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Directly Transmitted Infections Modeling Considering an Age-Structured Contact Rate

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Abstract—Mathematical models dealing with childhood viral infections consider the spread of disease according to the law of mass action. This law states that there are random encounters (or contacts) among susceptible and infectious individuals. Therefore, mathematical descriptions of the transmission of infections are heavily dependent on the assumptions concerning the contact rate. In order to develop an age-structured contact rate model, a pattern of contacts among individuals in a community is developed by stochastic processes.

Keywords—Contact rate, Age-structured model, Probability, Force of infection.

1. INTRODUCTION

Directly transmitted childhood infections, like rubella and measles, have been used as good examples for the application of mathematical models to the study and comprehension of the epidemiology of these diseases. For those infections, there are long-term incidence records in developed countries from Europe and North America [1–3] largely used by modelers as a reference to test their models [4,5], whose assumptions and simplifications proved to be rather good approximations to real data. But the picture is quite different when developing countries are considered, where the lack of a fully organized health system does not offer the same quality on case notification data. The most reliable information derived from these communities are seroprevalence surveys, from which the presence of specific antibodies against a given infectious agent in an individual is interpreted as a previous infection [6].

Mathematical models, based most conveniently on incidence records or seroprevalence data, can be a useful tool to estimate new cases per unit of time (incidence rate) per susceptible individuals, the so called force of infection [7]. Directly transmitted childhood infections models are formulated basically by taking the force of infection dependent on the contact rate, which is related to the pattern of contacts among susceptible and infectious individuals. Therefore, the assumptions on the contact rate lead to quite different approaches when one deals with models [8].

A first assumption, and also the simplest, is to consider a constant contact rate among individuals over all ages and time. Consequently, the force of infection becomes constant. The resulting mathematical model is described by a time-dependent system of differential equations without

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age structure. This assumption can generate nonrealistic outputs when modeling childhood diseases with a strong age dependent pattern. A second and better assumption is, therefore, to take into account the age variability in the pattern of contacts. A mathematical model with this assumption yields a time- and age-dependent system of differential equations (see [9] who was the first author to apply this formalism to epidemiology with constant contact rate), resulting in the well-established concept of the age dependent force of infection [7].

Two attempts of representing the age-structured contact rate can be found in the literature: a matrix with constant elements and a constant value for different age classes. Anderson and May [4] developed the concept they called *Who-Acquires-Infection-From-Whom* matrix (WAIFW). Briefly, this is a matrix where the elements of rows and columns are the contact rates constants over the discrete age classes of susceptible and infectious individuals. Schenzle [5] developed an age-structured contact pattern where constant values on several age intervals are assigned and, then, structured the dynamics in a coupled differential equations to estimate the contact rate from notified data. Although both methods represent good approaches to modeling the dynamics of direct transmitted diseases, they are applicable to the description of different kind of data collection: the WAIFW method is appropriate to analyze seroprevalence data while Schenzle's method is better applied to incidence records.

The purpose of this paper, is to develop a model with age-structured contact rate applicable whenever seroprevalence data are available. For instance, when the WAIFW method is considered, a large unknown matrix with n^2 elements is introduced, which gets even larger with increased accuracy. In this approach, it is necessary to devise a method of selecting an $n \times n$ contact matrix. Therefore, our goal is the development of an age-structured pattern of contacts based on stochastic processes. By doing this, we are reducing the n^2 unknown elements of the WAIFW matrix to a fewer number of parameters which are related to the continuous contact rate and, consequently, the total number of parameters of the model to be estimated is diminished greatly. However, as pointed by Tudor [10], data on contact rates do not exist, although most parameters related to the disease transmission can be estimated directly.

This fundamentally theoretical paper is divided as follows. In Section 2, the general model is presented. In Section 2.1, an age-structured pattern of contacts is developed, which is used to obtain the corresponding age-structured model in Section 2.2. In Section 3, we discuss and summarize our findings. The epidemiological applications are left to a further paper [11].

2. THE MODEL

Farrington [12] obtained an age dependent force of infection from cumulative distribution function of age at infection. Here, the age dependent force of infection will be obtained from a compartmental model taking into account an age-structured contact rate. In order to do this, we develop an age-structured pattern of contacts among individuals in Section 2.1, and the corresponding age-structured model is considered in Section 2.2.

The dynamics of directly transmitted infectious diseases models considering age-structured contact rates are described by a system of partial differential equations [13]. The scope of this paper is restricted to the steady state analysis [9].

