

# **The Effects of Immigration Policies on the Diffusion of Infectious Diseases: Demographic Balance and Disease Control**

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## **Abstract**

Epidemics that are either introduced into a population or fuelled by immigrating infective individuals are subject to dynamics that must include infectiveness external to the population itself. Immigration policies in developed countries are undertaken and modified according to socio-economic and demographic factors. Based on these facts and on the idea that a non-negligible portion of the inflowing individuals escapes official control, immigration dynamics are here introduced in an existing SIR model on the basis of the ability of a local population to control and select the admission of external individuals. Results show a peculiar behaviour of the epidemic dynamics and consequent public health measures for prevention or eradication of the infection are analyzed.

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## **1. Introduction**

The growing migration flows due to the socio-economic instability and enhanced by the globalization of mass transportation and by the constant (although not steady) growth of Western economies, have resulted in radical changes in the diffusion of infections, with direct effects on the contact mechanisms between the susceptible and the infective populations and a substantial modification of prevalences and incidences (see, among many others, [12], [10], [9], [8]). Some countries with a high immigration rate have appointed screening procedures on

perspective immigrants for infections with a long incubation time (see, as examples, [19] and [13]): such programs are, however, expensive and of limited efficacy and are put into effect only when there are clear emergencies (i.e., local epidemic outbreaks in countries of origin, avian pneumonia, SARS, BSE for cattle from high prevalence countries, etc.). Moreover, screening and health counselling recommendations, derived from local epidemiological studies, are often rather contradictory and far from being conclusive (compare, for instance, [14] for UK and [16] for Italy).

Until recently, theoretical models of infectious diseases essentially referred to populations which were either closed or had some “stable” relationship with the outer environment during an epidemic wave (known birth and death rates and known contact patterns) and their analysis and forecast results proved to be acceptable for most of the existing infections. However, starting around the mid 80’s with the insurgence of new “mysterious” infections, the theoretical knowledge and the validity of the available models proved increasingly inadequate and the scientific community had to start re-thinking principles, concepts and methodological structures. Infections such as HIV/AIDS, various forms of parenteral hepatitis, BSE and CJ disease have progressed, within a few years, to become global epidemics, most of them constituting a particularly serious threat to less developed countries, where entire generations of individuals have been put at risk of survival. Microbiological studies have isolated new agents and investigated their mechanisms of diffusion and interactions with the human host, but the diffusion of the epidemics in the population still presents unknown aspects to which mathematical models have not been able to provide global and satisfactory answers. Substantial improvements can be brought to the epidemic modelling schemes by including immigration flows, both susceptible and infectious, in the demographic dynamics; however, their patterns are difficult to keep within reasonable modelling boundaries, because of the several socio-economic aspects involved in their definition.

In the present paper, a balance between the modelling reduction of the actual complexity of the processes and the inclusion of factors external to the demographic and epidemiological dynamics is attempted. In particular, the model aims at describing and analyzing the situation, common to most Western countries, where the efforts to regulate the growing immigration from less developed countries is coupled with the impossibility of keeping under full control the flows of migrating individuals.

## **2. The Mathematical Model: Shaping the Immigration Flow**

Several models of infectious immigration are mentioned and commented in [15]: the demographic dynamics and the amount of resources available from time to time to the local population play a substantial role in determining the immigration flow. Therefore, in a more general setting, a model including an immigration flow should refer to time and other factors, such as local demographic and economic

dynamics, as influencing the quality and the quantity of the inflow, both directly and indirectly (a relevant analysis of these factors is in [6]). Under appropriate demographic and economic conditions, common to most developed countries, policies of expansive immigration are implemented and a quota  $w$  of “controlled”, or legal immigration is regularly admitted into the local population: this may be thought of as added to the “natural immigration”  $G(t)$  of the previous section. It seems reasonable to think that this legal immigration is entirely made out of healthy or screened individuals, whereas the natural immigration  $G(t)$ , otherwise called illegal, joins the local population as a mixture of  $(1 - p)G(t)$  healthy and  $pG(t)$  infectious individuals.

Studies on legal and illegal immigration, conducted by specialized organizations and agencies in the U.S. (the Immigration and Naturalization Service, the Census Bureau, the Center for Immigration Studies) and in other countries (see [7]; [17]; [18]; [1] and several reports of the Population Activities Unit of the United Nations), have explicitly reported the “intimate link between legal and illegal immigration” as “the legal immigration process embraces illegal immigration and encourages it” (see <http://www.cis.org>, but also “The Link: Legal and Illegal Immigration” by M. Krikorian in The New York Post, Feb. 16, 1997, among others). In this view, it seems then reasonable to divide the total immigration into a time-varying, natural immigration  $G(t)$  and a regular immigration as proportional to the natural one, thus providing the following categories of immigrants

