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SENSITIVITY OF PLAGUE LOCAL EPIDEMICS TO THE POPULATION SIZE AND SPATIAL DISTRIBUTION

Contribution of an individual based model, SIMPEST

Vincent LAPERRIERE, Dominique BADARIOTTI and Arnaud BANOS

ABSTRACT

This document presents some simulation results obtained on an epidemic model, SIMPEST, designed to simulate the dynamics of local bubonic plague epidemics. The individual-based approach allows to consider discrete individuals, their spatial behaviour, and the contingency of interactions among them. This approach allows to perform investigations about how various factors, such as sociological or geographical ones, may influence the development of a local epidemic. We performed some sensitivity analysis on two initial conditions: population density and habitat distribution. The influence of these input variables on the epidemic dynamics is investigated using some key output variables. Our simulation results can then be compared with some well-established elements of the theory of epidemics, more specifically the existence of an epidemic threshold on the population size and the influence of the contact network structure on the epidemic force.

KEYWORDS

Epidemic model, Individual-based, Sensitivity analysis, Replacement number, Contact network

INTRODUCTION

In the study of infectious diseases, the investigation of the causal factors which command the deployment of an epidemic is a crucial step to forecast, control and anticipate the outbreak. With the development of mathematical modelling, the last century has witnessed the emergence of a real theory of epidemics (Anderson, May, 1991) which led to the development of so-called “compartmental models”. The main contribution of classical compartmental models in this field was the definition of the threshold theorem, stating that the population which is susceptible to get infected must exceed a critical size for an epidemic to occur.

However, classical compartmental models often ignore the geographical context of the disease transmission, assuming that susceptible and infected individuals are homogeneously mixed. An individual-based approach, which considers discrete individuals and the contingency of interactions among them, provides an adequate framework to design more realistic epidemiological models and to enlarge investigations about how various factors, such as sociological or geographical ones, may influence the development of an epidemic (Bian, 2004 ; Dunham, 2005).

MODELLING OBJECTIVES

In this paper, we present SIMPEST, a model developed to simulate plague epidemics at a local scale in the context of the rural malagasy bubonic plague. Our task is to integrate in the model the spatial behaviour of the agents – namely fleas and rats – which are implied in the epidemiological cycle. This may help us to investigate the global dynamics of the disease arising at a populational level, taking into account the contingency of the disease transmission that results from the contacts between fleas and rats at an individual level. We consider some key output variables like:

- [1] The “**global force of the infection**”, given by the total number of rats and fleas infection cases over the simulation.

- [2] The “**potential force of infection to human**”, as an indicator of the disease potential to generate human cases, given by the total number of free-living infectious fleas over the simulation and thus susceptible to fix on and bite a human.
- [3] The “**replacement number of the infection**” R , traditionally considered in compartmental models, defined as the average number of secondary infections caused by a typical infectious individual during its entire period of infectiousness.
- [4] Some key properties of the emerging **contact network** between rats, as the **mean degree** and the **clustering coefficient**. In this network, nodes represent rats and edges represent rats that have shared some common fleas during the simulation, hence having been indirectly in contact.

Our modelling objective, here, is to perform some sensitivity analysis and compare our results with some elements of the theory of epidemics (Anderson, May, 1991 ; Keeling, 1999), or even previous experiences of plague modelling, mainly based on a mathematical formalism (Keeling, Gilligan, 2000). This is a way to validate our model in a first step, and to gain knowledge about factors influencing the epidemic dynamics in a second one, especially those related to the geographical context.

MODEL STRUCTURE

SIMPEST is implemented on the Netlogo platform, that enables us to define a schematical environment representing a malagasy village and its surroundings. Rats and fleas evolve in this environment. Humans, to which the disease can propagate, have not been explicitly represented since they cannot transmit the disease and thus don't influence the disease dynamics that we investigate. Instead, we consider the variable “potential force of infection to human” (defined in [2], see below).

The individuals' behaviour is determined by three kinds of rules. 1) Some demographic rules are introduced: rats and fleas are characterized by a life expectancy and a reproductive rate. 2) Specific mobility rules are implemented: rats move by night, leaving their nest and looking for some food. Free fleas wait for a host to fix on and stay fixed until the host dies. 3) Transmission rules between fleas and rats are stochastic and the epidemiological cycle follows a SEIR scheme (Susceptible-Exposed-Infectious-Removed): one susceptible flea may get exposed by biting an infectious rat. After the latent period has expired, the flea gets infectious and thus may transmit the disease to another susceptible host. At the end of the infectious period, a rat might recover and become immunized, whereas a flea automatically dies.

SIMULATION RESULTS

To assess the influence of the local geographical context on a bubonic plague epidemic, we stayed in a theoretical frame and performed some sensitivity analysis on two initial conditions, susceptible to vary from a local area to another: population density and habitat distribution.

Sensitivity analysis to the population density

We first paid attention on the initial population size of fleas and rats. The main reason is that it is the most studied input variable in epidemiological modelling (Keeling, Gilligan, 2000). The threshold theorem state that an epidemic can develop only if the susceptible population size exceeds some critical value, for which the replacement number R is one, otherwise it dies out.

Thus, to verify if such an epidemic threshold exist in our individual-based model, we defined the replacement number R as the product between the replacement number from flea to rat (R_{flea}) and the replacement number from rat to flea (R_{rat}). R is reported at the end of the simulation so that it traduces the average replacement number over the simulation, as defined in [3].

