Mathematical Study on the Social Situation-Dependent Prevalence in Epidemics

by

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CONTENTS

DECLARATION

I, Elza Firdiani Sofia, declare that this thesis has been composed solely by myself and that it has not been submitted, in whole or in part, in any previous application for a degree. Except where states otherwise by reference or acknowledgment, the work presented is entirely my own.

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- been composed entirely by myself;
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1

INTRODUCTION

1.1 A SHORT HISTORY OF EPIDEMICS

Epidemics have been a part of the long history of human civilization. One of the earliest records of an epidemic was found in the Amarna tablets which was an archive of correspondence between the king of Egypt and their ancient ambassadors in Canaan during 1300s BC. The tablet mentioned a failure in harvest, cities being attacked, and a plague that consumed the city of Megiddo [41].

An account by Thucydides on the Plague of Athens gave details on an epidemic that struck Greece in its antiquity during 430–426 BCE. In the modern era, now it is known that it was typhoid fever caused by *Salmonella enterica*. Spartan siege was the culprit for quick spread of the disease, as refugees from the countryside hurled into the city-state of Athens under insufficient fulfilment of nutrition and hygiene. [4]

The biggest epidemic that had struck humanity up until now is the black death, wiping 30-50% of medieval Europe's population during the plague in the 14th to 18th century and might even be higher in other parts of the world. This is the event in history that marked the birth of term 'epidemic' from the Greek word επιδημιοσ (*epidemios*) (from επι, epi 'upon', and δημοσ, demos, 'people', i.e. 'between people'), addressing the infectivity of the disease. Impoverished urban communities were the ones who got hit the hardest as they lived in such crowded small houses [4].

Paleobiological research and excavation of ancient tombs across Russia and Europe by Spyrou and her team concluded in the likely origin of *Yersinia pestis*, the cause of the Plague, to be around Volga river that empties into Caspian Sea. Genomic analysis found the strain that had infected the first patient in Barcelona in 1348 to be closely related with the original Volga ancestors [44]. As a flea-borne disease, its entry into Europe was purported by imports of goods coming from Central Asia which incidentally brought the flea-infested rats living in containers. The plague in the medieval era also brought about the modern term 'quarantine', from Italian *quaranta giorni* '40 days', the length of period required for ships to anchor themselves into the port before entering the port city of Venice to avoid newly-coming ships infecting the coastal cities. [9] The strain that infected Europe made its way to cause plague in China during the late 19th century [44].

Until then, there was no specific application of mathematical treatment for investigating disease spread until Daniel Bernoulli (1760) proposed a mathematical model based on statistical data of smallpox outbreak to calculate the life expectancy if smallpox were ruled out from a cause of death [6]. The work was originally conceived for addressing the actuarial issue as time annuities were being sold at that time, making his contribution to epidemiology was rather unintended [19]. The model itself was still premature for an actual epidemiological literature in addition to the limited understanding of biology and bacteriology in that period of time [15].

However, the advancement of bacteriology by the time of Spanish flu outbreak in 1918 ironically caused a misidentification of the cause of the pandemic [49]. German bacteriologist Richard Pfeiffer isolated *Haemophilus influenzae* from a patient and thought that it was the cause of the outbreak even though the bacteria were not always be found in every patients of influenza at that time, and his hypothesis ("Pfeiffer's doctrine") was much objected by other experts at that time [51]. Though the disease itself was unlikely originated from Spain, it had been named so because at that time only in Spain, a neutral country during the World War, media reportings were not restricted to censorship [50]. The disease spread quickly due to the advances in distant traveling by train and big carnival events, in addition to intentional information restriction by many governments during the war. It was one of the most devastating pandemic in the modern era, up until COVID-19 [29].

In the recent times, the global spread of COVID-19 [18] has been a constant fight for many communities, especially with the high human-to human transmissibility via respiratory droplets and airborne particles containing the virus [8]. Individuals infected with SARS-CoV-2 will show symptoms such as fever, sore throat, fatigue, and cough, within 4 to 19 days of recent exposure [56, 57]. Delayed onset of the symptoms and the ever-increasing mobility of human only promotes the spreading of the disease in global and local scales [28]. Additionally, there was no specific anti-SARS-Cov-2 treatment for patients with suspected or confirmed COVID-19 infection [45] during the conceptualization of this work, although in mid-2021, COVID-19 vaccine became readily available [30] The severity of COVID-19 vary according to the immune response of the infected individuals, from asymptomatic or mild symptomps that recover within two weeks to pneumonia, cytokine storm, acute cardiac and renal damage, and eventual death [45]. Even after the invention of COVID-19 vaccine, efficient control of disease spread still requires active efforts in non-pharmaceutical interventions (NPI) such as social distancing, masking, hand-washing habit, frequent testing for infection, real-time information update, and so on [11, 30].

1.2 EPIDEMIC OUTBREAKS AND HUMAN ACTIVITIES

The world's population started to double around mid-twentieth century and this was not without any consequences [12]. Advancements in transportation shortened the time required to cover a distance; as a result, infectious diseases spread faster especially in the recent history where international travels become easily accessible for most [21]. Humans as a species thrived so well that their travel and activities grow into something more massive that it was possible to grow food, do trade, have employment, wage war, and — in peaceful modern period — to spend leisure time [12]. Living in communities, humans associated together in groups. Communities interact with each other, building a certain network of society. This comes with a risk, however. Now a sick individual in the group may easily widespread his or her malady to the healthy especially in the absence of the understanding of disease and an effective public health infrastructure [42].

In the previous chapter, the Plague of Athens was mentioned and how refugees from the countryside contributed to the quick spread of typhoid fever. Thucydides described clearly that the epidemic entry was via the port trading route [4]. Moreover, the black death plague spread along the trade routes [44]. These examples in history have proved how human activity and travels were major factors in disastrous epidemics. [4, 12].

Current modern combination of growing population and ease of traveling accelerate the spread of infection communities across the world [12], such as in the case of COVID-19. Recently, it has been easy to track activities by using smartphones. Users carry their mobiles in their proximity, making phones to be an excellent platform for sensing human activity distribution [16]. Previously, it has been demonstrated that mobility data derived from phone can be utilized, among many, to investigate the relationship between travel, activity level, and disease spread. In Japan, mobile spatial statistics (MSS) were utilized to collect statistics of the actual population that are generated continuously from mobile terminal network operational data [36, 47]. Hara and his colleague [20] analyzed MSS to find for a trend in travel during and after the state of emergency during early wave of COVID-19. Their results showed that there was a significant reduction in trips and a decrease in population density index by 20% nationwide as people avoided traveling to densely populated areas. In Sapporo City, reduction of travel as much as 70-80% was reported after an analysis by Arimura and his colleagues [1]. In Chapter 3 of this work, the MSS data during the later waves of COVID-19 is used to analyze the relationship between the number of new COVID-19 cases and activity level.

Other works utilizing travel data in countries such as the USA [2, 26, 40, 53, 54] and Japan [33] during the early stages of COVID-19 pandemic drew correlation between social distancing and decreased

growth rate of COVID-19 case. Combination of phone signal and social media location tag data gave similar conclusion for UK [25] and Taiwan [10], as reduction in travel was uniform all over the countries. In Indonesia, the travel frequency in rural areas were still high compared to bigger cities with more awareness of the pandemic, and indeed the spread of COVID-19 in such rural areas was relatively quick [24].

It is clear that activity level and the risk of infection in communities have a tight relationship with each other. Activity level, reflected by the habit of travel, is a significant element to be analyzed in order to understand the course of disease spread and how communities react to such type of danger [39].

1.3 DISEASE PREVALENCE AND SOCIAL CHARACTERISTICS

Berkman and Kawachi defined social epidemiology as "the branch of epidemiology that studies the social distribution and social determinants of states of health" [5, 23]. It focuses especially on how social-structural factors affect the states of health. What societal characteristics affecting the pattern of disease and health distribution in a society as well as its mechanisms are the points of interest. Examples of the identifiable characteristics are social class, gender, race, ethnicity, discrimination, social network, social capital, income distribution, social policy, and so on [23]. The analysis may encompass the prevalence of both non-communicable and communicable diseases, though in this current work we will only focus on communicable diseases and an explanation of the mechanism on how the social factors affect the communicability would require a separate, careful study.

