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Basic Reproduction Number of Infectious Disease in Heterogeneous Environment: A Scoping Review

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Abstract. This paper overviews the recent advances how spatial-temporal heterogeneity affects the spread of epidemic diseases from the perspective of basic reproduction number. First part focuses upon the theoretical studies and applications of seasonal effects on infectious invasion. Then the investigations are introduced to demonstrate the role of spatial structure and spatial contact patterns in the prevention of disease outbreaks. Suggestions are presented for potential future researches.

AMS subject classifications: 92D30, 92C60

Key words: Epidemic, threshold, fluctuation, diffusion, stability.

1 Introduction

The basic reproduction number (R_0) is the most important quantity in epidemiology. It is defined as the expected number of second infections by a typical infectious individual during its infection period. Intuitively, a disease expands if its reproduction number is greater than unity and declines if it is less than unity. Therefore, it clearly describes a disease transmission risk and guides the control policy from public health officials, as indicated in the design of nonpharmaceutical public health interventions during pandemic COVID-19 [43, 56]. Furthermore, R_0 is also an important parameter to determine the minimal vaccination fraction p to achieve a herd immunity in a mass vaccination campaign because of the well known formula [9, 35]

$$p = 1 - \frac{1}{R_0}.$$

The concept of basic reproduction number (or ratio) was first rigorously defined by paper [19], where R_0 is identified as the spectral radius of next generation operator. (The

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earlier contributions to form the key concept can be found in paper [32].) This gives the fundamental framework to compute the quantity for mathematical models with heterogenous populations. For epidemic models in autonomous ordinary differential equations (ODE), paper [70] formulated a beautiful computation formula

$$R_0 = \rho(FV^{-1}),$$

where ρ means the spectral radius, F is the matrix of disease transmission rate at a disease-free equilibrium and V^{-1} is the matrix for the expected infection duration of an infected agent near the disease-free state. Equivalently, V is used to describe the decay rates and transition rates among infection compartments for infectious individuals. If $S(a)$ is the probability that an infectious individual remains in an infection compartment at infection time a, it was shown in [34] that the basic reproduction number can be expressed as

$$R_0 = \int_0^\infty F(a)S(a)da.$$

Paper [16] delineated the complexity in selecting the new infection rate matrix, which links closely to the interpretations of "infective" agent. This ambiguity in fixing infectious compartments for multiple hosts and multiple transmission routes could be avoided by type reproduction number, which focuses upon a specific host type and is the average number of secondary cases of that type produced by the primary cases of the same host type during the entire course of infection [33, 39, 62]. In particular, the type reproduction number is useful when control targets a specific type, and is generalized to target reproduction number [45, 64].

The previous mathematical theories are powerful for analyzing the invasion threshold of epidemic disease in homogenous conditions. Note that environmental fluctuation in time and spatial heterogeneity are ubiquitous and can shape infection dynamics. For example, seasonally varying contact rates in measles result from the closure and opening of schools [20]. Fluctuations in birth rates or seasonal vaccinations are also common [21, 50]. For vector-born diseases, biting rate and transmission probability are critically dependent on temperature which varies seasonally [53]. More biological details of seasonal influences on infectious diseases can be found in review papers [7,28] and the main advances in mathematical modeling are described in [11]. For spatial epidemic model with regional scale, geographic heterogeneity arises from mountains, rivers and cities which affect population mobility and disease transmissions [5, 10, 12, 15, 35, 44, 60, 61, 63]. Therefore, it is important to reveal how temporal fluctuations and spatial variations influence the spread of epidemic disease. The objective of this paper is to select some papers from recent advances to show how R_0 is affected by temporal-spatial heterogeneity. The next section considers the epidemic models with the fluctuating coefficients in time. Section 3 focuses upon the models with spatial variations. The last section highlights future research problems.