**Journal of Computer and Mathematical Sciences,** *Vol.10(1),146-150 January 2019* *ISSN 0976-5727 (Print)*
(An International Research Journal), *www.compmath-journal.org* *ISSN 2319-8133 (Online)*

# Spread of Contagious Diseases in Heterogenous Population

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(Received on: January 10, 2018)

**ABSTRACT**

In this paper we discuss in detail about the model of the invasion and spread of diseases in heterogeneous environments. This is organized in the form such that it contains the heterogeneous population structured into homogeneous subgroups, with the application modeling TB and HIV epidemics, then give a new approach to analyze epidemics in well-mixed populations in which individual-level variations in infectiousness is represented by a distributed reproductive number in particular the expected number of secondary cases due to each individual is drawn from a gamma distribution, yielding a negative binomial distribution after stochastisity in transmission is taken into account.

**Keywords:** Reproduction number, Tuberculosis (TB), Gamma Distribution, Human immunodeficiency virus (HIV).

## INTRODUCTION

Population heterogeneity can be given by dividing the host population into homogeneous subgroups based on spatial location, behavior, genetic of other factors. Analysis of basic epidemic properties in such multi-group or multi type populations is well developed.

Heterogeneous populations with homogeneous subgroups can broadly be divided into those with and those without inter-group transition on time scales relevant to the analysis. Another important distinction is whether transmission occurs among individuals in different groups or only among individuals within the same group. These two criteria define a basic taxonomy of multi-group disease models. For example a population may be structures into groups according to some unchanging social categorization (here no transitions among groups)

or all individual are potentially able to interact with another but interaction rates are much greater within than among groups.

## INTERCONNECTED HOMOGENEOUS SUBGROUPS IN TB AND HIV

Let us consider a critical source of heterogeneity in a population challenged into HIV is the presence of other disease that can act as cofactors, particularly veneral disease that enhance opportunities for the spread of HIV. Conversely because it destroys immunity, HIV infection can have dramatic impacts on host response to other disease, particularly the so-called opportunistic infection, one of the most important of which is tuberculosis (TB). For many years TB-infected individual the infection remains latent and has little effect on their health, but infectious become acute and deadly once individuals are immune compromised. From the TB epidemic point of view the course of the disease is going to be vastly different in individuals infected with I|HIV than in otherwise healthy individuals. When we consider the interaction of these two epidemics, we can formulate a full TB-HIV model in a homogeneous population where we include the transmission and progression dynamics of both diseases.

We can begin by modeling TB in a population in which a certain proportion of individuals have HIV without explicitly modeling the HIV epidemic itself. This approach is justified by the fact that the impact of TB on the epidemiology of HIV appears to be less dramatic than that of HIV on TB. Under these assumptions, the background HIV structure is not static because we allow individuals in different HIV groups to progress according to the WHO disease staying system of disease. The essential simplification is that we model the transition of susceptible to HIV stage I using a recruitment process, based on historical patterns obtained for empirical data rather than modeling HIV transmission in detail. We have a TB only epidemic model embedded within a population that is heterogeneous with respect to HIV stages.

Because one of our motivations for developing a TB-HIV model is to dete3rmine the potential impact of reducing treatment duration in TB infected patients in some areas of high HIV prevalence, the model outlines below has a strong focus on TB treatment classes progressing over a monthly time scale.

Specifically individuals newly infected with TB can progress to active disease at a slow or fast rate and those with an active infection  positive or negative. Among those individuals with an active infection a certain fraction can be detected and placed on treatment. Others are undetectable and will never become treated, while some can recover completely without ever being treated. At each time step individuals within the detectable category are placed on treatment at a given rate and can enter either a DOTS or non-DOTS regimen. (DOTS stands for Directly Observed Treatment Short-course.) Treated individuals can recover transiently or completely or relapse to active disease. Flows among these categories were chosen to reflect the most critical processes determining TB incidence, prevalence and mortality under different treatment regimens paying particular attention to case detection, default, relapse and re-infection.

**HETEROGENEITY IN WELL-MIXED POPULATIONS**

We have for above two decades made analysis of epidemic dynamics based on the basic reproduction number $R_0$ which is the expected number of new infections due to each infectious individual in a wholly susceptible population. Models of homogeneous population usually use a point estimate $O + R_0$ implicitly assuming that every individual has the same degree of infectiousness. But the infectiousness of each individual varies due to a complex blend of host, pathogen and environment factors. The factors lead to continuous variation in infectiousness and distinct risk groups often cannot be recognized prior, there by hampering our ability to represent this heterogeneity using group structure.

