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Low dimensional chaotic models for the plague epidemic in Bombay (1896–1911)

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ABSTRACT

A plague epidemic broke out in Bombay in 1896 and became endemic. From 1905 to 1911, the epidemic was closely monitored by an Advisory Committee appointed to investigate the causes of the disease in any way. An impressive quantity of information was gathered, analyzed and published. Published data include records of the number of people who died from plague, and of the two main populations of rodents which were infected by plague in Bombay city.

In the present paper, these data are revisited using a global modeling technique. This technique is applied to both single and multivariate observational time series. Several models are obtained for which a chaotic behavior can be observed. Obtaining such models proves that the dynamics of plague can be approximated by low-dimensional deterministic systems that can produce chaos. The multivariate models give a strong argument for interactive couplings between the epidemic and the epizootics of the two main species of rat. An interpretation of this coupling is given.

1. Introduction

Plague is a zoonoses that has already caused several historical pandemics. It has reappeared in several countries during the 1990s as a reemerging disease [1]. Plague is caused by the bacillus *Yersinia Pestis*, discovered in 1894 [2]. Bubonic plague is the most common form of plague. Clinically, bubonic plague is characterized by buboes that result from the infection of lymph nodes. Contrarily to pneumonic plague, which is a severe type of lung infection, bubonic plague is generally transmitted to people by the bite of a flea having transited onto a rat or another rodent infected by the disease and is not transmitted directly from person to person. Without adapted antibiotics, the disease can become septicemic and then lead to the death in the first 36 h, or even less. Plague zoonose may have different specificities from one source of plague to another, however, its dynamics always implies mammal hosts as reservoirs and fleas as vectors. Although the reservoirs, vectors and the main processes at work in the propagation of the disease are well identified, the persistence of this highly virulent disease remains poorly understood.

Since the first model introduced by Kermack and McKendrick in 1927 [3], most of the models for bubonic plague were focused onto the human aspect of the disease. Such models are based on the assumption that the number of cases can be simulated by models made of three compartments corresponding to the susceptible people x, the infected people y and the dead (or immune) people z respectively [3,4]. It was only recently that a plague model taking into

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account interactions between rat and flea populations was attempted [5–7]. Higher-dimensional models resulted from this approach. It is generally assumed in these recent models that the rat epizootic drives the epidemic, so that human cases can be modeled as a by-product of the progression of the disease in the rodent community; such an assumption allows notable simplifications. It is generally also assumed in such models that one single specie of rodent and one single specie of flea are enough to simulate plague progression.

However, it is also known that numerous species of rodents and fleas may carry the plague [8,9]. Black rat (*Mus rattus*) was identified in numerous cases as having a key role in plague epidemic (*e.g.* [10]), but other species of rat [9,11] and other rodents such as marmots, prairie dogs, gerbils, squirrels etc. are also known to be reservoirs of plague [8,12–14]. Moreover, it was observed that epizootics of different species may exhibit differences in their dynamical behavior; It was the case in the Bombay plague, for which a systematic delay in the epizootics of rats *Mus decumanus* and *Mus rattus* was observed [11] (p. 754). It was also found in some places that the immune response to infection may differ within the same endemic area, even for the same specie of rat [10]. Indeed, it was observed in Madagascar that, despite the undeniable susceptibility of rats to plague, populations of very resistant *M. rattus* and *M. norvegicus* could be found in Antananarivo city within the plague-endemic areas [15]. The effective interactions between the population dynamics of the different species in a same source of plague, as well as the relations between susceptible and resistant behaviors remain poorly understood.

It was observed at different geographical places in the world that the populations of different species of flea may exhibit different behaviors in relation to epizootics and epidemic [16,17]. In particular, it was found that some species of flea disliked biting man. However, it was shown that rat-fleas, which do not attack man under normal conditions, may attack once their natural hosts have died off [18]. Fortunately, from a modeling point of view, it was shown that the population of infected flea can be approximated by a stationary state due to the short average time required for a free-living flea to find a new host [7].

Historically, most of the epidemic models are analytically built based on an empirical knowledge of the processes at work. This is in particular the case for Kermack and McKendrick's model for plague. For this reason, as pointed out by its authors, a close fit between the model to the data cannot be expected [3]. Contrary to this, models based on selection techniques are directly obtained from the data and do not require strong *a priori* hypotheses, allowing for a better agreement between model and observational data. Nevertheless, data based approaches generally do not allow to explicit in a comprehensive way the interaction between the variables of the obtained model. A description of the underlying processes remains hard to provide.

In the present work, the Bombay plague epidemic is investigated as case study in order to investigate the possibility (1) to detect chaos from observed plague epidemic, that is, to provide arguments for both determinism and high sensitiveness to initial conditions, and (2) to identify the dynamical couplings existing between the infected people and the two rat epizootics.

The theory of nonlinear dynamical system is used as a background to investigate the problem. The global modeling technique provides deterministic dynamical models that reproduce the trajectory reconstructed from the observational time series. Two types of reconstructed space are mainly used for global modeling, either spanned by derivative coordinates leading to Ordinary Differential Equations [19] or by delay coordinates leading to difference equations [20,21]. In the present work, only the former formulation based on derivative coordinates is considered. Since its earlier applications [19,22], the global modeling technique mostly focused on modeling dynamical behaviors from a single scalar time series. During the last decade, the technique has proved to have the potential to deal with single observable from real world in quite various domains [23–26]. To the best of our knowledge, although several models were obtained from single time series, no dynamical system of Ordinary Differential Equations (that is numerically integrable) was ever obtained directly from multivariate observational data.

In the present paper, it is first attempted to obtain a global model from the multivariate time series collected during the Bombay plague epidemics. The obtained models are then used to explain the so determined couplings between the plague epidemic and the two epizootics of *M. rattus* and *M. decumanus* species. Model validation is based on predictability considerations.

The paper is constructed as follows: Context and data are presented in Section 2. The theoretical background and the methods are described in Section 3. Results are presented in Section 4 where univariate and multivariate models are first considered separately and then compared based on their forecasting ability. The algebraic structure of the multivariate models is then discussed in Section 5. Conclusions are drawn in Section 6.

2. Context and data

2.1. Context

The information that bubonic plague epidemic broke out in Bombay (now Mumbai) in 1896 was first reported by the *British Medical Journal* on October 3rd [27]. The outbreak was estimated *a posteriori* to have taken place in the month of August 1896. December 1896 was marked by a sudden rise of mortality that triggered the alarm; A first Plague Committee for the City of Bombay was appointed by the Bombay Government on the 2nd of March 1897 with the aim to stamp out the disease before the beginning of rains [28]. Once a peak of mortality was reached in January 1897, the mortality progressively decreased, but the epidemic became endemic and propagated year to year without stopping, exhibiting a marked seasonal signal in the mortality.

The first Indian Committees clearly considered the role of rat as a key element but disregarded the flea. The possibility for flea to transmit plague disease from rat to rat was first mentioned by Ogata in 1897 [29] and a first demonstration of its possible role as a vector was given by Simond in 1898 [30]. Probably due to the multiplicity and to the complexity of the situations met, this idea was not immediately accepted