Let a closed community be subdivided into four groups: $X(a)$, $H(a)$, $Y(a)$, and $Z(a)$ which are, respectively, the susceptible, the infected but not infectious (exposed), the infectious and the immune individuals, distributed according to age a. The steady state portrait is given by the set of differential equations

$$\begin{aligned} \frac{d}{da}X(a) &= -\left[\lambda(a) + \nu(a) + \mu\right] X(a), \\ \frac{d}{da}H(a) &= \lambda(a)X(a) - (\sigma + \mu)\, H(a), \end{aligned} \tag{1}$$

$$\begin{aligned}\frac{d}{da}Y(a) &= \sigma H(a) - (\gamma+\mu)Y(a),\\ \frac{d}{da}Z(a) &= \nu(a)X(a) + \gamma Y(a) - \mu Z(a),\end{aligned} \qquad (1)(\text{cont.})$$

where $\nu(a)$ is the vaccination rate, σ^{-1} and γ^{-1} are, respectively, the average incubation and recovery periods, μ is the natural mortality rate,

$$\lambda(a) = \int_0^L \beta(a,a')\,Y(a')\,da' \qquad (2)$$

is the force of infection with $\beta(a,a')$ being the age-structured contact rate among susceptible and infectious individuals, and L is the upper limit for the duration of human life. Here a constant mortality rate for all ages is used to describe the age distribution of developing countries [8], but the age distribution of developed countries is easily approximated by setting zero to the mortality rate up to the life expectancy and infinite thereafter [12].

Let us consider that all new-borns be susceptible, since the effect of maternally derived antibodies is not considered. Additionally, the loss of immunity [14] is not taken into account. Under these simplifications, system (1) is, then, submitted to the following initial conditions

$$\begin{aligned}X(0) &= X_b,\\ H(0) &= 0,\\ Y(0) &= 0,\\ Z(0) &= 0,\end{aligned} \qquad (3)$$

where X_b is the new-born rate.

The solutions for the first three equations of system (1), using the initial conditions given by (3), are

$$\begin{aligned}X(a) &= X_b e^{-[\mu a+\Lambda(a)+N(a)]},\\ H(a) &= X_b e^{-(\mu+\sigma)a}\int_0^a e^{\sigma\zeta-N(\zeta)}\lambda(\zeta)e^{-\Lambda(\zeta)}\,d\zeta,\\ Y(a) &= X_b e^{-(\mu+\gamma)a}\int_0^a \sigma e^{(\gamma-\sigma)s}ds\int_0^s e^{\sigma\zeta-N(\zeta)}\lambda(\zeta)e^{-\Lambda(\zeta)}\,d\zeta,\end{aligned}$$

where $\Lambda(\zeta)$ and $N(\zeta)$ are given by

$$\begin{aligned}\Lambda(\zeta) &= \int_0^\zeta \lambda(t)\,dt,\\ N(\zeta) &= \int_0^\zeta \nu(t)\,dt.\end{aligned}$$

Substituting the resulting $Y(a)$ into equation (2), and changing the upper limits of the integrations, we obtain the following integral equation for the force of infection

$$\lambda(a) = \int_0^L \beta(a,a')\,X_b e^{-(\mu+\gamma)a'}da'\int_0^L \sigma e^{(\gamma-\sigma)s}\theta(a'-s)\,ds\int_0^L e^{\sigma\zeta-N(\zeta)}\lambda(\zeta)e^{-\Lambda(\zeta)}\theta(s-\zeta)\,d\zeta, \qquad (4)$$

where $\theta(x)$ is the step or Heaviside function. An advantage in changing the upper limits of the integrations relies on letting L to be sufficiently high, and then, neglecting the expressions depending on L.

Equation (4) for the force of infection can be rewritten, by interchanging the integrations, as

$$\lambda(a) = \int_0^L B'(a,\zeta)\lambda(\zeta)e^{-\Lambda(\zeta)}\,d\zeta, \tag{5}$$

where the kernel $B'(a,\zeta)$ is given by

$$B'(a,\zeta) = e^{-N(\zeta)}X_b\sigma \int_\zeta^L e^{-\sigma(s-\zeta)}e^{\gamma s}\,ds \int_s^L \beta(a,a')\,e^{-(\mu+\gamma)a'}\,da'. \tag{6}$$

Equation (5) is a Hammerstein equation [15].

