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Modeling the Effectiveness of Face Coverings in Curtailing SARS-CoV-2 Spread

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The student author, whose presentation of the scholarship herein was approved by the program of study committee, is solely responsible for the content of this creative component. The Graduate College will ensure this creative component is globally accessible and will not permit alterations after a degree is conferred.

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DEDICATION

I would like to dedicate this creative component to my parents, Seema, and Vijay Koshti. Without their constant encouragement throughout my engineering education, this work would not have been possible.

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ABSTRACT

Face coverings by the general public are widely being recommended for limiting the spread of COVID-19. Several states have also mandated using masks in public places. However, its potential and correct use are still relatively unknown. Several studies have assessed the effectiveness of masks by implementing the SIR model of epidemics, but they have certain limitations. This thesis addresses these limitations by modeling for shortcomings in the practical use of masks. The primary limitation addressed is the high transmission rate through high-frequency areas in the closed settings. A multi-group Kermack-McKendrick-type compartmental mathematical model was used to model different scenarios and simulate them with actual data to evaluate the effectiveness of face coverings. It is established that perfect use of face coverings contributes significantly to lowering the spread, but its practical implementation is still highly dependent on strict compliance and effective surface-contact transmission rate.

CHAPTER 1. INTRODUCTION

Covid-19

After the first epidemic of severe acute respiratory syndrome (SARS), several SARS-related coronaviruses (SARSr-CoVs) continued to exist and mutate into different strains and variants [1] [2] [3] [4]. SARS is a deadly disease that presents with a wide variety of symptoms depending on an individual's underlying health. However, a high fever with temperature greater than 100.4°F [>38.0°C]) has been the most common symptom. Some other symptoms like headache, discomfort, and body aches are also common. Although, most SARS cases present with only mild symptoms, susceptible individuals may develop severe symptoms with pneumonia and may need hospitalization and intensive care [5]. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the virus responsible for coronavirus disease 2019 (COVID-19) which eventually propagated into COVID-19 pandemic.

The structure of coronaviruses is quite unique. They are enveloped in a shell with spike proteins and are positive single-stranded large RNA viruses [6]. The first occurrence of coronaviruses in literature is seen in 1966 study by Tyrell and Bynoe [7]. The viruses were cultured from the samples of patients affected with common cold. They were later termed coronaviruses upon resemblance to solar coronas in their appearance. There are seven subtypes of coronaviruses that can infect humans. Beta-coronaviruses cause severe infection that eventually results in hospitalization, long-term chronic symptoms and in many cases, death. Alpha-coronaviruses usually are present with mild symptoms that go away within a period of 7-14 days. Many cases of alpha-coronavirus infection are even asymptomatic in nature [6]. SARSCoV-2 poses a great concern as it belongs to a family of deadlier beta-coronavirus that have higher fatality rate.

Covid-19 was first identified in Hubei, China and was linked with consumption of bats. It has spread to many countries since then causing 2.64 million deaths as of March 13, 2021. People with COVID-19 have reported a wide range of symptoms – ranging from mild discomforts to severe illness – thus far. Covid-19 progresses after an initial incubation period, during which one has contracted an infection but not produced any symptoms. Symptoms usually occur after a period of 5-14 days and thus, a diagnostic test is recommended post 5 days instead of immediately after coming into a contact with an infected individual. Fever or chills, shortness of breath, fatigue, muscle or body weakness, headache, new loss of taste or smell, congestion or runny nose, sore throat, nausea or vomiting, diarrhea are possible symptoms of Covid-19 and warrant testing [8].

Chan et al. reported findings that were consistent with person-to-person transmission of SARS-CoV-2 in hospital and family settings, thus confirming the human-to-human transmission in January 2020 [9]. Initially, respiratory droplets from coughs, sneezes, or conversations were assumed to be primary routes of transmission, concerningly within a 6-feet distance. Not long after, other modes were examined and confirmed to transmit the infection. When people with COVID-19 cough, sneeze, sing, talk, or breathe they produce visible or invisible droplets varying greatly in size. Infections mainly occur through these droplets when someone is in close contact with a person infected with Covid-19. These droplets coming from the nose or the mouth of an infected person can only travel a certain length, before the larger particles fall off due to gravity. The smaller particles stay afloat in air and can settle down on surfaces. The spread through surfaces is also a possible mode of transmission but is uncommon [10]. Laser light scattering experiments suggest that speaking is an additional mode of transmission, especially in an indoor setting [11]. Same studies possibly suggest louder speech producing more droplets. Few studies