- natural susceptible:  $(1 - p)G(t)$
- natural infectious :  $pG(t)$
- controlled healthy:  $wG(t)$

i.e.,

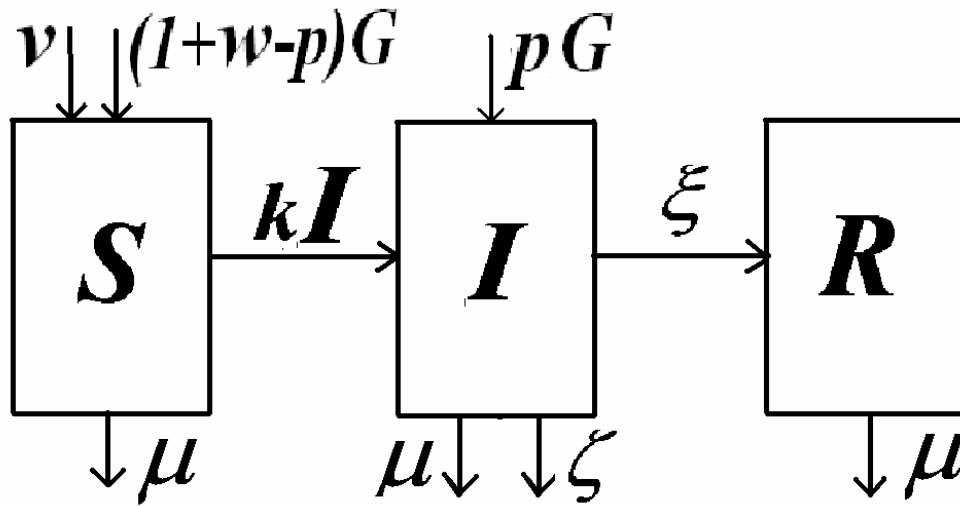
- total susceptible immigration:  $(1 - p)G(t) + wG(t) = (1 + w - p)G(t)$
- total infectious immigration:  $pG(t)$

so that we have a total number of immigrants given by  $Y(t) = (1 + w)G(t)$ .

Following these ideas, a modification of the SIR model already presented in [15] is here proposed:

$$\begin{cases} \frac{dS(t)}{dt} = -kS(t)I(t) - \mu S(t) + \nu N + (1 + w - p)G(t) \\ \frac{dI(t)}{dt} = kS(t)I(t) - (\mu + \xi + \varsigma)I(t) + pG(t) \end{cases} \quad (1)$$

whose flows are graphically represented in figure 1 (see further down for the model parameter definitions in Table 1).



**Figure 1:** schematic representation of the SIR epidemic model in (2) with inflows of susceptibles and infectives.

The proportion  $w$  of controlled immigration is here constant with time: this is a restrictive hypothesis based on the idea that  $w$  is subject to relatively small changes with respect to the other quantities in the model. This is true in particular with respect to the natural immigration  $G(t)$ ; in other words, we have that a supposedly time-dependent proportion  $W(t)$  of controlled immigration is such that  $W(t) = w + o(G(t))$  and, therefore, can be thought of either as being approximated with  $w$  or as being in a *quasi steady state*. As mentioned above, the quota  $w$  of controlled individuals admitted into a local population depends on various exogenous factors: in modelling terms this amounts to saying that  $w$  depends on a vector of time-dependent covariates  $X(t)$ , whose values can be obtained by applying appropriate estimation methods to Official Statistics; however, this issue is not addressed here, supposing that the *quasi-steady state* condition also applies to the variability of  $X(t)$ . Various further hypotheses can be made on the model quantities in order to analyze the dynamics of the epidemic:

- i) if  $\mu \approx \nu$ ,  $\zeta \approx 0$ ,  $w \approx 0$ , the “natural immigration”  $G(t)$  is “small” and approximately constant over time (say, a quasi steady state  $G(t) = g$  and estimated from external sources) and  $N \approx S(0) + I(0) + R(0)$ , then (1) can be reasonably approximated by

$$\begin{cases} \frac{dS(t)}{dt} = -kS(t)I(t) - \mu S(t) + \mu N + (1-p)g \\ \frac{dI(t)}{dt} = kS(t)I(t) - (\mu + \xi)I(t) + pg \end{cases}$$

if, following the hypotheses in the previous section, the “natural immigration” is given by

$$G(t) = \mu S(t) + (\mu + \varsigma)I(t) + \mu R(t) - \nu N(t)$$

with the total population  $N(t)$  corresponding to a variable saturation level. In this case the total population size  $N(t)$  varies according to

$$\frac{dN(t)}{dt} = w[(\mu - \nu)N(t) + \varsigma \cdot I(t)]$$

with  $N(t) = S(t) + I(t) + R(t)$ .

In case i) results from [3] can be usefully employed to study the equilibria of the system. In case ii) the whole epidemic system (1) is given by

$$\begin{cases} \frac{dS(t)}{dt} = -kS(t)I(t) - \mu S(t) + \nu(S(t) + I(t) + R(t)) + \\ \quad + (1 + w - p)[(\mu - \nu)(S(t) + I(t) + R(t)) + \varsigma \cdot I(t)] \\ \frac{dI(t)}{dt} = kS(t)I(t) - (\mu + \xi + \varsigma)I(t) + p[(\mu - \nu)(S(t) + I(t) + R(t)) + \varsigma \cdot I(t)] \\ \frac{dR(t)}{dt} = \xi \cdot I(t) - \mu R(t) \end{cases} \tag{2}$$

where the variable total population is given by  $N(t) = S(t) + I(t) + R(t)$ ; the model parameter definitions are in Table 1.

