Rev Saúde Pública 2006;40(2):205-6

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Comentário: The reversible catalytic model and the mathematical theory of epidemics

O modelo catalítico reversível e a teoria matemática de epidemias

In the early XXth century, mathematical models were introduced into infectious disease epidemiology with the hope that formal procedures so successfully applied to physics and chemistry would be helpful in untangling complex causal relationships in epidemic systems. This hope is clearly stated by Kermack & McKendrick[2] (1927) at the start of one of their papers on the mathematical theory of epidemics: "One of the most striking features in the study of epidemics is the difficulty of finding a causal factor which appears to be adequate to account for the magnitude of the frequent epidemics of diseases which visit almost every population."

The starting point for the mathematical modeling of infectious diseases is the analogy between the contagious process that lead to new infections and the process observed in autocatalytic reactions, where the conversion from substrate to product is driven by the contact between these two substances. In both systems, the product (or infective individuals in the case of epidemic systems) acts as a catalyst and the rate of reaction increases as the concentration of the product increases. The reaction slows down when the substrate (or the amount of susceptibles) reduces below a certain threshold. Based on this analogy, Muench[3] published a book in 1959 where he proposed the use of catalytic models to estimate the rate of conversion (that is, the infection or incidence rate) from susceptible to infective, based on the fitting of these models to prevalence data.

Since its publication, Muench's book has been source of inspiration for an ever growing series of papers (Figure). Part of this publications concerns the estimation of age-specific infection rate from serological data using catalytic models or their derivations. Rodrigues da Silva (1969) is one of the first examples of this type of application. He estimated the rate of Chagas disease infection in communities in Salvador city, Brazil, and compared them to a community in Venezuela. He found similar values in all three communities and concluded that the contagious process involved in Chagas transmission was similar in both regions. When presenting his results, however, he showed how concerned he was about the oversimplifying assumptions of the catalytic model. He listed them: the assumption that the dynamics of disease transmission is at an endemic steady-state, that is, during the whole period since the birth of the oldest member of the cohort until the last recorded data, the rate of infec-

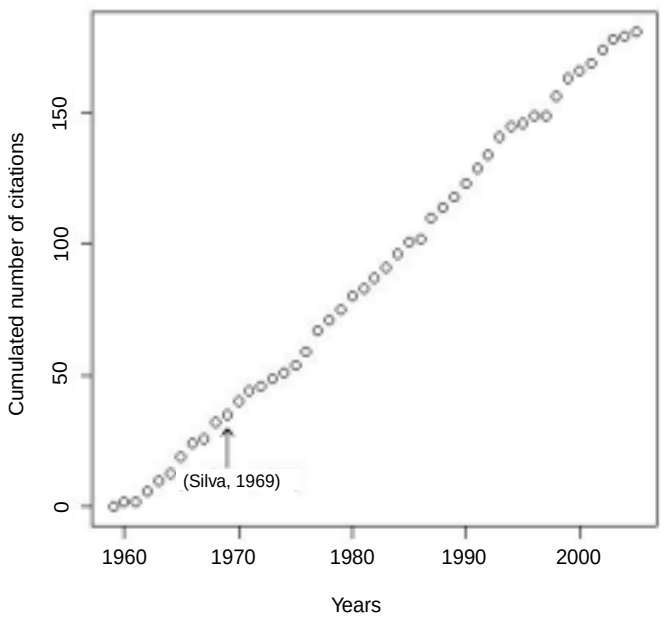

Figure - Cumulated number of papers citing Muench's book (1959) on the application of catalytic models in epidemiology.

tion is assumed constant; 2) that migration was negligible; 3) that mortality due to the disease was negligible; 4) the model did not consider other components of the transmission process, as the vector population or the other vertebrate hosts populations.

Even if these simplifications may have brought doubts about the conclusions of the work, the modeling exercise *per se* was successful in itself as way to make explicit arguments regarding the nature of the contagious process and allowing a mechanistic model-based approach to the estimation of the infection rate. Since then, the emphasis in mechanistic mathematical modeling has turned to the development of more refined models that seek to relax the strict assumptions present in formulations as those proposed by Muench and applied by Rodrigues da Silva. In the case of the Chagasic disease, for example, new mathematical models have been recently proposed to evaluate alternative control strategies, more specifically, the pros and cons of removing animals (specially dogs) from the domestic environment. With this aim in mind, Cohen & Gurtler[1] (2001) elaborated a mathematical model to represent the dynamics of infection within a household where humans, dogs, chickens and insect vectors coexist. In this environment, the parasite circulates among humans, dogs and bugs and the model seek to capture this contact process. Chickens, which are not susceptible, have two potential impacts on transmission. They may help to reduce human exposition by diverting part of bug biting activity. On the other hand, increased number of available hosts may increase the bug population, and consequently increase transmission of the parasite to humans. They find that having or not chickens at home have only a marginal impact on transmission while removing dogs from sleeping rooms would have a large beneficial effect.

Still, although models used today are much more complex than the reversible catalytic models used by Rodrigues da Silva, the most basic assumption of an autocatalytic process still holds in most models of infectious diseases dynamics. Moreover, the problem of estimating infectious diseases parameters from serological data is still an open issue and much work is expected in the future on the development of mathematical models for disease. An interesting idea would be to apply modern methods to Rodrigues da Silva's data.

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