As can be seen, the force of infection, given by the definition (2), is proportional to the number of infectious individuals. Nevertheless, to produce new infections there must have encounters of infectious individuals with susceptible individuals and, then, the infectious agent must pass to the latters from these encounters. Hence, the first feature of the contact rate is related to the pattern of contacts, i.e., the possible encounter of susceptible individuals with infectious individuals. The other is the transmissibility (infectivity) of the infectious agent during this contact. Based on these two features, we can construct an age-structured contact rate $\beta(a,a')$ and, then, the kernel, given by equation (6), can be specified.

2.1. An Age-Structured Pattern of Contacts

To develop a pattern of contacts, two basic assumptions are considered throughout this subsection. One is related to the fact that susceptible and infectious individuals are homogeneously mixed in the community, and the other, to the random encounters among all individuals in the community.

By the term pattern of contacts we mean the contacts among susceptible and infectious individuals which can lead to a new infection. Since there is no kind of available data on contact rate specified by age, it is not possible to obtain kind of statistical estimate. Therefore, there is not a completely specified (known) contact rate to be applicable directly in a model. On the other hand, however, the counts of contacts made by individuals of different ages can be achieved by considering the demographic distribution of the population and the social rules which characterize the relations among individuals. By doing this, it is possible to retrieve indirectly the information about the contact patterns by considering an appropriate model. For this reason, we would like to express a pattern of contacts in a community by the mean of probability theory which takes into account the effective contact among individuals.

Let us consider a probability function, denoted by $P_c(a,a')$, which relates all sufficiently close contacts made among susceptible (with age between a and $a+da$) and infectious (with age between a' and $a'+da'$) individuals. We will develop this effective probability function which describes the pattern of contacts by stochastic processes considerations. In this paper, we will deal with a very simple hypothesis about the pattern of contacts. First, we develop a probability density which relates the closely (potentially infective) contacts made by individuals, disregarding their health status, taking into account the demographic distribution. Thereafter, we develop a probability density which describes the infective contact made by infectious and susceptible individuals regarding to the social rules.

First, the probability of closely contacts among all individuals in the community irrespective of their health status, called $\pi(a)$, is treated. This probability can be obtained in two steps.

Let us consider the random variable Z as the age of occurring closely contacts of an individual with others (it is assumed that the contacts made during the birth, $a = 0$, can be disregarded). Suppose that the probability of contacts among individuals are independent of their age, and also, independent of the density of the individuals at a given age. Then, the distribution of the random variable Z follows an exponential distribution, me^{-ma}, where m is the general contact rate (dimension of time^{-1}). Therefore, the probability of k closely contacts during the semiopen age interval (0,a] is given by

$$p_k(a) = \frac{e^{-ma}(ma)^k}{k!}, \qquad \text{with } k = 0, 1, 2, \ldots, \tag{7}$$

which is the Poisson probability distribution.

The Poisson distribution, given by equation (7), was obtained by taking into account the demographic distribution. For this reason, if one individual has much more possibility of contact with others, as in urban areas, then the general contact rate must assume higher values. Conversely, if rural areas are dealt with, then the general contact rate must take lower values. On the other hand, the demographic distribution is related to how the individuals are occupying the space. This geo-distribution of the individuals shows that individuals who are living closely has higher number of contacts during a fixed period of time than the individuals who are living dispersed. In another words, if we consider the number of contacts made by individuals, instead of fixing the period of observation time, then a small period of time is spent to occur a fixed number of contacts for a highly concentrated population than overdispersed population. Calling this period of time as the togetherness period b_2 (dimension of time), then it is reasonable to relate inversely this parameter with the general contact rate, that is, $m = 1/b_2$. The contact rate m (with respect to age) is, then, related to period b_2 (with respect to time), because during this time interval, we must have an increasing in age given by the same period of time considered. By doing this, the togetherness parameter lets the contacts among individuals of different communities be appropriately described by the same Poisson process by shrinking (or expanding) the scale of the aging process.

However, the number of closely contacts is not enough to describe the potentially infective event. Note that some infectious diseases, like influenza, require much less number of closely contacts to infect a susceptible individual than others, like measles. For this reason, let us consider the period of time elapsed among closely contacts made by individuals. The probability density of A_{b_1+1}, the age elapsed from the birth until the occurrence of the $(b_1+1)^{\text{th}}$ closely contact, with $b_1 \geq 0$, can be calculated. The distribution of $A_{b_1+1} > a$ follows the same probability distribution as the random variable $Z_1 + \cdots + Z_{b_1+1}$, where $Z_{j+1} - Z_j$ is the period of time elapsed between closely contacts. This distribution can be obtained by [16]

$$P(A_{b_1+1} > a) \equiv P[k < b_1 + 1] = \sum_{i=0}^{b_1} p_k(a),$$

which is calculated using Poisson probability distribution, given by equation (7), with mean a/b_2. Then A_{b_1+1} has the probability density

$$g(a; b_1, b_2) = \frac{1}{b_2(b_1!)} \left(\frac{a}{b_2}\right)^{b_1} e^{-a/b_2} \tag{8}$$

which is the so called gamma probability density [17].

