Threshold behaviour for a chain-binomial S-I-S infectious disease

Claude Lefèvre

Université Libre de Bruxelles, Institut de Statistique, C.P. 210, Boulevard du Triomphe, B-1050 Bruxelles, Belgium

Abstract. The present paper is concerned with a chain-binomial deterministic model for an infectious disease of the S-I-S type. The model extends the Cooke et al. (1977) and Longini (1980) model in the sense that it accounts for the distribution of the number of contacts made by each susceptible during an infectious period and for the probabilities of infection at the different contacts with infectives. The aim of the work is to investigate under which conditions the disease becomes endemic or not. Some partial results are first derived, but a complete analysis of the threshold behaviour seems very intricate. A more detailed discussion is then presented for the specific case where at least \( K + 1 \) contacts with infectives, \( K \geq 1 \) fixed, are required to make a susceptible infected.

Key words: S-I-S infectious disease — Threshold behaviour — First-order difference equation — Stability analysis

1. Introduction

There is an important mathematical literature devoted to the description of the spread of infectious diseases. Relatively little general theory, however, exists for discrete time models. Most work in the field is concentrated on simple models of chain binomial type. In these models, the disease process is reduced to a succession of two periods: the latent period, supposed of fixed length, and the infectious period, considered to be contracted to a single point of time. While infectious, an individual transmits the disease agents to any susceptible with a constant probability \( p, 0 < p < 1 \). After that period, the individual either becomes immune against further infection or returns to the susceptible state. The former hypothesis is said of the S-I-R (susceptible-infectious-removed) type and corresponds to the classical Reed-Frost model (see, e.g. Bailey (1975)). The latter hypothesis is said of the S-I-S (susceptible-infectious-susceptible) type and corresponds to the model discussed by Cooke et al. (1977) and Longini (1980); in the sequel, this model will be referred as the C.-L. model.
The most outstanding result obtained for these models is a threshold theorem for the qualitative behaviour of the disease process. Roughly, its object is to state the conditions which lead, for the Reed-Frost model, to a major transient outbreak, and for the C.-L. model, to an endemic situation. Such a property holds for both deterministic and stochastic formulations, but of course in slightly different terms. For more information, see Von Bahr and Martin-Löf (1980), and Cooke et al. (1977) and Longini (1980).

The model considered in the present paper is an extended version of the C.-L. model. Two assumptions in the above description are modified. On the one hand, we suppose that during every infectious period, each susceptible makes a random number $R$ of contacts with other individuals of the population. On the other hand, we suppose that the $j$th contact, $j \geq 1$, of a not yet infected individual with an infective leads to infection with probability $\beta_j$. The former hypothesis, with $\beta_1 = 1$, has been introduced by Dietz and Schenzle (1985) to construct a more realistic and flexible version of the Reed-Frost model. These authors have shown that the usual assumption of a constant probability $p$ for the transmission of the infectious agents can be seen as corresponding to the particular case where $R$ has a Poisson distribution with parameter $(N-1)\ln[1/(1-p)]$. Obviously, the same conclusions remain valid for our similar generalization of the C.-L. model. Now, the hypothesis $\beta_1 = 1$ means that one contact with an infective is sufficient to make a susceptible infective. This assumption can appear too restrictive in practice, and it is then natural to extend it by introducing the probabilities of infection $\beta_j$, $j \geq 1$. For example, situations where the different contacts with infectives do not cause necessarily infection but weaken [or, more rarely, reinforce] the resistance of the susceptible may be modelled by considering a nondecreasing [resp. nonincreasing] sequence of $\beta_j$'s, $j \geq 1$.

The paper is structured as follows. In Sect. 2, we formulate the model stochastically and we deduce the associated deterministic version. Afterwards, we only deal with the deterministic model and we investigate under which conditions the disease becomes endemic. We derive some partial results in Sect. 3, but a complete analysis of the threshold behaviour seems impossible. In Sect. 4, we study with more details the specific case where $\beta_j = 0$, $1 \leq j \leq K$ and $\beta_{K+1} = 1$, $K \geq 1$ fixed, that is, when at least $K+1$ contacts with infectives are needed for infection to occur. One group of diseases that may satisfy this requirement as first approximation are those caused by some of the enteric agents (e.g. shigellosis, amebiasis, giardiasis), where the probability of infection is dose related.

### 2. Model formulation

The model is an extension of the C.-L. model. Let us consider a closed and homogeneously mixing population of $N$ individuals subdivided in two classes, the susceptibles and the infectives. After a susceptible has become infected, there is a latent period of fixed length. The subsequent infectious period is contracted...