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QUARANTINE MEASURES
UNDER ASYMMETRIC INFORMATION
ABOUT INFECTION RISK.**

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ABSTRACT

Escaping Epidemics Through Migration? Quarantine Measures Under Asymmetric Information About Infection Risk.*

This paper explores implications of the fact that individuals know more than the authorities about their risk of infection and can take migration decisions before their health status is publicly observable. In a 2-period model we study under which conditions the presence of quarantine measures may lead to inefficient outcomes as individuals' interest in migration to escape centres of disease may become stronger and generate negative externalities imposed on other uninfected individuals.

JEL Classification: I18, O15, O19 and R23

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1 Introduction

This paper studies the effect of public policies to restrict migration by individuals suspected of carrying disease, when those individuals have more information than the authorities about their probability of being carriers. It has long been known that migration affects the spread of disease, and this influence has for centuries been used to justify placing restrictions on the movement of individuals suspected of carrying infections¹. For example, immigrants to the United States were screened for disease on Ellis Island and sometimes kept in quarantine until considered safe for the rest of the population (for a detailed description of how migrants were screened when leaving their origin country and at their arrival on Ellis Island, see Markel and Stern, 2002). Epidemiological studies have addressed how individual behaviour, among other factors, affects the spread of infections. However, the study of how individual behaviour in turn changes in response to the new incentives created by the occurrence of a disease is much less developed. The principal contribution of our paper is to bring the study of strategic behavior under uncertainty into the domain of epidemiology, and to analyze its impact, in interaction with public policies, on the overall impact of epidemic disease.

To our knowledge the work that has been done to date on strategic behavior in the context of disease considers the economic determinants of preventive behaviour such as vaccination or the adoption of safe sex. In particular, Philipson (2000) and Geoffard and Philipson (1996) show that, if demand for prevention treatments such as vaccines is prevalence elastic, then initially successful public health efforts typically run into diminishing returns, not simply for technical reasons but because the decline of a disease discourages prevention. Similar considerations apply to the factors determining the adoption of means of contraception such as barrier methods for Sexually Transmissible Diseases or the choice of partners (see for example, Kremer, 1996), and a growing literature now focuses on the microeconomic determinants of such individual decisions, in order to reach a better understanding of epidemiological patterns.

However, migration as a form of preventive behaviour has received very little attention, although evidence has accumulated that migration behaviour and epidemics are intrinsically linked. Migration behaviour can respond very rapidly to changes in the health environment, in particular when it suddenly deteriorates through epidemics. There are numerous historical instances of people fleeing plague or other infectious diseases by migrating to distant areas (see McNeill, 1997). During the Black Death, for instance, inhabitants from infected villages migrated to less infected villages in the neighbourhood. Much more recently, after the SARS outbreak in China, workers in urban areas were returned in

¹The first international regulations on health were adopted by twelve European states during a conference held in Paris in 1851 following the failure of the early public health strategies using "quarantine and lazarettos" to prevent the importation of contagious diseases through the Mediterranean region. 137 regulations dealing with health issues for maritime traffic included articles that specifically mentioned plague, yellow fever and cholera, diseases for which international regulations and requirements continued until the late 20th century.

large numbers to live with their families in safer rural areas².

Understanding such behavioural responses is of importance for understanding the effectiveness of policy measures, in the context of an increasing demand for national and international regulation of disease control, especially given the very real fear that antibiotic resistance will soon lead to the recurrence of old diseases no longer curable by antibiotics and the emergence of new diseases that make regions such as Europe and North America more vulnerable to epidemics than they have been in the past.

Recently, the outbreak of SARS in China and its rapid transmission across the world (principally via air travel) demonstrated both the dramatic consequences of the lack of reporting by one state, and, subsequently, the effectiveness of strict health regulations applied quickly and simultaneously in different countries.

Even if it would be premature to draw conclusions regarding the long-term evolution of SARS, at least one question needs to be addressed urgently. How effective are quarantine measures and in what circumstances do they work best? Such measures have often been considered as the most efficient way to prevent individuals in an infected area from moving to a non infected area, and they continue to be used as a systematic response to epidemic outbreaks. For example, when the Black Plague arrived in Milan, three households were infected. The authorities immediately bricked up the doors and windows, leaving the human beings and the rats to their fate. "All perished and Milan was spared", as revealed by historical chronicles (for a review of these, see Benedictow, 2004).

But what happens if individuals have more accurate information on their risk probability than the state health authorities, and use this information asymmetrically strategically? Can economists warn policy makers about unexpected effects of quarantine regulations, taking into account that individual behaviour changes in response to policy regulations? The aim of the paper is to study the possibility of such effects, which, to our knowledge, have not yet been considered in the debates on health and migration³.

We develop a framework where migration responds to the prevalence of disease, to health regulations, and to the costs of migration. An important assumption is that individuals may know more about their risk characteristics than the health authorities. This assumption does not contradict the fact that health authorities have, or may have, considerable experience, as well as statistical information about earlier outbreaks of a disease, including its rate of propagation and incubation periods. We simply assume here that individuals know more about their idiosyncratic risk of being infected since they know more about their own characteristics (their past locations, personal habits, living conditions, networks etc...), whereas health authorities can only observe individuals' health status with a lag, once symptoms appear. This creates an asymmetry of information that has potentially important consequences for the effect of quarantine measures. It also means that the risk characteristics of individuals who migrate

²see *Le Point*, 16 Mai 2003 pp50-51

³See for example the review "Health and Migration" published by the International Organization for Migration

may differ in important ways from those of the general population, a fact that may have a significant impact on the subsequent evolution of the disease.