have also hinted at airborne transmission with infected aerosols being potent in air for upto 4-6 hours [12]. Human-to-human transmissions occur only after crossing a threshold of viral load, thus confirming that minimal exposure is no reason for a doom. Intervention even after contracting the virus can help significantly in reducing the viral load and limiting the severity, or even the infection in some cases. This threshold was identified on an average as 1000 infectious SARS-CoV-2 virions [13]. This study had interesting findings like fomites not being a major source of infection spread which was later confirmed by the Centers for Disease Control and Prevention (CDC), and that exceptionally high viral loads are not necessary to trigger a superspreading event. The study claimed superspreading events are principally a result of the accumulation of infective aerosols exhaled by an infected, regardless of symptoms, patient in a confined space. The accumulation was found to be amplified by crowded spaces and vocal activities like singing and high-pitch conversations. In these circumstances, viruses do not take much longer to spread and can cause sufficient infections in timespans of less than an hour. The same study suggested that social distancing and temperature screening may not strongly reduce COVID-19 transmission. The accumulation of infected aerosols has proved to be the most challenging factor.

Impact on economy

Covid-19 first broke out in late November 2019 in China. The first recorded case of the novel coronavirus in the US was reported on January 20, in a 35-year-old man from Washington who was traveling from the epicenter of virus – Wuhan, China [14]. A day later, on January 21, the Centers for Disease Control and Prevention (CDC) activated its emergency operations center to tackle the looming crisis. The virus spread quickly through the countries and asymptomatic infections made it difficult to stop that spread. In most places, the travel restrictions were too late

of an intervention and thus helped little. China went into a lockdown in late January, giving United States and other countries a blueprint of reactive strategies. Most states in United States went into a lockdown or stay at home orders were issued in the month of March. Businesses were ordered to be closed, at certain times initially, and completely later. The lockdown affected many small business owners and saw a shutdown of many small and medium scale enterprises.

A USC study claimed the United States could lose as much as $3.2 - $4.8 trillion in real gross domestic product owing to the Covid-19 pandemic [15]. Real GDP is an inflation-adjusted measure reflecting the monetary value of final goods and services produced by a nation's economy each year. According to USC's research, the extent of losses depends on infection and fatality rates, duration of lockdowns, gradual reopening timelines and pent-up consumer demands. According to the study, the direct losses from closures would make a dent of 22% in the GDP while the residual impact would be felt for next couple of years. Comparing to China, the total loss was enumerated to be at 4x as that of the China [15]. The total result was estimated to be 14.7% to 23.8% cuts in jobs affecting 36.5 million workers. A study detailing a survey of over 5,500 small businesses analyzed the financial implications of lockdowns and had some strong datapoints to make a case for the financial plight of these small businesses [16]. The survey was conducted in the months of March and April and thus gives a good insight into the expectations of businesses rather than the opinions and the feelings in the hindsight. The themes emerging from these results were mostly pessimistic and uncertain. Most small businesses had little to no cash-on-hand to survive the closure, several jobs were lost, and all hopes were pinned on government assistance programs. Out of the businesses in the survey sample, around 1.8% reported to be permanently closed because of the pandemic. Among the remaining businesses, the number of full-time and part-time employees had fallen by 32% and 57% respectively [16].

The study claimed that firms with fewer than 20 employees in January were more likely to be closed as they were operating on minimal days' cash on hand. The major problems faced by these businesses were employee sickness, disruption in supply chains, and reduction in consumer demands. The smaller firms had limited cash-on-hand; firms with monthly expenses totaling above $10,000 had cash-on-hand worth less than 15 days [16]. These numbers reflect the financial fragility of small businesses while dealing with the pandemic.

This survey was heavily represented by retail store owners which are worst affected by the pandemic. Hence, the results could potentially overstate the damages done by lockdowns on small businesses.