**Table 1:** Parameters of model (2)

Parameter	Definition
$k$	Transmission coefficient
$\nu$	Birth rate
$\mu$	Natural death rate
$\zeta$	Disease mortality rate
$\xi$	Infective removal rate
$p$	Proportion of infectious immigration
$w$	Quota (proportion) of controlled immigration

Setting to zero the derivatives in (2) and using  $\bar{R} = \frac{\xi}{\mu} \bar{I}$  from the last equation, the solution of the following system of ordinary equations

$$\begin{cases} -k\bar{S}\bar{I} - \mu\bar{S} + \nu\left(\bar{S} + \bar{I} + \frac{\xi}{\mu}\bar{I}\right) + (1+w-p)\left[(\mu-\nu)\left(\bar{S} + \bar{I} + \frac{\xi}{\mu}\bar{I}\right) + \zeta \cdot \bar{I}\right] = 0 \\ k\bar{S}\bar{I} - (\mu + \xi + (1-p)\zeta)\bar{I} + p(\mu-\nu)\left(\bar{S} + \bar{I} + \frac{\xi}{\mu}\bar{I}\right) = 0 \end{cases} \tag{3}$$

provides the critical points of (2).

In order to simplify the notation, let us divide the equations in (3) by  $\bar{N}(\mu + \xi + (1-p)\zeta)$ , where  $\bar{N} = \bar{S} + \bar{I} + \bar{R}$ ; we thus have the new equations in terms of new variables  $\bar{s} = \frac{\bar{S}}{\bar{N}}$ ,  $\bar{i} = \frac{\bar{I}}{\bar{N}}$  and  $\bar{r} = \frac{\bar{R}}{\bar{N}}$ :

$$\begin{cases} -K\bar{s}\bar{i} + \bar{i} + \frac{A}{\beta}(\beta\bar{s} + (1-\gamma)\cdot\bar{i}) + \delta\cdot\bar{i} = 0 \\ K\bar{s}\bar{i} - \bar{i} + \frac{\alpha}{\beta}(\beta\bar{s} - (1-\gamma)\cdot\bar{i}) = 0 \end{cases} \tag{4}$$

whose parameters are listed and defined in table 2.

**Table 2:** new parameter definitions for model (4)

New parameter	Original Parameters
$K$	$\frac{k\bar{N}}{\mu + \xi + (1-p)\zeta}$
$A$	$\frac{(w-p)(\mu-\nu)}{(\mu + \xi + (1-p)\zeta)}$
$\alpha$	$\frac{p(\mu-\nu)}{(\mu + \xi + (1-p)\zeta)}$
$\beta$	$\frac{\mu}{\mu + \xi + (1-p)\zeta}$
$\gamma$	$\frac{(1-p)\zeta}{\mu + \xi + (1-p)\zeta}$
$\delta$	$\frac{w\zeta}{\mu + \xi + (1-p)\zeta}$

By further setting  $\varphi = \frac{1-\gamma}{\beta} = \frac{\mu + \xi}{\mu}$ , equations (4) become:

$$\begin{cases} -K\bar{s}\bar{i} + \bar{i} + A(\bar{s} + \varphi \cdot \bar{i}) + \delta \cdot \bar{i} = 0 \\ K\bar{s}\bar{i} - \bar{i} + \alpha(\bar{s} - \varphi \cdot \bar{i}) = 0 \end{cases} \quad (5)$$

The solutions of (5) provide the equilibrium point of (2):

$$\begin{cases} \bar{i} = -\frac{A + \alpha(1 + \delta)}{K[(A + \alpha)\varphi + \delta]} \\ \bar{s} = \frac{A + \alpha(1 + \delta)}{K(A + \alpha)} = \frac{1}{K} \left( 1 + \frac{\alpha\delta}{A + \alpha} \right) \end{cases} \quad (6)$$

and the following admissibility lemma holds:

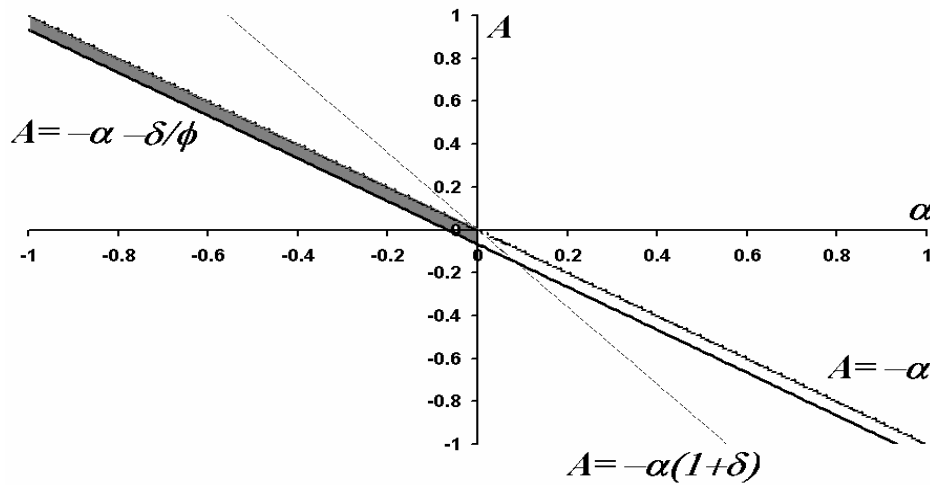
**Lemma 1** – *The steady state solutions  $(\bar{s}; \bar{i}) = [\bar{s}(A; \alpha); \bar{i}(A; \alpha)]$  of (7) are real, positive iff  $\alpha < 0$  and  $-\left(\alpha + \frac{\delta}{\varphi}\right) < A < -\alpha$ .*