At the population level γ has some probability distribution that can be fitted to database describing the observed distribution that can be fitted to datasets describing the observed distribution of secondary cases caused by each individual. The actual number of cases caused by each individual will vary stochastically around γ, so that the offspring distribution will be a compound distribution with firm Poisson (γ). In a completely homogeneous population where all the individuals have identical infectiousness $\gamma = R_0$, the offspring distribution will be Poisson. If all the individual transmit the same rate and exponentially distributed infectious periods then μ is distributed exponentially with mean $R_0$ and the offspring distribution is geometric.

To allow for a more flexible degree of individual variation in infectiousness. We propose a model with a gamma-distribution γ. Gamma distribution are a useful two-parameter family of distributions for forming epidemic theory in heterogeneous populations, since they are uni-model and non-zero only on (0,∞). More important for the development of branching process theory, the offspring distribution arising from a population with gamma distributed individual reproductive number γ is a negative binomial distribution. This model is a generalization of the two conventional models namely Poisson and geometric offspring distribution as given below.

The negative binomial distribution is typically expresses in terms of a scale parameter $p$ and $a$ dispersion parameter $K$. We deviate from this practice and use the notation $\phi_{R_{0,K}}^{N\beta}$ to denote a negative binomial offspring distribution with mean $R_0$ related to $p$ and $x$ by the equation

$$R_0 = K\left(\frac{1}{p} - 1\right) \tag{1}$$

The probability generating function for negative binomial distribution has the form

$$\phi_{R_{0,K}}^{N\beta} : g\ x\ = \left(1 + \frac{R_0}{K}\ 1 - z\ \right)^{-K} \tag{2}$$

The probability $R_0$ that an infectious individual in the parent generation will not transmit to anyone in the offspring generation in $g\ 0$ from which it follows for the negative binomial that

$$q_0 = \left(1 + \frac{R_0}{K}\right)^{-K} \tag{3}$$

The Poisson and geometric distributions are special cases of the negative binomial distribution with

$K \to \infty$ and $K = 1$ respectively.

For example, in the outbreak of SARS in Singapore in 2003, maximum likelihood methods that can be used to find parameters of the negative binomial, Poisson and geometric distributions that best fit these data of SARS evasion in Singapore.

But because of its extra parameter the negative binomial distribution will always provide a better fit for the SARS outbreak. But improvement in fit over the one-parameter Poisson or geometric models is sufficient to meet inclusion of the extra parameter.

A large degree of individual variation in infectiousness is evident in all most all of the datasets of SARS in Singapore (2003) $N = 57$, SARS in Beijing (2003) $N = 33$, Measles in US (1977-79) $N = 165$, Measles in Canada (1998-2001) $N = 49$, Smallpox in Europe (1958-73) $N = 32$, Smallpox in Benin (1967) $N = 25$, Smallpox in Pakistan $N = 17$, Variola minor in England (1990) $N = 25$, Monkey pox in Zaire (1980-84) $N = 147$, Pneumonic plague 6 outbreaks $N = 74$, Avian influenza in S.E. Asia (2004) $N = 33$, Rubella in Hawaii (1970) $N = 19$, Hanta virus in Argentina (1966) $N = 20$, Ebola HF in Uganda (2000) $N = 13$. For five of the disease data sets, the 90% confidence intervals for the negative binomial dispersion parameter $K$ are bounded below 1, indicating that only the negative binomial offspring distribution can represent the observed patterns. The best estimate of $K$ for 11 of the datasets suggests that heterogeneity is either greater or much greater than that arising from an exponential distribution of infectiousness (is best fit $K < 1$ ).

In half of the above mentioned epidemic the host estimate for $K$ was $K < 1/3$ . The estimated value of $R_0$ are also quite low. The low values of $K$, together with relatively low values of $R_0$ suggest that the great majority of individuals are unlikely to infect any other individual. For example, if $K = 1/3$ then from the equation $q_0 = \left(1 + \frac{R_0}{K}\right)^{-K}$, it follow that for each individual to have a greater than 50% chance of infecting another individual $R_0$ would have to exceed

$$R_0 > \frac{2^3 - 1}{3} = 2.33$$

which is outside the 90% confidence interval for all the datasets analyzed except for smallpox in Europe in the period 1958 to 1973.

Similarly our method allow us to estimate that 73% of SARS cause in Singapore were below that critical infectious level of $\gamma = 1$, which only 6% had infectiousness of $\gamma > 8$.

## CONCLUSION

This result is consistent with field reports from SARS afflicted regions indicating that infectiousness is highly over dispersed, but contrast sharply with many publishes SARS models that do not take heterogeneity into account.

Also we present that the assumption of heterogeneity over simply the analysis of most epidemic to the point of $R_0$ alone do not adequately describe disease dynamics.

Much work remains to be done to provide a more coherent theory on the spread of disease in heterogeneous populations.

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