Diseases are assumed to be byproduct of mutual interaction among important three factors: social, individual, and biological [42]. In social epidemiology, social factors can be risk factors of health by adding to or interacting with individual and biological factors [23]. Asking why a population has a particular distribution of risk is different from the etiological question of why a particular individual got sick [42]. Social epidemiology may contribute to public health by providing hints for social, environmental, and political intervention [23]. The threats posed by infectious diseases today are being amplified by social, behavioral and environmental factors that accelerate the natural phenomena that modify infectious disease patterns [21].

COVID-19, the cause of the 2020 pandemic, has infected over 500 million people worldwide, caused at least 6 million deaths [7, 52]. Social distancing and masking as a form of Non-Pharmaceutical Intervention (NPI) has been enacted in most of the countries around the world for reducing the spread of the virus, even after vaccines were widely distributed in the mid-2021 [30]. Many countries have implemented strict quarantine, isolation, mobility limitation in other forms, or social distancing policies early in the epidemic [35, 48]. Understanding social

situations will help us develop optimal strategies for both ''flatten the curve'' and ease the society burden due to economic stagnation, deaths, and changes in habit as well as culture [3, 46].

People adapt their behavior according to the perceived notion of social norms, and more often than not, their perceptions are often inaccurate. Underestimation of health-promoting behaviours and overestimation of unhealthy behaviors generally contribute to the habits against infection [27]. In addition, social networks can amplify the spread of behaviours that could be harmful and beneficial during an epidemic [27], even causing mass panic via rumors in social media [14].

Slowing viral transmission during pandemics requires significant shifts in behaviour. Various aspects of social and cultural contexts influence the extent and speed of behaviour change. Works by Dryhurst and Bhuiya showed how risk perception about COVID-19 differed between countries, and the personal risk perception citizens felt was influenced by both country and income [7, 17]. Therefore, it is useful to look at disease spread by using the lens of community, as commonly shared characteristics and habits could be determining factors on the prevalence of diseases.

2

A MODELING ON THE INFECTION RISK BY SHOPPING

2.1 INTRODUCTION

The COVID-19 pandemic has changed the mobility of people around the world in a significant way. More than a hundred countries had exercised different types of mobility restriction, from strict lockdowns to loose suggestions of activity restrictions in public sphere [39]. In the case of Japan, a lockdown especially a strict one is not an option because the Constitution of Japan states that citizens should be allowed to move freely in addition to the absence of law that enforces the citizens to stay at home [20]. Encouragements for NPIs such as social distancing, limitation of public facilities usage by a large group of people, and refraining from activities after the declaration of the state of emergency were instead observed [39].

In early 2020, Japanese public interest in COVID-19 was rather limited. As the docking of cruise ship Diamond Princess and the repatriation of its passengers made into national news in February 2020, COVID-19 became a more familiar issue[20]. On April 7th, seven sizable prefectures (Tokyo, Saitama, Chiba, Kanagawa, Osaka, Hyogo and Fukuoka prefectures) were placed under a state of emergency until May 6. Temporary closing of offices, schools, and limitations of commercial establishment such as restaurants and shops were recommended by Japanese Ministry of Health, Labour and Welfare following a suggestion for the citizens to avoid leaving their homes for non-essential activities [31]. This was the situation when the model in the following sections was conceptualized, as "stay-at-home" was the main strategy in place with certain limitation of activities being suggested.

2.2 ASSUMPTIONS AND MODELING

Let us assume a local community with two shops and the community is in a situation where going out is essentially not recommended due to an outbreak of an infectious disease. In this situation, we assume the situation and the setup of the community as follows (Figure 1):

- Two shopkeepers from two different shops, Shop 1 and 2;
- There are n customers;

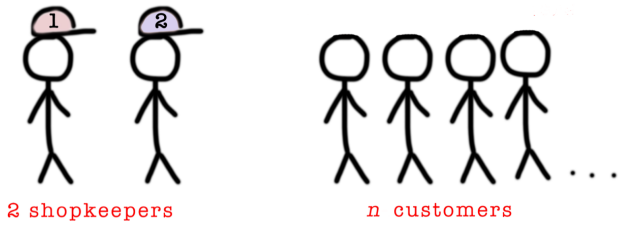

Figure 1: Set-up for the modeling in this chapter

- All are in the same community;
- Customers have preferences in choosing shops.

To factor the disease spread in the community, we set further assumptions:

- A shopkeeper and a customer interact with probability of infection β;
- Customer's preference is represented by the probability p of going to Shop 1 and $(1-p)$ to Shop 2;
- No interaction between shopkeepers is assumed;
- Customers can visit only opened shops;
- Closed shops provide delivery services to customers who stay at home, but opened shops do not provide such a service.

Let us consider the total expected number of infections E:

$$E = E_C + E_S$$

with the expected number of infections E_C and E_S for the customers and shopkeepers respectively. Let us have the probability of infection β when a shopkeeper and a customer interact. The interaction between customers bring a certain risk of infection with probability $s\beta$, where s is the average closeness of interaction between the customers compared to the interaction between the customer and shopkeeper. In the case of delivery, let us assign the probability $\epsilon\beta$ to be the risk of infection that arises from the interaction between the deliveryperson and customer. We assume $\epsilon < 1$ as the interaction between the deliveryperson and customer shall be brief and much shorter than the interaction in the physical shop. In this modeling, the probability β can be regarded as the infectiousness of the disease in question.

In our set-up, the shops may be opened like usual or closed but with delivery service. Such a set-up will result in three possible situations:

Case I: All shops are open.

In this situation there are interactions between the shopkeeper and customers and also between the customers.

The distribution of the customers depends on the shopping preference which is now introduced by pn and $(1-p)n$ for Shop 1 and 2, respectively. For a customer of Shop 1, he/she would need to escape from infections coming from other $pn-1$ customers with probability $(1-s\beta)^{pn-1}$ in addition to escaping the infection from the shopkeeper with probability $1-\beta$. Overall, the probability for a customer of Shop 1 to do shopping without getting infected is $(1-s\beta)^{pn-1}$ and thus the probability of infection is $1-(1-s\beta)^{pn-1}$. The risk of infection for a customer shopping in Shop 2 follows a similar fashion, that is, with probability pn: $1-(1-s\beta)^{(1-p)n-1}$. For the shopkeeper, the probability of escaping new infection after consecutive contacts with the customers is $(1-\beta)^{pn}$ for Shop 1 and $(1-\beta)^{(1-p)n}$ for Shop 2, and the probability of infection is given by $1-(1-\beta)^{pn}$ and $1-(1-\beta)^{(1-p)n}$, respectively. From those descriptions, we have the following expected numbers of new infections E_C and E_S for customers and shopkeepers in Case I:

$$\begin{aligned} E_C =& pn[1-\{(1-s\beta)^{pn-1}(1-\beta)\}] \\ &+ (1-p)n[1-\{(1-s\beta)^{(1-p)n-1}(1-\beta)\}]; \\ E_S =& \{1-(1-\beta)^{pn}\}+\{1-(1-\beta)^{(1-p)n}\}. \end{aligned} \tag{1}$$

Case II: One shop is open and the other is closed

In this case, the open shop — let us assign it as Shop A — can be treated similar to Case I but with a modified distribution of customers $p'n$. There are $(1-p')n$ customers who stay home and have items delivered to their homes from the closed shop. Interactions between customers, the customers and shopkeeper in Shop A, and between the customer and deliveryperson — the shopkeeper of Shop B, i.e. the closed shop — must be taken into account. As the interaction between the deliveryperson and customers at home is significantly shorter, the infection probability for customers receiving the delivery is now given by $(1-p')n\epsilon\beta$. For the deliveryperson, the infection probability for every delivery is $\epsilon\beta$, and the deliveryperson escapes infection with probability $(1-\epsilon\beta)^{(1-p')n}$ for all deliveries he/she does. Therefore, the probability of infection for the deliveryperson is $\{1-(1-\epsilon\beta)^{(1-p')n}\}$. For ϵ, it has to be $0<\epsilon<1$ as there will always be a probability to get an infection from the deliveryperson and we do not expect the case where the deliveryperson will absolutely infect the customer. The expected numbers of infection in Case II are given by

$$\begin{aligned} E_C &= p'n[1-\{(1-s\beta)^{p'n-1}(1-\beta)\}]+(1-p')n\epsilon\beta; \\ E_S &= \{1-(1-\beta)^{p'n}\}+\{1-(1-\epsilon\beta)^{(1-p')n}\}. \end{aligned} \tag{2}$$

Case III: All shops are closed.

