POPULATION DYNAMICS MODEL FOR THE EFFECT OF ISOLATION ON FINAL EPIDEMIC SIZE

ISHFAQ AHMAD
B9ID1501

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Tohoku University, Sendai, Japan

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CANDIDATE
ISHFAQ AHMAD

CANDIDATE ID
B9ID1501

THESIS
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SUPERVISOR
Professor Hiromi Seno

AFFILIATION
Department of Computer and Mathematical Sciences
Graduate School of Information Sciences
Tohoku University, Sendai, Japan

DISSERTATION REFEREES
Professor Toshiyuki Sugawa
Associate Professor Naoya Fujiwara

DECLARATION

I, ISHFAQ AHMAD, proclaim that the work provided in this thesis titled, " Population Dynamics Model for the Effect of Isolation on Final Epidemic Size " is my own. Here is what I confirm:

- This work was done mainly while as a candidate for a doctoral degree.
- It has been clearly stated if any part of this thesis has previously been submitted to this university for a degree or other qualification.
- I always give the source of quotes I have taken from other sources. It is entirely my own work, apart from such quotations.
- All sources of help have been acknowledged.
- I have specified exactly what others have done and what I myself have contributed..

ISHFAQ AHMAD
Sendai, September 2022

SUMMARY

In this thesis, we examine the effect of limited isolation capacity on the final epidemic size, defined as the total number of individuals who had the experienced of the disease. Many countries have faced the difficulty from the shortage of medical resource against the outbreak of SARS-COV-2. When the medical resource is much small, how does the final epidemic size depend on the limited isolation capacity?

Our analysis on a mathematical epidemic dynamics model derives a critical value of the isolation capacity below which the isolation reaches the capacity in a finite time in the epidemic season. In such a case, the final epidemic size necessarily becomes larger than that when the isolation capacity is beyond the critical value. Further, we find that the final epidemic size could have a discontinuous jump at the critical of the isolation capacity under a condition about the epidemic dynamics. In such an epidemic dynamics, the isolation capacity below the critical value causes a drastic increase in the final epidemic size, compared to that when the capacity is beyond the critical value. Such a jump in the final epidemic size does not appear under the other condition. Then the isolation capacity below the critical value could not result in much increase in the final epidemic size, so that the existence of the critical value of the isolation capacity may be little observable.

From the viewpoint of a policy for prevention of a transmissible disease spread, the isolation of detected infectives is one of the possible choices. Our result implies the necessity of a sufficient capacity of the isolation in order that the isolation works effective to suppress the final epidemic size.

Keywords: Epidemic dynamics; Mathematical model; Ordinary differential equations; Isolation capacity; Final epidemic size

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CONTENTS

1

INTRODUCTION

1.1 INFECTIOUS DISEASE

A disease is a condition in which a part of the body is affected and does not work properly. The term infectious disease refers to the disease produced by bacteria, fungi, viruses, protozoa, and other pathogenic microorganisms. A variety of agents can transmit such infectious diseases, including animals, humans, insects, and other organisms.

Epidemiology is a branch of medicine that investigates health and disease patterns as well as the factors that influence them. In other words, epidemiology is defined as the study of the link between the disease transmission and the factors related to the spread.

Epidemiology was concerned only with the health of the human population. According to some historians, Hippocrates (460–377 B.C.E.) is considered the father of epidemiology who first explained the concept of disease transmission through the environment [23]. According to Epidemiologia Espanola, the Spanish physician De Villalba used the term "epidemiology" for the study of epidemics in 1802 [9]. Disease such as strokes and coronary heart that cannot be transmitted from one individual to another and are focal of epidemiology, these diseases are the major cause of death in the world [32].

1.1.1 *Classification of infectious diseases*

Pathogenic microorganisms are capable of causing the disease. There can be a variety of microbial pathogenic agents like bacteria, viruses, fungi, and parasites, toxic proteins. Tuberculosis and pneumonia are diseases caused by bacterial infections. Influenza is an infectious disease that is caused by a virus pathogenic agent. Dermatomycoses infection is caused by a fungus pathogenic agent. Protozoa, trematodes, and cestodes infections are caused by parasite pathogenic agents.

Transmission of a disease from one individual to another is possible either in a direct or indirect way. Physical (touching or sexual contact) is the direct way to transmit a disease from one individual to another. Sexually transmitted diseases (STD) are diseases that spread via sexual interaction. Examples are gonorrhea, and AIDS etc. While the indirect transmission occurs for a disease which is transmitted with the contact of individual to contaminated objects like blood, vomit or excreta. The diseases transmitted through indirect contacts include influenza and COVID-19.

There are different categorizations for the infectious disease, based on the route of transmission :

- **Airborne disease:** This kind of disease is transmitted through inhalation of infected air such as influenza, smallpox, measles, tuberculosis, etc.
- **Food-and waterborne disease:** The transmission of a disease occurs when food or water is contaminated with pathogens. Salmonella and stomach flu are examples of food-borne diseases, whereas, waterborne diseases include cholera.
- **Vector-borne disease:** The transmission of a disease can occur through an arthropod including a mosquito, or a mollusk such as a snail. This kind of disease is called vector-borne disease. The disease transmitted by mosquitoes includes malaria, dengue, etc.
- **Vertical transmissible disease:** When an infected mother transfers the disease to her child through the placenta, this kind of transmission is called a vertical transmission and the disease is called a vertical transmissible disease. Rubella, HIV, and hepatitis B are examples of such vertical transmissible diseases.

The transmission is distinguished into four types on the bases of modeling [29]: direct transmission when an individual contracts the pathogen from another individual; vector transmission when an individual contracts the pathogen from a vector; environmental transmission when an individual contracts the pathogen from the environment; vertical transmission when the pathogen is transmitted to the child from mother at birth.

Diseases directly transmitted from one person to the other or spread by air are generally modeled as directly transmitted. Although it is necessary to make sexual contact for sexually transmitted diseases, in the case of airborne disease, physical closeness is sufficient without touching involved. It is important to include the dynamic of the vector along with that of the infected people in order to model a vector-borne transmissible epidemic dynamics.

Typically, environmental viruses are modeled separately from the transmission of free pathogens between individuals who are in contact with these viruses. A pathogen reservoir is an area where a pathogen may survive and reproduce. Most human pathogens are transmitted by humans, and humans are the primary reservoir for such pathogens. Most animal pathogens are transmitted among vertebrates, which act as reservoirs. It is important from an epidemiological perspective that some of these pathogens transmit the disease to humans through animals. Among vertebrate animals, zoonoses are infectious diseases that spread to humans.

There may different pathways with which an infectious disease is transmitted. For example, HIV can be transmitted vertically during pregnancy to a baby from an infected mother, also through sex, or by using shared needles. Direct contact with an infected bird is the most common route of spreading Avian influenza H5N1, while the transmission from human to human occurs very rarely. In addition, there are now substantial evidences that the H5N1 virus can survive

in the environment, and the route of environmental transmission becomes increasingly relevant.

1.1.2 *Important factors in epidemic dynamics*

In epidemiology, infectious diseases are closely linked to a number of concepts. As a result of these concepts, mathematical models are constructed by including several characteristics. Nelson and Williams [30] explained the detail of those concepts used until now. Below are some of the concepts used in the modeling of infectious diseases.

- **Susceptible individual:** Susceptible individuals are those individuals who are healthy and can get the disease.
- **Exposed individual:** Exposed individuals are those individuals who are healthy and have made potentially disease-transmitting contact after being vulnerable to a disease. The individual from this class may not spread the disease.
- **Infected individual:** Infected individuals are those individuals who got the disease and can transmit it in the community. The exposed individual becomes infected once the pathogen establishes itself.
- **Recovered individual:** Recovered individuals are those individuals who have gotten the immunity against the disease and cannot be reinfected again.
- **Latent individual:** Latent individuals are those individuals who have been infected but are not able to transmit the disease to the other individuals in the community. The latent period, in such a case, is referred to the period between the infection and the emergence of transmission ability to another individual.
- **Incubation Period:** The time span between being exposed to an infectious agent and the appearance of the symptoms. The symptoms occurs when the infectious agent multiplies until it reaches a threshold necessary to cause them. Both the incubation and latent periods do not necessarily coincide. For example, flu symptoms appear one day after an individual becomes infectious.
- **Incidence:** It determines how many individuals get sick within a certain period of time. However, sometimes it also determines how much proportion of individuals gets sick within a certain period of time. Most often, it can be calculated based on the number of clinical cases.
- **Prevalence:** It determines the number of patients at a given time.
- **Disease-Induced Mortality:** It determines the proportion of individuals died by the disease per unit time.

1.1.3 *Mathematical modeling for epidemic dynamics*

Athens was struck by the plague in (430-426B.C.E), the first serious epidemic traced by historians. In Thucydides' History of the Peloponnesian War (460–400B.C.E.), the scientific historian accurately described the plague. According to him, the symptoms and number of deaths are all based on personal experience. It is still unclear which agent caused Athens' plague [31, 33]. During 165–180 CE, Egypt and the Roman Empire suffered from smallpox. There were around ten millions of deaths [2].

In Europe, the Black Death was one of the most well-known epidemics. Around 50–100 million people died as a result of the Black Death in the Mediterranean and Europe during the years 1348–1350 [2]. Yersinia pestis is the bacterium that causes different forms of plague and based on DNA evidence, it is suspected that it spread throughout Europe [17]. In the 16th century, the Aztec populations in central Mexico were badly affected by the outbreak of smallpox disease and around 35 million people died. Moreover, in the nineteenth century, many countries in Europe were affected by the Black Death. Around 20 million people died due to the influenza pandemic in the early twentieth century. In the current situation, epidemics still occur on a regular basis: for example, the severe acute respiratory syndrome of 2003, and the 2009 pandemic swine flu. Epidemics and pandemics are constantly a threat because the viruses can be mutated rapidly, and they can cross species barriers to infect humans.

Despite the long history of epidemiology, it is around 350 years since mathematics was first used to study diseases and their spread. John Graunt (1620–1674) was the first epidemiologist who described the first statistical analysis of public health problems in his book "Natural and Political Observations Made upon the Bills of Mortality" published in 1663. After that, mathematical methods were used by Daniel Bernoulli to analyze the mortality due to the smallpox disease a century later, regarded as the first epidemiological model, published in 1766 [5]. According to Bernoulli, the vaccination with a mild case of smallpox virus would reduce the deaths even if the vaccine may sometimes be fatal. Diez and Heesterbeek [12] revisited Bernoulli's approach according to the modeling with equations.

A significant contribution to the research on the cause and prevention of the disease was made by Louis Pasteur in the mid of 19th century. He developed the first vaccines against rabies and anthrax as well as from puerperal fever. The germ theory of the disease was supported by his medical discoveries. Meanwhile, Robert Koch who is the founder of modern bacteriology discovered that tuberculosis, cholera, and anthrax are caused by specific agents, and he put experimental support behind the idea that infectious diseases exist. Scientists were finally able to explain how a sickness happens in the late 1800s. The concept of transmitting a bacterial disease from an infected individual to a healthy individual is now firmly established. This contributed to the development of mathematical models for the infectious diseases.