At this stage we do not study the effects of quarantine measures in general, many of which affect the mobility of goods (for a study of the effects of a ban on import of goods, see Anderson and James, 1998). But we study the effects of specific quarantine measures that affect the mobility of individuals and are taken by the health authorities of the country harbouring the focus of the disease, such as the ones taken recently by the Chinese authorities or, later on, by the Canadian authorities after the outbreak of SARS⁴. We show merely that behavioural responses to actual interventions may lead to perverse or undesirable effects, and that these are more likely when the period during which individuals have more information about their infection risk than the authorities is long relative to the speed of propagation of the disease. Such effects are therefore more likely for diseases such as HIV/AIDS than for Ebola or SARS, with tuberculosis being an intermediate case.

In Section 2 we set out the assumptions of a two-period model in which individuals face a choice of whether to migrate between two areas that differ in the prevalence of disease. The difference in disease prevalence provides the sole motive for migration in absence of quarantine regulations. Individuals choose whether or not to migrate according to the costs and benefits of doing so. In Section 3 we discuss the welfare implications of migration decisions and public policy using simple examples to illustrate. In Section 4 we use the model to study in more general cases welfare impacts of different types of quarantine measures, before concluding in Section 5.

Our model is very simple, and unrealistic in a number of ways that enable us to find analytical solutions. However, it illustrates three very useful and intuitive principles which apply much more generally than in this particular context, and which we believe will be helpful to analysts and policymakers in thinking about the impact of public policies towards disease. When disease incidence differs from one place to another, decisions to migrate involve private benefits and costs to the individual concerned which include changes in their risk of catching the disease. They also impose externalities on others, externalities that are negative and increasing in the number of uninfected individuals with which the migrant comes into contact, in the infectiousness of the disease, and in the probability that the migrant is a carrier. This much is uncontroversial. In our model we show that

- First, when the disease is concentrated in one place (the epicentre of an epidemic for instance), a decision to migrate away from the epicentre brings a potentially infected individual in contact with more uninfected individuals than she would have met had she remained where she was. Thus the typical migrant imposes a net negative externality as a result of her decision

⁴We acknowledge that, in the past, most measures taken at the national level to stop the spread of a disease have been taken by the authorities of the countries which people are trying to reach. But given the increased need of controlling diseases at the international level, pressures on the authorities of the country harbouring the infectious diseases have increased.

to migrate, and the marginal migrant (for whom, by definition, private benefits of migrating just equal the private costs of doing so) has a negative impact on social welfare. Laissez-faire will therefore lead to excessive migration. This provides a rationale for the frequent (and frequently justified) public policy response to epidemics, which is to attempt to restrict migration away from the epicentre by those who may be infected.

- Secondly, and less obviously, not all policies that aim to restrict migration in fact do so. In particular, we distinguish two effects of quarantine policies: they raise migration costs, which lowers migration, and they impose a welfare cost on those who suffer them, which tends to increase migration by those who are not currently subject to quarantine but fear they may become so if they remain where they are. This second effect will be stronger if individuals have more information than the authorities about their prior exposure to the disease. Policies implemented without taking this effect into account may therefore have effects that are opposite from those intended.
- Thirdly, even policies that actually reduce migration may have an adverse impact on social welfare if they reduce migration "too much", and specifically if they discourage those intra-marginal migrants whose private benefits from migration substantially exceed their private costs of migration, by enough to outweigh the negative externality they impose on others. Overall disease prevalence may even increase if in the name of avoiding negative externalities the authorities discourage relatively low-risk individuals from escaping the epicentre of the disease, thereby increasing the probability that they will catch the disease there from infected individuals.

The design of quarantine policies needs therefore to be sensitive to the actual incentives for or against migration created by those policies, as well as to the need to consider the impact on intra-marginal as well as on marginal migrants. These messages, we believe, have an importance and relevance that extends far beyond the rather special circumstances of our model.

2 The Model

There are two cities, 0 and 1, each containing a continuum of inhabitants. There are two time periods, $t = 0, 1$. The size of the population in period 0 is normalized to 1 in city 0 and N in city 1, with $N > 1$. Apart from their size and their number of infected individuals, the two cities are identical. At the outbreak of the epidemic at period 0, r_0 and r_1 inhabitants are infected by the disease in city 0 and city 1 respectively; we assume that $r_0 > r_1$, with minimal loss of generality since all it implies is that the initial population in which an epidemic is concentrated (the "epicentre") is small relative to the total potentially infected population. We also call r_0 and r_1 the initial "prevalence levels" of the disease in the two cities. However, individuals may choose to migrate between the cities

and this will affect prevalence levels in the second period. In equilibrium, and as a result of the various decisions affecting migration made by individuals and by the authorities, there will be N_0 and N_1 inhabitants infected by the disease in the two cities in period 1.

We write $s_{ijt} \in (h, s)$ for the state of health of individual i in city j in period t , where h is the healthy state and s is the sick state. An individual does not know her own state of health in period 0, learning this only in period 1. However, everyone (individuals plus the authorities) knows the prevalence rates of the disease r_0 and r_1 (we assume these are known through random sample testing methods).