In this case, that all the shops are closed and all customers stay at home, every customer has their item delivered by their preferred shop, following similar distribution as in Case I, with p and $(1-p)$ for Shop 1 and 2, respectively. The possible interaction is only between the customer and shopkeeper who is now the deliveryperson. The infection probability for a customer is $\epsilon\beta$. For the deliveryperson, the probability of escaping from the infection by a customer is $1-\epsilon\beta$, and the probability of escaping from the infection at all the deliveries becomes $1-(\epsilon\beta)^{pn}$ and $(1-\epsilon\beta)^{(1-p)n}$, respectively for Shop A and Shop B. Thus the probability for the deliverers to get the infection is $1-\{1-(\epsilon\beta)^{pn}\}$ and $1-\{1-(\epsilon\beta)^{(1-p)n}\}$. We have the following expected numbers of new infections case in Case III:

$$\begin{aligned} E_C &= n\epsilon\beta; \\ E_S &= \{1-(1-\epsilon\beta)^{pn}\}+\{1-(1-\epsilon\beta)^{(1-p)n}\}. \end{aligned} \tag{3}$$

2.3 ANALYSIS ON THE MODEL

We compare every two of cases in the previous section to find the least expected number of new infections in which situation the infection risk is considerably lower. The difference E_1-E_2 between the total expected numbers of new infections in Case I and Case II, E_1 and E_2, that is a way to find which case is the worse situation.

First let us consider a specific situation with $p'=1$, when all customers in Case II go to Shop 1. In this situation, $E_1-E_2<0$ for a sufficiently small β, while $E_1-E_2>0$ for sufficiently large β (Appendix A.1).

Next, let us consider the other specific situation with $p'=0$. All customers in Case II stay home, which can be regarded as equivalent to Case III. In this case for sufficiently large $\beta\leq 1$, we have $E_1-E_2>0$ for any value of $\epsilon\beta\leq 1$ (Appendix A.2). In contrast, if β is sufficiently small, we have $E_1-E_2<0$ for sufficiently large ϵ. This result indicated that, there is a critical value of β for which the sign of E_1-E_2 changes.

This result shows that it depends on the infectivity of the disease which is the worse, Case I and II. For the higher infectivity, Case I is worse than Case II, while the latter becomes worse even for the low infectivity if the delivery causes a high risk of infection for the customers.

2.4 DISCUSSION

We compared the infection risks in three possible situations making use of the expected numbers of new infections under a limitation of non-essential activity: no limitation at all (first situation), a certain limitation

(second situation), and finally a semi-lockdown with movements of limited persons (third situation). Although intuitively the first situation would have the highest infection risk, it is not necessarily so according to this model. The second situation could result in a higher infection risk for the community members given that the preference for visiting a public place, i.e. shops (or the awareness to stay home) represents a nature of the community. If the preference to visit a non-restricted facility is very high the infection risk could be higher than that with limitation for the activity.

Even in the first situation where there is no limitation of activity, the interaction between the customers or the customer and shopkeeper could be controlled by social distancing, masking, and using contactless payment. The only feasible option by the shop is to control the customer density. In the second situation, the awareness of customers to consciously choose to stay home and get their items delivered to their doors highly depends on the nature of community. A community with an open shop in close distance may have a considerable amount of people still visiting the shop in person, compared to where the opened shop is located farther from the residential area, for which the delivery is more convenient the customers. In this case, matching the open/closed strategy with the shopping habit of the customers may be an ideal approach to limit the risk of infection caused by shopping. Indeed, shopping to any actual brick-and-mortar shop is the activity which has the least reduction in frequency compared to other possible activities outside home [39].

In this research project, we do not consider any strategy for the community to reduce the epidemic prevalence, that is the infection risk. It may be the shopkeeper's choice whether the shop is opened or closed under an epidemic. The choice by the shopkeeper must depend also on the economic/commercial reason to maintain the business. It may depend on the reputation of the shop with the choice under a situation of disease spread in the community. Thus, the result of the choice could depend on the response of customers for the shop which represents a nature of the community/ Such a situation could be theoretically regarded as a game between the shop(s) and customers. The choice/preference of customers on which they use going to a shop or ordering by the delivery could be a strategy, while the strategy of shop could be to open, close, change to the delivery-based business. Our research is not to purpose any discussion on such an aspect of theoretical game, although it would be an intersting theoretical problem. Further, in contrast to our simplest assumption with a mean-field approximation, it would be possible to introduce a heterogeneity of customer's choice, for example, with a certain distribution of a parameters representing the individuality in the customers. In such a modeling, the distribution could be regarded as a representation of the nature of community. This one of future studies related to ours.

Shopping does take place in a public place with a chance of exposure to people from different neighborhood, which may or may not carry a significant risk of infection, unbeknownst to the individuals carrying out the activity. On the other hand, people also spend their time in their private space with family or neighbors in a residential area. This is not without any risk, and we will explore in the next chapter about how the social situation, in the context of human activity, affect the infection risk in the community.

3

A MODELING FOR THE DEPENDENCE OF INFECTION RISK ON COMMUNITY STRUCTURE

3.1 INTRODUCTION

In this chapter, we construct and analyze a mathematical model to the consider the correlation between the social activity and the infection risk in a community. In our modeling, the infection risk is indexed by the expected number of new cases. We take into account the activity levels and the sphere where the activity takes place, according to the types of social interaction held. We shall try to discuss the relation of the infection risk to the social activity level and activity sphere.

In the first wave of COVID-19 pandemic, Japanese government announced a state of emergency in April 2020 for seven prefectures with large populations [31]. During the state of emergency, restrictions of non-essential activities were suggested by the government. Schools were closed and many offices arranged the workers to do work from home (WFH). The trend of WFH, or teleworking, had increased between January and June 2020, peaking around May 2020 (25% average increase) nationwide in Japan when the state of emergency was in effect [37, 38]. Mobility control reduced long-distance travel by 90% [11] at that time. An analysis by Nagata and his colleagues [33] using mobile phone signal data showed a strong association between work, nightlife, and residential location mobility in Japanese metropolitan areas with COVID-19 incidences. Mobility changes in locations related to nightlife were more significantly associated with the outbreak than other types of location [33]. Similarly, we investigated the correlation between activity level reflected by the mobile phone signal density and the recorded number of new COVID-19 cases. We picked Shibuya crossing as the representative location since it is one of the busiest district in Tokyo area [13] and number of new COVID-19 cases recorded for the city of Tokyo.

By our calculation to get their correlation described in Appendix B, we found that there could a positive correlation between the rise of activity and number of new cases (Figure 2), in agreement with previous demonstrations that increased mobility positively contribute to the spread of COVID-19 [53]. There must be a lag between the peaks in the new cases and peaks in the crowd density, because of a necessary

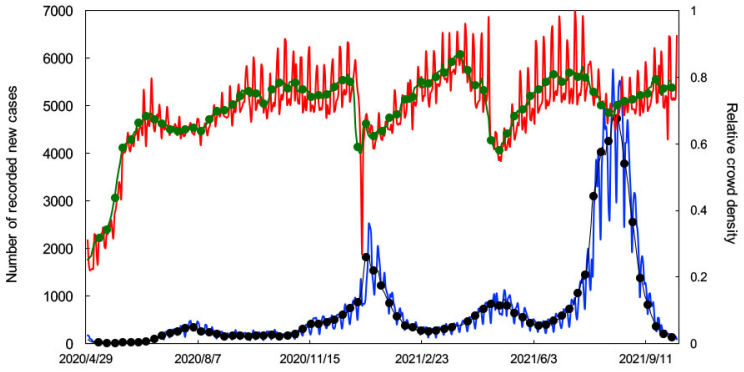

Figure 2: Graph of relative crowd density in Shibuya crossing from MSS data [36] and recorded new COVID-19 cases in Tokyo Metropolitan area from May 2020 to September 2021 [34]. The upper fluctuating graph indicates the relative crowd density where the weekly average is drawn together. The lower fluctuating graph indicates the number of new cases, where the weekly average is drawn together.