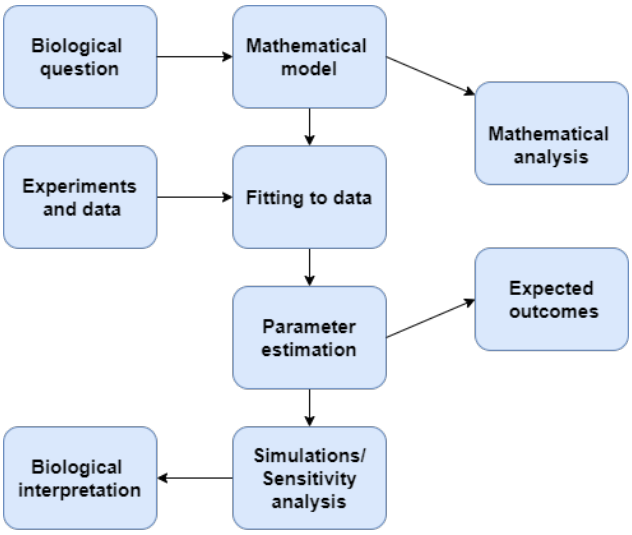

Figure 1.1: Diagram of research steps with mathematical model

During the early part of the twentieth century, William Hamer made major contributions to the mathematical modeling of infectious diseases. He wanted to know why measles recurred so frequently. However, it was Sir Ronald Ross, now regarded as a father of mathematical epidemiology, who discovered how the malaria disease can be transmitted between human and mosquito. The Nobel Prize was awarded to him for his work on the malaria in 1902. Despite his effort, he was unable to convince his contemporaries that the malaria could be eliminated by simply reducing the mosquito population. Using mathematical models to explain the malaria transmission, Ronald Ross derived a threshold quantity, which is now called the basic reproduction number [28]. In 1927, Kermack and McKendrick published a model on the spread of an infectious disease, which brought the mathematical epidemiology to the next stage [24]. In their first joint project "A contribution to the mathematical theory of epidemics", they used a deterministic epidemic model incorporating susceptible, infected, and removed populations. Part II and III of their first joint research project were published in 1932 and 1933. In 1991, their three consecutive fundamental research articles were reprinted due to their important contribution to mathematical epidemiology [25–27].

During the 1980's, the HIV outbreak brought an increase of the recognition about the importance of mathematical modeling for infectious diseases. Then, numerous models have been developed, analyzed, and employed to investigate the spread of a variety of infectious diseases. Currently, mathematical epidemiology occupies a substantial place in the research literature's. Mathematical modeling contributes significantly to public health and mathematics today [20, 21, 38].

The purpose of mathematical models is to study the relation of various components of a system as well as to predict the behavior of those components. A mathematical problem is formulated from a biological scenario as shown in Figure 1.1:

- **Dynamic verses Static model**. When a system changes its state in terms of time, it is called a dynamic model. Differential equations or difference equations are commonly used in dynamic models. In contrast, the static model focuses on a specific state established by a phenomenon, and to describe the structure of the state with the factors determining its nature. A variety of mathematical concepts have been applied, for example, the graph theory, the network dynamics, the game theory, the operation research, etc.
- **Deterministic verses stochastic model**. In the deterministic model, the transition from one variable state to the next state is uniquely determined, while the stochastic model is based on a probability theory, introducing randomness and stochastic variable states.

In order to construct a model, it is necessary to assume the disease severity. The disease can be acute or chronic. In an acute infection, pathogens are removed after a short period of time by a relatively rapid immune response. Flu, rabies, chickenpox, and rubella are examples of acute infection. The chronic infection persists for a long time (months or years), like herpes, chlamydia, etc.

The mathematical modeling of infectious disease can contribute to the discussion of how a disease can spread, the duration of the epidemic, the total number of infected, or the epidemiological indices including the basic reproductive number. The early work was done by Kermack and McKendrick in 1927 which is an origin of modern mathematical modeling of infectious diseases and has been widely applied for a variety of epidemic problems with necessary modifications [24].

1.2 KERMACK–MCKENDRICK SIR MODEL

Kermack–McKendrick introduced the mathematical model in the field of epidemiology, known today as the Kermack–McKendrick SIR model [6, 24]. The model consists of three classes, the susceptible class, the infective class, and the recovered class. To formulate a simplest version of the SIR model, we assume the followings:

- The total size of the population is constant, ignoring demographic change due to birth and death.
- The recovered individual cannot be infected again.
- The contact rate between susceptible and infective individuals is independent of the size of the total population.

Based on the above assumptions, we have the following model:

$$\begin{aligned} \frac{dS}{dt} &= -\beta \frac{I}{N} S; \\ \frac{dI}{dt} &= \beta \frac{I}{N} S - \gamma I; \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{1.1}$$

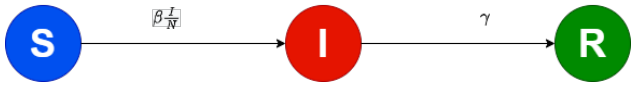

Figure 1.2: The state transition in the Kermack–McKendrick SIR epidemic model (1.1)

with the initial condition given by $S_0 > 0$, $I_0 > 0$ and $R_0 = 0$. Variables S, I, and R denote the *susceptible*, *infective* and *recovered* population sizes respectively. Every parameter is positive. The parameter γ denotes the recovery rate of infective individual. The transmission of the disease is determined by the frequency-dependent infection force with the infection coefficient β. The constant size of the total population is denoted by N, and it is satisfied that $S(t) + I(t) + R(t) = N$ for any $t \geq 0$. The state transition for the individual is shown in Figure 1.2.

1.2.1 *Properties of the model*

In the Kermack-McKendrick SIR model (1.1), the derivative of $S(t)$ is negative for all $t \geq 0$, so that the number of susceptible individuals is monotonically decreasing in terms of time irrespective of the initial value $S(0) = S_0 > 0$. For the monotonic and positive nature of $S(t)$, we have

$$\lim_{t\to\infty} S(t) = S_\infty \geq 0.$$

Moreover, $R'(t) > 0$ for all $t \geq 0$, the number of recovered individuals is monotonically increasing with the passage of time and cannot beyond the total population size. Therefore, we have

$$\lim_{t\to\infty} R(t) = R_\infty \leq N.$$

On the other hand, the number of infectives can be monotonically decreasing or increase to a certain level before decreasing. If $dI/dt|_{t=0} = (\beta S_0/N - \gamma) I_0 > 0$, the prevalence will increase at the initial time of epidemic dynamics. Therefore, the number of infectives will start to increase at the initial stage under the necessary condition $\mathscr{R}_0 := \beta/\gamma > 1$, where $\mathscr{R}_0$ is a threshold corresponding to the basic reproduction number that is an important index in epidemiology. It is used to characterize the disease transmission and can give a theoretical reference about whether an infection will spread or not at the initial stage of epidemic dynamics. If $\mathscr{R}_0 < 1$, the disease will die out, while, only if $\mathscr{R}_0 > 1$, the disease can spread in the population from the initial condition with sufficiently small number of infectives.

The equation to determine the final epidemic size is given by

$$S_\infty = S_0 \exp\left\{-\frac{\beta}{\gamma}\left(1 - \frac{S_\infty}{N}\right)\right\},$$

where S_∞ denotes the final size of susceptible subpopulation at the end of epidemic dynamics. The final epidemic size can be obtained as

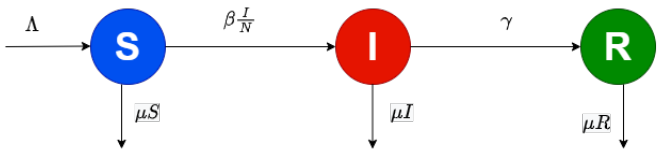

Figure 1.3: The scheme for the epidemic dynamics model (1.2).

$R_\infty = N - S_\infty$, since the infective individuals disappears at the end of epidemic dynamics governed by the SIR model (1.1).

1.2.2 *SIR model with demography*

In order to consider the longer-term persistence of an infectious disease and its endemic dynamics, the demographic process is important. There are diseases that take long time to develop, such as AIDS, tuberculosis, and hepatitis C. In such a case, it is not reasonable to ignore the demography of population, since the change of the population size over time has a relavent relation to the epidemic dynamics.

To construct an SIR model with the demographic effect, we must extend the model (1.1), for example, by assuming that all newborn individuals are healthy and can be infected. Then, we may consider the following model:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta\frac{I}{N}S - \mu S; \\ \frac{dI}{dt} &= \beta\frac{I}{N}S - \gamma I - \mu I; \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned} \tag{1.2}$$

with the initial condition $(S(0), I(0), R(0)) = (S_0, I_0, 0)$. The positive Λ denotes the recruitment/birth rate into the susceptible subpopulation, and μ denotes the removal rate of individuals from each state. The size of total population is given by N, and satisfies $N = S(t) + I(t) + R(t)$. It is easily seen from (1.2) that $dN/dt = \Lambda - \mu N$, and $N(t) \to \Lambda/\mu$ as $t \to \infty$ if Λ is a constant. This means that the population is not constant, while it asymptotically approaches a constant. The state transition of the individual is shown in Figure 1.3.

The dynamics of model (1.2) can be mathematically regarded as equivalent to the following two-dimensional system:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta\frac{I}{N}S - \mu S; \\ \frac{dI}{dt} &= \beta\frac{I}{N}S - \gamma I - \mu I. \end{aligned} \tag{1.3}$$

It is easily derived the following non-dimensionalized system from (1.3) with the transformation of variables such as $s(t) = S(t)/(\Lambda/\mu)$, $i(t) = I(t)/(\Lambda/\mu)$ and $r(t) = R(t)/(\Lambda/\mu)$, the dimensionless time

$\hat{t} = t/\tau$, where $\tau := 1/(\gamma + \mu)$ is the expected length of the effective infectivity.

$$\begin{aligned} \frac{ds}{d\hat{t}} &= \rho(1-s) - \mathscr{R}_0 is; \\ \frac{di}{d\hat{t}} &= (\mathscr{R}_0 s - 1)i, \end{aligned} \tag{1.4}$$

where $\mathscr{R}_0 = \beta/(\gamma + \mu)$ and $\rho = \mu/(\gamma + \mu)$. Although this model cannot be solved analytically, we can mathematically get some insights about its behavior. It is significant from an epidemiological perspective to determine how the solution would perform over time, in order to know whether the disease would die out or spread in the population to become endemic?

For this purpose, it is important to find the equilibrium and check its stability. Equilibrium is the point at which the system remains unchanged. We have the disease-free equilibrium $E_0(1, 0)$ at which there is no infective individual in the population. The other equilibrium usually called the endemic equilibrium is given as $E_\star\left(1/\mathscr{R}_0, \rho\left(1 - 1/\mathscr{R}_0\right)\right)$ for (1.4). For the existence of the endemic equilibrium, $\mathscr{R}_0 > 1$ is necessary and sufficient.

1.3 CONTROL STRATEGY FOR EPIDEMIC DYNAMICS

As human interaction comes, there is a risk of spreading infectious diseases. To reduce the risk of the spread of infectious diseases in the community, quarantine, vaccination, and treatment strategies are important policies. To control various kinds of infectious diseases like severe acute respiratory syndrome, plague, smallpox, Infectious Tuberculosis, Cholera, Yellow fever, influenza, and COVID-19, the quarantine and vaccination are the primary methods. Martcheva [29] gave a summary of such techniques used to control the spread of infectious diseases.

Treatment is the amplification of medication, a procedure, or bed rest in order to prevent the disease. It is now possible for the majority of infectious diseases to vanish with the medication in order to improve the quality of patient's life. Malaria and tuberculosis are diseases that can be cured through the medication [14]. Disease like AIDS cannot be cured, and the medication can give only a certain relief to the patient [36].

Vaccination is an established method for the protection of individuals in a population from the infection and for the reduction in mortality and morbidity. The vaccine can provide the acquired immunity against a specific infectious disease [4]. In a vaccination process, killed microorganism is introduced into body. Vaccine agents are recognized as foreign subjects by our immune system. As a result, antibodies are produced against the agent by triggering an immune response with the vaccine. Then the antibodies can disturb the multiplication of the microorganism more effectively when they enter the body. Those individuals who got the immunity may be protected from the infection. The outbreak of a disease may be suppressed once the number of

vaccinated individuals become sufficiently large in the population. It is referred as the herd immunity. The vaccination program has been a great success for the public health in history. The vaccination has almost eradicated polio in most countries and wiped out smallpox from the world.