Nevertheless, individuals know something important that the authorities do not know, namely the extent of their prior exposure to the disease, which we interpret simply as the probability that they have already caught the disease in period 0. We write e_{ij} for the exposure of individual i in city j (it will often be convenient to drop the subscript j without risk of confusion); in the analysis below we make a variety of different assumptions about how e_{ij} is distributed across individuals; let e_{\max} be the highest value of e_{i0} - that is, the highest exposure in city 0. In addition, exposure may or may not affect the risk of developing the disease in the future, as follows:

For what we call "type-insensitive" epidemics, the risk for a previously uninfected individual of catching the disease in period 1 depends only on the infectiousness ϕ of the disease, and on the number of infected individuals N_j in the city where she decides to live⁵. The natural interpretation of this is that the factors affecting previous exposure are purely random and have no bearing on the individual's future risk of catching the disease. Formally, we write this as follows:

$$pr(s_{ijt+1} = s | s_{ijt} = h) = \phi N_j \tag{1}$$

For a "type-sensitive" epidemic, however, the risk of future infection will depend on the same factors that determined past exposure. A good example would be a disease such as AIDS in which individual sexual behavior determines exposure, so that those whose behavior makes them more likely to have been infected in the past are also, if still uninfected, more likely to become infected in the future for given prevalence levels. For simplicity we suppose that the risk of future infection is simply proportional to past exposure (as well as to the disease- and city-specific factors just described):

$$pr(s_{ijt+1} = s | s_{ijt} = h) = e_{ij} \phi N_j \tag{2}$$

However, even without proportionality the qualitative insights of the model would remain unchanged so long as risk is increasing in past exposure.

⁵The equations would be more complex and non-linear if we assumed that the risk depended not on the number but on the proportion of infected individuals in the city in question. However, we have no reason to think that this would affect the qualitative insights of the model, and the absolute numbers assumption has a natural interpretation: in a city centre which every citizen passes, the risk of infection is a function of the total number of dangerous pathogens in the environment, not the proportion of dangerous ones to innocuous ones.

The fact that the risk of infection is increasing in the prevalence of the disease in the chosen city is important because it implies that individuals will choose whether or not to migrate between cities in response to differences in the prevalence levels⁶. However, not all individuals will make the same choices, not (in our simple framework) because of differences in their migration costs or in their ability to afford those costs, since we assume migration costs and incomes to be identical across individuals. Rather, their choices will differ because of their prior exposure to the disease. This happens in two ways. First, individuals are more likely to migrate, other things equal, if they have had lower prior exposure, since that increases the probability that they are healthy - only healthy individuals are at risk of catching the disease since infected individuals have caught it already. Secondly, individuals are more likely to migrate if their individual risk is higher, since then that risk is also more sensitive to the environment in which they live. Since under type-sensitive epidemics their individual risk is increasing in their past exposure, this means, other things equal, that migration is more likely given *higher* past exposure. The net impact of these two effects will be considered in more detail below.

3 Migration and welfare: general arguments

Individual migration decisions affect social welfare and not just individual welfare. This is because they generate externalities - in this framework because an infected individual creates a risk for the uninfected individuals that surround her (the migration decision of an uninfected individual creates no externalities). However, asymmetric information about this risk makes it impossible for the authorities to correct perfectly for these externalities, since they know neither the health status of individuals nor their prior exposure before they migrate. If individuals did not know these things either, there would be no public policy issue since there would be equal uncertainty faced by everyone. Conversely, if individuals knew everything - and specifically, if they knew their own health status - there would be no public policy issue either, since infected individuals, who are the only ones whose migration decision creates externalities, would have no incentive to migrate. It is the fact that individuals know some things the authorities do not know, but do not know everything, that creates the really interesting public policy problem in our framework.

To see this, consider the case of type-insensitive epidemics (the argument is similar but more elaborate for the case of type-sensitive epidemics). Denote by d the utility costs of being infected, m the migration costs, y the per capita income generated in each city, and n_s (n_h) the number of sick (healthy) individuals who

⁶We deliberately ignore here the possibility that cities may differ in the quality of preventive or curative medical care available. It is possible that cities with high disease prevalence may be more, not less attractive to uninfected individuals, for instance if they also offer vaccination whose effects outweigh those of greater disease prevalence, or if prior residence is a condition of access to treatment. This qualification should be borne in mind when interpreting our results, though historically we do not believe such phenomena have been common.

migrate from city 0 to 1. To ensure that at least one individual will be better off migrating we assume that

$$m < d\phi(r_0 - r_1) \quad (3)$$

The objective of public policy is to minimize I , the sum of the utility costs of infection plus the costs of migration. We can write $I = d(N_0 + N_1) + m(n_h + n_s)$ with $N_0 + N_1$ being the total number of infected individuals living in both cities.

As, after migration, $N_0 = (1 - r_0 - n_h)(r_0 - n_s)\phi - n_s + r_0$ and $N_1 = (N - r_1 + n_h)(r_1 + n_s)\phi + n_s + r_1$, it is easy to derive

$$\frac{dI}{dn_h} = d\phi(r_1 - r_0 + 2n_s) + m$$

and show that, for n_s small, $\frac{dI}{dn_h} < 0$ as condition (3) holds. Moreover, $\frac{dI}{dn_s} > 0$ as:

$$\frac{dI}{dn_s} = d\phi(N - 1 - r_1 + r_0 + 2n_h) + m$$

Therefore public policy should minimize the number of sick migrants, n_s , which optimally should be equal to 0, and maximize the number of healthy migrants, n_h .

These optimal migration flows come from the specification of the infection function (equation 1), which implies that the total number of infected individuals is minimized when healthy individuals go to the city with fewer sick individuals and when sick individuals stay in the city with a high number of infected individuals.

However, since the authorities do not know the health status of each individual, they can only apply policy measures (such as quarantine) that in principle affect both healthy and sick individuals. Furthermore, individuals will react to those measures on the basis of their own prior exposure, which affects their gains from migration. But there is asymmetric information about these prior exposure levels - individuals with different exposures may react differently to the same measure, although the authorities cannot tell the individuals apart. As in many contexts with asymmetric information this will give rise to inefficient outcomes.