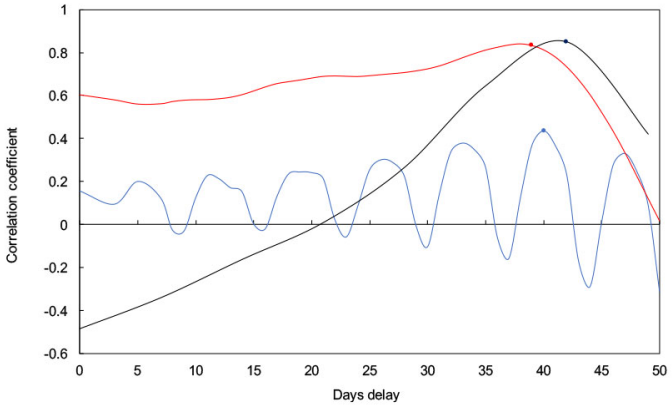

Figure 3: Graph of the correlation coefficient between the activity level and the number of new infection cases in Figure 2 calculated using raw (blue, peaked at exactly 40 days), weekly average (black, peaked at 42 days), and 7-days past average relative (red, peaked at 38 days) data.

latent period for the COVID-19 and a technical or behavioral lag for the detection of new cases. Our calculation on the data shows that about 40 days lag could result in the greatest correlation coefficient between them (Figure 3). Although this lag seems much longer than the averaged latent period, two weeks for the COVID-19, we could not find the reasonable explanation about it. However we consider that this result could be regarded as an example of the positive correlation of new cases to the social activity.

3.2 ASSUMPTIONS AND MODELING

Let us assume a community which is composed with two classes based on their activity level in daily life: less-active and active. The less-active class typically includes elderly and infants who do not spend a significant portion of their day outside their residential area. The active class members partake their activities both in the residential area and public places out of their residential area (Fig. 4).

We here assume only two different phases about the activity sphere as previously done by Seno [43]: private and social phases. Private phase is defined as the activity sphere mainly in the residential area with limited interactions with the family members, neighbors, and house staff. Traveling by personal car or bicycle is considered to be at the private phase. At the social phase, the interactions are with arbitrary members of the community in the public sphere which may include but not limited to work, school, shops, public transportations, etc. The less-active class members have activities at only at the private phase, and the active class members have those at both the private and social phases. At the private phase, the epidemiological contact between members of two classes is possible. In contrast, the epidemiological contact at the social phase happens only between members of the active class. The likelihood of infection is assumed to be different according to the interaction that takes place at each phase, contributed by both the active and less-active classes at the private phase and solely by the active class at the social phase.

The member of less-active class has a probability to get infected only at the private phase. The member of active class has a probability to get infected at the private or social phase. Higher risk of infection must result in a higher expected number of new cases.

Expected number of new cases for the less-active class

Let N denotes the population size of the community. The sizes of active and less-active classes are now given by qN and $(1-q)N$ respectively with the ratio q of active class in the community. Now we define the

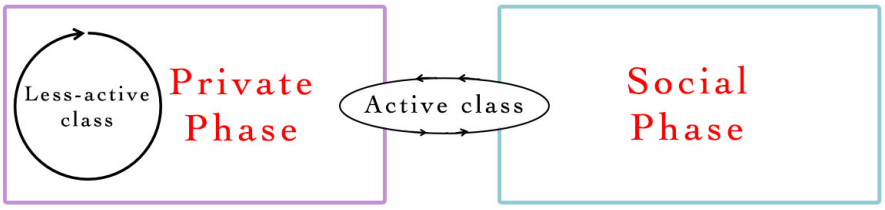

Figure 4: The active class and less active class and their activity sphere.

expected number of new cases in an appropriate time unit for the less-active class as

$$E_l(\alpha, q) = \beta_p(1-q)N, \tag{4}$$

where β_p is the probability of infection for an individual at the private phase. Since β_p may be contributed by may be contributed by active and less-active individuals, E_l is denoted here as a function of α and q in general.

Expected number of new cases for the active class

Let us introduce here a constant parameter α $(0 < \alpha < 1)$ that indexes the mean proportion of time at the social phase of the member of active class in a daily life. Then, let $\alpha\beta_s$ be the probability for an individual of active class to get infected at the social phase. The individual may get infected at the private phase with probability $(1-\alpha)\beta_p$. Thus, the individual of active class can avoid getting infected at the private phase and social phase to keep being uninfected with probability $\{1-(1-\alpha)\beta_p\}(1-\alpha\beta_s)$. Hence, the infection occurs for the individual of active class with probability $1-\{1-(1-\alpha)\beta_p\}(1-\alpha\beta_s)$. Therefore, we define the expected number of new cases for the active class as

$$E_a(\alpha, q) = [1-\{1-(1-\alpha)\beta_p\}(1-\alpha\beta_s)]qN. \tag{5}$$

Infection risk for the community

The infection risk for the community is now indexed by the expected number of new cases $E(\alpha, q) = E_l(\alpha, q) + E_a(\alpha, q)$ where $E_l(\alpha, q)$ and $E_a(\alpha, q)$ are given by (4) and (5). In this work, we introduce the following formulas for probabilities β_s and β_p:

$$\begin{aligned} \beta_s &= \beta_s(\alpha, q, N) = \sigma_s\alpha qN; \\ \beta_p &= \beta_p(\alpha, q, N) = \sigma_p\{(1-q)N + (1-\alpha)qN\}, \end{aligned} \tag{6}$$

where αqN corresponds to the expected population density at the social phase and $(1-q)N+(1-\alpha)qN$ does to that at the private phase. Positive constants σ_s and σ_p are the infection coefficients at the social and private phases, respectively. This formulation is based on the assumption that

the infection probability has a positive correlation to the population density. For the well-definition of probabilities β_s and β_p, our modeling leads to a confinement of parameters σ_s and σ_p such that $\sigma_s N \leq 1$ and $\sigma_p N \leq 1$.

In the following sections, we will consider the dependence of E to parameters q and α. These parameters characterize the community structure. Parameter q may correspond to the characteristic hardly changeable (age structure, for example) in the epidemic dynamics, while α may be changeable like the behavioral nature of the active class in the community,which could be influenced by governmental policy, social perception, campaign, education, and so on. Focusing on these two parameters, we will try to discuss how the infection risk depend on the community structure.

3.3 ANALYSIS ON THE MODEL

3.3.1 *Dependence of the infection risk on the class size*

We can get the following result about the q-dependence of the expected number of new cases E(Appendix C):

Theorem 3.3.1. *The expected number E is monotonically decreasing in terms of $q \in (0,1)$ if and only if $\alpha \leq \alpha_c$, where α_c is given by the unique root of the cubic equation*

$$-3\alpha^3 + 5\alpha^2 + 2A\alpha - \frac{2}{\sigma_s N} = 0$$

for $\alpha \in (0,1)$, where

$$A := \frac{1}{\sigma_s N} + \frac{1}{\sigma_p N} - 1. \qquad (\#\#)$$

Otherwise, when $\alpha > \alpha_c$, E has a unique extremal minimum at $q = q^* \in (0,1)$ where q^* is given by

$$q^* = \begin{cases} \dfrac{1}{3(1-\alpha)\alpha}\Big\{ -(A+\alpha) + \sqrt{(A+\alpha)^2 + \dfrac{6(1-\alpha)}{\sigma_s N}}\Big\} & \text{for } \alpha \in (\alpha_c, 1); \\ \dfrac{1/(\sigma_s N)}{1/(\sigma_s N) + 1/(\sigma_p N)} & \text{for } \alpha = 1. \end{cases} \qquad (7)$$

When the active class has the social phase sufficiently longer than the private phase, there is a certain proportion of active class for which the expected number of new cases becomes minimum. Otherwise, the expected number of new cases is smaller as the size of active class gets larger.

3.3.2 *Dependence of the infection risk on the activity*

We can derive the following result on the α-dependence of the expected number E (Appendix D):

Theorem 3.3.2. *The expected number of new cases E is monotonically decreasing in terms of $\alpha \in (0,1)$ if and only if $q \leq q_c$, where q_c is defined by*

$$q_c := \frac{3}{2} + A - \sqrt{(\frac{3}{2} + A^2) - \frac{2}{\sigma_s N}}. \tag{8}$$

Otherwise, when $q > q_c$, E has a unique extremal minimum at $\alpha = \alpha^*$ which is the unique root of

$$-4\alpha^3 + 3(1 + \frac{1}{q})\alpha^2 + \frac{2A}{q}\alpha - \frac{2}{\sigma_s N q^2} = 0 \tag{9}$$

for $\alpha \in (0,1)$.