However, a vaccinated person cannot be completely protected against all diseases. It is likely that such a person could become sick. Some pathogens can be mutated even if their hosts develop antibodies. Then the immune system may be unable to defeat such mutated pathogens. The level of protection of the vaccinated individual against the infection is known as the vaccine efficacy. In order to reduce the infection rate, a large number of susceptible individuals are targeted in the vaccination process. Vaccination necessitates the rapid identification of the infectious agent as well as the production and administration of a safe vaccine. These processes may take a long time, as shown in the SARS pandemic for 2003. Gokbulut et al. [16] considered the effect of the vaccine for COVID-19, and they found that the vaccination is effective in the presence of some precautions such as wearing a mask, social distance, etc.

Quarantine/Isolation is other kind of good strategy to control infectious diseases. Usually in the strategy, the exposed or infectious individuals are removed from the population for the purpose to supress the outbreak of the disease. In order to reduce the infection rate, a large number of infected individuals are targeted in the isolation process. In the past, a lot of work has been done using mathematical models with the isolation process for the purpose of a discussion on its efficiency to suppress the disease spread [7, 11, 15].

In the isolation, when the symptoms are unclear, the infected person may not be detected. It is crucial to trace contacts in such situations. Many dangerous diseases have been controlled by the isolation. Examples are severe acute respiratory syndrome, plague, smallpox, infectious tuberculosis, cholera, yellow fever, influenza virus, COVID-19 [7, 11, 15]. When SARS swept across the globe from 2002 to 2003, quarantines were implemented.

Using mathematical models with control strategies is important to understand the influence as well as the dynamics of infectious diseases for the purpose of discussion on an efficient way to suppress the disease spread [7]. Hethcote, Zhien, and Shengbing [19] has proposed SIR+Q and SIQS mathematical models with three forms of incidence, where Q indicates the isolation state. In their SIR+Q model with a quarantine-adjusted incidence, the endemic equilibrium is an unstable spiral for a set of parameter values, and a periodic solution arises with the Hopf bifurcation. Erdem, Safan, and Castillo-Chavez [13] considered the case of imperfect quarantine/isolation, and found a periodic solution or damp oscillation depending on the parameter value of quarantine effectiveness. Vivas-Barber, Castillo-Chavez, and Barany [37] considered an SIR+Q model with the perfect isolation and an asymptomatic compartment and got the result of damped oscillations that indicate a recurring epidemics. Castillo-Chavez, Castillo-Garsow, and Yakubu [10] considered the mathematical model for the purpose

of predicting whether the isolation/quarantine can control the SARS for a limited time frame within a single outbreak. Their model implied the isolation/quarantine drastically could reduce the size of the SARS outbreak. Amador and Gomez-Corral [3] considered a stochastic SIQS model that includes two quarantine states, susceptible and infected, and a quarantine compartment with a limited carrying capacity. Their results indicated that quarantine occupancy rates are influenced by the capacity of quarantine and the contact process.

Hu et al. [22] considered a SAIQR (SARS CoV-2) mathematical model with two patches to investigate the transmission dynamics of SARS-CoV-2 with a limited medical resource under the human migration between two regions. They took account of an asymptomatic state (A) in the modeling for the epidemic dynamics. Their results indicated that making the basic reproduction number below 1 is not sufficient in order to control the present COVID-19, and, it should be significantly below 1. Abdelrazec et al. [1] considered a deterministic compartmental model with a nonlinear recovery rate in order to discuss how available resources in the health system influence the dengue fever spread and control. They found an important feature such that the model shows a backward bifurcation, where an endemic equilibrium coexists with the disease-free equilibrium, which is an important result to consider for designing a control scheme. This means that the basic reproduction number less than 1 is enough to eliminate an epidemic when the number of infected cases is very small.

Zhao et al. [40] considered a deterministic model to investigate the influence of limited medical resources on the transmission and control of Zika. In order to understand how limited medical resources may affect the transmission and control of Zika, they included a piece wise smooth recovery rate with the treatment in the model. When the model with no treatment has a globally stable equilibrium, the model with treatment, can undergo a backward bifurcation, Hopf bifurcation, or Bogdanov–Takens bifurcation of codimension 2. Based on these dynamic patterns, Zika outbreaks may occur periodically as extinctions, reoccurrences, or multiple stable outbreaks. Their Zika model indicated the substantial dependence on parameters and initial conditions with respect to the control of Zika virus.

1.3.1 *A modeling with vaccination*

To construct an SIR model with vaccination, let us assume that the vaccine is perfect, and the susceptible individuals who got the vaccine cannot be infected. Moreover, a fraction p of vaccinated newborns is assumed to enter the population. The flux of $p\mu N$ from the newborns goes to the recovered class, while the flux of $(1-p)\mu N$ enters into the susceptible class [29]. Based these assumptions, we can consider the following model:

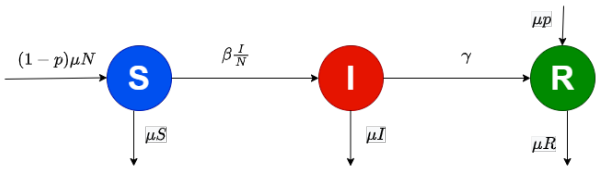

Figure 1.4: The scheme for the epidemic dynamics model (1.5).

$$\begin{aligned}\frac{dS}{dt} &= (1-p)\mu N - \beta\frac{I}{N}S - \mu S,\\ \frac{dI}{dt} &= \beta\frac{I}{N}S - \gamma I - \mu I,\\ \frac{dR}{dt} &= p\mu N + \gamma I - \mu R\end{aligned} \tag{1.5}$$

with the initial condition $(S(0), I(0), R(0)) = (S_0, I_0, 0)$. Parameter μ denotes the death rate of susceptible, infected, and recovered individual in the population. The size of total population is given by a constant N we have $S(t) + I(t) + R(t) = N$ for any $t \geq 0$. The state transition of the individual and the demographic flows for the model (1.5) are schematically shown in Figure 1.4.

The disease-free equilibrium is given by $E_0\left((1-p)N, 0, pN\right)$. For the model (1.5), $\mathscr{R}_0 = \beta/(\gamma+\mu)$ is the basic reproduction number with no vaccination, and $(1-p)\mathscr{R}_0$ is that with vaccination. The vaccination reduces the basic reproduction number by fraction p. In order to discuss how many fraction of individuals should be vaccinated to prevent the outbreak, we solve $(1-p)\mathscr{R}_0 < 1$ for p, and get $p > \hat{p}$ with $\hat{p} = 1 - 1/\mathscr{R}_0$. If a fraction of the population beyond $\hat{p}$ is successfully vaccinated, the disease will not spread in the population. It is referred to as herd immunity.

1.3.2 *A modeling with quarantine/isolation*

A mathematical model with the isolation can be constructed by an extension of the SIR model with demographic effect under the assumption that isolated individuals cannot contact any other [15]. We can consider the following model:

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \beta\frac{I}{N-Q}S - \mu S;\\ \frac{dI}{dt} &= \beta\frac{I}{N-Q}S - \gamma I - \sigma I - \mu I;\\ \frac{dQ}{dt} &= \sigma I - \gamma Q - \mu Q;\\ \frac{dR}{dt} &= \gamma I + \gamma Q - \mu R\end{aligned} \tag{1.6}$$

with the initial condition $(S(0), I(0), Q(0), R(0)) = (S_0, I_0, 0, 0)$. The variable Q denotes the isolated population size in the community at

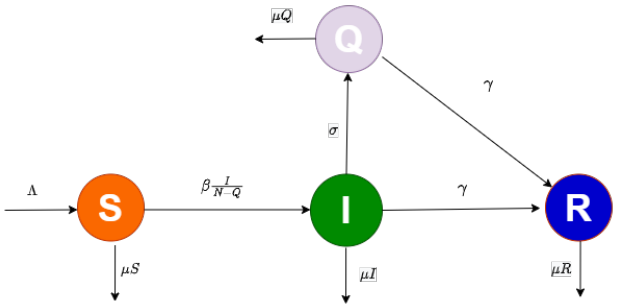

Figure 1.5: The scheme for the epidemic dynamics model (1.6).

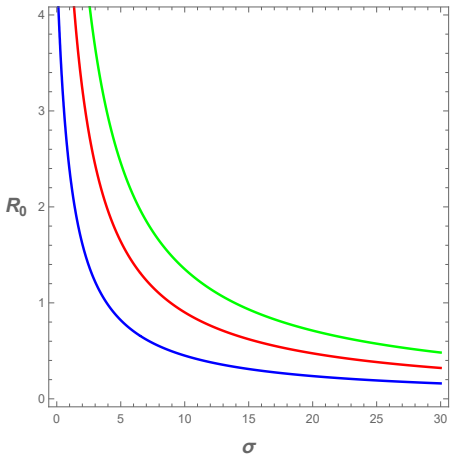

Figure 1.6: Dependence of the basic reproduction number $\mathscr{R}_0$ on the isolation rate σ. Numerically obtained for $\beta = 5$ (blue), 10 (red), 15 (green). Commonly used $\gamma = 0.6$, $\mu = 0.5$.

time t. Every parameter is positive. The parameter σ denotes the isolation rate of the infective individual. The transmission of the disease is determined by the frequency-dependent infection force with the infection coefficient β. Since the subpopulation size of free individuals is given by $N - Q$, the net incidence rate is given by $\beta SI/(N - Q)$. The state transition of the individual and the demographic flows for the model (1.6) are schematically shown in Figure 1.5.

The basic reproduction number $\mathscr{R}_0$ for the model (1.6) is defined as $\mathscr{R}_0 = \beta/(\gamma + \sigma + \mu)$. It can be seen in the Figure 1.6 that $\mathscr{R}_0$ is monotonically decreasing in terms of σ, which means that increasing the isolation rate can decrease the basic reproduction number of model (1.6).

2

EPIDEMIC DYNAMICS MODEL WITH LIMITED ISOLATION CAPACITY

In many countries, there has been a shortage of medical resources since the outbreak of SARS-COV-2. In recent, a lot of work has been done using mathematical models to investigate how the limited medical resources could affect the transmission and control of an infectious disease [1, 34, 35, 39]. The isolation requires a specific space with highly organized conditions to isolate the infected individuals from the others in the community. When the isolation capacity is much small, the isolation strategy may fail in a certain finite time on the way of the epidemic process. In such a case, how does the final epidemic size depend on the limited isolation capacity?

2.1 ASSUMPTIONS AND MODELING

We consider a model with a four-dimensional system of ordinary differential equations to investigate the influence of limited isolation capacity on the final epidemic size. The variables and parameters in the model are as follows:

$S(t)$: Susceptible subpopulation size at time t;

$I(t)$: Infective subpopulation size at time t;

$Q(t)$: Isolated subpopulation size at time t;

$R(t)$: Recovered subpopulation size at time t;

N: Total population size of the community;

$\sigma(Q)$: Per capita isolation rate;

β: Infection coefficient;

γ: Per capita recovery rate;

Q_{max}: Isolation capacity.

We consider an epidemic dynamics in a season, which consists of susceptible, infective, isolated, and recovered individuals. We assume the followings:

- The total population size of the community is constant, ignoring demographic change due to birth and death in a given season.
- Isolated individuals cannot contact any other.
- Any isolated individual is not discharged in the season.
- The isolation is limited by a capacity beyond which the isolation is ceased.

