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Author: Orlando Silva

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Black Death – model and simulation

Orlando Silva

Universidade de Évora, Portugal

Highlights:

- One continuous deterministic SI epidemic model is chosen and further developed to simulate the Black Death.
- A model for the interaction between short range dislocations and contagion is proposed that minimizes the need of a classical diffusion term for infected transport.
- One modified expression for the classical contagion rate law takes in account the saturation effect in contact rate.
- The results of the spatiotemporal simulation of Black Death are compared with historical data.

ABSTRACT

The Black Death analysis and data organizing have been performed in unifying manners, among others, by Byrne and Christakos. The mathematical modelling of Noble of 1974 did not originate full spatiotemporal 2D simulations. The present model incorporates a new term to calculate the effects of the interaction between contagion and short range dislocations. The contagion rate term is modified to account for saturation effects in the contact rate. The model and parameters used very nearly reproduce important features of the spatiotemporal evolution of the Black Death, the calculated global mortality attaining a very large fraction of the European population.

Keywords: Black Death, epidemiology, model, simulation

1. Introduction

Approximately at the autumn of 1347, one disease was introduced in Europe via sea ports, possibly by land too, that spread to all the continent by sea coast, river and land routes, resulting in the infection of almost all European regions. It came to extinction by 1351, meanwhile killing a large part of the inhabitants, usually estimated between one fourth and half the population.

Attributed to the Italian notary Gabrielle de Mussis is the claim that the disease was transported by boat after the infection of Caffa, in Crimea, till Messina and then to Italy. Differently, Wheelis [31] discards the role of Caffa in the contagion of the European ports.

In the following decades, some other epidemics led to much lower mortalities in Europe, possibly by the rise of the fraction of the immune population or due to the change of the pathogenic agent. Most authors support the disease was caused by the bacteria Yersinia pestis while others reject this hypothesis. More detailed information can be found in [13] and references there.

According to [4] and most authors, the transfer of the pathogen to humans is made by infected flees from rats. Others sustain the transfer is made by infected fleas and lice between humans (see [16]), the goods in trade routes possibly carrying infected fleas even in the absence of rats.