The probability distribution (8) was obtained assuming the Poisson process with b_1 closely contacts until age a. Let us now relate this parameter with the average number of closely contacts needed to generate, successfully, a new infection, if an individual of the community

enters in contact with infectious individual. Then, the distribution (9) can be slightly modified as

$$\pi(a) = \frac{1}{b_2\Gamma(b_1+1)}\left(\frac{a}{b_2}\right)^{b_1} e^{-a/b_2}, \tag{9}$$

where $\Gamma(x)$ is the gamma function, to related the contacts occurring in the community taking into account the demographic distribution. The distribution (10) assumes an average number b_1 (not necessarily integer) of closely contacts occurred during an age interval $(0, a]$.

Second, the probability density of contacts among susceptible and infectious individuals, $\zeta(a, a')$, is considered. This probability density is determined by assuming a Poisson distribution and is based on the social rules.

Let us considered an infective event, that is, a susceptible individual of age a contacting, after an average of b_1 closely contacts, an infectious individual of age a'. We define the random variables Z and Z' being related to ages a and a', respectively, when the encounter have occurred. Observe that the probability of contact between susceptible individual of age a and infectious individual of age a' must equal the probability of contact between susceptible individual of age a' and infectious individual of age a, from the symmetry of mixing processes. For this reason, we assume that both random variables refer to a Poisson process with same distribution parameter, resulting in the exponential distribution $b_3 e^{-b_3 a}$, where b_3 is the infective contact rate (dimension of time^{-1}). Hence, the probability density of contacts among susceptible and infectious individuals, called $\zeta(a, a')$, is described by the random variable $Y = Z - Z'$, which follows the Laplace probability density [16],

$$\zeta(a, a') = \frac{b_3}{2} e^{-b_3|a-a'|}, \tag{10}$$

with the properties: mean value a' (or a, because both vary) and standard deviation $\sqrt{2}/b_3$. From the fact that the susceptible individuals reached the age a after b_1 contacts, hence, the distribution density (10) is conditioned to those made by susceptible individuals among all contacts.

In the former case, it was developed the pattern of contacts among all individuals in the community irrespective of their health status. Now, the probability distribution given by equation (10) describes the preferential route of infection, that is, the contacts among susceptible and infectious individuals follow a privilege encounter, i.e., at same age $a = a'$, $\zeta(a, a')$ assumes the highest value. This interpretation was corroborated by the observation that the introduction of one primary case generated several localized epidemics [18], that is, the primary case was a nurse who infected the children in the baby nursery, and those infectious children, in turn, infected other children of the same age in their home and neighbors. This kind of rule of social obligations (children at school, for instance) may be obeyed by young and adult individuals [19].

Finally, we will join the above two distributions to obtain the effective probability function $P_c(a, a')$. Observe that $\pi(a)$ is the $(b_1+1)^{\text{th}}$ closely contact occurring at age a, and $\zeta(a, a')$ is the probability of this aged individual contacting an infectious individual with age a'. However, $\pi(a)$ does not consider the health status. To be potentially infective, the contact among all individuals must be done between susceptible and infective individuals. Suppose that a fraction $\chi(a)$ of $\pi(a)$ is regarded to the contacts made by the susceptible individuals. Hence, the effective probability function can be written as

$$P_c(a, a') = \pi(a)\chi(a)\zeta(a, a'), \tag{11}$$

where $\pi(a)\chi(a)$ represents the contacts made by an susceptible individual.