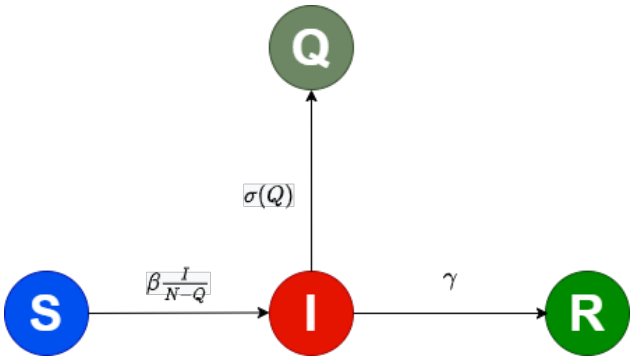

Figure 2.1: The scheme for the epidemic dynamics model (2.1).

Following the last assumption, the epidemic dynamics may contain two phases: the isolation effective phase and the isolation incapable phase. In the isolation effective phase, the isolation works, while in the isolation incapable phase, the isolation is ceased since the isolation already reached the capacity.

With the above assumptions, we consider the following SIR+Q model:

$$\begin{aligned} \frac{dS}{dt} &= -\beta\frac{I}{N-Q}S; \\ \frac{dI}{dt} &= \beta\frac{I}{N-Q}S - \gamma I - \sigma(Q)I; \\ \frac{dQ}{dt} &= \sigma(Q)I; \\ \frac{dR}{dt} &= \gamma I, \end{aligned} \tag{2.1}$$

with

$$\sigma(Q) = \begin{cases} \sigma_0 & Q < Q_{max}; \\ 0 & Q = Q_{max}, \end{cases}$$

and the initial condition $(S(0), I(0), Q(0), R(0)) = (S_0, I_0, 0, 0)$. The variables S, I, Q, and R denote the susceptible, infected, isolated and recovered population sizes respectively. The total population size of the community is denoted by N, and it is satisfied that $S(t) + I(t) + Q(t) + R(t) = N$ for any $t \geq 0$. The state transition of the individual is schematically shown in Figure 2.1.

Every parameter is positive. The parameter γ denotes the recovery rate of infective individual. The transmission of the disease is determined by the frequency-dependent infection force with the infection coefficient β. Since the subpopulation size of isolated free individuals is given by $N - Q$, the net incidence rate is given by $\beta SI/(N - Q)$. The piece-wise function $\sigma(Q)$ denotes the isolation rate of infected individual. Parameter σ_0 is a positive constant that is the isolation rate at the isolation effective phase. The parameter Q_{max} denotes the isolation capacity. Once the isolation reaches its limit Q_{max}, the isolation

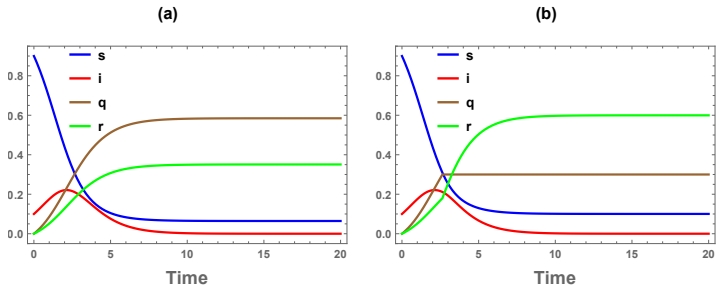

Figure 2.2: Numerical examples of temporal variation of the system (2.2). (a) $q_{max} = 0.7$; (b) $q_{max} = 0.2$. Commonly $\mathscr{R}_0 = 1.5$, $\gamma = 0.3$, $\sigma_0 = 0.5$, (s_0, i_0, q_0, r_0)=$(0.9, 0.1, 0.0, 0.0)$.

becomes ceased, and the epidemic dynamics enter the isolation incapable phase with $\sigma(Q) = 0$, as numerically demonstrated in Figure 2.2(b).

It is easily derived the following non-dimensionalized system from (2.1) with the transformation of variables such as $s(t) = S(t)/N$, $i(t) = I(t)/N$, $q(t) = Q(t)/N$ and $r(t) = R(t)/N$. The dimensionless time is given by $\hat{t} = t/\tau$, where $\tau := 1/(\gamma + \sigma_0)$ is the expected length of the effective infectivity.

$$\begin{aligned} \frac{ds}{d\hat{t}} &= -\mathscr{R}_0 \frac{is}{1-q}; \\ \frac{di}{d\hat{t}} &= \mathscr{R}_0 \frac{is}{1-q} - \hat{\gamma} i - \hat{\sigma}(q) i; \\ \frac{dq}{d\hat{t}} &= \hat{\sigma}(q) i; \\ \frac{dr}{d\hat{t}} &= \hat{\gamma} i, \end{aligned} \tag{2.2}$$

with $\mathscr{R}_0 := \beta/(\gamma + \sigma_0)$ and

$$\hat{\sigma}(q) = \begin{cases} \hat{\sigma}_0 & q < q_{max}; \\ 0 & q = q_{max}, \end{cases}$$

where $\hat{\gamma} = \gamma/(\gamma + \sigma_0)$, $\hat{\sigma}_0 = \sigma_0/(\gamma + \sigma_0)$ and $q_{max} = Q_{max}/N$. The initial condition becomes $(s(0), i(0), q(0), r(0)) = (s_0, i_0, 0, 0)$ where $s_0 := S_0/N$ and $i_0 := I_0/N$. It is satisfied that $s(\hat{t}) + i(\hat{t}) + q(\hat{t}) + r(\hat{t}) = 1$ for any $\hat{t} \geq 0$.

2.2 BASIC REPRODUCTION NUMBER

The parameter $\mathscr{R}_0$ corresponds to the basic reproduction number which is an important index in epidemiology. It is used to characterize the disease transmission and can give a theoretical reference about whether an infection will spread or not in the community. The definition of basic reproduction in a biological context is the expected

number of new infections, produced by a single infective individual in a community where the infective individual contacts only susceptible individuals until the infectivity is lost [18].

For the mathematical derivation of the basic reproduction number for our model (2.1), we may use a fundamental way used in [8], and can get $\mathscr{R}_0 = \beta/(\gamma + \sigma_0)$, where β corresponds to the supremum of the expected number of new cases produced by an infective per unit time, and $1/(\gamma + \sigma_0)$ is the expected duration of the effective infectivity. If $\mathscr{R}_0 < 1$, the disease will die out, and, if $\mathscr{R}_0 > 1$, the disease can spread in the population from the initial condition with sufficiently small number of infectives.

2.3 CONSERVED QUANTITY

2.3.1 *At the isolation effective phase*

At the isolation effective phase, the model (2.2) becomes:

$$\begin{aligned} \frac{ds}{d\hat{t}} &= -\mathscr{R}_0 \frac{is}{1-q}; \\ \frac{di}{d\hat{t}} &= \mathscr{R}_0 \frac{is}{1-q} - \hat{\gamma} i - \hat{\sigma}_0 i; \\ \frac{dq}{d\hat{t}} &= \hat{\sigma}_0 i; \\ \frac{dr}{d\hat{t}} &= \hat{\gamma} i. \end{aligned} \tag{2.3}$$

From the first and second equations of (2.3), we can derive the following differential equation:

$$\frac{di}{ds} = -1 + \frac{1-q}{\mathscr{R}_0 s}. \tag{2.4}$$

Where we used $\hat{\sigma}_0 + \hat{\gamma} = 1$. Moreover, from the third and fourth equations of (2.3), we can derive the following as well:

$$\frac{dq}{dr} = \frac{\hat{\sigma}_0}{\hat{\gamma}}. \tag{2.5}$$

Solving the ordinary differential equation (2.5), we can obtain the following relation between $q(\hat{t})$ and $r(\hat{t})$ which holds for any $\hat{t}$ at the isolation effective phase:

$$q(\hat{t}) = \frac{\sigma_0}{\gamma} r(\hat{t}), \tag{2.6}$$

where we used $q(0) = r(0) = 0$. Now, with $s + i + q + r = 1$, we can find the following equation from (2.6):

$$1 - q = \frac{s + i + \gamma/\sigma_0}{1 + \gamma/\sigma_0}. \tag{2.7}$$

By substituting (2.7) for (2.4), we can derive the following ordinary differential equation:

$$\frac{d}{ds}\left(is^{-\sigma_0/\beta}\right) = s^{-\sigma_0/\beta}\left(-1+\frac{1}{\beta/\sigma_0}\right)+\frac{s^{-1-(\sigma_0/\beta)}}{\beta/\gamma}.$$

We can solve this ordinary differential equation, and get

$$is^{-\sigma_0/\beta} = -s^{1-(\sigma_0/\beta)} - \frac{\gamma}{\sigma_0}s^{-\sigma_0/\beta} + C$$

with a constant C. We can easily get the expression for C from the initial conditions $i(0) = i_0$, $s(0) = s_0$, and $i_0 + s_0 = 1$. Then, as a result, we obtain the following equation which holds for any $\hat{t}$ at the isolation effective phase:

$$i(\hat{t}) + s(\hat{t}) = -\frac{\gamma}{\sigma_0} + \left(\frac{s_0}{s(\hat{t})}\right)^{-\sigma_0/\beta}\left(1+\frac{\gamma}{\sigma_0}\right). \tag{2.8}$$

Equation (2.8) gives the conserved quantity at the isolation effective phase.

If the isolation never reaches the capacity at any time $\hat{t} > 0$, then we have the following equation for $s(\hat{t}) \to s_\infty^-$ as $\hat{t} \to \infty$ from (2.8):

$$(s_\infty^-)^{-\sigma_0/\beta}\left(s_\infty^- + \frac{\gamma}{\sigma_0}\right) = (s_0)^{-\sigma_0/\beta}\left(1+\frac{\gamma}{\sigma_0}\right), \tag{2.9}$$

where we used a mathematical feature of (2.3) such that $i(\hat{t}) \to 0$ as $\hat{t} \to \infty$.

Next from $s + i = 1 - (q + r)$ and (2.6), we have

$$s + i = 1 - q\left(\frac{\gamma}{\sigma_0} + 1\right). \tag{2.10}$$

Substituting (2.10) for (2.8), we get

$$q(\hat{t}) = 1 - \left(\frac{s_0}{s(\hat{t})}\right)^{-\sigma_0/\beta}. \tag{2.11}$$

As $t \to \infty$, the equation (2.11) becomes

$$q_\infty^- = 1 - \left(\frac{s_0}{s_\infty^-}\right)^{-\sigma_0/\beta}, \tag{2.12}$$

where $q(\hat{t}) \to q_\infty^-$ and $s(\hat{t}) \to s_\infty^-$. The value q_∞^- gives the final size of the isolated subpopulation at isolation effective phase as $\hat{t} \to \infty$. Since $q(\hat{t})$ is monotonically increasing in terms of $\hat{t}$ at the isolation effective phase. If value of q_∞ is not beyond q_{max}, the epidemic dynamics can follows only the isolation effective phase, while, if not, the dynamics have a moment to switch from the isolation effective phase to the isolation incapable phase. When the isolation never reaches the capacity for any $\hat{t} > 0$, we have $s_\infty^- = 1 - q_\infty^- - r_\infty^-$, since $i(\hat{t}) \to 0$ as $\hat{t} \to \infty$. Hence, from (2.12), we get

$$1 - q_\infty^- - r_\infty^- = s_0(1 - q_\infty^-)^{\beta/\sigma_0}. \tag{2.13}$$

Substituting (2.13) for (2.9), we can derive the equation

$$1 - q_\infty^- \left(1 + \frac{\gamma}{\sigma_0}\right) = s_0 \left(1 - q_\infty^-\right)^{\beta/\sigma_0}. \tag{2.14}$$

Equation (2.14) determine the proportion of final isolated subpopulation at the end of epidemic dynamics when the isolation never reaches the capacity for any $\hat{t} > 0$.