**Proof**

From (6), the conditions  $\bar{i} > 0$  and  $\bar{s} > 0$  are equivalent to  $-\alpha(1 + \delta) < A < -\left(\alpha + \frac{\delta}{\varphi}\right)$  and  $-\alpha < A$  for  $\alpha > \frac{1}{\varphi}$ ;  $-\left(\alpha + \frac{\delta}{\varphi}\right) < A < -\alpha(1 + \delta)$  and  $-\alpha < A$  for  $0 < \alpha < \frac{1}{\varphi}$ ;  $-\left(\alpha + \frac{\delta}{\varphi}\right) < A < -\alpha(1 + \delta)$  and  $A < -\alpha$  for  $\alpha < 0$ .

The thesis follows by putting together all these inequalities. ■

A graphical summary of all the conditions above in the  $\alpha \times A$ -plane is sketched in figure 5 in the text.



**Figure 5:** graphic representation of the admissibility region (grey area) for the endemic equilibrium of the system (2) on the  $\alpha \times A$ -plane, where  $\varphi = 12$  and  $\delta = 0.5$ . The equilibria, although admissible, are unstable everywhere on the  $\alpha \times A$ -plane. (see lemma 1).

The quality and the stability of  $(\bar{s}; \bar{i})$  in (6) is determined by the eigenstructure of the jacobian matrix of (5):

$$J = \begin{bmatrix} -K\bar{i} + A & -K\bar{s} + 1 + A\varphi + \delta \\ K\bar{i} + \alpha & K\bar{s} - 1 + \alpha\varphi \end{bmatrix} \quad (7)$$

and a complete characterization of the steady states of the model is provided by the following theorem

**Theorem 1** – *The endemic steady state (6) is unstable for all admissible  $\alpha$  and  $A$ .*

*Proof*

The determinant of the jacobian matrix in (7), written at the equilibrium point (6), is given by  $D(J) = A + \alpha(1 + \delta)$  and, following the conditions in lemma 1, we have  $D(J) < 0$  for  $A > 0$  and  $\alpha > 0$  (for  $\alpha = A = 0$  see the proof of theorem 2); i.e., any admissible equilibrium point is a saddle point and, therefore, unstable. ■

In spite of the results of theorem 1, the system (2) can be further manipulated in order to attain a “partial equilibrium”, as stated in the next theorem.



**Theorem 2:** Consider a solution vector  $[S(t), I(t); R(t)]'$  of (2) and define

$$Q(t) = \frac{I(t)}{R(t)}.$$

i) If  $\mu = \nu$  then the “quotient system”

$$\begin{cases} \frac{1}{R(t)} \frac{dS(t)}{dt} = -kS(t)Q(t) + \mu(Q(t) + 1) + (1 + w - p)\zeta \cdot Q(t) \\ \frac{dQ(t)}{dt} = Q(t)[kS(t) - \mu - \xi - (1 - p)\zeta - \xi Q(t) + \mu] \end{cases} \quad (8)$$

has a locally asymptotically stable equilibrium at

$$\begin{cases} \bar{S} = \frac{1}{k} \left[ \mu \left( 1 + \frac{1}{\bar{Q}} \right) + (1 + w - p)\zeta \right] \\ \bar{Q} = \frac{\mu + w\zeta - \xi + \sqrt{(\mu + w\zeta - \xi)^2 + 4\xi\mu}}{2\xi} \end{cases} \quad (9)$$

ii) If  $\mu \neq \nu$  then the “quotient system”

$$\begin{cases} \frac{1}{R(t)} \frac{dS(t)}{dt} = -kS(t)Q(t) + \nu(Q(t) + 1) + \\ \quad + (1 + w - p)[(\mu - \nu)(Q(t) + 1) + \zeta \cdot Q(t)] + (w - p)(\mu - \nu) \frac{S(t)}{R(t)} \\ \frac{dQ(t)}{dt} = kS(t)Q(t) - (\mu + \xi + \zeta)Q(t) + \\ \quad + p[(\mu - \nu)(Q(t) + 1) + \zeta Q(t)] - Q(t)(\xi Q(t) - \mu) + p(\mu - \nu) \frac{S(t)}{R(t)} \end{cases}$$

has an approximate, locally asymptotically stable equilibrium at

$$\begin{cases} \bar{S} = \frac{1}{k} \left( \mu \left( 1 + \frac{1}{\bar{Q}} \right) + (w - p)(\mu - \nu) \left( 1 + \frac{1}{\bar{Q}} \right) + (1 + w - p)\zeta \right) \\ \bar{Q} = \frac{\mu + w(\mu - \nu + \zeta) - \xi + \sqrt{(\mu + w(\mu - \nu + \zeta) - \xi)^2 + 4\xi(\mu + w(\mu - \nu))}}{2\xi} \end{cases} \quad (10)$$

where the approximation is  $o([R(t)]^{-1})$  as  $t \rightarrow \infty$ .

