather than to remain
3 in their city of origin can be written as $V_{it}^M(H) - V_{it}^N(H) \geq 0$, or written out in
4 full as

$$0 \leq U(Y - r_{jt}, H) - U(Y - r_{it}, H) - m + \gamma\alpha_j p_{jt} W_{jt+1}(S) - \gamma\alpha_i p_{it} W_{it+1}(S) \\ + \gamma(1 - \alpha_j p_{jt}) W_{jt+1}(H) - \gamma(1 - \alpha_i p_{it}) W_{it+1}(H). \quad (13)$$

5 Similarly, the condition for sick individuals to migrate, $V_{it}^M(S) - V_{it}^N(S) \geq 0$, can
6 be written as out in full as

$$0 \leq U(Y - r_{jt}, S) - U(Y - r_{it}, S) - m + \gamma\pi (W_{jt+1}(H) - W_{it+1}(H)) \\ + \gamma(1 - \pi) (W_{jt+1}(S) - W_{it+1}(S)) \quad (14)$$

7 and we note that the conditions (13) and (14) are more likely to be fulfilled as
8 $r_{it} - r_{jt}$ increases and as m decreases, as we would expect.

9 4.2. Migration Flows

10 Denote by $s_{jt}(h_{jt})$, the proportion of the sick (healthy) who migrate from j to i
11 in period t .

12 The proportions s_{it} , s_{jt} , h_{it} , and h_{jt} of individuals of each type migrating from
13 one city to the other result from aggregating individual migration decisions shown
14 by the equations (13) and (14). Hence, they are determined by the level of migration
15 costs, by the differential in rental rates, which clear the market for land and by the
16 differential in disease prevalences. Specifically, if inequality (14) does not hold,
17 $s_{it} = 0$, while if it holds with strict inequality then $s_{it} = 1$. Likewise, if inequality
18 (13) does not hold, $h_{it} = 0$, while if it holds with strict inequality then $h_{it} = 1$.

19 We know that those falling sick in any period on a given city consist of those
20 previously healthy in the same city who have not migrated outwards and have fallen
21 sick, plus any previously healthy in the other city who have migrated inwards and
22 have fallen sick, plus those previously sick in the other city who have migrated
23 inwards and have not recovered, plus those who were previously sick in the same
24 city who have not migrated and who have not recovered. We can therefore write the
25 equations governing the evolution of prevalence rates in the two cities as follows:

$$p_{it+1} = (1 - \pi)p_{it}(1 - s_{i.}) + \alpha_i p_{it}(1 - p_{it})(1 - h_{it}) \\ + p_{jt}(1 - \pi)s_{jt} + \alpha_i p_{it}(1 - p_{jt})h_{jt}, \quad (15)$$

26 where $(1 - \pi)p_{it}(1 - s_{i.})$ represents the sick in period t who do not migrate and
27 remain ill; $\alpha_i p_{it}(1 - p_{it})(1 - h_{it})$ are the healthy in t who do not migrate and
28 become sick; $p_{jt}(1 - \pi)s_{jt}$ are the sick in period t who migrate from j to i and

1 remain ill; $\alpha_i p_{it}(1 - p_{jt})h_{jt}$ are the healthy who migrate from j to i and become
 2 sick.

3 For the land market to clear requires that net migration is zero, which, with
 4 equally sized cities, implies

$$s_{it}p_{it} + h_{it}(1 - p_{it}) = s_{jt}p_{jt} + h_{jt}(1 - p_{jt}). \quad (16)$$

5 A final useful piece of notation is to define $r_t^H(m)$ as the value of r_t such that
 6 inequality (13) binds for $i = 1$. Intuitively, $r_t^H(m)$ is the value of rental in the
 7 high-rent city in period t that is just high enough, given the level of migration cost
 8 m , to dissuade healthy individuals from moving there from the zero-rent city. We
 9 define $r_t^S(m)$ analogously as the value of rental in the high-rent city in period t
 10 that is just high enough, given the level of migration cost m , to induce sick individuals
 11 to move away from there to the zero-rent city. As we shall see, when migration
 12 occurs it is only of the healthy to the high-rent city and of the sick to the low-rent
 13 city.

14 The next section characterises the different Equilibria that may occur, a subset
 15 of which may represent steady states.

16 4.3. Equilibrium

17 An equilibrium of the model at time t given prevalence rates p_{1t}, p_{2t} , is a set of
 18 values $r_t, p_{1t+1}, p_{2t+1}, h_{1t}, h_{2t}, s_{1t}$, and s_{2t} such that

- 19 1) p_{1t+1}, p_{2t+1} are generated by equation (15);
- 20 2) Equation (16) is satisfied (i.e. the land market clears).

21 The Appendix establishes by Lemma 1 that, in equilibrium, there cannot be
 22 migration in both directions by individuals of a given type. This necessary property
 23 of any equilibrium is important to establish in Lemma 2 that an equilibrium
 24 exists.¹⁴ We now consider the properties of equilibria in steady state.

25 5. STEADY STATE EQUILIBRIA

26 5.1. Properties of Steady State Equilibria

27 In the steady state, the proportions of individuals in each health category remain the
 28 same across periods in each city, so we can write: $p_{it} = p_{it+1} = p_i$ for $i = 1, 2$.
 29 Therefore, the rental rate which clears the market remains constant in steady
 30 state: $r_t = r_{t+1} = r$. Similarly, we can write the steady state values of $r_t^H(m)$ and
 31 $r_t^S(m)$ as $r^H(m)$ and $r^S(m)$ respectively.

32 Lemmas 3 and 4 in Appendix show interesting properties of the continuation
 33 values for sick and healthy individuals to live in different cities in steady states,
 34 which allow to establish the following Proposition:

1 **Proposition 1** *In steady state equilibrium with $m > 0$ healthy individuals, if*
 2 *they migrate, will do so from the unhealthy city to the healthy city, while sick*
 3 *individuals, if they migrate, will do so in the opposite direction.*

4 With minimal loss of generality, let $\alpha_2 p_2 < \alpha_1 p_1$ and call 2 the “healthy city”
 5 in steady state and 1 the “unhealthy city”.¹⁵ Therefore, $s_1 = h_2 = 0$. We ignore
 6 the case where $\alpha_2 p_2 = \alpha_1 p_1$ since migration would not take place for any positive
 7 migration cost.

8 5.2. Existence, Uniqueness, and Stability of Equilibria

9 We now turn to studying the existence of different types of steady state equilibria.

10 It is straightforward to establish Proposition 2.

11 **Proposition 2** *There exists a steady state equilibrium with $p_1 = p_2 = 0$ and*
 12 *zero gross migration.*

13 This simply states that a disease cannot spread if it does not arise in the first
 14 place, and follows from the fact that healthy individuals become infected with a
 15 probability that is proportional to the prevalence of the city in which they choose
 16 to live. The fact that there is zero gross migration follows trivially from the fact
 17 that if prevalence is zero there is no motivation for migration from one city to the
 18 other.