Varying the initial population size of both fleas has a positive and a more or less linear effect on the global force of the epidemic as defined in [1]. Now, to go beyond this expected result, we can study the correlation between the global force of the infection and the mean replacement number.

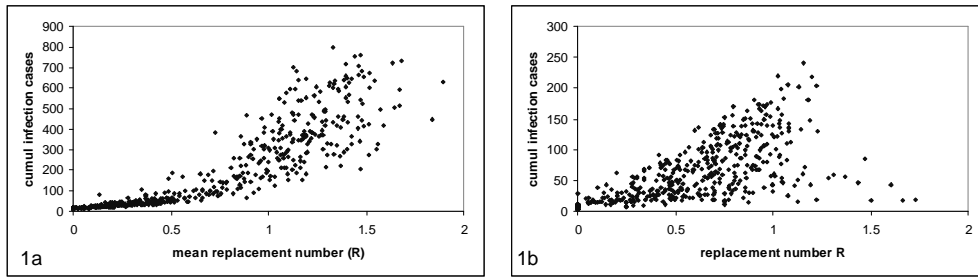


Figure 1: OAT sensitivity analysis results to the initial flea index (1a) and to the rat initial population size (1b). For each simulation realized, we plotted the “mean replacement number of the infection R ” [3] against the “global force of the infection” [1]. To take into account stochasticity, 20 simulations are run for each value of the tested parameter.

This way, we can compare our simulation results with the theoretical threshold of unity for a disease to develop. Figure 1 suggests that the threshold effect is more pronounced for the flea population size (Figure 1a) than for the rat one (Figure 1b). Our results show that the initial flea index must exceed a critical value (around 2) for the replacement number R to reach unity, and for an epidemic to develop. This is a slightly upper value than the empirically-observed threshold of one for the flea index (Pollitzer, 1954). A same value for the replacement number corresponds to various sizes of the epidemic, especially when it is high. This is due to the contact structure that is different from a simulation to another, because of their stochasticity.

Sensitivity analysis to the rat habitat distribution and mobility

In a second series of simulations, we studied the effect of varying both the rat initial habitat distribution and mobility. We tested four theoretical scenarios, presented in figure 2. As output variables, we considered the mean replacement number R [3] and an intermediary one which is the contact network between rats, resulting from a simulation, as defined in [4].

	<p>Scenario A: Totally grouped, never moving Rats never move and stay fixed in one nest. This is close to the unrealistic assumption of the simplest compartmental model that the population is constantly mixed in an homogeneous way.</p>
	<p>Scenario B: Totally grouped during night, randomly moving during day Rats stay grouped in one nest during the night and move randomly during the day.</p>
	<p>Scenario C: Multi-grouped during night, randomly moving during day Rats are grouped in several dispersed nests during the night and move randomly during the day. This is the closest scenario to the reality, where the rat population is structured in family groups.</p>
	<p>Scenario D: Never grouped, always randomly moving Rats constantly move randomly. No distinction is done between night and day.</p>

Figure 2: Scenarios tested for the sensitivity analysis to the rat habitat distribution and mobility.

Simulation results, presented below, show that the more grouped the population is, the highest the potential for the disease to develop is (Figure 3a). Hence, the social structure of the rat population, more or less distributed in spatially dispersed family groups, plays an important role in the epidemic development.

To better understand this result, we can study the effect of the different scenarios on the contact network structure traduced by two essential properties: the mean degree, that is the average number of neighbour nodes per node (Figure 3b) and the average clustering coefficient (Figure 3c), measuring the interconnectedness of the network. Defined for one node, it is the number of edges existing among the neighbourhood of this node, divided by the total number of possible nodes.

When the dispersal of individuals increases, rats enter in contact (*via* sharing common fleas) with less other rats and thus the mean degree decreases. Moreover, spatial correlations between rats explain that the interconnectedness of the contact network increases. It means that there are more local connections and less long-range connections than in the totally grouped scenario, where individuals have the same probability to encounter everyone else. We verify Keeling's statements obtained on a network model (Keeling, 1999), that is to say: the potential of the disease to develop is reduced by having few neighbours (the mean degree is small) or a highly interconnected network structure (the clustering coefficient is large).

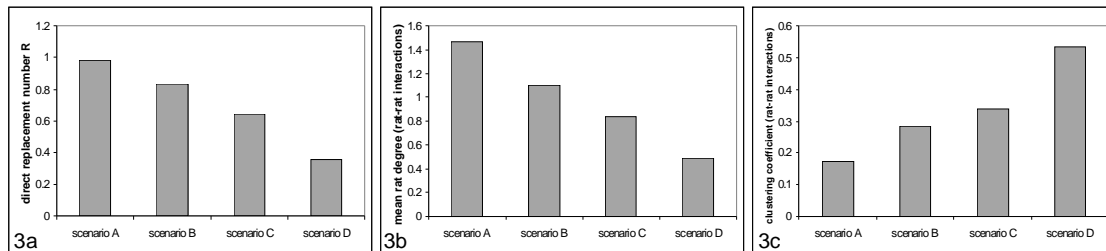


Figure 3: Sensitivity analysis to four scenarios of rat habitat distribution and mobility for three output variables: the mean replacement number of the infection R (3a), the mean degree (3b) and the clustering coefficient (3c) of the rat contact network. Results are averaged over 20 simulations.

These results show that besides the total population size of both fleas and rats, the rat social structure and mobility are factors of huge importance when understanding conditions for plague epidemics to develop. Such investigations on the population composition and spatial distribution are difficult to be led with traditional mathematical models and the individual based approach may be the most appropriate one.

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