**Proof**

i) If  $\mu = \nu$  then (2) becomes

$$\begin{cases} \frac{dS(t)}{dt} = -kS(t)I(t) + \mu(I(t) + R(t)) + (1 + w - p)\zeta \cdot I(t) \\ \frac{dI(t)}{dt} = kS(t)I(t) - (\mu + \xi + \zeta)I(t) + p\zeta \cdot I(t) \\ \frac{dR(t)}{dt} = \xi \cdot I(t) - \mu R(t) \end{cases} \quad (11)$$

By dividing the first equation by  $R(t)$  we have the following “quotient system” of two equations

$$\begin{cases} \frac{1}{R(t)} \frac{dS(t)}{dt} = -kS(t) \frac{I(t)}{R(t)} + \mu \left( \frac{I(t)}{R(t)} + 1 \right) + (1 + w - p)\zeta \cdot \frac{I(t)}{R(t)} \\ \frac{d}{dt} \frac{I(t)}{R(t)} = \frac{R(t)I'(t) - R'(t)I(t)}{R^2(t)} \end{cases}$$

which, by setting  $Q(t) = \frac{I(t)}{R(t)}$ , using (11) and rearranging the terms, becomes

$$\begin{cases} \frac{1}{R(t)} \frac{dS(t)}{dt} = -kS(t)Q(t) + \mu(Q(t) + 1) + (1 + w - p)\zeta \cdot Q(t) \\ \frac{d}{dt} \frac{I(t)}{R(t)} = Q(t)[kS(t) - \xi - (1 - p)\zeta - \xi Q(t)] \end{cases} \quad (12)$$

When the left-hand sides vanish, we have the following system of ordinary equations

$$\begin{cases} -k\bar{S}\bar{Q} + \mu(\bar{Q} + 1) + (1 + w - p)\zeta \cdot \bar{Q} = 0 \\ \bar{Q}[k\bar{S} - \xi - (1 - p)\zeta - \xi\bar{Q}] = 0 \end{cases}$$

whose real and positive solutions provide the admissible equilibria of (12); the “infection-free” equilibrium  $\bar{Q} = 0$  is here not admissible, as it leads to an undetermined solution for  $\bar{S}$ , while the endemic equilibrium (real, positive solution) is given by

$$\begin{cases} \bar{S} = \frac{1}{k} \left[ \mu \left( 1 + \frac{1}{\bar{Q}} \right) + (1 + w - p)\zeta \right] \\ \bar{Q} = \frac{\mu + w\zeta - \xi + \sqrt{(\mu + w\zeta - \xi)^2 + 4\xi\mu}}{2\xi} \end{cases} \quad (13)$$

The jacobian matrix of (8) is given by

$$J = \begin{bmatrix} -k\bar{Q} & -k\bar{S} + \mu + (1+w-p)\zeta \\ k\bar{Q} & k\bar{S} - \xi - (1-p)\zeta - 2\xi\bar{Q} \end{bmatrix}$$

whose trace and determinant, computed at (13), are given by  $T(J) = -k\bar{Q} + \mu + \frac{\mu}{\bar{Q}} + w\zeta - \xi - 2\xi\bar{Q}$  and  $D(J) = k\bar{Q}(\xi + 2\xi\bar{Q} - \mu - w\zeta)$ . The local stability conditions  $T(J) < 0$  and  $D(J) > 0$  are then combined into  $\bar{Q} > \max\left\{\frac{\mu}{\bar{Q}(k+2\xi)} + \frac{\mu+w\zeta-\xi}{k+2\xi}; \frac{\mu+w\zeta-\xi}{2\xi}\right\}$  and, by noticing that  $k \ll 2\xi$ ,

the resulting condition  $\bar{Q} > \frac{\mu}{\bar{Q}(k+2\xi)} + \frac{\mu+w\zeta-\xi}{k+2\xi}$  becomes

$$(k + \xi)\bar{Q}^2 + \xi\bar{Q}^2 - \mu - (\mu + w\zeta - \xi)\bar{Q} > 0$$

Using the equation generating  $\bar{Q}$ :  $\xi \cdot \bar{Q}^2 - (\mu + w\zeta - \xi)\bar{Q} - \mu = 0$  leads to  $(k + \xi)\bar{Q}^2 > 0$ , which is always met.