The unknown fraction $\chi(a)$ in the effective probability function $P_c(a, a')$ can be determined as follows. Note that the total number of contacts of susceptible individuals of age a with infectious individuals of age a' must equal the total number of contacts of susceptible individuals of age a'

with infectious individuals of age a. This conservation of contacts can be achieved if we have

$$\int_{a'=0}^{L}\int_{a=0}^{L}\zeta(a,a')\,\pi(a)\chi(a)\,da\,da'=1. \tag{12}$$

Observe that $\int_0^\infty \pi(a)\,da = 1$ and $\int_{-\infty}^{\infty}\zeta(a,a')\,da' = 1$. However, we are dealing with variable which is a nonnegative age. Hence, if we let $L \to \infty$, which is reasonable due to the quick decaying of both probability densities, then the Laplace distribution (10) must be normalized as $\int_0^\infty \zeta(a,a')\,da' = 1$. The above expression follows if $\chi(a) = 2/\left(2 - e^{-b_3 a}\right)$. This expression, being the contacts made by susceptible individuals, is a decreasing function of age as expected.

Summarizing the above results, the pattern of contacts in a community can be described by the probability function

$$P_c(a,a') = f_1(a)e^{-b_3|a-a'|}, \tag{13}$$

where the function $f_1(a)$ (dimension of time^{-2}) is

$$f_1(a) = \frac{b_3}{b_2\Gamma(b_1+1)}\frac{(a/b_2)^{b_1}e^{-(a/b_2)}}{2-e^{-b_3 a}}. \tag{14}$$

Hence, the infection propagates following probabilistic events, that is, a new infection can occur according to the pattern of contacts of susceptible individuals with age between a and $a+da$ and infectious individuals with age between a' and $a'+da$ according to the probability function (13).

2.2. An Age-Structured Model

Up to now an age-structured pattern of contacts was described by the mean of probability function. Another aspect to be considered is the transmissibility of the infectious agent [20]. When there are contacts, the occurrence of a new infection will depend on the transmissibility of the virus from infectious to susceptible individuals. The contact rate must capture both features.

Assuming that the effective age-structured contact rate is proportional to the effective probability function (pattern of contacts), and setting the infectivity of the virus as the constant of proportionality, then the contact rate takes the form

$$\beta(a,a') = \beta_0 f_1(a)e^{-b_3|a-a'|}, \tag{15}$$

where the period of exposure β_0 (dimension of time) encompasses the infectivity of virus. This period is the average period of time that a virus circulates in the environs until reaches a susceptible individual.

In this section, we can calculate the kernel, given by equation (6), by considering the effective age-structured contact rate, given by equation (15), developed in the preceding section. The first integration results in

$$\int_s^L \beta(a,a')\,e^{-(\mu+\gamma)a'}\,da' = \beta_0 f_1(a)\left\{\frac{e^{b_3 a}e^{-(\mu+\gamma+b_3)s}}{\mu+\gamma+b_3}\theta(s-a) + \left[\frac{e^{-b_3 a}e^{-(\mu+\gamma-b_3)s}}{\mu+\gamma-b_3} + \frac{2b_3 e^{-(\mu+\gamma)a}}{(\mu+\gamma)^2-(b_3)^2}\right]\theta(a-s)\right\},$$

neglecting the exponentials depending on the upper limit of integration (L). The next integration, that is, the previous solution multiplied by $e^{-\sigma(s-\zeta)}e^{\gamma s}$ and integrated to s from ζ to L, depends on the relative position between ζ and a. If $\zeta > a$ then we have

$$\int_\zeta^L e^{-\sigma(s-\zeta)}e^{\gamma s}\,ds\int_s^L \beta(a,a')\,e^{-(\mu+\gamma)a'}\,da' = \beta_0 f_1(a)\int_\zeta^L e^{-\sigma(s-\zeta)}e^{\gamma s}\frac{e^{b_3 a}e^{-(\mu+\gamma+b_3)s}}{\mu+\gamma+b_3}\,ds,$$

and if $\zeta < a$ then we have

$$\int_{\zeta}^{L} e^{-\sigma(s-\zeta)}e^{\gamma s}\,ds \int_{s}^{L} \beta\,(a,a')\,e^{-(\mu+\gamma)a'}\,da' = \beta_0 f_1(a)\left\{\int_{a}^{L} e^{-\sigma(s-\zeta)}e^{\gamma s}\frac{e^{b_3 a}e^{-(\mu+\gamma+b_3)s}}{\mu+\gamma+b_3}\,ds + \int_{\zeta}^{a} e^{-\sigma(s-\zeta)}e^{\gamma s}\left[\frac{e^{-b_3 a}e^{-(\mu+\gamma-b_3)s}}{\mu+\gamma-b_3} + \frac{2b_3 e^{-(\mu+\gamma)a}}{(\mu+\gamma)^2-(b_3)^2}\right]ds\right\}.$$