19 However, the zero-prevalence steady state is not stable, in the sense that once
 20 an infection arises it will spread until the rate of new infections equals the rate at
 21 which sick individuals recover from the disease. Our next propositions examine
 22 the properties of such positive-prevalence steady states and show that they depend
 23 on migration costs. One Interpretation of such migration costs is the severity of
 24 restrictions on international migration, but other interpretations are possible as
 25 well; the important point of such an interpretation is that migration costs may be
 26 influenced by public policy.

27 We now examine the elementary case of steady states where an infection arises
 28 and the costs of migration are high enough to discourage all migration within
 29 any relevant neighborhood of the steady state ($h_{it} = s_{it} = 0 \forall i, t$). Where gross
 30 migration is zero, we know that those falling sick in any period consist of those
 31 previously healthy who fall sick in the same city, and in the steady state, these will
 32 exactly match the numbers recovering from the disease.

33 Indeed, the prevalence rate of disease in city i in period $t + 1$ will be equal to
 34 the proportion of healthy individuals in period t who fell sick plus the proportion
 35 of sick individuals in t who have not recovered from the disease. This can also
 36 be seen easily after rewriting equation (15) in the case where $h_{it} = s_{it} = 0 \forall i, t$,
 37 which yields $p_{it+1} = \alpha_i p_{it}(1 - p_{it}) + p_{it}(1 - \pi)$. Substituting the steady state
 38 conditions that $p_{it+1} = p_{it} = p_i$ implies that $p_i = 1 - \pi/\alpha_i$ for $i = 1, 2$. We
 39 have thus established the following proposition:

1 **Proposition 3** *When migration is impossible there exists a steady state in which*
 2 *$p_i = 1 - \pi/\alpha_i$ for $i = 1, 2$.*

3 This defines a unique equilibrium and implies cities with lower α (as for example
 4 low degree of humidity for the case of malaria) have lower steady state levels of
 5 disease prevalence and diseases with higher rates of natural recovery have lower
 6 prevalence in the steady-state. We show in the Appendix that the steady state
 7 prevalence rate in each city is locally stable.

8 Next, we consider whether there exist steady states that are compatible with
 9 positive levels of gross migration. We first establish some properties of such a
 10 steady state, if it exists, and we consider the conditions for its existence later.

11 Proposition 1 has established that it is not possible in the steady state to have
 12 flows of healthy individuals migrating to city 1 ($h_2 = 0$) and flows of sick individ-
 13 uals migrating to city 2 ($s_1 = 0$). Where gross migration is not zero, the zero net
 14 migration implied by clearing of the rental market implies that some proportion
 15 h_1 of the healthy migrate from high prevalence to low prevalence cities, and those
 16 who migrate in the other direction are a proportion s_2 of the sick (who, unless
 17 they recover from disease, have nothing to fear from high prevalence). We specify
 18 “a proportion” because of our assumption that there are more healthy than sick
 19 individuals, so complete migration by both populations will not be feasible.

20 Let the value ψ and ϕ be the the values taken in equilibrium by the variables h_1
 21 and s_2 respectively. Note that $\phi > \psi$ when migration is strictly positive, because
 22 $p_{1t} + p_{2t} < 1$ which implies that the sick in city 2 are less numerous than the
 23 healthy in city 1.

24 We use the dynamics governing the evolution of diseases in the two cities open
 25 to migration and the properties of steady state equilibria established earlier to
 26 characterize the steady state equilibria with non-zero gross migration out of the
 27 equilibrium as follows.

28 From [equation \(15\)](#) and given $h_2 = 0$, $s_1 = 0$ the steady state prevalence rates
 29 in city 1 and city 2 will respectively satisfy

$$p_1 = (1 - p)(1 - \psi)\alpha_1 p_1 + \phi p_2(1 - \pi) + p_1(1 - \pi); \quad (17)$$

$$p_2 = \alpha_2 p_2(1 - p_2) + \psi(1 - p_1)\alpha_2 p_2 + (1 - \phi)p_2(1 - \pi). \quad (18)$$

31 Moreover, the “adding up condition” has to hold in steady state, which implies
 32 that the proportion ϕ of the sick who migrate in the steady state yields the same
 33 absolute number of migrants as the proportion ψ of the healthy who migrate, so
 34 that

$$\phi p_2 = \psi(1 - p_1). \quad (19)$$

35 And the “behavioral condition” implies that the demand for migration by propor-
 36 tion ϕ of the sick is generated by the same r in city 2 as generates the demand for
 37 migration by a proportion ψ of the healthy.

Figure 1 - Colour online, B/W in print

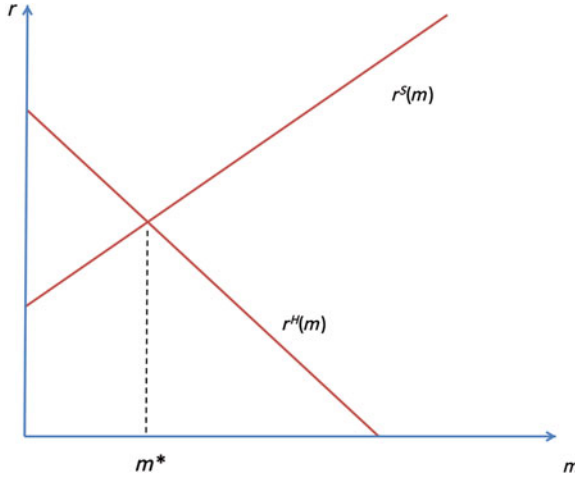


FIGURE 1

1 Replacing equation (19) into equation (17) and equation (18) gives the following
 2 conditions characterizing these steady state equilibria:

$$p_1 = (1 - p_1)(1 - \psi)\alpha_1 p_1 + \psi(1 - p_1)(1 - \pi) + p_1(1 - \pi); \quad (20)$$

$$p_2 = \frac{\alpha_2 - \pi - \phi(1 - \pi)}{\alpha_2(1 - \phi)} \text{ or } p_2 = 0. \quad (21)$$

4 Studying condition (21), we can show easily that there will no longer be migra-
 5 tion at the steady state if $p_2 = 0$ (Indeed $1 - p_1 > 0$ and equation (19) imply that
 6 $\psi = 0$). Substituting $\psi = p_2 = 0$ into equation (20), we show that $p_1 = 1 - \frac{\pi}{\alpha_1}$.

7 The question now is whether such steady states exist. To explore this question,
 8 recall that $r^H(m)$ and $r^S(m)$ denote respectively, for any m , the rental rate at which
 9 healthy individuals in the high-prevalence city are just deterred from migrating
 10 to the low prevalence city, and the rental rate at which sick individuals in the
 11 low-prevalence city can just be induced to migrate to the high-prevalence city. It
 12 is evident that $r^H(m)$ is decreasing in m and $r^S(m)$ is increasing in m . Let m^* be
 13 the value of m such that $r^H(m^*) = r^S(m^*)$.