To see how this might happen, consider a simple example (still in the context of type-insensitive epidemics). Assume that individuals migrate from one city to another on the basis of current prevalence levels in the two cities. This is an over-simplification because it ignores the way in which individuals anticipate future evolution of prevalence levels, and we shall relax this assumption in the model below, but it is useful for understanding the kinds of perverse effect that can result from well-intentioned public policy interventions.

The utility if an individual of type e_i chooses to migrate is written as:

$$U_m = -m + e_i(y - d) + \phi r_1(1 - e_i)(y - d) + (1 - e_i)(1 - \phi r_1)y$$

The utility if an individual of type e_i chooses not to migrate is:

$$U_n = e_i(y - d) + \phi r_0(1 - e_i)(y - d) + (1 - e_i)(1 - \phi r_0)y$$

So individual i will wish to migrate as long as:

$$m < \phi(1 - e_i)(r_0 - r_1)d \tag{4}$$

Equation (4) shows that individual i will wish to migrate to the low prevalence city as long as the migration cost, m , is smaller than the migration benefit (on the right hand side of the inequality). This benefit is higher the lower is prior exposure e_i and the higher the difference between prevalence rates between the two cities. From this it follows that all individuals having an exposure factor below $e^* = 1 - \frac{m}{\phi d(r_0 - r_1)}$ migrate to the low infected city.

Consider a policy intervention such as quarantine. What kinds of effect might this have? One thing it might do would be to raise the migration cost, thus lowering e^* and hence decreasing the number of migrants. We call this a "type 1" quarantine measure. Another result might be to impose a utility cost on individuals of remaining in city 0, since they face a chance of being subjected to awkward and possibly dangerous restrictions on their movements. We call this a "type 2" quarantine measure. Writing this utility cost as q we can define the post-quarantine threshold as $e^{**} = 1 - \frac{m - q}{\phi d(r_0 - r_1)}$, and we see that $e^{**} > e^*$ so that the number of migrants increases.

In fact quarantine measures could actually lower social welfare if inappropriately implemented. Suppose that prior exposure levels are distributed discretely so that there are two types of individual, the high-risk and the low-risk types. Formally, $e_i \in \{e_L, e_H\}$. Assume also that prequarantine, the marginal migrant is such that $e_L < e^* < e_H$.

Without quarantine measures, low risk individuals migrate to city 1 whereas high risk individuals stay in city 0. This is optimal provided low-risk individuals are sufficiently low-risk: the benefit to them of escaping exposure to the high-risk individuals in city 0 outweighs the negative externality they impose on the larger numbers of uninfected individuals in city 1 than there are in city 0.

If, after quarantine measures of type 2 are implemented, the marginal migrant is such that $e_L < e_H < e^{**}$, then high risk individuals are also encouraged to migrate, which may increase the total number of infected individuals and, hence, decreases total welfare. So, in this case, social welfare is higher when the authorities do not intervene than when they do.

We are not, of course, suggesting that all policy interventions in the presence of epidemic diseases will be self-defeating. On the contrary, it is likely that laissez-faire will be inefficient because of the externalities imposed by the sick on the healthy. However, our example has shown that devising policies that actually improve matters is not a straightforward task. The next section will illustrate this in a more general framework.

4 Migration and welfare under rational expectations

Unlike in our example, we assume that migrants have rational expectations about prevalence rates in the two cities in period 1. That is, when they make their migration decisions in period 0 they do not assume that current prevalence rates will continue to hold in the future; they take into account that others are reasoning as they do. We now use the model to look in more detail at welfare questions, beginning with type-insensitive diseases.

4.1 Type-insensitive infections

We begin by setting out individual migration incentives.

The utility if an individual of type e_i chooses not to migrate is written as:

$$U_m = -m + e_i(y - d) + \phi(1 - e_i)(r_1 + n_s)(y - d) + (1 - e_i)(1 - \phi(r_1 + n_s))y$$

The utility if an individual of type e_i chooses not to migrate is:

$$U_n = e_i(y - d) + \phi(1 - e_i)(r_0 - n_s)(y - d) + (1 - e_i)(1 - \phi(r_0 - n_s))y$$

So individual i will wish to migrate as long as:

$$m < \phi(1 - e_i)(r_0 - r_1 - 2n_s)d \quad (5)$$

Note that this differs from equation 4 by the inclusion of the term $2n_s$ - the migrant takes into account that others like herself will be migrating between periods 0 and 1.

To ensure that at least one individual will wish to migrate we assume that

$$m < \phi(1 - e_{\min})(r_0 - r_1 - 2n_s)d \quad (6)$$

We can study the impact of quarantine measures on migration incentives in general, without solving for the migration equilibrium level, n_s , by defining the marginal migrant by the exposure level setting migration incentives, defined by equation (5), equal to 0 as:

$$e^* = 1 - \frac{m}{\phi d(r_0 - r_1 - 2n_s)}$$

a) if quarantine measures of type 1 are implemented, so that migration cost is now $m' > m$, the marginal migrant is now defined by $e^{**} = 1 - \frac{m'}{\phi d(r_0 - r_1 - 2n_s)}$ and we see that $e^{**} < e^*$ so that migration has decreased.

b) if quarantine measures of type 2 are implemented, so that there is a utility cost q to remaining in city 0, the marginal migrant is now defined by $e^{**} = 1 - \frac{m - q}{\phi d(r_0 - r_1 - 2n_s)}$ and we see that $e^{**} > e^*$, so that migration has increased.