2.3.2 *At the isolation incapable phase*

If the capacity of isolation is sufficiently small, the isolated subpopulation size $q(\hat{t})$ reaches the capacity q_{max} at a certain moment $\hat{t} = t^\star > 0$ on the way of epidemic process, and the dynamics switches from the isolation effective phase to the isolation incapable phase. At the isolation incapable phase, model (2.2) becomes

$$\begin{aligned} \frac{ds}{d\hat{t}} &= -\mathscr{R}_0 \frac{is}{1 - q_{max}}; \\ \frac{di}{d\hat{t}} &= \mathscr{R}_0 \frac{is}{1 - q_{max}} - \hat{\gamma} i; \\ \frac{dr}{d\hat{t}} &= \hat{\gamma} i \end{aligned} \tag{2.15}$$

for $\hat{t} \geq t^\star$. From the first and second equations of (2.15), we can derive the following differential equation:

$$\frac{di}{ds} = -1 + \frac{1 - q_{max}}{\mathscr{R}_0 \left(1 + \sigma_0/\gamma\right) s},$$

where we used $\hat{\gamma} = \gamma/(\gamma + \sigma_0)$. We can easily solve this ordinary differential equation, and can get the relation

$$i(\hat{t}) = -s(\hat{t}) + \frac{1 - q_{max}}{\beta/\gamma} \ln s(\hat{t}) + C \tag{2.16}$$

with a constant C. For $\hat{t} = t^\star$, we have

$$C = i(t^\star) + s(t^\star) - \frac{1 - q_{max}}{\beta/\gamma} \ln s(t^\star). \tag{2.17}$$

Substituting (2.17) for (2.16), we get the following equation that gives the conserve quantity at the isolation incapable phase:

$$s(\hat{t}) + i(\hat{t}) = i(t^\star) + s(t^\star) + \frac{\gamma}{\beta}(1 - q_{max}) \ln \frac{s(\hat{t})}{s(t^\star)}. \tag{2.18}$$

Since the epidemic dynamics remains at the isolation incapable phase once the isolation reaches the capacity, we have the following equation of $s(\hat{t}) \to s_\infty^-$ as $\hat{t} \to \infty$:

$$s(t^\star) + i(t^\star) = -\frac{\gamma}{\sigma_0} + \left(\frac{s_0}{s(t^\star)}\right)^{-\sigma_0/\beta} \left(1 + \frac{\gamma}{\sigma_0}\right), \tag{2.19}$$

where we used the feature that $i(\hat{t}) \to 0$ as $\hat{t} \to \infty$ for (2.15).

2.4 CRITICAL VALUE OF THE ISOLATION CAPACITY q_c

We have the following theorem for the isolation to reach the capacity in a finite time on the way of epidemic dynamics:

Theorem 2.4.1. *The isolation reaches the capacity in a finite time on the way of epidemic dynamics if and only if $q_{max} < q_c$, where q_c is the critical value of the isolation capacity and uniquely defined as the positive root of the following equation:*

$$1 - q_c \left(1 + \frac{\gamma}{\sigma_0}\right) = s_0 \left(1 - q_c\right)^{\beta/\sigma_0}. \tag{2.20}$$

Proof. From (2.9), if the isolation never reaches the capacity, we have

$$s_\infty^- = F(s_\infty^-) := -\frac{\gamma}{\sigma_0} + \left(\frac{s_\infty^-}{s_0}\right)^{\sigma_0/\beta} \left(1 + \frac{\gamma}{\sigma_0}\right). \tag{2.21}$$

We have $F(0) = -\gamma/\sigma_0$, $F(s_0) = 1$, and $F'(s) > 0$ for $s \in (0, s_0)$. Thus $F(s)$ is a monotonically increasing continuous and differential function of $s \in (0, s_0)$, and satisfies that $F(0) < 0$ and $F(s_0) > s_0$. Further, we can easily find that $F(s)$ is linear if $\sigma_0/\beta = 1$, and otherwise it is alternatively convex or concave in $(0, s_0)$. Therefore, we find that the equation (2.21) has a unique root $s_\infty^- \in (0, s_0)$, and $F(s) < s$ for $s \in (0, s_\infty^-)$ while $F(s) > s$ for $s \in (s_\infty^-, s_0)$.

On the other hand, with respect to the final sizes s_∞^- and q_∞^- in this case, we have (2.12). Now it must be satisfied that $q_\infty^- \leq q_{max}$, because this is the case where $q(\hat{t})$ never reaches q_{max} for any $\hat{t} > 0$. Since $q(\hat{t})$ is monotonically increasing in terms of $\hat{t}$, if $q_\infty^- \leq q_{max}$, the isolation does not reach the capacity for any $\hat{t} > 0$. Therefore, if and only if $q_\infty^- \leq q_{max}$, the isolation does not reach the capacity for any $\hat{t} > 0$. Consequently, we find that, if and only if $q_\infty^- > q_{max}$, the isolation reaches the capacity at $\hat{t} = t^\star < \infty$.

Now (2.12) and (2.21), we can derive the following condition equivalent to $q_\infty^- > q_{max}$:

$$s_\infty^- < 1 - q_{max} \left(1 + \frac{\gamma}{\sigma_0}\right). \tag{2.22}$$

Since $s_\infty^- > 0$, we note that this inequality holds only if $q_{max} < 1/(1 + \gamma/\sigma_0)$. Consequently, from the nature of the function $F(s)$ shown in the above, the condition (2.22) is equivalent to the condition $F(s) > s$ for $s = 1 - q_{max}(1 + \gamma/\sigma_0)$. This leads to $q_{max} < q_c$ which is the condition for the isolation to reach the capacity in a finite time. □

If $q_{max} < q_c$ is satisfied, the system (2.2) changes the phase from isolation effective to incapable phase on the way of epidemic dynamics. Otherwise, the system remains at the isolation effective phase until the end of epidemic dynamics.

2.5 FINAL EPIDEMIC SIZE

The final epidemic size for the system (2.2) is defined as the proportion of recovered and isolated individuals in the community at the end of the epidemic dynamics.

2.5.1 *Final size equation for $q_{max} \geq q_c$*

When the isolation never reaches the capacity at any time due to the sufficient capacity of isolation, the final epidemic size is determined only by the isolation effective phase. In this case $s_\infty^- = 1 - (q_\infty^- + r_\infty^-)$, and the final epidemic size is given by $z_\infty^- = q_\infty^- + r_\infty^-$, where z_∞^- denotes the final epidemic size. Therefore, making use of s_∞^- for (2.9), we get

$$\left(1 - z_\infty^-\right)^{-\sigma_0/\beta} \left(1 + \frac{\gamma}{\sigma_0} - z_\infty^-\right) = (s_0)^{-\sigma_0/\beta} \left(1 + \frac{\gamma}{\sigma_0}\right), \tag{2.23}$$

the root of equation (2.23) gives the final epidemic size for the case when the isolation never reaches the capacity at any time.

Theorem 2.5.1. *The final epidemic size z_∞^- is uniquely determined by the equation (2.23) when the isolation never reaches the capacity.*

Proof. We define the left-hand side of (2.23) is a function of z

$$A(z) := (1 - z)^{-\sigma_0/\beta} \left(1 + \frac{\gamma}{\sigma_0} - z\right),$$

while the right-hand side of (2.23) represents a horizontal line and denoted by B_0, therefore, the final size equation becomes

$$A(z) = B_0. \tag{2.24}$$

The function $A(z)$ is continuous and differentiable for $z \in (0, 1)$, and satisfies that $A(0) < B_0$ and $\lim_{z \to 1-} A(z) > B_0$. From these arguments, there exist at least one root.

Further, the behavior of function $A(z)$ shows that it is monotonically increasing or may have an extremal minimum in the range of z. If $A(z)$ is monotonically increasing in the range of z, then by intermediate value theorem, it has a unique intersection with the horizontal line B_0. However, if $A(z)$ may have an extremal minimum in the range of z, then from condition $A(0) < B_0$, it has a unique intersection with horizontal line B_0. In both cases, the final size equation (2.24) has a unique root in between range $(0, 1)$. As a result, the final epidemic size z_∞^- is uniquely determined by the equation (2.23) when the isolation never reaches the capacity. □

2.5.2 *Final size equation for $q_{max} < q_c$*

When the isolation reaches the capacity in a finite time on the way of epidemic dynamics due to insufficient capacity. There is a certain

moment $\hat{t} = t^\star$ at which the isolation reaches the capacity, before this moment $\hat{t} < t^\star$, the dynamic follows the isolation effective phase, and after this moment $\hat{t} > t^\star$, the dynamic follows the isolation incapable phase. The connection of these two dynamics gives the equation which determine the final epidemic size for the case of isolation reaches the capacity in a finite time during epidemic process. At $\hat{t} = t^\star$, we have $i(\hat{t}) = i(t^\star)$, $s(\hat{t}) = s(t^\star)$ and $q = q_{max}$, therefore, the equation (2.10) becomes

$$s(t^\star) + i(t^\star) = 1 - q_{max}\left(1 + \frac{\gamma}{\sigma_0}\right). \tag{2.25}$$

Making use of (2.19) and (2.25), we can explicitly obtain the value of $s(t^\star)$ and $i(t^\star)$ that are given as

$$s(t^\star) = s_0\left(1 - q_{max}\right)^{\beta/\sigma_0}; \tag{2.26}$$

$$i(t^\star) = 1 - q_{max}\left(1 + \frac{\gamma}{\sigma_0}\right) - s_0\left(1 - q_{max}\right)^{\beta/\sigma_0}. \tag{2.27}$$

Next, we consider the epidemic dynamic after $t^\star$, and after $t^\star$ the epidemic dynamics follow the isolation incapable phase. Therefore, making use of (2.26) and (2.27) for (2.18) and $\hat{t} \to \infty$, we get the following equation

$$\begin{aligned} s_\infty^+ =& 1 - q_{max}\left(1 + \frac{\gamma}{\sigma_0}\right) \\ &+ \frac{\gamma}{\beta}(1 - q_{max})\left[\ln s_\infty^+ - \ln\left\{s_0\left(1 - q_{max}\right)^{\beta/\sigma_0}\right\}\right], \end{aligned} \tag{2.28}$$

where we used $i_\infty = 0$. In the case of isolation reaches the capacity, $s_\infty^+ = 1 - (q_{max} + r_\infty^+)$, where the the epidemic size is given by $z_\infty^+ = q_{max} + r_\infty^+$, therefore, making use of the value of s_∞^+ for (2.28), we can obtain the following equation

$$\begin{aligned} &\frac{\beta}{\sigma_0}\left\{\frac{q_{max}\left(1 + \sigma_0/\gamma\right)}{1 - q_{max}} + \ln\left(1 - q_{max}\right)\right\} \\ &\qquad = \ln(1 - z_\infty^+) - \ln s_0 + \frac{(\beta/\gamma)z_\infty^+}{1 - q_{max}}. \end{aligned} \tag{2.29}$$

The root of equation (2.29) gives the final epidemic size for the case when isolation reaches capacity in a finite time during the epidemic process.