The inconclusive data on face coverings

Face coverings have been widely recommended and mandated in many places as a response to Covid-19 outbreak. They are a good preventive mechanism as respiratory particles are the leading route of transmission of COVID-19. Masks also help with outward protection since asymptomatic people can spread the infection without knowing themselves that they are infected [17]. As such, masks are an effective barrier that can prevent aerosols containing virus particles from being inhaled. They have been widely used as a protection from respiratory infections in hospital setting and naturally, were one of the primary recommendations from official agencies. The evidence around face coverings though is conflicting at best. The practical execution of masks exposes them to vulnerability against tiny virus aerosols. There have been studies proving inability of masks in stopping superspreading events [13], but the bigger concern is the lack of practical constraints modeled in either of these studies.

In his work aimed at controlling 1910 Manchurian Plague, also acclaimed as “a milestone in the systematic practice of epidemiological principles in disease control”, Wu ascertained a cloth mask as “the principal means of personal protection.” [18]. Wang et al., analyzed the

impact of face coverings on community spread in China [19]. They looked at the reduction of secondary transmission of SARS-CoV-2 in Beijing households by face mask use, social distancing, and disinfection. They found out that wearing a mask before onset of any symptoms was 79% effective in reducing the spread, while mask use after the onset of symptoms did not make any difference. This study had several limitations. Firstly, the data came from a questionnaire answered by families with primary cases. The questionnaire included demographic, subject-knowledge, and behavioral questions. Questionnaires are prone to subjectivity, personal bias, inaccuracy, and inconsistency in reporting. Another systematic review sponsored by World Health Organization (WHO) looked at face coverings, social distancing, and eye coverings to prevent person-to-person transmission of SARS-CoV-2 [20]. However, the review included just three studies of face mask use outside the hospital setting and none of them pertained to SARS-CoV-2 exclusively. Moreover, the conclusions in one of those studies were statistically insignificant to draw any conclusions [17]. The last study ascertained mask use as strongly protective, with a risk reduction of 70% for those that diligently followed the mask mandate, but the study did not include the outward protection from masks in their design and hence did not consider the spread from the wearer [17]. None of the studies assessed the relative effectiveness of different types of masks. MacIntyre et al., in their study on respiratory infections in the Australian winters of 2006 and 2007 discovered that masks have protective efficiency of over 80% from viruses but only when wearing protocols were strictly followed [21]. The study had inconclusive findings which were described to be heavily reliant on compliance. The authors believed that since compliance is dependent on perceived risk, they would expect more compliance during a pandemic. Additionally, the data for this study came from a questionnaire which makes it less compelling. Not considering for unavoidable slip-offs

from consistent mask use and assuming everyone will always wear the mask perfectly highly distorted the results of many studies. Addressing these assumptions is the main premise of this thesis.

Jefferson et al., tested mask use as a standalone intervention to prevent the spread of respiratory viruses in their systematic review [22]. They conducted randomized controlled trials (RCTs) and cluster-RCTs to probe common modes of intervention like temperature-screening at airports, home isolation, quarantine, social distancing, personal protective gear, hand hygiene and sanitization) to prevent transmission of coronavirus. Observational studies were excluded from this review. The authors concluded that the pooled results of randomized trials concluded that the use of face coverings during seasonal flu did not lessen the spread of viral infection. Additionally, the N95/P2 respirators fared no better than medical masks when tested in healthcare setting [22]. Furthermore, they posited that the hand hygiene was likely to be the best way to reduce the burden of respiratory illness. McIntyre et al., conducted a review evaluating effectiveness of masks in curbing community spread and breaking the infection chain but only found modest evidence. Even in those studies supporting the finding, majority were either conditioned on continuous use of N-95 grade mask or found no statistical significance for efficacy of intervention [23].

Thus, several reviews have probed mask use during non-pandemic outbreaks of influenza and other respiratory infections, and it remains fairly unknown whether SARS-CoV-2 will act or progress in similar fashion to regular respiratory viruses [17]. To ascertain a strong evidence, or a link of one thing with other, it is best to rely on randomized controlled trials (RCT), a suitably operated metanalysis of RCTs, or a systematic review of unbiased, peer-reviewed studies. To note, there are no such studies done until today evaluating the effectiveness of masks. The

epidemiological studies have shown many assumptions, shortcomings and thus cannot be relied upon. One of the primary issues with several studies analyzed in this literature review is considering perfect mask use and not accounting for fatigue-led, unavoidable slippages in community use of face coverings. Especially when mandated for long durations, the general population tends to get fatigued and stop wearing masks [24].