14 Figure 1 illustrates as follows: It shows $r^H(m)$ and $r^S(m)$, which will cross
 15 at a strictly positive value of m , which we have defined as m^* , provided that
 16 $r^H(0) > r^S(0)$.¹⁶

17 Appendix shows that $r^H(0) > r^S(0)$, such that we can establish the following
 18 Proposition:

1 **Proposition 4** *There exists $m^* > 0$ such that, for all $m < m^*$, an equilibrium*
 2 *exists with $p_1 = 1 - \frac{\pi}{\alpha_1}$ and $p_2 = 0$. In this equilibrium, there is non-zero gross*
 3 *migration outside the steady state but not at the steady state.*

4 The intuition behind the proof is relatively straightforward: because the healthy
 5 are more willing than the sick to pay to live in the low-prevalence city, there will
 6 be a rental rate that compensates the sick for moving to the high-prevalence city
 7 and does not deter the healthy from migrating to the low-prevalence city, provided
 8 migration costs are low enough.

9 Note how the possibility of migration out of the steady state makes the crucial
 10 difference between the steady states described in Propositions 3 and 4 *even though*
 11 *in the steady state no actual migration takes place*. This is because any infected
 12 individuals who arise in city 2, instead of remaining in city 2 where they progres-
 13 sively infect the rest of the population, migrate out immediately to city 1. This
 14 keeps the prevalence rate at zero in city 2. It has no lasting effect on the prevalence
 15 in city 1, though, because in the steady state there is no further in-migration and
 16 the prevalence in city 1 is determined in exactly the same way as it was in the
 17 non-migration steady state.

18 Moreover, we show in the Appendix that the Low migration costs characterized
 19 above generate an unstable root, which pushes the prevalence rate in the healthy
 20 city to a corner. This type of corner steady state is the only one compatible with
 21 positive migration flows along the transition path and is robust to small changes
 22 of parameter values.

23 It is straightforward to show that there is no steady state equilibrium with non-
 24 zero gross migration in steady state (such that $\varphi > 0$, $\psi > 0$), because to do so
 25 would require the two migration inequalities (13) and (14) to bind at the same
 26 level of r , which is impossible as shown in the proof of Proposition 1.¹⁷

27 Having established the existence of these equilibria, we now turn to highlighting
 28 an interesting property of the steady state with migration in Proposition 5.

29 **Proposition 5** *In the equilibrium with $p_2 = 0$, the healthy city may be the*
 30 *city with the more disease-prone environment, that is, $p_2 < p_1$ is compatible with*
 31 *$\alpha_2 > \alpha_1$.*

32 The proof of this proposition follows immediately from Proposition 1 and noting
 33 that if $p_2 = 0$, $\alpha_2 p_2 = 0 < \alpha_1 p_1$ whatever the value of α_2 .

34 This shows that, in the case of Low migration costs, there is a possibility of
 35 multiple equilibria because of strategic complementarities in the utility function.
 36 Indeed the probability of becoming infected is a positive function of the degree of
 37 disease-proneness in city i , α_i , and of the proportion of sick individuals in city i ,
 38 p_i , but the latter is determined in equilibrium. This generates a clear coordination
 39 problem and underlines an important role of expectations in the model. A city
 40 can be established as the more healthy city, and therefore become a destination
 41 for healthy individuals, with higher rents that drive out sick individuals, simply
 42 because it is expected to be more healthy, in spite of having a more disease-prone

1 environment. However, it is unlikely that it would become so established if mi-
 2 gration costs are initially very high and are gradually reduced, since the autarky
 3 prevalence of the more disease-prone city will be higher, so that initial migration
 4 by the healthy is likely to be from the more disease-prone to the less disease-prone
 5 environment.

6 Finally, we can show that the range of values of m for which a steady state
 7 exists with migration by the healthy to the city with the more disease-prone
 8 environment is strictly smaller than the range of values of m for which a steady
 9 state exists with migration by the healthy to the city with the less disease-prone
 10 environment. A consequence of the proof of Proposition 4 is that $r^H(0) - r^S(0)$ is
 11 strictly decreasing in the value of α_2 as well as in the value of π . Thus, the more
 12 disease prone the environment of the city to which the healthy are migrating and
 13 the higher the recovery rate, the smaller the value of the m^* for which a migration
 14 equilibrium exists. This is rather intuitive since a more disease prone environment of
 15 the healthy city and a higher recovery rate make migration less attractive as a
 16 way of escaping from endemic diseases.

17 This allows us to state our final proposition which characterizes existence
 18 conditions of steady state equilibria for all values of m .

19 **Proposition 6** *For any value of π , there exists two strictly positive values of m ,*
 20 *m^* , and m' , with $m' < m^*$ such that, the following steady state equilibria exist:*

- 21 1) *when migration costs exceed m^* , there is no migration; $p_1 = 1 - \frac{\pi}{\alpha_1}$ and $p_2 =$*
 22 *$1 - \frac{\pi}{\alpha_2}$.*
- 23 2) *when migration costs lie between m' and m^* , the only equilibrium has migration out*
 24 *of steady state by the healthy from the city with the more disease-prone environment*
 25 *to the city with the less disease-prone environment; $p_1 = 1 - \frac{\pi}{\alpha_1}$ and $p_2 = 0$.*
- 26 3) *when migration costs lie below m' , there are two steady state equilibria in each of*
 27 *which $p_1 = 1 - \frac{\pi}{\alpha_1}$ and $p_2 = 0$. In the first steady state, city 2, the city to which the*
 28 *healthy migrate out of steady state, is the one with the less disease-prone environment*
 29 *and, in the second, city 2 is the one with the more disease-prone environment.*

30 **Figure 2** illustrates the relation of m' to m^* .

31 5.3. Welfare Comparisons

32 Now, we can consider the comparative welfare properties of the steady states with
 33 and without the possibility of migration. It is straightforward to see that the steady
 34 states with migration are Pareto-superior to the steady state without migration.
 35 Prevalence in city 1 is the same in the steady states defined by Propositions 3 and
 36 4; only that in city 2 differs (and is strictly lower in the migration steady states).
 37 Given that there is no migration (and therefore no migration costs incurred) in
 38 all steady states, this means that the steady states with migration have fewer
 39 sick individuals and incur no offsetting costs. We have therefore established the
 40 following:

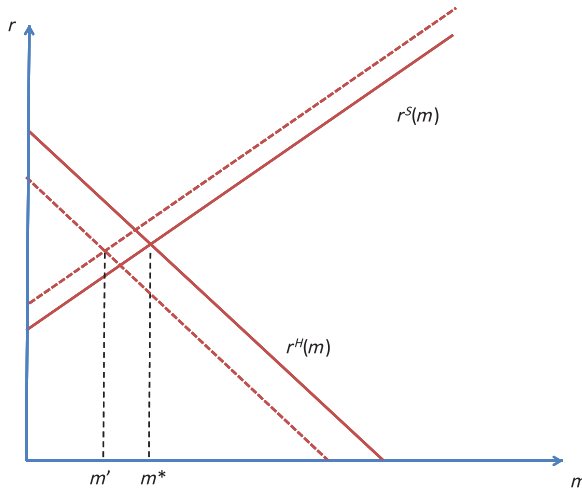


FIGURE 2

1 **Proposition 7** *The steady states with migration are Pareto-Superior to the*
 2 *steady state without migration.*

3 However, we can also Pareto-rank the two steady states that exist when migration
 4 costs lie below m' , since they differ in their values of α_1 , and therefore in the
 5 prevalence rates in the high-prevalence city. It is immediate that

6 **Proposition 8** *The second steady state, that exists only when migration costs*
 7 *are lower than m' , has lower prevalence than, and is therefore Pareto Superior to,*
 8 *the first steady state.*

9 Proposition 6 has merely characterized the steady states according to whether or
 10 not migration costs are low enough for gross migration to occur out of steady state.
 11 The fact that the steady states with non-zero gross migration out of equilibrium
 12 Pareto-dominate that with zero migration has important implications for policy. In
 13 this model, it is a good thing for there to be outmigration of sick individuals from
 14 city 2 (driven by the higher rents due to competition from individuals in-migrating
 15 from city 1). The reason for this is that outmigration of such individuals removes
 16 them from where they would contribute to new infections and places them in a city
 17 in which the disease is already established and to which their presence will bring
 18 no lasting deterioration in the prevalence. In these circumstances, action by the
 19 authorities should not be to discourage migration (which may be a by-product of
 20 quarantine measure or of other types of migration restrictions) but rather actively
 21 to encourage it. In the steady state, there will in fact be no migration, but out of
 22 the steady state such migration is an important means of reducing the risk that the
 23 disease established in city 1 also establishes itself in city 2.

6. CONCLUSION

Our analysis indicates that differences in disease prevalence rates can emerge as the equilibrium outcome of more fundamental differences in environment, with migration behavior acting as a means of arbitrage between locations with different prevalence levels. We have also shown that whether migration takes place out of the steady state has important implications for steady state prevalence levels even if there is no migration at the steady state. In particular, it is desirable for infected individuals to migrate away from low-prevalence localities since these are the ones in which they create the greatest negative externalities. This has potentially important implications for policy since it suggests that, far from seeking to discourage voluntary migration in conditions of endemic disease, it may sometimes be desirable to encourage it.

A key mechanism in our model is that the willingness to pay of healthy individuals to live close to other healthy individuals exceeds that of sick individuals, which leads to sorting by health and higher costs of living in healthier areas. This, however, is true only under certain conditions. As we discussed, under certain alternative assumptions, it could be that sick individuals would have a higher willingness to pay to live in low-prevalence environments, which would act against segregation: recovery rates may, for example, be higher in rich cities due to better health infrastructures and income dynamics or family decisions may make sick people less likely to migrate to poor cities with high prevalence rates. Moreover, as highlighted in Mesnard and Seabright (2009), under different assumptions about the distribution of past exposure to the disease and the asymmetric information individuals may have on their own risk, migrants to low-prevalence destinations may include a significant proportion of asymptomatic individuals likely to become sick, thereby mitigating segregation effects in the short run even if they do not wholly offset them.

Finally, we have shown that expectations may matter in this model with fully rational individuals, which leads to the possibility of multiple equilibria and generates an interesting coordination problem. However, it is also possible that individuals are not fully rational nor perfectly informed on their health risks when considering their location decisions, which may lead to the existence of other equilibria.

The conditions described in our model are thus not general but they do constitute an important class of cases for public policy to bear in mind. They warn policy makers to take into account positive externalities generated by migration in the presence of endemic diseases. Public policy needs to model very carefully the interactions between disease and migration in order to ensure that policy interventions do not have counter-productive consequences in the short run (Mesnard and Seabright 2009) and in the long run, as highlighted in the present paper. We also believe that segregation by disease-prevalence of neighborhoods within cities, and of regions within countries, has been a phenomenon of historical significance which models of this kind can help us to understand. Given the startling differences

1 in disease prevalence between different regions of the world, it remains of real
 2 significance today. And given the likely emergence of new forms of antibiotic-
 3 resistant infections in years to come, some of which may become endemic in
 4 certain parts of the world, the problem can only grow in importance in the future.

5 **APPENDIX A: PROOFS**

6 **Lemma 1** *There cannot be migration in both directions in equilibrium by individuals of a*
 7 *given type.*

8 **Proof of Lemma 1.**

9 The proof is by contradiction. Suppose that if $r_t = r_t^H(m)$, inequality (13) is weakly
 10 satisfied for $i = 2$.

11 Then, inserting $r_t^H(m)$ in inequality (13) for $i = 1$ yields

$$U(Y, H) - U(Y - r_t^H(m), H) = Z_t - m, \tag{22}$$

12 where

$$\begin{aligned} Z_t &= \gamma\alpha_2 p_{2t} W_{2t+1}(S) - \gamma\alpha_1 p_{1t} W_{1t+1}(S) \\ &\quad + \gamma(1 - \alpha_2 p_{2t}) W_{2t+1}(H) - \gamma(1 - \alpha_1 p_{1t}) W_{1t+1}(H). \end{aligned}$$

13 Setting $i = 2$ in inequality (13) yields

$$0 \leq U(Y, H) - U(Y - r_t^H(m), H) - m - Z_t$$

14 and substituting equation (22) in inequality (13) yields

$$0 \leq Z_t - m - m - Z_t$$

15 which is a contradiction as $m > 0$. This shows that inequality (13) cannot be satisfied
 16 for both $i = 1$ and $i = 2$ if $r_t = r_t^H(m)$. For all $r_t > r_t^H(m)$, inequality (13) will not be
 17 satisfied for $i = 1$, while for all $r_t < r_t^H(m)$ inequality (13) will not be satisfied for $i = 2$.
 18 Thus, inequality (13) cannot be simultaneously satisfied for $i = 1$ and $i = 2$ at any value
 19 of r_t . Analogous arguments when inequality (14) binds for $i = 2$ inequality (14) cannot be
 20 simultaneously satisfied for $i = 1$ and $i = 2$ at any value of r_t . ■

21 Lemma 2 establishes that an equilibrium exists:

22 **Lemma 2** *For any p_{1t}, p_{2t} and for any s_{1t}, s_{2t} implied by p_{1t}, p_{2t} , there exist h_{it} , for*
 23 *$i = 1, 2$ such that equation (16) is satisfied with $0 \leq h_{it} \leq 1$.*

24 **Proof of Lemma 2.**

25 From Lemma 1, we know that if $s_{it} > 0, s_{jt} = 0$ and that if $h_{it} > 0, h_{jt} = 0$. Thus,
 26 equation (16) implies

$$s_{it} p_{it} = h_{jt} (1 - p_{jt}) \text{ for } i = 1, 2$$

27 which can be rewritten

$$h_{jt} = \frac{s_{it} p_{it}}{(1 - p_{jt})} \text{ for } i = 1, 2.$$

Since [equation \(1\)](#) implies that $0 \leq p_{it} < (1 - p_{jt})$ and since $0 \leq s_{it} \leq 1$, it follows that $0 \leq h_{it} \leq 1$. ■

Properties of Steady State Equilibria

We write $W_i(H) = W_{it}(H) = W_{it+1}(H)$ and $W_i(S) = W_{it}(S) = W_{it+1}(S)$ for the continuation value of living in city i for a healthy and a sick individual respectively.