Of course, in the definition of these thresholds the number of migrants, n_s , is endogenous. We now take this endogeneity into account in order to establish the following propositions. Proposition 1 defines parameter values for which there exist no welfare-improving interventions:

Proposition 1: If exposure levels are discretely distributed between two types, so that the exposure factor $e_i \in \{e_L, e_H\}$ with p the proportion of individuals with low exposure level, and if $e_L < 1 - \frac{m}{\phi d(r_0 - r_1 - 2pe_L)} < e_H$, then:

a) the unique laissez-faire equilibrium has migration by all and only the low-exposure individuals, and there exists a threshold t such that if $e_L < t$ this outcome is second-best optimal⁷, where t is the lower root of the quadratic equation $(A + Be_L + Ce_L^2) = 0$, where

$$\begin{aligned} A &\equiv mp - dp\phi(r_0 - r_1); \\ B &\equiv d\phi(N - 1 + 2p + 2(r_0 - r_1)); \\ C &\equiv 2pd\phi; \end{aligned}$$

b) if a quarantine intervention of type 1 raises migration cost to m' such that $e_L > 1 - \frac{m'}{\phi d(r_0 - r_1)}$, the unique equilibrium has zero migration by both exposure types, which if $e_L < t$ is second-best sub-optimal since low-risk types are not separated from high-risk types;

c) if a quarantine intervention of type 2 imposes a disutility cost of q on sick individuals in city 0 such that $e_H < 1 - \frac{m-q}{\phi d(r_0 - r_1 - 2pe_L)}$, there exists no equilibrium in pure strategies, and the sole equilibrium has a proportion $\pi < 1$ of high-risk types migrating, with π defined by $1 - \frac{m-q}{\phi d(r_0 - r_1 - 2pe_L - 2\pi(1-p)e_H)} = e_H$. If $e_L < t$ this is also suboptimal since a proportion of high-risk types are not separated from low-risk types.

Proof: Appendix

Migration entails two types of change in welfare. First, migration imposes a negative externality as migrants will encounter a larger number of healthy individuals in the destination area than in their area of origin, thereby increasing the overall exposure of others to infection risk. Secondly, all migrants have a net positive "private" gain from migration as the distribution of exposure levels is discrete (so that migrants are all infra-marginal individuals, who gain a strictly positive private benefit from migrating). Proposition 1a simply says that, if low type individuals are low risk enough, the negative externality is more than compensated by the positive "private" gains, and migration of low type individuals increases overall social welfare. From this it follows naturally that, in this case, discouraging low type individuals by quarantine measures of type 1 worsens social welfare (Proposition 1b) and that, encouraging high type individuals to migrate by quarantine measures of type 2 worsens social welfare

⁷What we mean by "second-best" optimality is optimal "in the set of feasible policies where the government can control m and q ".

(Proposition 1c), as high risk individuals who migrate entail excessive negative externalities.

Proposition 2 establishes conditions under which welfare-improving interventions may exist:

Proposition 2: If exposure levels are discretely distributed between two types, so that the exposure factor $e_i \in \{e_L, e_H\}$ with p the proportion of individuals with low exposure level, then:

a) if $1 - \frac{m}{\phi d(r_0 - r_1 - 2pe_L)} < e_L < e_H$, no individuals migrate, which is sub-optimal if $e_L < t$, but a second-best optimal migration can be induced by a quarantine intervention of type 2 such that $m - \phi d(r_0 - r_1 - 2pe_L)(1 - e_H) > q > m - \phi d(r_0 - r_1 - 2pe_L)(1 - e_L)$;

b) if $e_L < e_H < 1 - \frac{m}{\phi d(r_0 - r_1 - 2pe_L)}$, there exists no equilibrium in pure strategies, and the sole equilibrium has a proportion $\pi < 1$ of high-risk types migrating, with π defined by $1 - \frac{m}{\phi d(r_0 - r_1 - 2pe_L - 2\pi(1-p)e_H)} = e_H$.

If $e_L < t$, second-best optimal migration can be induced by a quarantine intervention of type 1 such that $\phi d(r_0 - r_1 - 2pe_L)(1 - e_L) > m > \phi d(r_0 - r_1 - 2pe_L)(1 - e_H)$;

c) if $e_L \geq t$ the second-best optimum has zero migration, which can be induced by a quarantine intervention of type 1 such that $m > \phi d(r_0 - r_1 - 2pe_L)(1 - e_L)$.

Proof: Follows immediately from Proposition 1.

Note that even where welfare-improving policy interventions exist they must be carefully designed to encourage only low type individuals to migrate. The quarantine measure should not "overshoot", moving from a situation in which neither type of individual migrates to one in which both types migrate, or vice versa - unless even the low-risk types are sufficiently high-risk that it is better for society that they do not migrate at all.

Once again, the details of the policy matter, and policies devised with good attention but without attention to detail may end up ineffective or making things worse.

We now consider the more interesting case where the exposure factor e_i is uniformly distributed over $[0, \bar{e}]$.

Without quarantine measures, the total number of migrants is $n_s + n_h = \frac{e^*}{\bar{e}}$ and $n_h = \frac{e^*}{\bar{e}}(1 - \frac{e^*}{2})$ and $n_s = \frac{e^{*2}}{2\bar{e}}$ where $\frac{e^*}{2}$ is the average exposure in the pool of migrants.

From (5) we can derive the threshold exposure factor defining the marginal migrant:

$$e^* = 1 - \frac{m}{\phi d(r_0 - r_1 - \frac{e^{*2}}{\bar{e}})}$$

e^* is the only real solution such that $\phi \neq 0$ and $d \neq 0$ of the following cubic equation :

$$e^* \phi d \left(r_0 - r_1 - \frac{e^{*2}}{\bar{e}} \right) = \left[\phi d \left(r_0 - r_1 - \frac{e^{*2}}{\bar{e}} \right) - m \right] \quad (7)$$

Public policy will aim to minimize $I = d(N_0 + N_1) + m \frac{e^*}{\bar{e}}$.