CHAPTER 2. LITERATURE REVIEW

Kermack-McKendrick model developed in 1927 is one of the primary models used to model the infectivity and spread during a pandemic. The model was developed to obtain detailed understandings of various factors and their consequences in order to get some foresight on progression of the pandemics [25].

This model is a system of two ordinary differential equations:

$$S' = -\beta SI$$

$$I' = (\beta S - \alpha)I$$

The population in the model is divided into three classes: Susceptible (S), Infected (I), and Removed (R). The model works on three principal assumptions:

i) Infected individual (or multiple) is initiated into the population susceptible to the infection. The contact occurs with βN others per unit time, with N representing the total population.

ii) The disease spreads from infected to the non-infected through close contact

iii) Every infected person undergoes the disease and is removed from infected pool either by recovery or by death at the rate of α per unit time

Though this is the basic model, various other questions have been raised and suitable modifications have been made to the model. One fundamental finding shared by all these models is that there is a certain threshold of both – (a) the cases before an outbreak of a pandemic and (b) the number of recoveries before the pandemic can be said to be passed leaving remaining population uninfected [26]. Chowell et al., used the variation called SEIJR model to discover parameter for the past SARS outbreak in Canada, Hong Kong in China, and Singapore [27]. The SEIJR in this model stands for susceptible, exposed, infective, diagnosed, and recovered classes,

respectively. This model considers varied susceptibility by introducing two classes of susceptible population: S_1, which is more susceptible and S_2, which is less susceptible. The class E represents those exposed to the virus but did not display an onset of symptoms. These are called asymptomatic cases and have reduced transmission rate which is accounted for in the model. Another important assumption in this study was that the diagnosed class are managed with care; thus, implying reduced transmission rate. The final model was represented with a system of non-linear differential equation that goes as follows [27]:

$$S_1 = -\beta S_1(I + qE + lJ)N$$

$$S_2 = -\beta p S_2(I + qE + lJ)N$$

$$E = \beta(S_1 + pS_2)(I + qE + lJ)N - kE$$

$$I = kE - (\alpha + \gamma_1 + \delta)I$$

$$J = \alpha I - (\gamma_2 + \delta)J$$

$$R = \gamma_1 I + \gamma_2 J$$

Where β is the transmission rate per day, q is the relative infectiousness of the asymptomatic class *E,* l is the relative measure of reduced risk upon infection, p is the reduction in risk of infection for class S_2, k is the rate of infection measured per day, α is the rate of progression from infective to diagnosed per day, γ_1 is the rate of recovery from infectious class, γ_2 is the rate of recovery from diagnosed class, δ is the disease-induced death rate per day and ρ is the initial high-risk population.

Another modified version of the model was studied by Gumel et al., in their paper [28]. They formulated a model considering six sub-classes of population, viz., susceptible (S), asymptomatic (E), quarantined (Q), symptomatic (I), isolated (J) and recovered (R) individuals. It was a deterministic model aimed at assessing the long-term progression of an epidemic and did

not consider the initial outbursts due to the superspreading events. This model goes a step further than other models by considering a net addition of new population into the region at a rate Π per unit time. This parameter includes new births, immigrations, and emigrations. The final model is represented with the following system of differential equations:

$$\frac{dS}{dt} = \Pi - \frac{S(\beta I + \varepsilon_E \beta E + \varepsilon_Q \beta Q + \varepsilon_J \beta J)}{N} - \mu S$$

Where, β = transmission coefficient for symptomatic class

$\varepsilon_E\beta$ = transmission coefficient for asymptomatic class

$\varepsilon_Q\beta$ = transmission coefficient for quarantined class

$\varepsilon_J\beta$ = transmission coefficient for isolated class

μ = natural death rate

$$\frac{dE}{dt} = p + \frac{S(\beta I + \varepsilon_E \beta E + \varepsilon_Q \beta Q + \varepsilon_J \beta J)}{N} - (\gamma_1 + k_1 + \mu)E$$