Therefore, the resulting kernel is a $C_2\,[0,L]$ function (two times continuously differentiable), given by

$$B'(a,\zeta) = \beta B(a,\zeta)e^{-N(\zeta)}, \tag{16}$$

where

$$\beta = \beta_0 X_b \tag{17}$$

is the transmission coefficient, a dimensionless parameter. The quasikernel $B(a,\zeta)$ is given by

$$B(a,\zeta) = f_1(a)\,[f_2(a,\zeta)\theta(\zeta-a) + f_3(a,\zeta)\theta(a-\zeta)]\,, \tag{18}$$

where the auxiliary functions $f_2(a,\zeta)$ and $f_3(a,\zeta)$ are given by

$$f_2(a,\zeta) = \frac{\sigma e^{-\mu\zeta}e^{-b_3(\zeta-a)}}{(\mu+\gamma+b_3)\,(\mu+\sigma+b_3)} \tag{19}$$

and

$$f_3(a,\zeta) = \frac{\sigma e^{-\mu\zeta}e^{-b_3(a-\zeta)}}{(\mu+\gamma-b_3)\,(\mu+\sigma-b_3)} - \frac{2\sigma b_3 e^{-\mu a}}{\left[(\mu+\gamma)^2-b_3^2\right](\sigma-\gamma)}\left\{e^{-\gamma(a-\zeta)} - \frac{\left[(\mu+\sigma)^2-b_3^2-(\sigma-\gamma)\,(2\mu+\gamma+\sigma)\right]e^{-\sigma(a-\zeta)}}{\left[(\mu+\sigma)^2-b_3^2\right]}\right\}. \tag{20}$$

This kernel is called quasikernel due to the fact that the terms β and $e^{-N(\zeta)}$ were extracted from $B'(a,\zeta)$. Observe, again, that the exponentials depending on the upper bound of the integration (L) do not appear in the functions (19) and (20) because they are negligible compared to the other values. Also, we have $f_2(a,a) = f_3(a,a)$, as required.

The Hammerstein equation (5) in terms of the quasikernel (18) can be rewritten as

$$\lambda(a) = \beta\int_{0}^{L} B(a,\zeta)e^{-N(\zeta)}\lambda(\zeta)e^{-\Lambda(\zeta)}\,d\zeta. \tag{21}$$

This equation provides with analysis of situations with and without vaccination strategy. When there is not vaccination strategy, then $\beta B(a,\zeta)$ is the kernel.

The integral equation (21) describes the steady state of directly transmitted infections in different communities. Each childhood disease has its own incubation σ and recovery γ rates, and period of exposure β_0. Different communities can be described by their own population size given by new-borns rate X_b and pattern of contacts given by average number of effective contacts b_1, togetherness period b_2 and infective contact rate b_3. Therefore, this model shows a wide range of applications, whenever the unknown parameters could be estimated.

3. DISCUSSION

The application of mathematical models is increasingly proving its usefulness as a tool for the understanding of epidemiological problems particularly those related to control and/or eradication strategies of infectious diseases.

With the intention of creating a wide applicable method, an age-structured contact rate model was developed for the directly transmitted infections. This approach considering stochastic considerations for the age-structured pattern of contacts resulted in a model with only a few parameters. This approach, like the method of Anderson and May [4], is applicable to seroprevalence data. When considerations due to Tudor [10] are taken into account, then the present approach is an improvement of the WAIFW method since it uses only four parameters (of gamma and Laplace distributions plus virus transmissibility).

Observe that an age-structured pattern of contacts was developed taking into account the demographic distribution and social rules. From the former we obtained a probability which describes the contacts among all individuals irrespective of their health status. Also, a probability which describes the contacts among susceptible and infectious individuals was obtained by considering assumptions of symmetry. This distribution showed that there is a preferential infective contact when the susceptible and infectious individuals have same age, which describes roughly the social obligations. The effective distribution of contacts was completely determined considering assumption of conservation of contacts.

In this paper, we presented only theoretical development of an age-structured model. We would like to stress that Greenhalgh [21] and Inaba [22] provided a useful tool to analyze age-structured modeling, but they failed to give a practical example due to lack of an age-structured contact rate. Indeed, in [21], although presented an analysis tool of an age-structured contact model, Greenhalgh considered a constant contact rate application. For this reason, in a further paper [11], a model considering the age-structured pattern of contacts developed here is analyzed regarded to the epidemiological parameters, which are the force of infection and its correlated variables. A preliminary analysis was already done by Coutinho *et al.* [23].

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