When the active class is sufficiently larger than the less-active class, there is a certain length of the social phase for which the expected number of new cases becomes minimum. Otherwise, the expected number of new cases becomes smaller as the length of the social phase gets longer.

3.3.3 *Social situation to minimize the infection risk*

First we find the following result from the results obtained in the previous section:

Lemma 3.3.3. *The expected number of new cases E cannot become minimum for $\alpha = 0$ or $q = 0$.*

The case of $\alpha = 0$ is the situation in which the members of active class always stay at the private phase, in other words, every individual in the community is of the less-active class. Such a situation could be regarded as the community under the complete lockdown. Hence, this result implies that the complete lockdown could not minimize the infection risk in the community. Therefore, the expected number of new cases E may become minimum when (α, q) is one of the following cases: $(1,1); (1,q^*); (\alpha^*,1); (\alpha^*,q^*)$ with $\alpha^* \in (0,1)$ and $q^* \in (0,1)$.

The case of $(\alpha, q) = (1,1)$ could be taken into account only when $\alpha \leq \alpha_c \in (0,1)$ and $q \leq q_c \in (0,1)$ because this is the case when E is monotonically decreasing in terms of $\alpha \in (0,1)$ and $q \in (0,1)$ as shown in the previous lemmas. Thus the case when $\alpha = 1$ and $q = 1$ is contradictory to the condition. Therefore, this case cannot make the value of E minimum. The case of $(\alpha, q) = (1,1)$ is corresponding to the situation such that every individual belongs to the active class and always has activities at the social phase. This result matches our

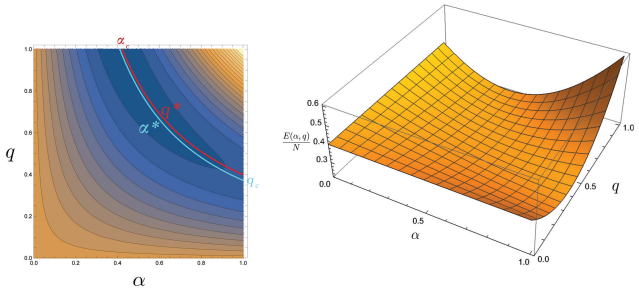

Figure 5: Numerically obtained (α, q)-dependence of $E(\alpha, q)$ with $\sigma_s N = 0.6$, $\sigma_p N = 0.4$, $q_c = 0.371967$, and $\alpha_c = 0.42152$. Contour map (left) and three dimensional graph (right).

intuition that the highest active situation would be worse with respect to infection risk.

For the other cases of $(1, q^*)$ and (α^*, q^*), we can prove that they cannot make E minimum either, and consequently we have the following result (Appendix E):

Theorem 3.3.4. *The expected number of secondary cases E becomes minimum for $(\alpha, q) = (\alpha^*, 1)$ such that $0 < \alpha^* \leq \alpha_c < 1$.*

The result of Theorem 3.3.4 is visualized by the numerical calculation for the value of E in Fig. 5. Theoretically, it is implied that the situation with no less-active class minimizes the infection risk. This may be translated as the less-active class having a higher density at the private phase, which raises the infection risk, whereas such a situation in an established community could not be realistic in general. On the other hand, a specific situation in a temporarily organized village like that in the Olympiad may be applicable. Then, Theorem 3.3.4 implies that the infection risk could be minimized by controlling the daily schedule in the village to an appropriate extent about the activity there.

As for the dependence of $E(\alpha^*, 1)$ on parameters $\sigma_s N$ and $\sigma_p N$, we find that $E(\alpha^*, 1)/N$ becomes larger as $\sigma_s N$ or $\sigma_p N$ gets larger. This is an intuitively expected result because the larger $\sigma_s N$ or $\sigma_p N$ means the higher risk at the social or private phase.

From the results about the q-dependence and the α-dependence of E obtained in Lemmas 3.3.1 and 3.3.2, we find that the management of infection risk is significantly restricted by the community structire, and get the following result:

Lemma 3.3.5. *The expected number of new cases E becomes minimum for $\alpha = 1$ when $q \leq q_c$, while it becomes minimum for $q = 1$ when $\alpha \leq \alpha_c$.*

The situation of $\alpha = 1$ means that the case when the active class is always at social phase. That is, the individual of the active class is never at private phase. This may be regarded as the complete separation of the active class from the less-active one, or of the less-active class from the

active one. This could be adapted only for some specific situations, for example in a temporarily organized village of social event like Olympics or carvinals.

The situation of $\alpha = 1$ means that the active class is always at the social phase. That is, the member of active class is never at the private phase. This may be regarded as a complete separation of the active class from the less-active one, or of the less-active class from the active one. However, it cannot be adapted for most of community-level risk management about the spread of a transmissible disease because such a complete separation of active and less-active classes can be hardly realized. In general, the less-active class may consist of elders, infants, and people with disabilities in the community. Thus, the proportion of active class q is hardly changed. On the other hand, it would be possible to control the proportion of the social phase α. For example, limiting the office work, the school time, or prohibiting from going out for a certain period may be imposed to control the infection risk.

3.3.4 *Classification of community according to the infection risk*

As every community is characterized by its own α and q, we can classify communities according to the infection risk. Based on the results obtained for our mathematical model, we can categorize the community structure into three types as shown in Figure 7. Type I community is for $q \leq q_c$, Type II is for $q > q_c$ and $\alpha \leq \alpha_c$, Type III is for $q > q_c$ and $\alpha > \alpha_c$. As shown in Lemma 3.3.1 and 3.3.2, α_c and q_c are uniquely determined by ($\sigma_s N$ and $\sigma_p N$), as numerically seen in Figure 5. They always exist in terms of $\sigma_s N$. Since parameters $\sigma_s N$ and $\sigma_p N$ are factors to define the infection probability at social and private phases respectively in the infection probability at social and phases respectively in our modeling, this dependence of α_c and q_c can be regarded as the dependence on the infection probability at each phase, which must reflect the characteristics of community according to the epidemics. This dependence is quantitative and dependent on the modeling of the infection probability, whereas it does not change the qualitative result of the dependence of E on (α, q). Thus, we consider hereafter only the qualitative aspect.

In Type I community, the expected number of new cases becomes smaller as the proportion of the social phase gets larger. Therefore, the reduction of time at the social phase could not be appropriate to reduce the infection risk in such a community, which could be regarded as a modern aged community with a large proportion of aged people.

In Type II community, there is a relatively large active class. which has a sufficiently small proportion of the social phase. It would be characterized by a specific custom of social activities including working, which provides a daily life with a sufficiently short time for the social activity. For example, this may be regarded as a community with sufficiently effective telecommunication and teleworking which

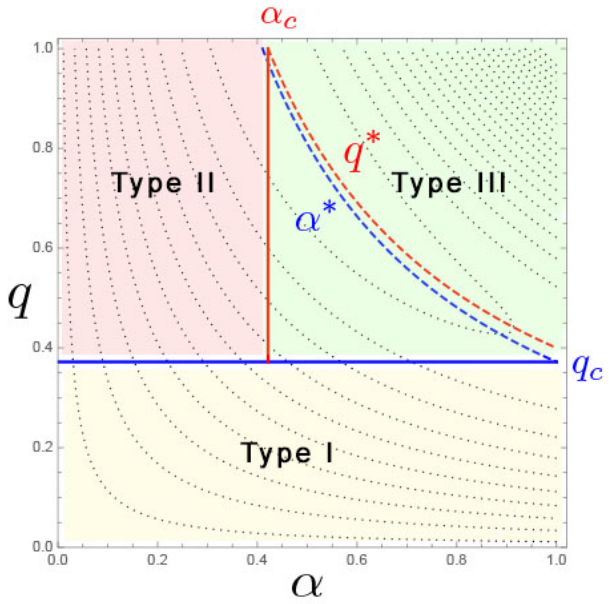

Figure 6: Classification of the community structure indexed by (α, q). Numerically drawn with parameter values, same with those for Figure 5.