Where, p = rate of entry of asymptomatic travelers

γ_1 = rate of quarantining of asymptomatic individuals

k_1 = rate of onset of symptoms in asymptomatic individuals

$$\frac{dQ}{dt} = \gamma_1 E - (k_2 + \mu)Q$$

Where, k_2 = rate of onset of symptoms in quarantined individuals

$$\frac{dI}{dt} = k_1 E - (\gamma_2 + d_1 + \sigma_1 + \mu)I$$

Where, γ_2 = rate of isolation of symptomatic individuals

d_1 = rate of disease induced death of symptomatic individuals

σ_1 = rate of recovery of symptomatic individuals

$$\frac{dJ}{dt} = \gamma_2 I + k_2 Q - (\sigma_2 + d_2 + \mu)J$$

Where, σ_2 = rate of recovery of isolated individuals

d_2 = rate of disease induced death of isolated individuals

$$\frac{dR}{dt} = \sigma_1 I + \sigma_2 J - \mu R$$

This model also integrates reproduction numbers. R_o is defined as the "*expected number of secondary infections produced by a primary infection*" [29]. When $R_o < 1$, it is assumed that an epidemic will not develop from a minor exposure of infectious class, while $R_o > 1$ is a necessary but not sufficient condition for an epidemic to develop [28]. The model uses R_c which is the controlled reproduction number, which takes into account the control measures applied unlike R_o.

Acemoglu et al., developed a multi-risk SIR model (MR-SIR) where infection, hospitalization and fatality rates vary between groups. The different groups are considered according to the age to represent the underlying risk factor. Elderlies are considered to be more susceptible to the fatality from infection [30]. The study established that policies designed by considering the risk factors of different age groups suggestively outperform uniform policies designed for everyone. The best possible way of intervention according to the study was isolation of older population which is at higher risk.

Tracht et al., analyzed the effectiveness of N-95 grade masks on an H1N1 influenza pandemic [31]. The model was built assuming a closed system with no births, immigrations, or natural deaths. The population was divided into eight classes viz., susceptible (S), susceptible and wearing a mask (S_m), exposed (E), exposed and wearing a mask (E_m), infected (I), infected

and wearing a mask (I_m), recovered (R), and dead (D). The model is described by the set of following differential equations [31]:

$$\frac{dS}{dt} = -(\varphi_S m + \lambda)S + \varphi_S S_m$$

$$\frac{dE}{dt} = -(\varphi_E m + \omega)E + \varphi_E E_m + \lambda S$$

$$\frac{dI}{dt} = -(\varphi_I m + \delta + \mu)I + \varphi_I I_m + \omega E$$

$$\frac{dS_m}{dt} = -(\varphi_S + \lambda_m)S_m + \varphi_S m S$$

$$\frac{dE_m}{dt} = -(\varphi_E + \omega)E_m + \varphi_E m E + \lambda_m S_m$$

$$\frac{dI_m}{dt} = -(\varphi_I + \delta + \mu)I_m + \varphi_I m I + \omega E_m$$

$$\frac{dR}{dt} = \delta(I + I_m)$$

$$\frac{dD}{dt} = \mu(I + I_{m)}$$

Where, ψ_i = movement rate between classes

λ / λ_m = force of infection in the respective group

ω = incubation relative rate

δ = recovery relative rate

μ = death relative rate

The secondary infections are modelled using a controlled reproduction number calculated using "the next generation operator" approach. In a perfectly closed system, the reproduction rate is correlated to the infection rate and the duration of infection. Nevertheless, for complex designs with multiple classifications into the infected class, the simple heuristic value of R_o is inadequate

[32]. Hence, reproduction number can be intuitively defined as the number of new infections caused by an infected individual in a population at an equilibrium in terms of cases and recoveries. It is given by the equation:

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

Where, ρ = spectral radius of the given matrix

F = rate of appearance of new infections

V = rate of net transfer of individuals in and out of the system

The (*i*,*k*) entry of the product FV^{-1} gives the rate of secondary transmission. Here, i and k represent two different compartments [32].

CHAPTER 3. METHODS

Baseline Mathematical Models

Model with no mask use

We use the model formulated by Eikenberyy et al., [33] as a baseline model to establish epidemiological parameters and transmission rates in an uncontrolled non-healthcare setting. Here, we assume that no masks were used at all while the epidemiological parameters and transmission rates undergo a natural progression. Equivalent transmission rates from perfect and imperfect mask use as defined in detail in further sections, are also calculated using this model.