We can therefore write the migration conditions [\(13\)](#) and [\(14\)](#) in their steady state forms as

$$\begin{aligned} 0 \leq & U(Y - r_j, H) - U(Y - r_i, H) - m + \gamma \alpha_j p_j W_j(S) - \gamma \alpha_i p_i W_i(S) \\ & + \gamma(1 - \alpha_j p_j) W_j(H) - \gamma(1 - \alpha_i p_i) W_i(H) \end{aligned} \quad (23)$$

and

$$\begin{aligned} 0 \leq & U(Y - r_j, S) - U(Y - r_i, S) - m + \gamma \pi (W_j(H) - W_i(H)) \\ & + \gamma(1 - \pi) (W_j(S) - W_i(S)). \end{aligned} \quad (24)$$

We can then show two properties of the continuation values summarized in [Lemmas 3](#) and [4](#).

Lemma 3 *In steady state equilibrium, the additional continuation value of being healthy rather than sick in city i , $W_i(H) - W_i(S)$, is strictly positive and decreasing in the prevalence rate p_i , in the unhealthiness parameter α_i , and in the recovery rate π .*

Proof. Subtracting [equation \(11\)](#) from [equation \(9\)](#) yields

$$\begin{aligned} W^i(H) - W^i(S) = & U(Y - r_i, H) - U(Y - r_i, S) \\ & + \gamma(1 - \alpha_i p_i - \pi) W^i(H) - \gamma(1 - \alpha_i p_i - \pi) W^i(S) \end{aligned}$$

which implies that

$$W^i(H) - W^i(S) = \frac{U(Y - r_i, H) - U(Y - r_i, S)}{1 - \gamma(1 - \alpha_i p_i - \pi)}$$

which is strictly positive by [assumption \(6\)](#) and strictly decreasing in α_i , p_i , and π . ■

Lemma 4 *In steady state equilibrium, if $\alpha_j p_j > \alpha_i p_i$ the difference between the additional continuation values for healthy and sick individuals of living in city i rather than in city j , $(W_i(H) - W_j(H)) - (W_i(S) - W_j(S))$, is strictly positive and decreasing in the prevalence rate p_i , in the unhealthiness parameter α_i , and in the recovery rate π .*

Proof.

From [equation \(9\)](#) we can write

$$\begin{aligned} W^i(H) - W^j(H) = & U(Y - r_i, H) - U(Y - r_j, H) + \gamma \alpha_i p_i W^i(S) - \gamma \alpha_j p_j W^j(S) \\ & + \gamma(1 - \alpha_i p_i) W^i(H) - \gamma(1 - \alpha_j p_j) W^j(H). \end{aligned} \quad (25)$$

Similarly,

$$\begin{aligned} W^i(S) - W^j(S) = & U(Y - r_i, S) - U(Y - r_j, S) + \gamma \pi [W^i(H) - W^j(H)] \\ & + \gamma(1 - \pi) [W^i(S) - W^j(S)]. \end{aligned} \quad (26)$$

1 First, note that $U(Y - r_i, \theta) - U(Y - r_j, \theta)$ is independent of θ by [equation \(8\)](#), thus
 2 $[U(Y - r_i, H) - U(Y - r_j, H)] - [U(Y - r_i, S) - U(Y - r_j, S)] = 0$.

3 Therefore, subtracting [equation \(26\)](#) from [equation \(25\)](#) and re-arranging yields

$$\begin{aligned} W^i(H) - W^j(H) - [W^i(S) - W^j(S)] \\ = W^i(H)[\gamma(1 - \alpha_i p_i - \pi)] - W^i(S)[\gamma(1 - \alpha_i p_i - \pi)] \\ \dots - W^j(H)[\gamma(1 - \alpha_j p_j - \pi)] + W^j(S)[\gamma(1 - \alpha_j p_j - \pi)] \end{aligned}$$

4 which in turn yields

$$\begin{aligned} W^i(H) - W^j(H) - W^i(S) + W^j(S) \\ = \frac{1}{1 - \gamma(1 - \pi)} [\alpha_j p_j (W^j(H) - W^j(S)) - \alpha_i p_i (W^i(H) - W^i(S))]. \end{aligned}$$

5 The RHS expression is strictly positive because $\alpha_j p_j > \alpha_i p_i$ and

$$\alpha_i p_i (W^i(H) - W^i(S)) = \alpha_i p_i \frac{U(Y - r_i, H) - U(Y - r_i, S)}{1 - \gamma(1 - \alpha_i p_i - \pi)}$$

6 which is strictly increasing in $\alpha_i p_i$ because $\frac{x}{1+c+bx}$ is strictly increasing in x for all x if
 7 $1 + c \geq 0$. ■

8 **Proof of Proposition 1.**

9 Define $r^*(S)$ as the value of r_2 such that inequality [\(24\)](#) binds for $i = 1$. Intuitively, $r^*(S)$
 10 is the value of rental in the low-prevalence city that is just high enough to dissuade sick
 11 individuals from moving there from the high-prevalence city. This yields

$$U(Y, S) - U(Y - r^*(S), S) = Z' - m, \quad (27)$$

12 where

$$Z' = \gamma\pi (W^2(H) - W^1(H)) + \gamma(1 - \pi) (W^2(S) - W^1(S)).$$

13 Subtracting Z' from Z yields

$$\begin{aligned} Z - Z' = W^2(H)[\gamma(1 - \alpha_2 p_2 - \pi)] - W^1(H)[\gamma(1 - \alpha_1 p_1 - \pi)] \\ + W^2(S)[\gamma(\alpha_2 p_2 + \pi - 1)] - W^1(S)[\gamma(\alpha_1 p_1 + \pi - 1)]. \end{aligned}$$

14 Simplifying yields

$$Z - Z' = \gamma(1 - \alpha_2 p_2 - \pi) [W^2(H) - W^2(S)] - \gamma(1 - \alpha_1 p_1 - \pi) [W^1(H) - W^1(S)].$$