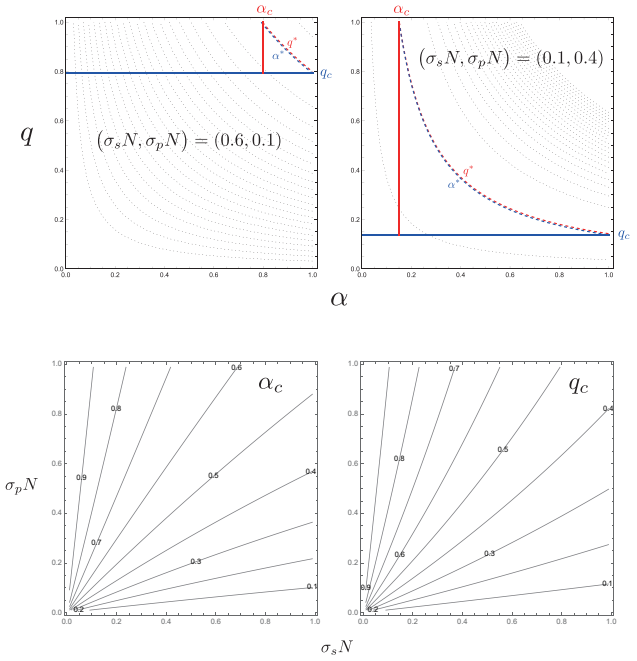

Figure 7: Dependence of α_c and q_c on $(\sigma_s N, \sigma_p N)$. Upper figures correspond to the left figure of Figure 4 with different $(\sigma_s N, \sigma_p N)$. Lower figures numerically shows the dependence of α_c and q_c on $(\sigma_s N, \sigma_p N)$.

do not necessarily require the direct contact with the others for a long duration in the daily life. In such a community, many people could be regarded as being at the private phase for relatively longer duration in the daily life. Our analysis implies that, the same as Type I, the longer time spent at the social phase makes the smaller number of new cases in such a community of Type II. Therefore, for Type II community, in order to reduce the infection risk, it would be effective to promote the social activity out of the private phase. If such a promotion is rather successful, the community of Type II may change to Type III with the longer time spent at the social phase.

Differently from Type I and II, the community of Type III has a specific length at social phase to minimize the infection risk. Hence for such a community, an appropriate control of the duration at the social phase would be successful to make the infection risk lower.

3.4 DISCUSSION

Let us think of three different communities: one located in an urban metropolis with a high mobility and relatively young population, one located in a semi-urban city with relatively young population but limited options of activity, and another one in a rural area with low mobility and aging population. In a situation without pandemic, we can imagine that the urban and semi-urban communities have larger size of active class compared to their rural counterparts. The active class in the urban population may have a significantly more option of public settings to spend their proportion of activity sphere in daily life, i.e. at the social phase. Meanwhile, the semi-urban community limits their time spent at the social phase as the activity of its active class are limited only to school/work in different locations and go home afterwards. The rural community will be content to spend their time at the private phase, socializing among neighbors and only a handful chances to have a huge event where all members of the community come together. It is obvious that the urban community belongs to the Type III, semi-urban one belongs to the Type II, while the rural community one may falls into Type I community.

In the event of pandemic, we can turn our attention back to the urban community of Type III, where there may be a need to control the duration at the social phase into a specific one tailored to reduce the infection risk. Applying policies such as closing shopping center and restaurants early to limit activities, teleworking, online classes, and so on may be appropriate in this type of community to shorten their length of social phase. Periodic closure of public facilities may have a special effect in minimizing a disease spread [22]. However, we need to recall that there must be a possibility of infection at the private phase. With the active class members crowding inside their residential area, the infection may spread still, even worse, putting elderly or infant members

of less-active class. For the semi-urban community of Type II, it is much easier to control the spread as they have already a limited duration at the social phase voluntarily due to lifestyle. However, similar to infection spread within homes in the Type I communities, promoting activities at the social phase may be necessary to manage the infection risk.

4

CONCLUDING REMARKS

Current situation regarding COVID-19 serves as a impetus for a deeper understanding of the community structure and its social characteristics to recognize the risk of infection, which is connected to the epidemic prevalence. Such a huge scale of epidemic is not the first and may not be the last time in history, thus the knowledge of social epidemiology would still be relevant in the distant future.

Disease spread is influenced by people's willingness to adopt preventative public health behaviors, which are often associated with public risk perception. Risk perception is correlated significantly with adoption of preventative health behaviors in ten countries as previously studied [17]. Even for the same disease, the response of communities in each country are dependent on the distinctive institutional arrangements and cultural orientation, affecting the community behavior as a whole[55]. For example, facing COVID-19, governments of the world employ different policies such as "nudge" in Sweden, "mandate" in China, "decree" in France, and "boost" in Japan, with different level of enforcement and types of strategy[55].

At first, we consider the modeling in which we focused on the local mobility in a regional community and what situations would have the least infection risk. The state of emergency in Japan during April – May 2020 served as an impetus for the consideration of such a modeling. The assumptions were set as such in a state of emergency or semi-lockdown situation where the mobility of people was limited only in a neighbor region that they belong to. According to the report by Parady et al. [39], shopping is one of the activities with the least reduction in frequency, so setting up the model to consider only the effect of shopping on disease prevalence could be reasonable, as a primitive theoretical consideration on the relation of the infection risk to the social situation.

Next, on the modeling for the dependence of the infection risk on community structure, the assumptions were considering the situation, for example, when the lockdown has been relaxed. People are freer to move, however their mobility is dependent on the social structure in the communnity. The community is divided by the activity level and activity phases [43], and these two factors comprises the social structure in the community. Our analysis of activity level versus the

number of new cases served as an initial point to exemplify the tight correlation between these two.

Even though these two models are independent, they are complimentary of each other in that they are considering the effect of social situation on the prevalence. What situation would give the larger prevalence? What kind of factors in terms of the community structure would be considered to get the estimation of the social situations according to the severity of an epidemic process? These two questions are common for building the assumptions for the models.

In Chapter 2, the characteristics of social situation were determined by the preference of shopping. We compared the infection risks in three possible situations: no limitation at all, a certain limitation, and a semi-lockdown with the mobility of limited persons. Semi-lockdown and activity limitation may not necessarily correlate with a lower infection risk, as is common with the event of superspreader.

In Chapter 3, the social characteristics of a community were introduced by the distribution of the community member's activities in terms of the activity level and phase. Communities with young and highly active members will have different challenges from aging communities, although appropriate recognition of the social structure is necessary for both cases. Activity center of the community, for example shops, offices, schools, and so on, may benefit from recognizing the activity patterns of its patrons. In such a case, a theoretical game between the activity center and the community members for the least prevalence may be an interesting topic to explore as an extension of this study.

As for the modeling in Chapter 2, the theoretical consideration would only work for small communities as large ones would not have only two shops serving the community. The modeling in Chapter 3 may work for big communities, and only activities in the localities were considered. Risks from visitors outside the communities were not taken into account. Only very simple modeling for the infection risk in the private and social phases were used. It was introduced by the simplest functions of the mean density. Disease transmissions do not necessarily require the presence of people, such as the case of droplet transmission.

Further study of the models used in work may examine:

- a more extensive division of activity phases
- the infection probability as a function of other demographic factors
- the dependence of infection probability on the disease prevalence

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A

SIGN OF $E_1 - E_2$

A.1 $E_1 - E_2$ FOR $p' = 1$

Consider the difference in the expected number of new cases $E_1 - E_2$ with $p' = 1$:

$$\begin{aligned} E_1 - E_2 = 1 - (1-\beta)^{pn} - (1-\beta)^{(1-p)n} + (1-\beta)^n \\ - n(1-\beta)\{p(1-s\beta)^{pn-1} + (1-p)(1-s\beta)^{(1-p)n-1} \\ - (1-s\beta)^{n-1}\}. \end{aligned}$$

We have $(E_1 - E_2)_{\beta=0} = 0$ and $(E_1 - E_2)_{\beta=1} = 1 > 0$. Then we can find that there is a unique value of β, $\beta_c \in (0,1)$, such that $E_1 - E_2 < 0$ for $\beta < \beta_c$, and $E_1 - E_2 > 0$ for $\beta > \beta_c$. This can be proved from the following mathematical features of $E_1 - E_2$ in terms of β:

- $E_1 - E_2$ is differentiable for $\beta \in [0,1)$;
- The derivative $d(E_1 - E_2)/d\beta$ has the value $-2s(1-p)pn^2 < 0$ at $\beta = 0$;
- The equation $d(E_1 - E_2)/d\beta = 0$ has a unique root of β in $[0,1)$.