Proposition 3: When exposure levels are uniformly distributed over $[0, \bar{e}]$:

- a) All individuals below the threshold exposure factor defined implicitly by $e^* = 1 - \frac{m}{\phi d (r_0 - r_1 - \frac{e^{*2}}{\bar{e}})}$ will migrate to the low prevalence city.
- b) there exists a unique exposure threshold e_{\min} , which minimizes I .
- c) $e_{\min} < e^*$, so that migration under laissez-faire is too high as compared to this optimum.

Proof: Appendix

The intuition of the results for a continuous distribution of exposure levels follows the intuition for the discrete case with one important difference: the marginal migrant has, by definition, no private gains from migration. Therefore the marginal migrant entails, in this case, net overall social welfare losses, as migration still entails negative externalities and private gains are zero.

It follows immediately that type 1 quarantine measures may be able to implement the optimum while type 2 measures cannot, though once again the type 1 measures must not be too stringent or they will "overshoot".

4.2 Type sensitive infections

We now turn to type-sensitive infections. These make the migration incentives more complicated, so we first solve for the new migration thresholds.

Using equation 1 instead of equation 2, the gains from migration to individual i become

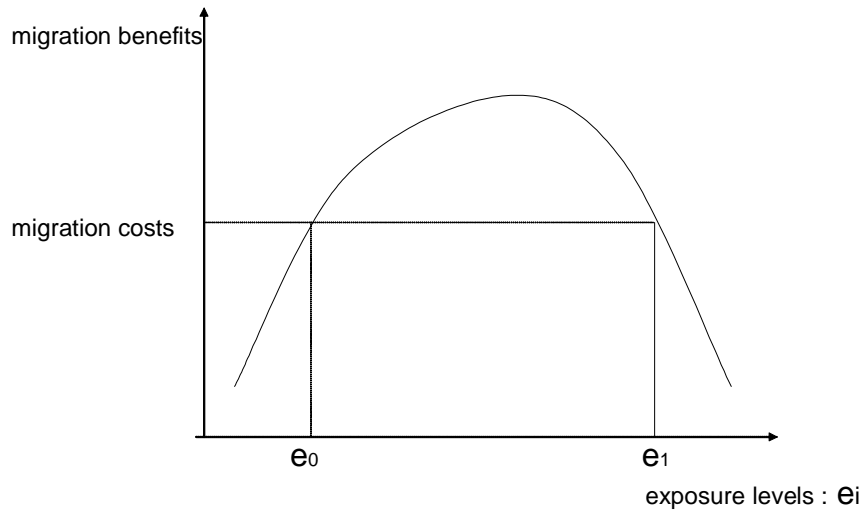
$$-m + e_i(1 - e_i)(r_0 - r_1 - 2n_s)d \quad (8)$$

where n_s is endogeneous, and will depend, once again on the distribution of exposure levels.

From this we can state:

Proposition 4: When epidemics are type-sensitive, migrants have rational expectations and exposure levels are discretely distributed, so that $e_i \in \{e_L, e_H\}$ with a proportion p of individuals of low type in city 0:

- a) for any given migration cost m and migration level n_s , there exist two thresholds e_0 and e_1 , which are solutions to the quadratic equation $e_i - e_i^2 = m/(r_0 - r_1 - 2n_s)$, such that any individual of risk type $e_i \in [e_0, e_1]$ chooses to migrate;
- b) therefore either type, no type or both types may migrate in equilibrium, depending on where the two risk types are situated with respect to these thresholds.



An illustration of the migration thresholds is given in the figure above.

The intuition of the welfare analysis for type insensitive epidemics when exposure levels were discretely distributed continues to hold in this case : there are some cases where policy measures may be welfare improving and other cases where policy measures may be welfare decreasing. Once again this will depend on where the low and high type individuals are positioned compared to the two migration thresholds; on how low low type individuals are; and on how policy measures are designed to affect migration costs or/and the disutility of living in the epicentre of the disease.

When types are continuously distributed, any change in migration cost will change the number of migrants in the neighborhood of both the low and the high thresholds. At laissez faire equilibrium, marginal migrants at both ends of the distribution will impose negative externalities through encountering a larger number of healthy individuals in the destination area than in their area of origin, thereby increasing the overall exposure of others to infection risk. At the same time, private gains of migration are zero at both migration thresholds. So, overall, there is always "too much" migration under laissez-faire when the distribution of exposure levels is continuous and the same policy implications follow as for a continuous distribution in the case of type insensitive epidemics.

The point remains that policy needs to be designed with a careful attention to detail - not only can quarantine measures encourage instead of discourage migration, but their marginal impact may affect individuals with quite different categories of risk exposure, with consequently quite different impacts on the welfare of others.

5 Discussions and conclusion

Our model has highlighted the fact that optimal policies to influence migration in the presence of epidemics are highly sensitive both to the type of the disease and to the distribution of prior exposure levels.

We began by looking at "type-insensitive infections", for which the future risk of being infected does not depend on factors that determined an individual's past exposure to infection, but only on disease- and city-specific factors. These have the characteristic that migrants are those who are less likely to be infected than non-migrants - that is, there is a threshold of prior exposure below which individuals migrate and above which they do not.

We show that optimal policy will depend crucially on the distribution of prior exposure levels. If it is discrete - with high-exposure and low-exposure types - and if the exposure of the low types is sufficiently low, the optimal policy involves migration by the low-exposure and non-migration by the high-exposure types. Thus optimal policy will depend on where the laissez-faire migration threshold lies with respect to the exposure levels of the two types. If the exposure of the low types is sufficiently low and if the migration threshold lies between the two types, nothing should be done. If it lies above the high-exposure level, then migration should be made more difficult, by raising its cost, while if it lies below the low-exposure level then migration should be made easier, by lowering its cost.