15 This must be strictly greater than zero since $(1 - \alpha_2 p_2 - \pi) > (1 - \alpha_1 p_1 - \pi)$ and
 16 $W^2(H) - W^1(H) \geq W^2(S) - W^1(S)$ from [Lemma 4](#). Therefore, from [equations \(22\)](#) and
 17 [\(27\)](#) we can see that

$$U(Y, H) - U(Y - r^*(H), H) > U(Y, S) - U(Y - r^*(S), S)$$

18 which implies that $r^*(H) > r^*(S)$ given [equation \(8\)](#). Thus, sick individuals will be dis-
 19 suaded from migrating to the low-prevalence city at a lower rental rate than will dissuade
 20 healthy individuals. Thus, for any rental rate at which sick individuals want to migrate to
 21 the low-prevalence city, healthy individuals also want to migrate in this direction. Since
 22 by [Lemma 1](#), there cannot be individuals of either health status simultaneously wishing

1 to migrate in the opposite direction, this cannot be an equilibrium satisfying the zero
 2 net migration condition. Thus, in equilibrium sick individuals, if they migrate at all in
 3 equilibrium, must migrate only to the high-prevalence city. Analogous arguments show
 4 that sick individuals will choose to migrate from the high-prevalence city at a lower rental
 5 rate than healthy individuals.

6 Therefore, there is no rental rate at which only sick individuals migrate to the low-
 7 prevalence city and healthy individuals to the high-prevalence city. Thus, healthy individ-
 8 uals, if they migrate, will do so from the high-prevalence to the low-prevalence city while
 9 sick individuals, if they migrate, will do so in the opposite direction. ■

10 **Proof of Proposition 4.**

11 Using equation (13), we define $r_t^H(m)$ implicitly as follows:

$$m = U(Y - r_t^H(m), H) - U(Y, H) + \gamma \alpha_j p_{jt} W_{jt+1}(S) - \gamma \alpha_i p_{it} W_{it+1}(S) \\ + \gamma(1 - \alpha_j p_{jt}) W_{jt+1}(H) - \gamma(1 - \alpha_i p_{it}) W_{it+1}(H). \quad (28)$$

12 Substituting $i = 1$ and $j = 2$, taking steady state values and noting that $p_1 = 1 - \frac{\pi}{\alpha_1}$ and
 13 $p_2 = 0$ yields

$$m = U(Y - r^H(m), H) - U(Y, H) \\ + \gamma(\pi - \alpha_1) W_1(S) \\ + \gamma W_2(H) - \gamma(1 - \alpha_1 + \pi) W_1(H). \quad (29)$$

14 Using equation (14), we define $r_t^S(m)$ implicitly as follows:

$$m = U(Y, S) - U(Y - r_t^S(m), S) + \gamma \pi (W_{jt+1}(H) - W_{it+1}(H)) \\ + \gamma(1 - \pi) (W_{jt+1}(S) - W_{it+1}(S)). \quad (30)$$

15 Substituting $i = 2$ and $j = 1$, taking steady state values and noting that $p_1 = 1 - \frac{\pi}{\alpha_1}$ and
 16 $p_2 = 0$ yields

$$m = U(Y, S) - U(Y - r^S(m), S) + \gamma \pi (W_1(H) - W_2(H)) \\ + \gamma(1 - \pi) (W_1(S) - W_2(S)). \quad (31)$$

17 Equation (8) implies that $r^H(0) > r^S(0)$ if $U(Y, H) - U(Y - r^H(0), H) > U(Y, S) -$
 18 $U(Y - r^S(0), S)$.

19 Define $R \equiv (U(Y, H) - U(Y - r^H(0), H)) - (U(Y, S) - U(Y - r^S(0), S))$. Then,
 20 setting $m = 0$ and using equations (29) and (31) yields

$$R = \gamma W_2(H) - \gamma(1 - \alpha_1 + \pi) W_1(H) + \gamma(\pi - \alpha_1) W_1(S) \\ + \gamma \pi (W_1(H) - W_2(H)) + \gamma(1 - \pi) (W_1(S) - W_2(S)). \quad (32)$$

21 This can be rewritten as $R = A\gamma(1 - \pi) + B(\alpha_1 - \pi)$, where

$$A = (W_2(H) - W_1(H)) - (W_2(S) - W_1(S))$$

22 which, since $\alpha_1 p_1 > \alpha_2 p_2$, is strictly positive by Lemma 4, and

$$B = (W_1(H) - W_1(S))$$

23 which is strictly positive by Lemma 3. Therefore, $R > 0$, which implies that $r^H(0) > r^S(0)$.

1 Using the definition of m^* this implies that $m^* > 0$.
 2 Lemmas 3 and 4 also imply that R , and therefore $r^H(0) - r^S(0)$, is decreasing in α_2 and
 3 in π . ■

4 **APPENDIX B**

5 **Local Stability of the Equilibrium without Migration**

$$p_{it+1} = u(p_{it}) = \alpha_i p_{it}(1 - p_{it}) + p_{it}(1 - \pi).$$

6 The steady state equilibrium in each city is a fixed point such that $u'(p_i) = p_i$.
 7 We can derive the function $u(\cdot)$ as $u'(p_{it}) = 1 - \pi + \alpha_i - 2\alpha_i p_{it}$ and note that the steady
 8 state p_i is stable if $|u'(p_{it})| < 1$ around the steady state.
 9 This condition is equivalent to

$$2(\alpha_i p_{it} - 1) < -\pi + \alpha_i < 2\alpha_i p_{it}.$$

10 We can show easily that $2(\alpha_i p_{it} - 1) < -\pi + \alpha_i$ since $-\pi + \alpha_i > 0$ by assumption and
 11 $\alpha_i p_{it} < 1$.
 12 Since $p_i = 1 - \pi/\alpha_i$ for $i = 1, 2$ we show easily that, close to the steady state, $2\alpha_i p_{it} \cong$
 13 $2(\alpha_i - \pi)$, which is clearly larger than $\alpha_i - \pi$.
 14 Therefore, the steady state equilibrium is locally stable.

15 **Local Stability of the Equilibria with Migration**

16 We have the following dynamic system:

$$p_{1t+1} = f(p_{1t}, p_{2t}) = [(1 - p_{1t})(1 - h_{1t})\alpha_1 + (1 - \pi)] p_{1t} + s_{2t} p_{2t}(1 - \pi);$$

$$p_{2t+1} = g(p_{1t}, p_{2t}) = h_{1t}(1 - p_{1t})\alpha_2 p_{2t} + \alpha_2 p_{2t}(1 - p_{2t}) + (1 - s_{2t})p_{2t}(1 - \pi).$$

17 We note

$$J = \begin{pmatrix} (1 - \pi) - (1 - h_{1t})\alpha_1 p_{1t} + (1 - h_{1t})\alpha_1 & s_{2t}(1 - \pi) \\ -h_{1t}\alpha_2 p_{2t} & \alpha_2 - \alpha_2 p_{2t} + h_{1t}(1 - p_{1t})\alpha_2 + (1 - s_{2t})(1 - \pi) \end{pmatrix}.$$

18 After writing $p(\chi) = |J - \chi I| = \begin{vmatrix} f_{p_1} - \chi & f_{p_2} \\ g_{p_1} & g_{p_2} - \chi \end{vmatrix}$, we can study the Eigenvalues of J ,
 19 roots of the equation $p(\chi) \equiv \chi^2 - (\text{tr}J)\chi + \det J = 0$.