From the second feature, we find that $E_1 - E_2$ must negative for sufficiently small β since $(E_1 - E_2)_{\beta=0} = 0$. On the other hand, since $(E_1 - E_2)_{\beta=1} = 1 > 0$, $E_1 - E_2$ must have at least one value of $\beta \in (0,1)$ such that $E_1 - E_2 = 0$ because of its continuity in terms of $\beta \in [0,1]$. Then from the third feature and the continuity of $E_1 - E_2$, we can conclude that such a value of $\beta \in (0,1)$ must be unique.

A.2 $E_1 - E_2$ FOR $p' = 0$

In the case of $p' = 0$,

$$\begin{aligned} E_1 - E_2 = 1 - (1-\beta)^{pn} - (1-\beta)^{(1-p)n} + (1-\epsilon\beta)^n \\ + n\{1 - p(1-s\beta)^{pn-1}(1-\beta) - (1-p)(1-s\beta)^{(1-p)n-1} \\ - (1-\beta) - \epsilon\beta\}. \end{aligned}$$

For a sufficiently large $\beta \leq 1$, $E_1 - E_2$ is positive for any value of ϵ with $\epsilon\beta \leq 1$.

To see the function behavior for a sufficiently small β near zero, applying 0–th degree Taylor approximation will yield

$$E_1 - E_2 \approx n\beta - 2\epsilon n\beta + (1-\beta)ns\beta\left[p(pn-1) - (1-p)\left\{-(1-p)n+1\right\}\right].$$

For a sufficiently small ϵ, $E_1 - E_2$ is positive. However, if we consider large value of ϵ as $\epsilon\beta \approx 1$, then $E_1 - E_2$ becomes negative. In that case we will have

$$E_q - E_2 \approx 1 - (1-\beta)^{pn} - (1-\beta)^{(1-p)n}.$$

Sufficiently large β i.e. $1-\beta \approx 0$ will result in positive $E_1 - E_2$. However, sufficiently small β i.e. $1 - \beta \approx 1$ will result in negative $E_1 - E_2$.

B

ANALYSIS ON A DATA OF NEW CASES AND SOCIAL ACTIVITY

We utilized a population estimation dataset created by the NTT DOCOMO mobile spatial statistics (MSS) which estimates population density using the operation data from mobile phone network. The data estimated hourly population in grids on the basis of phone signal [36]. The recorded densities at 15:00 were used as a reference. The relative density at the Shibuya crossing, one of the most crowded spot in Tokyo, Japan, was used as a representative. The daily recorded new case in Tokyo Metropolitan area was taken from the reported cases by the Japan Broadcasting Cooperation[34]. Datasets from May 2020-October 2021 was used. Steps to estimate an appropriate value of the correlation coefficient are as follows:

1. The daily data of phone signal was transformed into the relative crowd density by normalizing the daily density with the most crowded day between May 2020 to October 2021. It provides the original relative density data. The data of new infection cases was not modified for the original dataset.

2. For the original relative crowd density and the new infection cases, each dataset was used to obtain the weekly average and seven-days average datasets as well. The weekly average is counted by averaging Monday to Sunday data, while seven-days average is counted as an average from the past seven days including the calculated day (for example, a Wednesday data was obtained by averaging the data from Thursday of its previous week until the Wednesday in question). By now we have the original (raw), weekly average, and seven-days average data for both phone signal and daily case record for crowd density and new cases.

3. From the datasets, we focused on the periods when the new cases had been increasing. Periods between December 2020-January 2021, March-April 2021, and June-July 2021 were sampled.

4. The periods with increasing new cases were matched with the crowd density data of the corresponding periods but shifted from zero (recorded cases on the same day) to sixty days.

5. The correlation coefficients between the reported new cases and crowd density for shifted days were calculated using Pearson's correlation coefficient

$$r = \frac{\sum_{i=1}^{n}(x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_{i=1}^{n}(x_i - \bar{x})^2}\sqrt{\sum_{i=1}^{n}(y_i - \bar{y})^2}}.$$

   As a result, number of shifted days with the highest correlation coefficient was obtained as the optimal day(s) delay.

The analysis for optimal delay resulted in 40 days for original data ($r = 0.43$), 6 weeks (=42 days) for weekly average ($r = 0.85$), and 38 days for seven-days average ($r = 0.84$), as shown in Fig. 3.

C

PROOF OF THEOREM 3.3.1

From (4), (5), and (6), we have

$$
\begin{aligned}
E_l(\alpha, q) =& \sigma_p N(1-\alpha q)(1-q)N = N \cdot \sigma pN\{1-(1+\alpha)q+\alpha q^2\};\\
E_a(\alpha, q) =& N \cdot \sigma_p N \sigma_s N\left[(1-\alpha)\alpha^3 q^2 + \left\{-\frac{1}{\sigma_s N}(1-\alpha)\alpha + \frac{1}{\sigma_p N}\alpha^2\right\}q + \frac{1}{\sigma_s N}(1-\alpha)\right]q.
\end{aligned}
\tag{C.10}
$$

Let us consider

$$
\psi(\alpha, q) := \frac{E(\alpha, q)/N}{\sigma_p N \sigma_s N} = (1-\alpha)\alpha^3 q^3 + \sigma_s N(A+\alpha)\alpha^2 q^2 - \frac{2}{\sigma_s N}\alpha q + \frac{1}{\sigma_s N},
\tag{C.11}
$$

where A is defined by ##, and satisfies that

$$
A > \max\left[\frac{1}{\sigma_s N}, \frac{1}{\sigma_p N}\right] \geq 1,
$$

since $\sigma_s N < 1$ and $\sigma_p N < 1$ from the confinement for our modeling.

In order to investigate q-dependence of E, let us differentiate (C.11) in terms of q:

$$
\begin{aligned}
\psi_q(\alpha, q) &:= \frac{\partial \psi(\alpha, q)}{\partial q} = \alpha\left\{3(1-\alpha)\alpha^2 q^2 + 2(A+\alpha)\alpha q - \frac{2}{\sigma_s N}\right\}\\
\psi_{qq}(\alpha, q) &:= \frac{\partial^2 \psi(\alpha, q)}{\partial q^2} = \alpha\left\{6(1-\alpha)\alpha^2 q + 2(A+\alpha)\alpha\right\}.
\end{aligned}
\tag{C.12}
$$

Since $\psi_{qq} > 0$ for any $\alpha \in (0,1)$, ψ_q is monotonically increasing in terms of $q \in (0,1)$. Further, since $\psi_q(\alpha, 0) = -2\alpha \leq 0$, there are two distinct cases depending on the sign of $\psi_q(\alpha, 1)$. If $\psi_q(\alpha, 1) \leq 0$, then $\psi_q(\alpha, q) < 0$ for $q \in (0,1)$ so that ψ is monotonically decreasing for any $q \in 0$, while, if $\psi_q(\alpha, 1) > 0$, then $\psi_q(\alpha, q)$ changes its sign from negative to positive as q gets larger in $(0,1)$ so that ψ has a unique extremal minimum at a value of $q = q^* \in (0,1)$. In such a case, the value q^* is the root of the equation $\psi_q(\alpha, q)/\alpha = 0$ in terms of q as given by (7), where q^* depends on α.

Further, we can find that

$$
\frac{\partial}{\partial \alpha}\left\{\frac{\psi_q(\alpha, 1)}{\alpha}\right\} = 3(2-3\alpha)\alpha + 2(A+2\alpha) = 9\alpha(1-\alpha) + \alpha + 2A > 0
$$

for $\alpha \in (0,1)$, and

$$\frac{\psi_q(\alpha,1)}{\alpha}\Big|_{\alpha=0} = -\frac{2}{\sigma_s N} < 0; \quad \frac{\psi_q(\alpha,1)}{\alpha}\Big|_{\alpha=1} = 2(A+1) - \frac{2}{\sigma_s N} = \frac{2}{\sigma_p N} > 0$$

. Thus, there is a unique value $\alpha_c \in (0,1)$ such that $\psi_q(\alpha,1) < 0$ for $\alpha < \alpha_c$ and $\psi_q(\alpha,1) > 0$ for $\alpha > \alpha_c$. We can easily find that the critical value α_c is given by the unique root of the cubic equation (7) in terms of $\alpha \in (0,1)$. These results conclude Lemma 3.3.1.