Theorem 2.5.2. *The final epidemic size z_∞^+ is uniquely determined by the equation (2.29) when the isolation reaches the capacity in a finite time on the way of epidemic dynamics.*

Proof. In order to prove that the final epidemic size z_∞^+ is uniquely determined by the equation (2.29) when the isolation reaches the capacity in a finite time on the way of epidemic dynamics, we define the (2.18)

$$G(s) = s - (i(t^\star) + s(t^\star)) - \frac{\gamma}{\beta}(1 - q_{max}) \ln \frac{s}{s(t^\star)}. \tag{2.30}$$

The function $G(s)$ is continuous and differentiable for $s \in (0, s(t^\star))$. Moreover, it satisfies $\lim_{s \to 0+} G(s) > 0$, and $G(s(t^\star)) < 0$. From these facts, the function $G(s)$ has at least one root. Further, the behavior of function $G(s)$ shows that it is monotonically decreasing or may have an extremal minimum. If $G(s)$ is monotonically decreasing in the range of s then by the intermediate value theorem it has a unique root in the range of s. However, if $G(s)$ may have an extremal minimum then from condition $G(s(t^\star)) < 0$ it has a unique root. Hence in both cases, $G(s)$ has a unique root s_∞^+ in between the range $(0, s(t^\star))$.

The above arguments show that the final epidemic size z_∞^+ is uniquely determined by the equation (2.29) when the isolation reaches the capacity in a finite time on the way of epidemic dynamics □

Lemma 2.5.3. *It holds that* $z_\infty^+ \geq q_c \left(1 + \frac{\gamma}{\sigma_0}\right) \geq z_\infty^-$.

Proof. The proof is given straightforward from the arguments in the proof for Theorem 2.4.1. From (2.22), we note that the condition $q_\infty^- \leq q_{max}$ is equivalent following:

$$s_\infty^- \geq 1 - q_{max} \left(1 + \frac{\gamma}{\sigma_0}\right), \tag{2.31}$$

where s_∞^- is the root of (2.9), and subsequently q_∞^- is given by (2.12). Thus, when and only when the condition (2.31) is satisfied, the isolation never reaches the capacity, so that the epidemic dynamics is always at the isolation effective phase. Inversely, when and only when the condition (2.31) is unsatisfied, the epidemic dynamics enters in the isolation incapable phase at a finite time.

Thus, for the value $s(t^\star)$ at the moment when the isolation incapable phase begins, it must hold that

$$s(t^\star) < 1 - q_{max} \left(1 + \frac{\gamma}{\sigma_0}\right).$$

The value $s(\hat{t})$ is monotonically decreasing in terms of time since $ds/d\hat{t}$ is negative for any $\hat{t} > 0$. Hence, we have $s_\infty^+ < s(t^\star)$ where s_∞^+ is the root of (2.28) at the isolation incapable phase. Therefore, we have

$$s_\infty^+ < 1 - q_{max} \left(1 + \frac{\gamma}{\sigma_0}\right). \tag{2.32}$$

Since $z_\infty^- = 1 - s_\infty^-$, these arguments indicate that, when and only when the isolation never reaches the capacity, we have

$$z_\infty^- \leq q_{max} \left(1 + \frac{\gamma}{\sigma_0}\right)$$

from (2.31). Since this condition must hold for any $q_{max} \geq q_c$ from Theorem 2.4.1, and since z_∞^- is independent of q_{max}, we find that

$$z_\infty^- \leq q_c \left(1 + \frac{\gamma}{\sigma_0}\right).$$

On the other hand, when the isolation reaches the capacity at a finite time with $q_{max} < q_c$, we have

$$z_\infty^+ > q_{max} \left(1 + \frac{\gamma}{\sigma_0}\right)$$

from (2.32). Since this condition must hold for any $q_{max} < q_c$, we have

$$z_\infty^+ \geq q_c \left(1 + \frac{\gamma}{\sigma_0}\right).$$

□

Lemma 2.5.4. *It holds that* $z_\infty^- = q_c \left(1 + \frac{\gamma}{\sigma_0}\right)$.

Proof. Substituting $z_\infty^- = q_c\,(1 + \gamma/\sigma_0)$ for (2.23), and taking account of (2.20) in Theorem 2.4.1, we can easily find that the equation (2.28) holds. Since z_∞^- is uniquely determined as the root of (2.23) from Theorem 2.5.1, we can result in this lemma. □

2.5.3 *Dependence on the isolation capacity*

We can obtain the following theorem for the dependence of the final epidemic size z_∞^+ on the isolation capacity q_{max} when the isolation reaches the capacity in a finite time during the epidemic dynamics.

Theorem 2.5.5. *The final epidemic size* z_∞^+ *is monotonically decreasing in terms of the isolation capacity* q_{max}.

Proof. To prove the Theorem 2.5.5, we need the following two lemmas.

Lemma 2.5.6. $\lim_{q_{max} \to 0+} \partial z_\infty^+ / \partial q_{max} < 0.$

The proof of Lemma 2.5.6 is given in Appendix A.

Lemma 2.5.7. *There exists no extremal value of* $z_\infty^+ = z_\infty^+(q_{max})$ *for* $q_{max} \in (0, q_c)$.

Proof. From Lemma 2.5.6, it is easily found that $\partial z_\infty^+ / \partial q_{max} = 0$ if and only if $z_\infty^+ = 1 + (\gamma/\sigma_0) q_{max}$, therefore, there exists no extremal value for $q_{max} \in (0, q_c)$ at which $\partial z_\infty^+ / \partial q_{max} = 0$. □

From Lemmas 2.5.6 and 2.5.7, $\partial z_\infty^+ / \partial q_{max} < 0$. Hence, the final epidemic size z_∞^+ is monotonically decreasing in terms of the isolation capacity q_{max}. □

Figure 2.3 shows the dependence of the final epidemic size on the isolation capacity q_{max}. The blue curve denotes the final epidemic size z_∞^+ that is monotonically decreasing in terms of the isolation capacity $q_{max} \in [0, q_c]$. It is easily seen that increasing the isolation capacity makes the final epidemic size z_∞^+ smaller. The blue horizontal line denotes the final epidemic size z_∞^- for the case of isolation never reaches the capacity. When the isolation capacity is beyond the critical value, the isolation never reaches the capacity at any finite time.

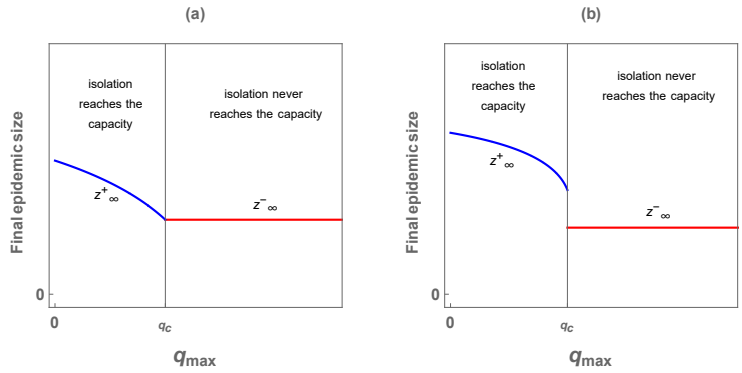

Figure 2.3: Dependence of the final epidemic size on parameter q_{max}. (a) $\gamma/\beta = 1.25$, $\beta/\sigma_0 = 0.8$; (b) $\gamma/\beta = 0.8$, $\beta/\sigma_0 = 1.25$. Commonly $s_0 = 0.9$, $\gamma/\sigma_0 = 1$.

2.5.4 *Severity of insufficient isolation capacity*

We can obtain the following theorem for the final epidemic size becomes drastically large if the isolation reaches the capacity at a finite time (Appendix B).

Theorem 2.5.8. *The final epidemic size has a discontinuous jump at $q = q_c$:*

$$z^\dagger_\infty := \lim_{q_{max} \to q_c - 0} z^+_\infty > z^-_\infty$$

if and only if

$$\frac{\gamma}{\beta} < 1 \ \ and \ \ s_0 > \frac{\gamma}{\beta}\left(1 + \frac{1 - \gamma/\beta}{\gamma/\sigma_0}\right)^{\beta/\sigma_0 - 1}. \tag{2.33}$$

Otherwise, it holds that $z^\dagger_\infty = z^-_\infty$.

When the condition (2.33) is not satisfied, the final epidemic size has no discontinuous jump at the critical value of the isolation capacity, as shown in Figure 2.3(a). In contrast, when the condition (2.33) is satisfied, the final epidemic size has a discontinuous jump at the critical value of the isolation capacity, as numerically demonstrated in Figure 2.3(b).

Figure 2.4 shows the parameter region $(\beta/\sigma_0, \gamma/\sigma_0)$ for the discontinuous jump of the final epidemic size at critical isolation capacity. It is easily seen that for sufficiently small β such that $\beta > \gamma$, such a discontinuous jump of the final epidemic size occurs at the critical value of the isolation capacity. Sufficiently small β means the small infection coefficient, that is the spread of disease is very slow. At the same time, the discontinuous jump requires sufficiently small γ, the recovery rate. Thus, such a discontinuous jump of the final epidemic size could be expected only when the recovery from the disease takes sufficiently long time. Therefore, the severity of insufficient isolation capacity appears especially for the epidemic dynamics of a transmissible disease such that the infectivity is weak while the disease is hardly treated to the recovery.

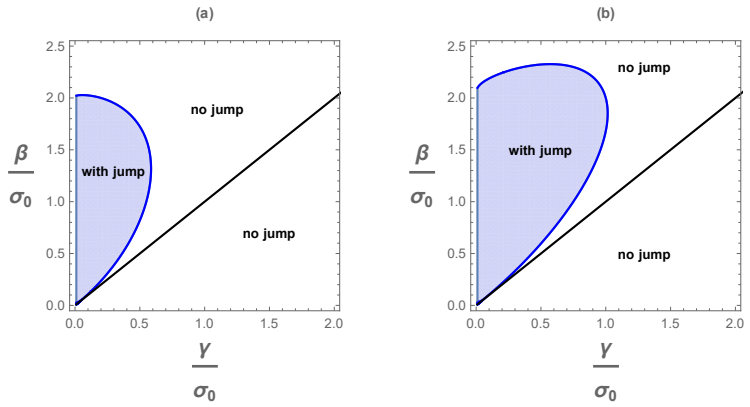

Figure 2.4: Classification of the region $(\gamma/\sigma_0, \beta/\sigma_0)$ for the discontinuous jump of the final epidemic size at $q = q_c$. Numerically obtained for (a) $s_0 = 0.55$; (b) $s_0 = 0.75$.

2.6 PARAMETER DEPENDENCE OF THE CRITICAL ISOLATION CAPACITY

In this section, we discuss the dependence of critical isolation capacity on the characteristics of the epidemic dynamics such as β/σ_0, γ/σ_0 and $1/\sigma_0$.

2.6.1 *On the infection coefficient*

The equation (2.20) can be written as

$$\frac{\beta}{\sigma_0} = \frac{\ln\{1 - q_c\,(1+\gamma/\sigma_0)\} - \ln s_0}{\ln(1-q_c)}. \tag{2.34}$$

It is easily seen that when $q_c = (1-s_0)/(1+\gamma/\sigma_0)$ then $\beta/\sigma_0 = 0$, and when q_c approaches to $1/(1+\gamma/\sigma_0)$ from the left hand side then $\beta/\sigma_0 \to \infty$. We can easily find the derivative of (2.34)

$$\frac{d(\beta/\sigma_0)}{dq_c} = \frac{D(q_c)}{\{1 - q_c\,(1+\gamma/\sigma_0)\}\,(1-q_c)\ln^2(1-q_c)}, \tag{2.35}$$

where

$$D(q_c) := \{1 - q_c\,(1+\gamma/\sigma_0)\}\,[\ln\{1 - q_c\,(1+\gamma/\sigma_0)\} - \ln s_0] \\ -\,(1+\gamma/\sigma_0)\,(1-q_c)\ln(1-q_c).$$

The sign of the right-hand side of (2.35) can be determined uniquely by the sign of $D(q_c)$, and it is easily proved that $D(q_c) > 0$ for $q_c \in (0, 1/(1+\gamma/\sigma_0))$, so that, $d(\beta/\sigma_0)/dq_c > 0$. Hence, q_c is monotonically increasing in terms of β/σ_0.