Eikenberry et al., use a deterministic susceptible, exposed, symptomatic infectious, hospitalized, asymptomatic infectious, and recovered modeling framework, with these classes respectively denoted S(t), E(t), I(t), H(t), A(t), and R(t); they also include D(t) to track cumulative deaths [33]. The model is given by following set of equations:

$$\frac{dS}{dt} = -\beta(t)(I + \eta A)\frac{S}{N}$$

Where β = infectious contact rate

η = relative infectiousness of asymptomatic carriers in comparison to symptomatic carriers

This equation states the susceptible population decreases owing to two factors – the susceptible population meeting – i) symptomatic and ii) asymptomatic population at the rate of β. The η is a fraction of 1 as the asymptomatic individuals are less infectious than symptomatic ones.

$$\frac{dE}{dt} = \beta(t)(I + \eta A)\frac{S}{N} - \sigma E$$

Where σ = the transition rate from exposed to infectious class (1/σ is the disease incubation period)

The equation states the exposed population increases in response to symptomatic and asymptomatic contact, and decreases as individuals move from exposed to infected class at the rate of σ.

$$\frac{dI}{dt} = \alpha\sigma E - \varphi I - \gamma_I I$$

Where α = fraction of cases that are symptomatic

ψ = rate of hospitalization of symptomatic individuals

γ_I = recovery rate of infected population

The equation describes the change in infected population. It increases with increase in exposed class population, while decreases as infected people either recover or are hospitalized.

$$\frac{dA}{dt} = (1 - \alpha)\sigma E - \gamma_A A$$

Where γ_A = recovery rate of asymptomatic population

The asymptomatic class population increases with increase in exposed class population. Out of the total exposed population, α% are symptomatic while (1-α) % are asymptomatic. The population reduces as people recover. Asymptomatic individuals are not hospitalized.

$$\frac{dH}{dt} = \varphi I - \delta H - \gamma_H H$$

Where γ_H = recovery rate of hospitalized population

δ = disease-induced death rate

This equation gives the rate of change in hospitalized population. It increases as infected people are hospitalized and decreased as people recover or die.

$$\frac{dR}{dt} = \gamma_I I + \gamma_A A + \gamma_H H$$

The total recovered population is the summation of recovered population from each class.

$$\frac{dD}{dt} = \delta H$$

This equation states that only hospitalized population may die. Those dying before being hospitalized are not considered for simplicity.

$$N = S + E + I + A + R$$

The hospitalized individuals do not contribute to infection as they are not present outdoor.

Model with perfect general population mask use

Model with perfect general population mask use assumes that some population in outdoor setting uses a face covering and takes perfect care of cross-contamination, while the other fraction does not. Those using the face covering are represented with subscript m (masked), while those who are not are represented with subscript u (unmasked). Some examples of cross-contamination can be touching the mask after touching a fomite, improper/loose placement resulting in airgaps exposing the skin, lowering of mask to eat/drink/relax. It is crucial to consider these conditions as COVID-19 can spread even from minimal contact.

We assume that masks have uniform inward efficiency (i.e., primary protection against catching disease) of ε_i, and outward efficiency (i.e., source control/protection against transmitting disease) of ε_o. The model for this scenario is given by the following set of equations [33]:

$$\frac{dS_u}{dt} = -\beta(I_u + \eta A_u)\frac{S_u}{N} - \beta((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m)\frac{S_u}{N}$$

This equation gives the rate of change in susceptible population not using a mask. The unmasked susceptible group can get infected from masked as well as unmasked population. The infection from infected and asymptomatic unmasked is similar to that in the model with no mask use while the infectiousness from masked group is reduced by an amount of ε_o (outward infection).

$$\frac{dE_u}{dt} = \beta(I_u + \eta A_u)\frac{S_u}{N} + \beta\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_u}{N} - \sigma E_u$$

This equation states the rate of change in exposed masked population. The rate of increase in exposed population corresponds to the rate of decrease in susceptible population, but it also accounts for the disease incubation period. Which means that it would take $1/\sigma$ days for susceptible population to move into exposed population upon the occurrence of a contact.