If prior exposure levels are continuously distributed, on the other hand, we show that migration is always too high relative to the optimum as the negative externalities it imposes are greater than its zero net private benefits. Policy should therefore aim to discourage migration, by raising its cost. It should avoid perversely encouraging migration (for example by raising the cost to individuals of becoming infected in the origin city). We show also that quarantine measures should not be too stringent, or they will overshoot.

We then turned to "type-sensitive" infections, for which the risk of infection depends also on factors that determined individual past exposure to infection (such as individual sexual behaviour in the case of STDs), and for which migration incentives become more complicated. As in the type-insensitive case, high-exposure individuals do not migrate since they are likely already to have the disease. However, low-exposure individuals do not migrate either, since their low exposure is linked to behavior that makes them less likely to acquire the disease in the future. We show that those who migrate are of intermediate exposure levels - those who lie between a lower and an upper threshold.

We also show that the intuition of the welfare analysis and the resulting

policy implications continue to hold in the case of type sensitive epidemics. In the case where exposure is discretely distributed, optimal policy will depend on where the high- and low-type exposure levels lie with respect to the two migration thresholds and on whether the exposure of the low types is sufficiently low; either encouragement or discouragement of migration could be desirable depending on the circumstances. When exposure is continuously and uniformly distributed, discouraging migration discourages additional migrants at both the high-and low-risk parts of the distribution, but, at both parts of the distribution, the marginal migrant entails "too much migration" under *laissez-faire*.

In a nutshell the results of the welfare analysis show that, whether future risk is unrelated or related to past exposure, the migration threshold will be too high if exposure levels are continuously distributed, leading to excessive migration under *laissez-faire*. Otherwise migration may be either too high or too low depending on circumstances.

Simple as it is, this two-period model indicates that asymmetries of information between individuals and the authorities about their health risks may encourage perverse responses to policy measures such as quarantine. It has shown that certain kinds of quarantine measure can have some intriguingly perverse consequences. Although quarantine of individuals who have been identified as sick reduces (obviously) the propensity *of these individuals* to migrate and spread the disease, the threat of quarantine increases the propensity to migrate of other individuals who have not yet been fallen sick but who know themselves to be at risk.⁸ This is surely one of the reasons why the health authorities in Toronto encouraged self-applied quarantine measures after the SARS outbreak. The idea was to encourage individuals voluntarily to adopt quarantine measures and go to the hospitals to get preventive treatment in case they were at risk of having been infected.

How important these perverse effects are in practice will clearly depend on the degree of information asymmetry between individuals and the authorities, which will depend on the nature of the disease. This will also depend on how long is the incubation period of the disease during which such asymmetries can be expected to last. That diseases differ greatly in the extent of such asymmetries explains, among other reasons, why quarantine may be comparatively effective at halting the spread of a disease such as SARS, while it would be ineffective or even counter-productive at halting the spread of HIV/AIDS.

Similar considerations apply to many of the other "new" pathogens that have emerged in the last 25 years, including not only the Ebola virus, HIV and Hepatitis C but also lesser known pathogens such as *Legionella pneumophila*, *E coli* 0157:H7, *Borrelia burgdorferi*, *Helicobacter pylori*, Hantavirus, Cryptosporidia, Ehrlichiosis, H5N1 (or Avian flu), and Nipah. Most of these pathogens have incubation periods greater than standard travel times. For example, after the

⁸For an account of the reactions of individuals to this type of quarantine restrictions during the SARS epidemic in China, including ways in which restrictions were evaded, see "In Liaoning" by Jon Cannon, *London Review of Books*, 25(11), 5 June 2003.

SARS virus enters the body, it requires 3-10 days incubation period before the disease appears, which is much longer than the duration of any trip by air. Hence an adequate response to the outbreak is indeed to encourage individuals to go to health centers when they suspect they may have been infected with SARS, instead of applying traditional non-voluntary quarantine measures to restrict migration away from the epicentre by those who may be infected⁹.

We may also want to consider the case of re-emerging infectious diseases such as malaria, tuberculosis, dengue, yellow fever or cholera. However, for these diseases, it may be important to consider disease prevalence as a more long-term characteristic of a given area and, hence, a determinant of both the long-term location of individuals and the development process, as we study in a companion paper on migration and endemic disease, Mesnard and Seabright (2007).

We cannot claim to have done more than indicate in a simple and stylized context the complexity of the considerations that public health policies must take into account, but we hope to have shown that when individuals have private information about their exposure to disease, their strategic behaviour may have important effects on public health outcomes that policymakers cannot afford to ignore. Furthermore, some much simpler and more general messages can be derived than simply the conclusion that everything is complicated and the authorities need to take the specific circumstances of the disease into account. As we stated in the introduction, three principles in particular apply much more widely than in the specific circumstances of our model. First, when the disease has an epicentre, the marginal migrant imposes a net negative externality. Secondly, quarantine policies may encourage migration instead of discouraging it, specifically on the part of individuals who are not currently sick but fear they may become so in the future. Thirdly, even when they succeed in discouraging migration, quarantine policies may lower social welfare, and even increase overall disease incidence, if they go too far, thereby discouraging those intra-marginal migrants for whom private benefits substantially exceed private costs, by enough to outweigh the negative externality they impose on others. These principles are ones that could help in designing better quarantine policies even in circumstances to which the assumptions of our model do not literally apply.

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⁹Moreover, according to current data, infected people do not pass on the SARS virus to others during the incubation period, but become infectious only when the first symptoms appear-like cough or sneezing which spread droplets containing virus particles.