D

PROOF OF THEOREM 3.3.2

We have the following partial derivatives of $\psi(\alpha, q)$ defined by (C.11) in terms of α:

$$\begin{aligned}\psi_\alpha(\alpha,q) &:= \frac{\partial\psi(\alpha,q)}{\partial\alpha} = q^3\Big\{-4\alpha^3+3(1+\frac{1}{q})\alpha^2+\frac{2A}{q}\alpha-\frac{2}{\sigma_s N q^2}\Big\};\\ \psi_{\alpha\alpha}(\alpha,q) &:= \frac{\partial^2\psi(\alpha,q)}{\partial\alpha^2} = 2q^3\Big\{-6\alpha^2+3(1+\frac{1}{q})\alpha+\frac{A}{q}\Big\}.\end{aligned} \tag{D.13}$$

Since $\psi_{\alpha\alpha}(0,q) = 2q^2A > 0$ and $\psi_{\alpha\alpha}(1,q) = 2q^3\{-3+(3+A)/q\} > 0$, we can easily find that $\psi_{\alpha\alpha}(\alpha,q) > 0$ for $\alpha \in (0,1)$. Hence, ψ_α is monotonically increasing for $\alpha \in (0,1)$.

From (D.13), we have $\psi_\alpha(0,q) = -2q < 0$ and

$$\frac{\psi_\alpha(1,q)}{q} = -q^2+(3+2A)q-\frac{2}{\sigma_s N},$$

and then,

$$\frac{\psi_\alpha(1,q)}{q}\Big|_{q=0} = -\frac{2}{\sigma_s N} < 0; \quad \frac{\psi_\alpha(1,q)}{q}\Big|_{q=1} = 2\Big(1+A-\frac{1}{\sigma_s N}\Big) = \frac{2}{\sigma_p N} > 0. \tag{D.14}$$

From (D.14), we can easily find that $\psi_\alpha(1,q)$ is negative for $q < q_c \in (0,1)$ and positive for $q > q_c$, where q_c is given by YY as the smaller root of the equation $\psi_\alpha(1,q)/q = 0$.

Consequently, since ψ_α is monotonically increasing for $\alpha \in (0,1)$, we have $\psi_\alpha(\alpha,q) \le 0$ for any $\alpha \in (0,1)$ if and only if $q \le q_c$, while $\psi_\alpha(\alpha,q)$ changes the sign from negative to positive as α gets larger if and only if $q > q_c$. Therefore, $\psi(\alpha,q)$ is monotonically decreasing in terms of $\alpha \in (0,1)$ if and only if $q > q_c$.

E

PROOF OF THEOREM 3.3.4

First, we prove the following lemma:

Lemma E.0.1. *The expected number of new cases E cannot be minimum for* $(\alpha, q) = (1, q)$ *for any* $q \in (0, q_c\,]$.

The case of $(\alpha, q) = (1, q^*)$ could be valid only when $\alpha > \alpha_c \in (0, 1)$ and $q \leq q_c \in (0, 1)$. Thus, it is necessary that $q^* \leq q_c$, otherwise this case is invalid for minimizing E. From the proof of Theorem 3.3.2 in Appendix D, the condition that $q^* \leq q_c$ is equivalent to that $\psi_\alpha(1, q^*) \leq 0$, that is, from (D.14),

$$-{q^*}^2 + (3 + 2A)q^* - \frac{2}{\sigma_s N} \leq 0.$$

Since q^* is given by (7) for $\alpha = 1$, this condition becomes

$$\begin{aligned}
&-\left\{\frac{1/(\sigma_s N)}{1/(\sigma_s N) + 1/(\sigma_p N)}\right\}^2 + (3 + 2A)\frac{1/(\sigma_s N)}{1/(\sigma_s N) + 1/(\sigma_p N)} - \frac{2}{\sigma_s N} \\
&\quad = -\left\{\frac{1/(\sigma_s N)}{1/(\sigma_s N) + 1/(\sigma_p N)}\right\}^2 + \Big(\frac{2}{\sigma_s N} + \frac{2}{\sigma_p N} + 1\Big)\frac{1/(\sigma_s N)}{1/(\sigma_s N) + 1/(\sigma_p N)} - \frac{2}{\sigma_s N} \leq 0.
\end{aligned}$$

With some calculation about this inequality, we can find the equivalent inequality such that $1/(\sigma_p N) \leq 0$. This is impossible. Hence the condition $q^* \leq q_c$ cannot be satisfied when $\alpha = 1$. Therefore, it has been shown that $q^*|_{\alpha=1} > q_c$. Therefore, the case of $(\alpha, q) = (1, q^*)$ cannot be valid. As a result, we can get Lemma E.0.1

Next, the case of $(\alpha, q) = (\alpha^*, 1)$ could be valid only when $\alpha \leq \alpha_c \in (0, 1)$ and $q > q_c \in (0, 1)$. Thus, it is necessary that $\alpha^* \leq \alpha_c$ for $q = 1$, otherwise this case is invalid for minimizing E. From Theorem 3.3.1, the condition that $\alpha^* \leq \alpha_c$ is equivalent to that

$$-3{\alpha^*}^3 + 5{\alpha^*}^2 + 2A\alpha^* - \frac{2}{\sigma_s N} \leq 0. \tag{E.15}$$

Then, from (9) in Theorem 3.3.2, we have α^* for $q = 1$ which satisfies the following equation:

$$-4{\alpha^*}^3 + 6{\alpha^*}^2 + 2A\,\alpha^* - \frac{2}{\sigma_s N} = 0, \tag{E.16}$$

that is,

$$-3{\alpha^*}^3 + 5{\alpha^*}^2 + 2A\,\alpha^* - \frac{2}{\sigma_s N} = {\alpha^*}^3 - {\alpha^*}^2.$$

Substituting (E.16) for (E.15), we can get the condition that $\alpha^{*3} - \alpha^{*2} = \alpha^{*2}(\alpha^* - 1) \leq 0$. This condition is necessarily satisfied if there exists $\alpha^* \in (0,1)$ when $q = 1$. Indeed, from (E.16), it can be easily seen that there exists uniquely $\alpha^* \in (0,1)$ even when $q = 1$. From these arguments, we have proved that it is necessarily satisfied that $\alpha^* \leq \alpha_c$ when $q = 1$. As a result, we can get the following lemma:

Lemma E.0.2. *The expected number of new cases E becomes minimum for $(\alpha, q) = (\alpha^*, 1)$ in the region $\{(\alpha, q) \in (0,1) \times (0,1) \mid \alpha \leq \alpha_c \in (0,1)$ and $q > q_c \in (0,1)\}$.*

Lastly, the case of $(\alpha, q) = (\alpha^*, q^*)$ could be valid for minimizing E only when $\alpha > \alpha_c \in (0,1)$ and $q > q_c \in (0,1)$. Now suppose that it could be valid. We have q^* for $\alpha = \alpha^*$ satisfying the following from (**??**) with Theorem 3.3.1:

$$-3\alpha^{*3} + (3 + \frac{2}{q^*})\alpha^{*2} + \frac{2A}{q^*}\alpha^* - \frac{2}{\sigma_s N q^{*2}} = 0, \tag{E.17}$$

while α^* for $q = q^*$ satisfies the following from (9) in Lemma 3.3.2:

$$-4\alpha^{*3} + (3 + \frac{3}{q^*})\alpha^{*2} + \frac{2A}{q^*}\alpha^* - \frac{2}{\sigma_s N q^{*2}} = 0,$$

that is,

$$-3\alpha^{*3} + (3 + \frac{2}{q^*})\alpha^{*2} + \frac{2A}{q^*}\alpha^* - \frac{2}{\sigma_s N q^{*2}} = \alpha^{*3} - \frac{\alpha^{*2}}{q^*}. \tag{E.18}$$

From these equations (E.17) and (E.18), we can immediately get the equation that $\alpha^{*3} - \alpha^{*2}/q^* = 0$, which results in $\alpha^* q^* = 1$. This is inconsistent for $\alpha^* \in (0,1)$ and $q^* \in (0,1)$. This means that there does not exist mathematically reasonable (α^*, q^*) such that $\alpha^* \in (0,1)$ and $q^* \in (0,1)$ about E. Finally we can conclude the following lemma:

Lemma E.0.3. *There is no definite point (α, p) to minimize E in the region $\{(\alpha, q) \in (0,1) \times (0,1) \mid \alpha > \alpha_c \in (0,1)$ and $q > q_c \in (0,1)\}$.*

This lemma indicates that the expected number of new cases E cannot be minimum when $\alpha = \alpha^*$ or $q = q^*$, and further implies that it becomes minimum only when $\alpha = 1$ or $q = 1$. Consequently, we can derive Theorem 3.3.4.