Rest of the equations are similar to the no-mask model, but just disaggregated based on mask-use (the subscript 'm' and 'u')

$$\frac{dI_u}{dt} = \alpha\sigma E_u - \varphi I_u - \gamma_I I_u$$

$$\frac{dA_u}{dt} = (1-\alpha)\sigma E_u - \gamma_A A_u$$

$$\frac{dH_u}{dt} = \varphi I_u - \delta H_u - \gamma_H H_u$$

$$\frac{dR_u}{dt} = \gamma_I I_u + \gamma_A A_u + \gamma_H H_u$$

$$\frac{dD_u}{dt} = \delta H_u$$

$$\frac{dS_m}{dt} = -\beta(1-\varepsilon_i)(I_u + \eta A_u)\frac{S_m}{N} - \beta(1-\varepsilon_i)((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m)\frac{S_m}{N}$$

$$\frac{dE_m}{dt} = \beta(1-\varepsilon_i)(I_u + \eta A_u)\frac{S_m}{N} + \beta(1-\varepsilon_i)\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_m}{N} - \sigma E_m$$

$$\frac{dI_m}{dt} = \alpha\sigma E_m - \varphi I_m - \gamma_I I_m$$

$$\frac{dA_m}{dt} = (1-\alpha)\sigma E_m - \gamma_A A_m$$

$$\frac{dH_m}{dt} = \varphi I_m - \delta H_m - \gamma_H H_m$$

$$\frac{dR_m}{dt} = \gamma_I I_m + \gamma_A A_m + \gamma_H H_m$$

$$\frac{\delta D_m}{dt} = \delta H_m$$

$$N = S_u + E_u + I_u + A_u + R_u + S_m + E_m + I_m + A_m + R_m$$

Model with imperfect general population mask use

Model with imperfect general population mask use divides the population into four classes based on their mask use. The first group is of people those who do not use any face covering at all, while the second group of those using face covering is further divided into three subclasses. The first subclass is of those using perfect face covering, the second is of those using perfect face covering but have come in contact with an infected fomite and the third subclass is of those improperly wearing a face covering. These sub-classes are represented with following subscripts: m (masked), u (unmasked), f (fomite-contact), x (improperly covered). The probability of a fomite being infected is assumed for simplicity of the model. The rationale behind this formulation is to take into account a secondary mode of transmission and evaluate the mask mandates. It is significant to note the difference between the effectiveness of masks versus the effectiveness of mask mandates. Even if masks reduce the spread when evaluated in perfectly conducive scenarios, the claim becomes invalid pretty soon in real world. Practically,

the infection can spread through different mechanisms and as such, it becomes imperative to assess the effectiveness of masks when subjected to these spread mechanisms. The introduction of fomite class helps capture this idea, especially in the closed settings. Whether it is an administrative building, or restaurants, the spread through human contact can be minimized by maintaining the social distancing. The problem arises at the high-frequency areas like door handles, purchase counters, and other commonly accessed areas. When there is no other way to enter a building without touching the door handle to open the door, everyone entering is inadvertently in contact with everyone else. If an infection is present at that point, the spread is then more dependent on handwashing and fomite contact and less on face coverings. Thus, incorporating a class representing those who have come in contact with high-frequency fomites will provide us with better assessment of mask mandates in closed settings. The model can be represented with the following set of equations:

$$\frac{dS_u}{dt} = -\beta(I_u + \eta A_u)\frac{S_u}{N} - \beta\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_u}{N} - \beta\big(I_f + \eta A_f\big)\frac{S_u}{N} - \beta(\Big(1-\frac{\varepsilon_o}{2}\Big)I_x + \Big(1-\frac{\varepsilon_o}{2}\Big)\eta A_x)\frac{S_u}{N}$$

The important parameter to note here is the outward mask efficiency (ε_o) for the fomite-contact (f) and improper-mask (x) sub-class. The ε_o is considered as 0 and $\frac{1}{2}\varepsilon_o$ for 'f' and 'x' sub-class, respectively. It is assumed for simplicity that a mask wore improperly is only 50% effective, while the persons who have come in contact with infected fomites carry the infection via the contact points and thus, their transmission rate is independent of mask use.