ii) If  $\mu \neq \nu$ , by dividing the first equation of (2) by  $R(t)$  we have the following “quotient system” of two equations

$$\left\{ \begin{array}{l} \frac{1}{R(t)} \frac{dS(t)}{dt} = -kS(t) \frac{I(t)}{R(t)} - \mu \frac{S(t)}{R(t)} + \nu \left( \frac{S(t)}{R(t)} + \frac{I(t)}{R(t)} + 1 \right) + \\ \quad + (1+w-p) \left[ (\mu - \nu) \left( \frac{S(t)}{R(t)} + \frac{I(t)}{R(t)} + 1 \right) + \zeta \cdot \frac{I(t)}{R(t)} \right] \\ \frac{d}{dt} \frac{I(t)}{R(t)} = \frac{R(t)I'(t) - R'(t)I(t)}{R^2(t)} \end{array} \right.$$

which, by setting  $Q(t) = \frac{I(t)}{R(t)}$ , using (2) and rearranging the terms, becomes

$$\left\{ \begin{array}{l} \frac{1}{R(t)} \frac{dS(t)}{dt} = -kS(t)Q(t) + \nu(Q(t)+1) + \\ \quad + (1+w-p)[(\mu-\nu)(Q(t)+1) + \zeta \cdot Q(t)] + (w-p)(\mu-\nu) \frac{S(t)}{R(t)} \\ \frac{dQ(t)}{dt} = kS(t)Q(t) - (\mu + \xi + \zeta)Q(t) + \\ \quad + p[(\mu-\nu)(Q(t)+1) + \zeta Q(t)] - Q(t)(\xi Q(t) - \mu) + p(\mu-\nu) \frac{S(t)}{R(t)} \end{array} \right.$$

By setting to 0 the derivatives of the left-hand sides of the equations and expressing the last term of the right-hand side by the symbol  $o$ , we have a system of ordinary equations

$$\left\{ \begin{array}{l} -k\bar{S}\bar{Q} + \nu(\bar{Q}+1) + (1+w-p)[(\mu-\nu)(\bar{Q}+1) + \zeta \cdot \bar{Q}] + o\left(\frac{1}{R(t)}\right) = 0 \\ k\bar{S}\bar{Q} - (\mu + \xi + \zeta)\bar{Q} + p[(\mu-\nu)(\bar{Q}+1) + \zeta \cdot \bar{Q}] - \bar{Q}(\xi\bar{Q} - \mu) + o\left(\frac{1}{R(t)}\right) = 0 \end{array} \right. \quad (14)$$

Neglecting the  $o$  terms when solving for  $\bar{S}$  and  $\bar{Q}$  corresponds to an approximation error of order of magnitude equal or less than  $[R(t)]^{-1}$  which tends to 0 as  $t$  increases, since, by theorem 1, there is no stability in the system (2). The solutions of (14) are then approximated by

$$\left\{ \begin{array}{l} \bar{S} = \frac{1}{k} \left( \mu \left( 1 + \frac{1}{\bar{Q}} \right) + (w-p)(\mu-\nu) \left( 1 + \frac{1}{\bar{Q}} \right) + (1+w-p)\zeta \right) \\ \xi\bar{Q}^2 - (\mu + w(\mu-\nu + \zeta) - \xi)\bar{Q} - \mu - w(\mu-\nu) = 0 \end{array} \right.$$

The solutions of the quadratic equation

$$\bar{Q}_{\pm} = \frac{\mu + w(\mu-\nu + \zeta) - \xi \pm \sqrt{(\mu + w(\mu-\nu + \zeta) - \xi)^2 + 4\xi(\mu + w(\mu-\nu))}}{2\xi}$$

are admissible only if real and positive and are therefore subject to both

1.  $(\mu + w(\mu-\nu + \zeta) - \xi)^2 + 4\xi(\mu + w(\mu-\nu)) \geq 0$
2.  $\mu + w(\mu-\nu + \zeta) - \xi \pm \sqrt{(\mu + w(\mu-\nu + \zeta) - \xi)^2 + 4\xi(\mu + w(\mu-\nu))} \geq 0$

These conditions are met in the following cases:

1. since we have that  $\mu \gg w(\mu - \nu)$  then  $\bar{Q}_{\pm}$  are both always real
- 2.1 if  $\mu + w(\mu - \nu + \varsigma) \geq \xi$  then  $\bar{Q}_{+}$  (with + sign before the root) is positive
- 2.2 if  $\mu + w(\mu - \nu + \varsigma) < \xi$  then

$$0 < |\mu + w(\mu - \nu + \varsigma) - \xi| < +\sqrt{(\mu + w(\mu - \nu + \varsigma) - \xi)^2 + 4\xi(\mu + w(\mu - \nu))}$$

and  $\bar{Q}_{+}$  (with + sign before the root) is positive

The jacobian matrix is here given by

$$J = \begin{bmatrix} -kQ & -kS + \nu + (1-p)(\mu - \nu + \varsigma) + w(\mu - \nu + \varsigma) \\ kQ & kS - (\xi + \varsigma) + p(\mu - \nu + \varsigma) - 2\xi Q \end{bmatrix}$$