The results from the analysis shows that the critical value of the isolation capacity q_c is monotonically increasing in terms of infection coefficient, so that, the larger infection coefficient requires a larger isolation capacity in order in order to avoid the isolation reaches the

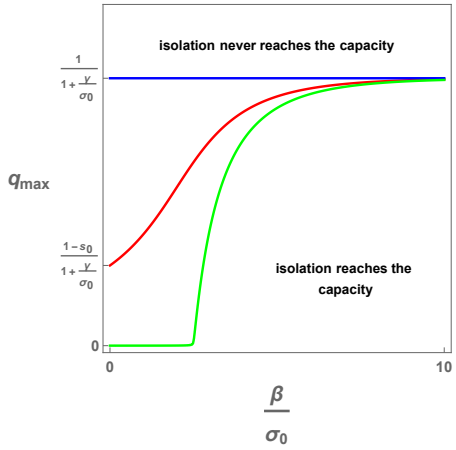

Figure 2.5: $(\beta/\sigma_0, q_{max})$-dependence of the isolation reaches the capacity. Numerically obtained for $s_0 = 0.0$ (blue), 0.7 (red), 0.9999 (green). Commonly $\gamma/\sigma_0 = 1.5$.

capacity at a finite time during the epidemic process, as demonstrated in Figure 2.5. Therefore, it is good strategy to increase the isolation capacity when the spread of the disease is fast in order to avoid the isolation reaches the capacity during epidemic process where the isolation is ceased,

2.6.2 *On the recovery rate*

The equation (2.20) can be written as

$$\frac{\gamma}{\sigma_0} = \frac{1 - q_c - s_0 (1 - q_c)^{\beta/\sigma_0}}{q_c}. \tag{2.36}$$

It is easily seen that when $q_c = 1$ then $\gamma/\sigma_0 = 0$, and when $q_c \to 0+$ then $\gamma/\sigma_0 \to \infty$. We can easily derive the derivative of (2.36)

$$\frac{d(\gamma/\sigma_0)}{dq_c} = \frac{-1 + s_0 (1 - q_c)^{\beta/\sigma_0 - 1} \{1 + q_c (\beta/\sigma_0 - 1)\}}{q_c^2}. \tag{2.37}$$

As $\lim_{q_c \to 0+} d(\gamma/\sigma_0)/dq_c < 0$, and the sign of right-hand side of (2.37) can be uniquely determined by the sign of numerator, therefore, we define as a function of q_c

$$E(q_c) := -1 + s_0 (1 - q_c)^{\beta/\sigma_0 - 1} \{1 + q_c (\beta/\sigma_0 - 1)\}.$$

It is easily found that $E(0) < 0$, $\lim_{q_c \to 1-0} E(q_c) = -1$ if and only if $\beta/\sigma_0 > 1$, and $\lim_{q_c \to 1-0} E(q_c) = \infty$ if and only if $\beta/\sigma_0 < 1$. We can easily derive the derivative of $E(q_c)$

$$\frac{dE}{dq_c} = -s_0 \frac{\beta}{\sigma_0} q_c (1 - q_c)^{\beta/\sigma_0 - 2} \left(\frac{\beta}{\sigma_0} - 1 \right).$$

As $dE/dq_c|_{q_c=0} = 0$, and the sign of dE/dq_c can be positive or negative depending on the value of parameter β/σ_0.

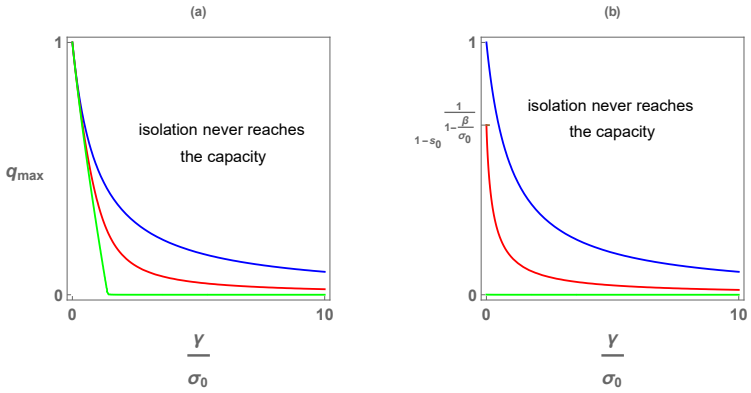

Figure 2.6: $(\gamma/\sigma_0, q_{max})$-dependence of isolation reach the capacity. (a) $\beta/\sigma_0 = 2.4$; (b) $\beta/\sigma_0 = 0.8$. Commonly $s_0 = 0.0$ (blue), 0.7 (red), 0.9999 (green).

When $\beta/\sigma_0 > 1$, $dE/dq_c < 0$ for positive q_c, so that, $E(q_c)$ is monotonically decreasing for positive q_c. Also at lower edge value $E(q_c)$ is negative, therefore, $E(q_c)$ is negative for every range of q_c including zero. As a result, $d(\gamma/\sigma_0)/dq_c < 0$, so that, q_c is monotonically decreasing in terms of γ/σ_0, as numerically demonstrated in Figure 2.6.

When $\beta/\sigma_0 < 1$, the $dE/dq_c > 0$ for positive q_c, so that, $E(q_c)$ is monotonically increasing for positive q_c. As $E(0) < 0$, it means that $E(q_c)$ changes the sign from negative to positive at a point q_c.

As the right-hand side of (2.37) changes the sign from negative to positive at a certain point q_c, therefore, the curve (2.36) has a certain extremal minimum for the positive q_c. It is easily found that the right-hand side of equation (2.36) has two roots $q_c = 1 - s_0^{1/(1-\beta/\sigma_0)}$, 1. Where $1 - s_0^{1/(1-\beta/\sigma_0)}$ is positive and less than one. On the other hand, the curve (2.36) has a certain extremal minimum for positive q_c, and at edge value it is positive. So from the continuity and edge values of the curve (2.36), the extremal minimum for q_c is in the range of $(1 - s_0^{1/(1-\beta/\sigma_0)}, 1)$. Hence, the curve (2.36) exist only for the positive γ/σ_0 in the range $(0, 1 - s_0^{1/(1-\beta/\sigma_0)})$, and q_c is monotonically decreasing in terms of γ/σ_0 with an upper bound less than 1, as demonstrated in Figure 2.6(b).

The results from the analysis shows that the critical value of the isolation capacity q_c is monotonically decreasing in terms of γ/σ_0, that is the larger recovery rate requires a smaller isolation capacity in order to avoid the isolation reaching the capacity at a finite time during the epidemic process, and the supremum of q_c is $1 - s_0^{\max(0,1/(1-\beta/\sigma_0))}$, as demonstrated in Figure 2.6.

2.6.3 *On the isolation rate*

We can obtain the following theorem to maximize the critical value of the isolation capacity q_c for a finite value of $1/\sigma_0$ (Appendix C).

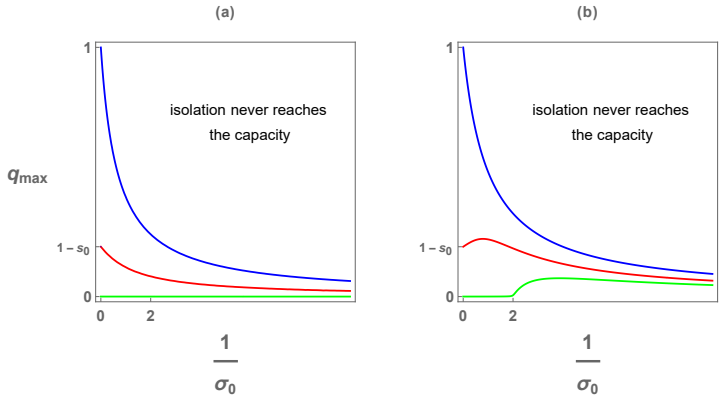

Figure 2.7: $(1/\sigma_0, q_c)$-dependence of isolation reach the capacity. (a) $\beta/\gamma = 0.66$; (b) $\beta/\gamma = 1.5$. Commonly $s_0 = 0.0$ (blue), $s_0 = 0.8$ (red), $s_0 = 0.9999$ (green).

Theorem 2.6.1. *There exists a finite value of* $1/\sigma_0$ *to maximize* q_c *if*

$$\frac{\beta}{\gamma} > \frac{s_0 - 1}{s_0 \ln s_0}. \tag{2.38}$$

Corollary 2.6.2. *There exists a finite value of* $1/\sigma_0$ *to maximize* q_c *only if* $\beta/\gamma > 1$.

Proof. It is easily seen that the right-hand side of (2.38) is greater than 1 for any $s_0 \in (0, 1)$. When $\beta/\gamma \leq 1$, the condition (2.38) cannot satisfied, so inversely it means that $\beta/\gamma > 1$ is the necessary condition to satisfy the condition (2.38). □

When Theorem 2.6.1 is satisfied, there exist at least an extremal maximum for q_c and the numerical calculation implies that the extremal maximum would be unique, as shown in Figure 2.7(b). The numerical calculation imply that when Theorem 2.6.1 is not satisfied, the critical isolation capacity q_c is monotonically decreasing in terms of $1/\sigma_0$, as shown in Figure 2.7(a).

3

CONCLUDING REMARKS

It is obvious that human interactions promote the risk of the spread of an infectious disease in the community, and to reduce the risk of the spread of infectious diseases in the community, "isolation/quarantine" is an important strategy because it can suppress the final epidemic size. The isolation requires a specific space with highly organized conditions to isolate the infected individuals from the others in the community. When the isolation capacity is much small, the isolation strategy may fail in a certain finite time on the way of the epidemic process. In such a case, how does the final epidemic size depend on the limited isolation capacity.

The results from our analysis imply that it is necessary to increase the isolation capacity in order to suppress the final epidemic size. Under the assumption that any isolated individual is not discharged during the season, the isolation affects the infection force. The infection force become larger under such a kind of permanent isolation. At the same time, the isolation can certainly reduce the risk of infection in the community, which must be a positive effect to suppress the spread of the disease. Our theoretical consideration on a mathematical model clearly indicates that the increase of the isolation capacity makes the final epidemic size smaller, while there is such a counteracting effect of the isolation on the epidemic dynamics.

Further, once the isolation reaches the capacity and the isolation becomes incapable, the final epidemic size becomes much large. The occurrence of such a much large final epidemic size depends on the characteristic of epidemic dynamics. When the spread of disease is very slow, the final epidemic size could become much large if the isolation becomes incapable due to the limited capacity. Such a drastic increase in the final epidemic size requires a sufficiently small recovery rate too. This means that such a drastic increase in the final epidemic size could be expected only when the recovery from the disease takes a sufficiently long time. Therefore, the severity of insufficient isolation capacity appears especially for the epidemic dynamics of a transmissible disease such that the infectivity is weak while the disease is hardly treated to the recovery.

The importance of the isolation capacity depends on the social situation, and the isolation capacity is important in such a situation where such a drastic increase could occur in the final epidemic size. On the other hand, if such a drastic increase in the final epidemic size could not occur, the increase of isolation capacity must be effective, while it works as a reduction of the final epidemic size by a certain finite magnitude only. The smaller critical value for the isolation capacity would be better for the controlling of the epidemic dynamics. That is, the smaller critical value for the isolation capacity makes the isolation operation expected to be effective. In contrast, the larger

critical value for isolation capacity indicates a harder situation for the efficiency of the isolation since its mean that a sufficiently large capacity is necessary to make the final epidemic size at a low level.