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7 Appendix

Proof of Proposition 1:

a) From the conditions on e_L and e_H it follows immediately that all and only low-exposure individuals migrate. Thus $n_s = pe_L$ and $n_h = p(1 - e_L)$ and we can define I^* , the "intermediate migration" social disutility level, by

$$I^* = mp + d[(1 - r_0 - p(1 - e_L))(r_0 - pe_L)\phi + r_0 - pe_L + (N - r_1 + p(1 - e_L))(r_1 + pe_L)\phi + r_1 + pe_L]$$

We first show that this is lower than the no-migration level, defined by :

$$I^0 = d[(1 - r_0)(r_0)\phi + r_0 + (N - r_1)(r_1)\phi + r_1]$$

Subtracting yields:

$$I^* - I^0 = p(A + Be_L + Ce_L^2), \text{ where}$$

$$A \equiv m - d\phi(r_0 - r_1);$$

$$B \equiv d\phi(N - 1 + 2p + 2(r_0 - r_1));$$

$$C \equiv -2pd\phi.$$

Noting that equation 5 implies that $A < 0$, that $B > 0$ because $N > 1$ and $r_0 > r_1$, and that $C < 0$, it follows that $A + Be_L + Ce_L^2$ is a quadratic function with a negative intercept, which admits two positive real roots. Substituting $e_L = 1$ and noting that $A + B + C > 0$, we can show that, for $e_L = 1$, $I^* - I^0$ is positive. So only one of the two roots lies between 0 and 1 . We denote this root t .

Therefore $I^* - I^0$ is negative if $e_L < t$ where t is the lower root of the quadratic equation $A + Be_L + Ce_L^2 = 0$.

To see that I^* is also lower than the all-migration level I^a in which all individuals leave city 0 for city 1, note that as $n_h + n_s = 1$ and, after migration, $N_1 = (N - r_1 + (1 - r_0))(r_1 + r_0)\phi + r_0 + r_1$, we can write

$$I^a = dN_1 + m = d[DE\phi + E] + m$$

where $D \equiv (N - r_1 + (1 - r_0))$ and $E \equiv r_0 + r_1$

We can write

$$I^* = d[FG\phi + HI\phi + E] + mp$$

where $F \equiv (1 - r_0 - p(1 - e_L))$,

$G \equiv (r_0 - pe_L)$,

$H \equiv (N - r_1 + p(1 - e_L))$,

$I \equiv (r_1 + pe_L)$.

Therefore we obtain easily: $I^a - I^* = m(1 - p) + d\phi(DE - FG - HI)$.

Noting that $D = H + F$ and $E = G + I$, we can write:

$I^a - I^* = m(1 - p) + d\phi(HG + FI)$, which is necessarily strictly positive.

b) follows immediately from the definition of the migration threshold;

c) follows from noting that if all individuals in city 0 migrated to city 1 there would remain no infected individuals in city 0, so migration would no longer be an equilibrium.

Proof of Proposition 3:

Let's denote $J = N_0 + N_1$

$$\frac{dI}{de^*} = d \left[\frac{dJ}{dn_s} * \frac{dn_s}{de^*} + \frac{dJ}{dn_h} * \frac{dn_h}{de^*} \right] + \frac{m}{\bar{e}}$$

$$\frac{dI}{de^*} = d \left[\phi(N - 1 - r_1 + r_0 + 2n_h) \frac{e^*}{\bar{e}} + \phi(r_1 - r_0 + 2n_s) \frac{(1 - e^*)}{\bar{e}} \right] + \frac{m}{\bar{e}} \quad (9)$$

Substituting $n_h = \frac{e^*}{\bar{e}}(1 - \frac{e^*}{2})$ and $n_s = \frac{e^{*2}}{2\bar{e}}$ into (9), we find:

$$\frac{dI}{de^*} = d \frac{\phi}{\bar{e}} \left[e^*(N - 1 - 2r_1 + 2r_0) + r_1 - r_0 + 3 \frac{e^{*2}}{\bar{e}} - 2 \frac{e^{*3}}{\bar{e}} \right] + \frac{m}{\bar{e}}$$

Let's define

$$A(e^*) = d \frac{\phi}{\bar{e}} \left[e^*(N - 1 - 2r_1 + 2r_0) + r_1 - r_0 + 3 \frac{e^{*2}}{\bar{e}} - 2 \frac{e^{*3}}{\bar{e}} \right] + \frac{m}{\bar{e}}$$

$$\frac{dA}{de^*} = d \frac{\phi}{e} \left[(N - 1 - 2r_1 + 2r_0) + 6 \frac{e^*}{e} (1 - e^*) \right]$$

$$A(0) = d \frac{\phi}{e} [r_1 - r_0] + \frac{m}{e} \text{ and } A(1) = d \frac{\phi}{e} \left[(N - 1 - r_1 + r_0) + \frac{1}{e} \right] + \frac{m}{e}$$

Given condition (3) holds, we can show easily that $A(0) < 0$. Moreover $A(1) > 0$ and $\frac{dA}{de^*} > 0$.

Therefore there is a unique threshold of exposure factor, $e_{\min} \in [0, 1]$ which minimises I . This threshold is implicitly determined by $\frac{dI}{de^*} = 0$.

To show that the laissez faire migration threshold, e^* , defined by equation (7) is larger than the optimal threshold, e_{\min} , it is sufficient to show that $\frac{dI}{de^*} > 0$.

From equation (9) above, and using the definition of e^* as being the threshold such that migration gains are zero, we can show that

$$\frac{dI}{de^*} = d \left[\phi (N - 1 - r_1 + r_0 + 2n_h) \frac{e^*}{e} \right]$$

Therefore $\frac{dI}{de^*} > 0$.