20 We know that in Equilibrium $s_2(=\phi) = h_1(=\psi) = 0$, $p_1 = 1 - \pi/\alpha_1$, and $p_2 = 0$ such
 21 that $J = \begin{pmatrix} 1 & 0 \\ 0 & \alpha_2 + 1 - \pi \end{pmatrix}$ and $p(\chi)$ can be rewritten as

$$p(\chi) = \chi^2 - (2 - \pi + \alpha_2)\chi + (\alpha_2 + 1 - \pi).$$

22 The two Eigenvalues χ_1 and χ_2 are as follows:

$$\chi_1 = \alpha_2 - \pi + 1;$$

$$\chi_2 = 1.$$

23 Since $|\chi_1| > 1$ and $|\chi_2| = 1$ such equilibria are locally unstable.

1 NOTES

2 1 See for example Boily (2002) on migration patterns of HIV infected sex-workers or Lurie
3 et al. (2003) on migrant couples in South-Africa with higher rates of HIV infection than non-
4 migrant.

5 2 During the Black Death, inhabitants from infected villages frequently migrated to less infected
6 neighboring villages. More recently, after the SARS outbreak in China, numerous workers in urban
7 areas returned to live with their families in safer rural areas (Le Point, 2003).

8 3 Alexis de Tocqueville arrived in Detroit in 1831 and was very troubled by mosquitoes during his
9 travels (he speaks in his journal of “inexpressible torment caused by mosquitoes” ; Tocqueville 1981,
10 p.140). The initiative shown by Americans in organizing to drain wetlands impressed Tocqueville
11 and was one of the features he contrasted with the French dependence on central government. See
12 <http://www.mackinac.org/article.asp?ID=25>

13 4 Downloadable from <http://www3.who.int/whosis>

14 5 For example, losses associated to the SARS outbreak have been estimated between US\$10 and
15 US\$30 billion, as compared to the 1994 outbreak of plague in India, the costs of which were estimated
16 at around US\$2 billions (Robertson 2003).

17 6 It may be more realistic for certain epidemics to consider the possibility of individuals fleeing
18 high-prevalence cities to stay with friends or relatives in lower-prevalence cities, implying an aggregate
19 temporary population shift between cities without any adjustment on the land market. This is captured
20 by Mesnard and Seabright (2009) in an epidemic framework. Here, by contrast, we consider longer-
21 run location decisions where capacity constraints may play a significant role. In the model, total
22 capacity of each city is fixed and cannot be changed by (for instance) construction, but less stringent
23 constraints would preserve the qualitative features of our results. Total capacity constraints also make
24 it easier to define and solve for a steady state as population size in each city remains constant over
25 time.

26 7 For instance, decisions of individuals to become sex workers, or within the commercial sex sector
27 to move between street prostitution and the formal brothel-based sector, are likely to be influenced by
28 what is known about relative risks of sexually-transmitted diseases.

29 8 The two cities could also be interpreted as countries or regions, or even in some circumstances
30 as different sectors of the economy. For instance, decisions of individuals to become sex workers, or
31 within the commercial sex sector to move between street prostitution and the formal brothel-based
32 sector, are likely to be influenced by what is known about relative risks of sexually-transmitted disease.

33 9 The case where population sizes can change together with the types of migrants after the outbreak
34 of an epidemic disease is already studied in Mesnard and Seabright (2009).

35 10 Introducing heterogeneity in income level would only complexify the model without adding
36 much insight to the results. Rich individuals will have higher willingness to pay for high rents and live
37 in the low prevalence area than poor individuals and unless they are systematically more likely to be
38 sick, this will not affect the properties of the sorting equilibria according to health status we describe
39 below.

40 11 In contrast to Mesnard and Seabright (2009), our model does not need to assume private
41 information of healthy individuals on their ex-ante risk of infection to generate unexpected effects of
42 policy measures.

43 12 We could extend the model by assuming that this probability depends on health care avail-
44 ability and quality, which may be higher where costs of living are higher. This would introduce two
45 counteracting effects: first, the differential in prevalence rates across cities in steady state equilibrium
46 would increase as the high prevalence city with low costs will have a lower rate of recovery than the
47 low-prevalence city with high costs of living. Second, this would give an incentive for sick individuals
48 to stay in the high costs city where they are more likely to recover. As long as there is a range of
49 parameters' values such that the second effect does not fully off-set the incentives for sick individuals
50 to live in the low costs city, the results would remain qualitatively the same.

51 13 With the exception of Finkelstein et al. (2013), who use data on elderly people in the US
52 struck by chronic diseases, there is remarkably very little empirical work on how marginal utility of

1 consumption depends on health, and we see no compelling reason to think that the relationship for
 2 communicable diseases runs in one way rather than the other.

3 14 Proofs of all lemmas and propositions not shown in the text are in Appendix.

4 15 The healthy city can be the one with the more disease prone environment ($\alpha_2 > \alpha_1$ is compatible
 5 with $\alpha_2 p_2 < \alpha_1 p_1$).

6 16 The linearity of the functions $r^H(m)$ and $r^S(m)$ is for illustration only. The only requirement
 7 for the demonstration is that these functions are monotonic, as stated above.

8 17 In earlier variations of this paper, we experimented with assuming heterogeneous migration
 9 costs, which we conjectured led to equilibria with non-zero gross migration in steady state but made
 10 the model analytically intractable for little additional insight.

11 REFERENCES

12 Acemoglu, Daron, Johnson Simon and James Robinson (2001) Colonial origins of comparative devel-
 13 opment: An empirical investigation. *American Economic Review* 91, 1369–1401. Q6

14 Auld, M.C. (2003) Choice, beliefs, and infectious disease dynamics. *Journal of Health Economics*
 15 22(3), 361–377.

16 Bauch, C., A.d. Onofrio and P. Manfredi (2013) Behavioral epidemiology of infectious diseases: An
 17 overview. In A. d.Onofrio and P. Manfredi (eds.), *Modeling the Interplay between Human Behavior*
 18 *and Spread of Infectious Diseases*. New York: Springer.

19 Barreca, Alan, Price Fishback and Shawn Kantor (2011) The impact of migration on malaria deaths
 20 in the early 20 century United States. mimeo, University of Tulane.

21 Bell, Clive, Shantayanan Devarajan and Hans Gersbach (2006) The long-run economic costs of AIDS:
 22 A model with an application to South Africa. *World Bank Economic Review* 20, 55–89.

