## Modelling the Effect of Human Heterogeneity on Infectious Disease Transmission Dynamics

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**Abstract.** Human heterogeneity is a critical issue in infectious disease transmission dynamics modelling, and it has recently received much attention in COVID-19 studies. In this article, a general human heterogeneous disease model with mutation is proposed to comprehensively study the effects of human heterogeneity on basic reproduction number, final epidemic size and herd immunity. We show that human heterogeneity may increase or decrease herd immunity level, strongly depending on some convexity of the heterogeneity function, which gives new insights and extends the results in [Britton *et al.*, Science, 369:846–849, 2020]. Moreover, human heterogeneity may decrease the basic reproduction number but increase the level of herd immunity, implying the unreliability of the basic reproduction number in characterizing the spread and control of infectious diseases with human heterogeneity.

AMS subject classifications: 35J55, 35B32

**Key words**: SEIR model, human heterogeneity, basic reproduction number, herd immunity level, final epidemic size.

## 1 Introduction

Human heterogeneity is ubiquitous and shares tremendous popularity in the study of social science [39] and epidemiology [4–9, 13–17, 19, 23–25, 28, 30, 37]. There are many heterogeneities in human societies that will influence virus transmission, such as social activity level, age structure, incubation period, individual susceptibility or exposure to infection. Recently, the effects of human heterogeneities on infectious disease are back to spotlight in modeling and precise control of COVID-19 spread. Questions of interest

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to mathematical epidemiologists and public health members are how the herd immunity level is affected by human heterogeneities such as susceptibility and infectivity [7,19], the limitations of basic reproduction number and what human heterogeneity can tell [31], why final epidemic size of COVID-19 is so different from the classic SIR model, and why ODE models might fail in modelling COVID-19 and how human heterogeneity shapes the evolution of infectious disease [20]. These questions imply the necessity to incorporate human heterogeneities in disease models to profoundly understand the transmission mechanism of infectious diseases.

Many studies have shown that under certain conditions, human heterogeneities will decrease herd immunity level [6,7,19,25]. It is found in [6,25] that when susceptibility is the only variable property, the final size of the epidemic is always smaller for a heterogeneous population than for a homogeneous population with the same reproduction number. A recent study on COVID-19 showed that population heterogeneity can significantly impact disease-induced immunity [19]. They estimated that if the basic reproduction number  $\mathcal{R}_0 = 2.5$  in an age-structured community with mixing rates adapted to social activity, then the disease-induced herd immunity level can be around 43%, which is substantially less than the classical herd immunity level of 60% obtained through homogeneous immunization of the population. Another study [7] showed that if the coefficient of heterogeneity variation increases from 0 to 4, the herd immunity threshold decreases from greater than 60% to less than 10%.

However, some researchers is probably impossible even with vaccination efforts in full force and the theoretical threshold for vanquishing COVID-19 looks to be out of reach, due to the reasons such as vaccination-induced human behaviour change and mutation of viruses [2]. The effects of human heterogeneity on the spread of infectious disease are comprehensive and repay many more studies from theoretical insights. In this paper, we will consider some SEIR model with mutation and heterogeneous populations, in order to investigate the effect of human heterogeneities such as activity level, susceptibility, exposure to infection on basic reproduction number, final epidemic size and herd immunity.

The rest of the paper is organized as follows. In Section 2, we will introduce the general human heterogeneous SEIR disease model with mutation, define the basic reproduction number, and investigate the threshold dynamics. The effect of human heterogeneities on disease persistence, herd immunity threshold and final epidemic size will be explored in Section 3. Applications to COVID-19 can be found in Section 4. Discussion and conclusion will be in Section 5.

## 2 A general disease model of human heterogeneities

The population is divided into four classes: susceptible (S), exposed (E, latently infected without symptoms), infected (I, infected with symptoms) and removed (R, recovered or disease-induced death). The susceptible individuals are infected by exposed and infec-