Consequently, if the spread of a disease is slow and the recovery from the disease takes a long time, then the isolation capacity must be prepared sufficiently large. However, if the epidemic dynamics have a characteristic such that the critical value for the isolation capacity is small, the increase of isolation capacity would have a partial effect to reduce the final epidemic size, and the improvement of another operation against the epidemic dynamics could be more effective for the purpose to make the final epidemic size smaller.

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A

PROOF OF LEMMA 2.5.6

The final epidemic size equation for the case of isolation reaches the capacity can be written as $P(q_{max}, z_\infty^+) = 0$ where

$$P(q_{max}, z_\infty^+) := \frac{\beta}{\sigma_0}\left\{\frac{q_{max}\,(1+\sigma_0/\gamma)}{1-q_{max}} + \ln(1-q_{max})\right\} - \ln(1-z_\infty^+) + \ln s_0 - \frac{(\beta/\gamma)z_\infty^+}{1-q_{max}}. \quad \text{(A.1)}$$

We have

$$\frac{\partial z_\infty^+}{\partial q_{max}} = -\frac{P_{q_{max}}(q_{max}, z_\infty^+)}{P_{z_\infty^+}(q_{max}, z_\infty^+)}.$$

It is easily derived the partial derivatives of (A.1)

$$P_{q_{max}}(q_{max}, z_\infty^+) = \frac{\beta/\gamma}{(1-q_{max})^2}\left(1 + \frac{\gamma}{\sigma_0}q_{max} - z_\infty^+\right)$$

and

$$P_{z_\infty^+}(q_{max}, z_\infty^+) = \frac{-(\beta/\gamma)\left\{1-(\gamma/\beta)(1-q_{max}) - z_\infty^+\right\}}{(1-z_\infty^+)(1-q_{max})}.$$

Hence, we get

$$\frac{\partial z_\infty^+}{\partial q_{max}} = \frac{(1-z_\infty^+)\left\{1+(\gamma/\sigma_0)q_{max} - z_\infty^+\right\}}{(1-q_{max})\left\{1-(\gamma/\beta)(1-q_{max}) - z_\infty^+\right\}}.$$

Then we can obtain

$$\lim_{q_{max}\to 0+}\frac{\partial z_\infty^+}{\partial q_{max}} = \frac{(1-z_\infty^0)^2}{1-\gamma/\beta - z_\infty^0}, \quad \text{(A.2)}$$

where z_∞^0 is the value of z_∞^+ as $q_{max} \to 0+$. The sign of the right-hand side of (A.2) can be determined uniquely by the sign of the denominator. For $\beta/\gamma \le 1$, it is necessarily negative.

For $\beta/\gamma > 1$, we use the final size equation for case when isolation reaches the capacity for $q_{max} = 0$, which is now denoted by $H(z) = 0$ where

$$H(z) := \ln(1-z) - \ln s_0 + (\beta/\gamma)z.$$

It is easily found that $H(1-s_0) > 0$ and $\lim_{z\to 1-} H(z) < 0$ for $z \in (1-s_0, 1)$. We can easily find the unique critical point $z_c = 1-\gamma/\beta$ that is the root of $H'(z) = 0$.

When $\beta/\gamma > 1$, the critical point is positive. If it is not located in $(1-s_0, 1)$ then $H'(z) < 0$, so that $H(z)$ is monotonically decreasing in $(1-s_0, 1)$. Therefore, $1-\gamma/\beta < z_\infty^0$ always holds. Hence, the right-hand side of (A.2) is negative. However, if z_c is located in $(1-s_0, 1)$, then $H(z)$ has an extremal maximum in $(1-s_0, 1)$. Therefore, $1-\gamma/\beta < z_\infty^0$ always holds under $H(1-s_0) > 0$. As a result, the right-hand side of (A.2) is negative. These arguments prove the lemma.

B

PROOF OF THEOREM 2.5.8

The right-hand side of (A.1) can be rewritten as

$$q_{max}\left(1+\frac{\gamma}{\sigma_0}\right)-z_\infty^+=\frac{\gamma}{\beta}(1-q_{max})\ln\frac{1-z_\infty^+}{s_0(1-q_{max})^{\beta/\sigma_0}}.$$

Thus, taking the limit as $q_{max}\to q_c$, we have the following equation with respect to $z_\infty^\dagger$ from (2.20) and Lemma 2.5.4

$$J(z_\infty^\dagger):=z_\infty^--z_\infty^\dagger-\frac{\gamma}{\beta}(1-q_c)\ln\frac{1-z_\infty^\dagger}{1-z_\infty^-}=0. \tag{B.1}$$

It is easily found that $J(z_\infty^-)=0$ and $\lim_{z\to1-0}J(z)=\infty$. Further, if

$$J'(z_\infty^-)=-1+\frac{\gamma}{\beta}\frac{1-q_c}{1-z_\infty^-}\geq 0,$$

then $J(z)>0$ for any $z\in(z_\infty^-,1)$, while, if $J'(z_\infty^-)<0$, there exists a unique value $\zeta\in(z_\infty^-,1)$ such that $J(\zeta)=0$. The former result indicates that, if $J'(z_\infty^-)\geq 0$, the root of $J(z)=0$ in $[z_\infty^-,1]$ is only $z=z_\infty^-$. On the other hand, from (2.29), we can derive

$$\frac{\partial z_\infty^+}{\partial q_{max}}=\frac{1+(\gamma/\beta)\ln\left[(1-z_\infty^+)/\left\{s_0(1-q_{max})^{\beta/\sigma_0}\right\}\right]}{1-(\gamma/\beta)(1-q_{max})/(1-z_\infty^+)}.$$

Then we have

$$\frac{\partial z_\infty^+}{\partial q_{max}}\Big|_{(q_{max},z_\infty^+=(q_c,z_\infty^-)}=\frac{1}{1-(\gamma/\beta)(1-q_c)/(1-z_\infty^-)}=\frac{1}{J'(z_\infty^-)}. \tag{B.2}$$

Hence we find that, if $J'(z_\infty^-)<0$, the derivative (B.2) becomes positive. Thus, if $z_\infty^\dagger=z_\infty^+$ with $J'(z_\infty^-)<0$, z_∞^+ must be smaller than z_∞^- for q_{max} less than and sufficiently near q_c because z_∞^+ is continuous and differentiable for $q_{max}\in(0,q_c)$ and the derivative (B.2) is positive. This is contradictory to the result of Lemma 2.5.3. Therefore, if $J'(z_\infty^-)<0$, $z_\infty^\dagger$ must be ζ which is greater than z_∞^-.

The condition $J'(z_\infty^-)<0$ is equivalent to the following:

$$\frac{\gamma}{\beta}<1 \quad\text{and}\quad q_c<q_{cc}:=\frac{1-\gamma/\beta}{1-\gamma/\beta+\gamma/\sigma_0}. \tag{B.3}$$

From $q_{max}<q_c$ and (2.20), the second inequality of (B.3) is equivalent to

$$1-q_{cc}\left(1+\frac{\gamma}{\sigma_0}\right)>s_0\left(1-q_{cc}\right)^{\beta/\sigma_0}.$$

This inequality results in the second condition of (2.33). If $J'(z_\infty^-)\geq 0$, $z_\infty^\dagger$ must be z_∞^-, since the equation $J(z)=0$ has the unique root $z=z_\infty^-$ in $[z_\infty^-,1]$ and the derivative (B.2) is non-positive with no contradiction. These arguments prove the theorem.

C

PROOF OF THEOREM 2.6.1

The equation (2.20) can be written as $K\left(1/\sigma_0, q_c\right) = 0$ where

$$K\left(1/\sigma_0, q_c\right) := 1 - q_c\left(1 + \gamma\frac{1}{\sigma_0}\right) - s_0\left(1 - q_c\right)^{\beta(1/\sigma_0)}. \tag{C.1}$$

When $1/\sigma_0 = 0$ then $q_c = 1 - s_0$. We have

$$\frac{\partial q_c}{\partial(1/\sigma_0)} = -\frac{K_{1/\sigma_0}\left(1/\sigma_0, q_c\right)}{K_{q_c}\left(1/\sigma_0, q_c\right)}.$$

It is easily derived the partial derivatives of (C.1)

$$K_{(1/\sigma_0)}\left(1/\sigma_0, q_c\right) = -\gamma q_c - s_0\beta\left(1 - q_c\right)^{\beta(1/\sigma_0)}\ln(1 - q_c)$$

and

$$K_{q_c}\left(1/\sigma_0, q_c\right) = -\left(1 + \gamma\frac{1}{\sigma_0}\right) + s_0\beta\frac{1}{\sigma_0}\left(1 - q_c\right)^{\beta(1/\sigma_0)-1}.$$

Hence, we get

$$\frac{\partial q_c}{\partial\left(1/\sigma_0\right)} = \frac{\gamma q_c + \beta\left[1 - q_c\left\{1 + \gamma(1/\sigma_0)\right\}\right]\ln(1 - q_c)}{-\left(1 + \gamma/\sigma_0\right) + \beta(1/\sigma_0)\left[1 - q_c\left\{1 + \gamma(1/\sigma_0)\right\}\right]\left(1 - q_c\right)^{-1}}. \tag{C.2}$$

At boundary edge values

$$\frac{\partial q_c}{\partial\left(1/\sigma_0\right)}_{(1/\sigma_0=0,\ q_c=1-s_0)} \leq 0 \ \text{ if and only if } \ \frac{\beta}{\gamma} \leq \frac{(s_0 - 1)}{s_0\ln s_0},$$

and

$$\frac{\partial q_c}{\partial\left(1/\sigma_0\right)}_{(1/\sigma_0=0,\ q_c=1-s_0)} > 0 \ \text{ if and only if } \ \frac{\beta}{\gamma} > \frac{(s_0 - 1)}{s_0\ln s_0}. \tag{C.3}$$

The right-hand side of (C.2) can be written as

$$\frac{\partial q_c}{\partial\left(1/\sigma_0\right)} = \frac{\sigma_0^2\gamma q_c + \sigma_0\beta\left\{\sigma_0 - q_c\left(\sigma_0 + \gamma\right)\right\}\ln(1 - q_c)}{-\sigma_0\left(\sigma_0 + \gamma\right) + \left[\beta\left\{\sigma_0 - q_c\left(\sigma_0 + \gamma\right)\right\}\right]/\left(1 - q_c\right)}, \tag{C.4}$$

In order to find the sign of the right-hand side of (C.4) for sufficiently large value of $1/\sigma_0$, we use the Maclaurin expansion formula by defining the right-hand side as a function of σ_0

$$L(\sigma_0) := \frac{\sigma_0^2\gamma q_c + \sigma_0\beta\left\{\sigma_0 - q_c\left(\sigma_0+\gamma\right)\right\}\ln(1-q_c)}{-\sigma_0\left(\sigma_0+\gamma\right) + \left[\beta\left\{\sigma_0 - q_c\left(\sigma_0+\gamma\right)\right\}\right]/(1-q_c)}.$$

The Maclaurin expansion formula is given as

$$L(\sigma_0) = L(0) + L'(0)\sigma_0 + o(\sigma_0).$$

It is easily found that $L(0) = 0$, and $L'(0) = (1-q_c)\ln(1-q_c)$. Therefore, we get

$$L(\sigma_0) = (1-q_c)\ln(1-q_c)\sigma_0 + o(\sigma_0). \tag{C.5}$$

The right-hand side of (C.5) has a negative coefficient in the first order of σ_0 , therefore, the sign of $dq_c/d(1/\sigma_0)$ is negative for sufficiently large value of $1/\sigma_0$. So as a consequence, the curve (C.1) is monotonically decreasing for sufficiently large value of $1/\sigma_0$.

Moreover, the curve (C.1) is continuous, and under condition (C.3) with edge value is positive, therefore, the curve is monotonically increasing for a certain range near to $1/\sigma_0 = 0$. Hence, the curve has at least an extremal maximum.