with trace and determinant, computed at  $\bar{S}$  and  $\bar{Q}_{+}$ , given by

$$T(J) = -k\bar{Q} + \mu + [(w-p)(\mu - \nu) + \mu] \frac{1}{\bar{Q}} + w(\mu - \nu + \varsigma) - \xi - 2\xi Q$$

and  $D(J) = k\bar{Q}[\xi + 2\xi Q - \mu - w(\mu - \nu + \varsigma)]$ . The local asymptotical stability conditions  $T(J) < 0$  and  $D(J) > 0$  are then combined into

$$\bar{Q} > \max \left\{ \frac{(w-p)(\mu - \nu) + \mu}{\bar{Q}(k + 2\xi)} + \frac{\mu + w(\mu - \nu + \varsigma) - \xi}{k + 2\xi}, \frac{\mu + w(\mu - \nu + \varsigma) - \xi}{2\xi} \right\}$$

If  $(w-p)(\mu - \nu) + \mu < 0$  the condition becomes  $\bar{Q} > \frac{\mu + w(\mu - \nu + \varsigma) - \xi}{2\xi}$ , which

is always met (recall the expression of  $\bar{Q}_{+}$ ).

If  $(w-p)(\mu - \nu) + \mu > 0$  the condition becomes

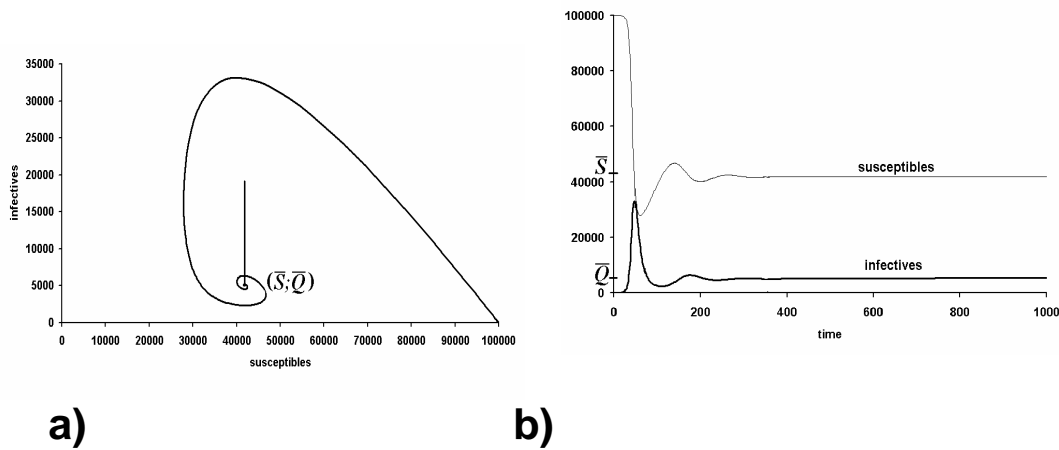
$$\bar{Q} > \frac{(w-p)(\mu - \nu) + \mu}{\bar{Q}(k + 2\xi)} + \frac{\mu + w(\mu - \nu + \varsigma) - \xi}{k + 2\xi}$$

i.e.,  $(k + \xi)\bar{Q}^2 + \xi\bar{Q}^2 - w(\mu - \nu) + p(\mu - \nu) - \mu - (\mu + w(\mu - \nu + \varsigma) - \xi)\bar{Q} > 0$ . By recalling the equation generating  $\bar{Q}$ :

$$\xi\bar{Q}^2 - (\mu + w(\mu - \nu + \varsigma) - \xi)\bar{Q} - \mu - w(\mu - \nu) = 0$$

it is equivalent to  $\bar{Q}^2 > -\frac{p(\mu - \nu)}{k + \xi}$ , which is always met, since, in general, we have that  $k + \xi > p|\mu - \nu|$ . ■

Theorem 2 states the approximate stability of a partial equilibrium of (2): if the natural demography of the population is at equilibrium, then the stability in (9) is exact, while, if the natural demography is imbalanced, then an approximation, in the sense of a *quasi-steady state*, is stated in (10): in fact, neglecting a term of the form  $C \frac{S(t)}{R(t)}$ , with  $C$  constant (see (14) in the proof of theorem 2), corresponds to making an error which tends to 0 as  $t \rightarrow \infty$ . In other words, as  $t$  increases, the trajectory of (2) in the  $S \times I$  phase plane tends to the partial equilibrium point  $(\bar{S}; \bar{Q})$ , after which the number of susceptibles  $\bar{S}$  remains constant, while the number of infectives  $I(t)$  keeps increasing with time, as in figure (6). Thus, although the majority of the immigrant individuals enters the system as controlled/screened for the disease under analysis, the increase of the population tends to be made out almost entirely of infective (and removed) individuals: this constitutes a relevant public health issue which cannot be ignored when shaping immigration policies.



**Figure 6:** an example of a trajectory (graph a) ) of the system (2) in the  $S \times I$  phase plane (see theorem 2) and its time series curves of infectives and susceptibles (graph b) ). Note the different Y-axis scale in the two graphs and the ordinates corresponding to the equilibrium  $(\bar{S}; \bar{Q})$  in the time series graph; moreover, the time lag in the second graphs is shorter than the first one in order to better visualize the oscillations of the time series.