23 Bell, Clive and Hans Gersbach (2006) Growth and enduring epidemic diseases. *CESifo Working Paper*
 24 *Series*, CESifo GmbH.

25 Bénabou, Roland (1996a) Equity and efficiency in human capital investment: The local connection.
 26 *Review of Economic Studies* 63, 237–64.

27 Bénabou, Roland (1996b) Heterogeneity, stratification, and growth: Macroeconomic implications of
 28 community structure and school finance. *American Economic Review* 86, 584–609.

29 Boily, Marie-Claude (2002) The impact of migration patterns of female sex workers on a slow spreading
 30 HIV epidemic: Implications for prevention. Meeting on “Phase Specific Aspects of STD and HIV
 31 epidemiology and Prevention”. Q7

32 Chen, Frederik and Flavio Toxvaerd (2014) The economics of vaccination. *Journal of Theoretical*
 33 *Biology* 363, 105–117.

34 Crosby, Alfred (1986) *Ecological Imperialism: The Biological Expansion of Europe 900–1900*, New
 35 York: Cambridge University Press.

36 Duncan, G.J., M.C. Daly, P. McDonough and D.R. Williams (2002) Optimal indicators of socioeco-
 37 nomic status for health research. *American Journal of Public Health* 92(7), 1151–1157.

38 Dupas, Pascaline, Esther Duflo and Michael Kremer (2014) Education, HIV, and early fertility: Ex-
 39 perimental evidence from Kenya. forthcoming *American Economic Review*. Q8

40 Ellickson, Bryan (1971) Jurisdictional fragmentation and residential choice. *American Economic*
 41 *Review* 61, 334–39.

42 Epple, Dennis (2003) Modeling population stratification across locations : An overview. *International*
 43 *Science Review* 26(2), 189–196.

44 Fenichel, E.P. (2013) Economic considerations for social distancing and behavioral based policies
 45 during an epidemic. *Journal of Health Economics* 32(2), 440–451.

46 Fenichel, E.P. and X. Wang (2013). The mechanism and phenomenon of adaptive human behavior
 47 during an epidemic and the role of information. In A. d.Onofrio and P. Manfredi (eds.), *Modeling*
 48 *the Interplay between Human Behavior and Spread of Infectious Diseases*. New York: Springer.

49 Fernandez, Raquel and Richard Rogerson (1996) Income distribution, communities, and the quality of
 50 public education. *Quarterly Journal of Economics* 111, 135–64.

- 1 Finkelstein, Amy, Enzo F. P. Luttmer and Matthew J. Notowidigdo (2013) What a good is wealth
2 without health? The effect of health on the marginal utility of consumption. *Journal of the European*
3 *Economic Association* 11, 221–258.
- 4 Geoffard, Pierre Yves and Thomas Philipson (1996) Rational epidemics and their public control.
5 *International Economic Review* 37(3), 603–624.
- 6 Gersovitz, Mark and Jeffrey S. Hammer (2003) Infectious diseases, public policy and the marriage of
7 economics and epidemiology. *World Bank Research Observer* 18, 129–157.
- 8 Gersovitz, Mark and Jeffrey S. Hammer (2004) The economical control of infectious diseases. *Eco-*
9 *nomics Journal* 114, 1–27.
- 10 Hurd, Michel, Peter Adams, Daniel McFadden, Angela Merrill, and Tiago Ribeiro (2003) Healthy,
11 wealth and Wise? Tests for direct causal paths between health and socioeconomic status. *Journal of*
12 *Econometrics* 112(1), 3–56.
- 13 Kremer, Michael (2000a) Creating markets for new vaccines. Part I: rationale. In *Innovation policy*
14 *and the Economy*, vol. 1, pp. 35–72. National Bureau of Economic Research.
- 15 Kremer, Michael (2000b) Creating markets for new vaccines, Part II: design issues. In *Innovation*
16 *Policy and the Economy*, vol. 1, pp. 73–118. National Bureau of Economic Research.
- 17 Lurie, M., B. Williams, K. Zuma, D. Mkaya-Mwamburi, G.P. Garnett, M.D. Sweat, J. Gittelsohn, and
18 S.S. Abdool Karim (2003) Who infects whom? HIV concordance and discordance among migrant
19 and non-migrant couples in South Africa. *AIDS* 17, 2245–2252.
- 20 McNeill, William H. (1977) *Plagues and people* Doubleday, New York.
- 21 Marmot, Michael G. (2002) The influence of income on health: Views of an epidemiologist. *Health*
22 *Affairs* 21(2), 31–46.
- 23 Medlock, J., P.M. Luz, C.J. Struchiner, and A.P. Galvani (2009) The impact of transgenic mosquitoes
24 on dengue virulence to humans and mosquitoes. *The American Naturalist* 174(4), 565–577.
- 25 Mesnard, Alice and Paul Seabright (2009) Escaping epidemics through migration? Quarantine mea-
26 sures under asymmetric information about infection risk. *Journal of Public Economics* 93, 931–938.
- 27 Montalvo, Jose G. and Marta Reynal-Querol (2007) Fighting against malaria: prevent wars while
28 waiting for the miraculous vaccines. *The Review of Economics and Statistics* 89(1), 165–177.
- 29 Pattanayak, Subhrendu, Christine Poulos and Jui-Chen Yang (2007) Modeling self-protection against
30 infectious disease: Towards the theory of the economics of environmental epidemiology, RTI Inter-
31 national, mimeo.
- 32 Philipson, Thomas (2000) *Handbook of Health Economics*, Culyer and Newhouse, eds.: North-Holland.
- 33 d’Onofrio, A., P. Manfredi and E. Salinelli (2013) Vaccinating behavior and the dynamics of vaccine
34 preventable infections. In A. d’Onofrio and P. Manfredi (eds.), *Modeling the Interplay between*
35 *Human Behavior and Spread of Infectious Diseases*. New York: Springer.
- 36 Reluga, T.C. (2010) Game theory of social distancing. *PLoS Computational Biology* 6(5), e1000793.
- 37 Sethi, S.P. (1978) Optimal quarantine programmes for controlling an epidemic spread. *Journal of the*
38 *Operational Research Society* 29, 265–268.
- 39 Tiebout, Charles M. (1956) A pure theory of local expenditures. *Journal of Political Economy* 64(5),
40 416–424.
- 41 Timmins, Christopher (2005). Estimable equilibrium models of locational sorting and their role in
42 development economics. *Journal of Economic Geography* 5, 83–100.
- 43 Tocqueville, Alexis de (1981) *Journey to America*, translated by George Lawrence and edited by
44 J.P. Mayer, revised in collaboration with A.P. Kerr, Greenwood Press Publishers, Westport, CT.
- 45 Wong, Grace (2008) Has SARS infected the property market? Evidence from Hong Kong. *Journal of*
46 *Urban Economics* 63(1), 74–95.