### 3. Public Health, Local Demography and Immigration Policies

A mixture of politics, public health, economic and demographic policies is encountered when dealing with immigration. A whole repertoire of measures in each of these fields is, in turn, applied to migrations to analyze and to variously control the flows of individuals into a local population. In particular, the existence of a current, major epidemic in the host population and/or in the immigrants is often a reason for policy adjustment measures. If, on the one hand, economic and political issues have a say on the immigration regulation that is often beyond epidemiological reasons (the calibration of the quota  $w$  of controlled immigration or the amount of local resources attracting individuals from outside) and determined on an emotional rather than an analytical basis, on the other hand, demography and public health are directly involved in the epidemiological process and dynamics. A small, relatively isolated community in a poor area, when hit by epidemic outbursts, can experience a significant reduction in the number of individuals (see [15]) and new entries contribute to sustain the epidemics either directly (infected immigration) or indirectly (healthy immigrants, susceptible to infection by locals). An open community, subject to exogenous demographic pressure, is likely to accept selected and controlled immigration for several reasons (fresh labour forces, refugees, other); here again support to a local epidemic is provided through two separated routes: the infectious immigration (unavoidable, under the hypothesis that part of the immigrants escape public health control) and the susceptible immigration. However, in this case the epidemic dynamics follow a very peculiar pattern, since, after a period of demographic and epidemiological instability, the population steadily increases and the increase is almost only made up of present or former infectives.

In details, the number of susceptible individuals in the population tends to approximately stabilize at  $\bar{S}$ , while the number of infectives exponentially increases. Analyzing the path followed by the trajectories of (2), a critical time

instant for the system can be detected at  $\hat{t}$ , such that, for some  $\varepsilon > 0$ ,  $\left| \frac{dS(t)}{dt} \right| > \varepsilon$

$\forall t < \hat{t}$  and  $\left| \frac{dS(t)}{dt} \right| < \varepsilon \quad \forall t \geq \hat{t}$ : a straightforward, graphical/heuristic proof of the

existence and uniqueness of  $\hat{t}$  (under weak conditions) is omitted, as it does not substantially affect the whole argument of this section..

The value of  $\hat{t}$  thus provides a time threshold for planning direct policy interventions on infectious immigration: usual public health control measures for immigration must be carefully calibrated, in order to avoid inefficacies and, moreover, to avoid investments with a negative cost-benefit balance. The role of the virulence of the infection becomes now irrelevant with respect to the

demographic balance of the local, healthy population (as a matter of fact, in this case, it seems not possible to determine the fundamental parameter  $R_0$ ) and the epidemic spread can be divided into two clearly separated phases: a first one, with a pattern similar to the model in [15] and a second one where the diffusion pattern becomes rather rigid and very little can be directly done to reduce the infection. While, in the first phase of the spread, the screening and cure of local infectives and some forms of quarantine and screening of immigrations may prove effective (see [15] for more details), in the second phase, even when the proportion of infectious immigration is vanishing, the number of infectives in the population increases and the number of healthy individuals remains more or less constant (see figure 6). Thus, it is important to act promptly, before the epidemic reaches the second phase at  $\hat{t}$ : after that, a decrease in the spread of the infection can be reached only by promoting a decrease of the total population (obtained by immigration and birth control). It becomes, therefore, essential to determine  $\hat{t}$ , i.e., the threshold when the phase transition of the epidemic spread occurs.

Unfortunately, the vector function  $(S(t); I(t))'$  does not have a closed inverse form to derive  $\hat{t} = \min\{t : S(t) \approx \bar{S}\}$  corresponding to the quasi-steady level of susceptibles in (10). and the determination of  $\hat{t}$  (and of related issues such as the velocity of increase of  $I(t)$ ,  $\forall t > \hat{t}$ ) must, therefore, proceed by numerical approximations

Once  $\hat{t}$  is reached, quarantine, screening or any other attempt to act directly on the infectious immigration becomes ineffective and the infection tends to exponentially invade the population: only unlikely and radical control measures on birth and total immigration may have some effects on the local diffusion of the infection.

#### 4. Conclusions

The epidemiologically meaningful parameter  $R_0$ , called basic reproduction number, determines a bifurcation point at  $R_0 = 1$  between an infection-free and an endemic equilibrium in epidemic models that are closed or whose demography only depends on local dynamics. However, if an immigration flow is introduced in the modelling scheme, then  $R_0$  is no longer alone in determining the bifurcation (as an example see [15]) and if, furthermore, the total population varies with time (according to all the factors listed in this paper), then no infection-free equilibrium is admissible and  $R_0$  completely loses its epidemiological relevance. In this latter case a peculiar behaviour of the system dynamics is observed and other factors must be taken into consideration to analyze the course of the epidemics, such as the proportion of susceptibles entering the population and the point-wise



progression of the infective time-series: a thorough investigation of these factors becomes therefore essential before undertaking public health policies.

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