$$\frac{dE_u}{dt} = \beta(I_u + \eta A_u)\frac{S_u}{N} + \beta\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_u}{N} + \beta\big(I_f + \eta A_f\big)\frac{S_u}{N} + \beta(\Big(1-\frac{\varepsilon_o}{2}\Big)I_x + \Big(1-\frac{\varepsilon_o}{2}\Big)\eta A_x)\frac{S_u}{N} - \sigma E_u$$

$$\frac{dI_u}{dt} = \alpha\sigma E_u - \varphi I_u - \gamma_I I_u$$

$$\frac{dA_u}{dt} = (1-\alpha)\sigma E_u - \gamma_A A_u$$

$$\frac{dH_u}{dt} = \varphi I_u - \delta H_u - \gamma_H H_u$$

$$\frac{dR_u}{dt} = \gamma_I I_u + \gamma_A A_u + \gamma_H H_u$$

$$\frac{\delta D_u}{dt} = \delta H_u$$

$$\frac{dS_m}{dt} = -\beta(1-\varepsilon_i)(I_u + \eta A_u)\frac{S_m}{N} - \beta(1-\varepsilon_i)\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_m}{N}$$
$$- \beta\big(I_f + \eta A_f\big)\frac{S_m}{N} - \beta(1-\varepsilon_i)\left(\left(1-\frac{\varepsilon_o}{2}\right)I_x + \left(1-\frac{\varepsilon_o}{2}\right)\eta A_x\right)\frac{S_m}{N}$$

$$\frac{dE_m}{dt} = \beta(1-\varepsilon_i)(I_u + \eta A_u)\frac{S_m}{N} + \beta(1-\varepsilon_i)\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_m}{N}$$
$$+ \beta\big(I_f + \eta A_f\big)\frac{S_m}{N} + \beta(1-\varepsilon_i)\left(\left(1-\frac{\varepsilon_o}{2}\right)I_x + \left(1-\frac{\varepsilon_o}{2}\right)\eta A_x\right)\frac{S_m}{N} - \sigma E_m$$

$$\frac{dI_m}{dt} = \alpha\sigma E_m - \varphi I_m - \gamma_I I_m$$

$$\frac{dA_m}{dt} = (1-\alpha)\sigma E_m - \gamma_A A_m$$

$$\frac{dH_m}{dt} = \varphi I_m - \delta H_m - \gamma_H H_m$$

$$\frac{dR_m}{dt} = \gamma_I I_m + \gamma_A A_m + \gamma_H H_m$$

$$\frac{\delta D_m}{dt} = \delta H_m$$

$$\frac{dS_f}{dt} = -\beta(1-\varepsilon_i)(I_u + \eta A_u)\frac{S_f}{N} - \beta(1-\varepsilon_i)\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_f}{N} - \beta\big(I_f + \eta A_f\big)\frac{S_f}{N}$$
$$- \beta(1-\varepsilon_i)\left(\left(1-\frac{\varepsilon_o}{2}\right)I_x + \left(1-\frac{\varepsilon_o}{2}\right)\eta A_x\right)\frac{S_f}{N}$$

The inward mask efficiency (ε_i) is considered 1 against the masked, unmasked, and improperly masked sub-classes. The ε_i is considered zero against fomite-contact sub-class as the spread in this sub-class is independent of mask.

$$\frac{dE_f}{dt} = \beta(1-\varepsilon_i)(I_u + \eta A_u)\frac{S_f}{N} + \beta(1-\varepsilon_i)\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_f}{N} + \beta\big(I_f + \eta A_f\big)\frac{S_f}{N}$$
$$+ \beta(1-\varepsilon_i)\left(\left(1-\frac{\varepsilon_o}{2}\right)I_x + \left(1-\frac{\varepsilon_o}{2}\right)\eta A_x\right)\frac{S_f}{N} - \sigma E_f$$

$$\frac{dI_f}{dt} = \alpha\sigma E_f - \varphi I_f - \gamma_I I_f$$

$$\frac{dA_f}{dt} = (1-\alpha)\sigma E_f - \gamma_A A_f$$

$$\frac{dH_f}{dt} = \varphi I_f - \delta H_f - \gamma_H H_f$$

$$\frac{dR_f}{dt} = \gamma_I I_f - \gamma_A A_f - \gamma_H H_f$$

$$\frac{\delta D_f}{dt} = \delta H_f$$

$$\frac{dS_x}{dt} = -\beta\left(1-\frac{\varepsilon_i}{2}\right)(I_u + \eta A_u)\frac{S_x}{N} - \beta\left(1-\frac{\varepsilon_i}{2}\right)\big((1-\varepsilon_o)I_m + (1-\varepsilon_o)\eta A_m\big)\frac{S_x}{N}$$
$$- \beta\big(I_f + \eta A_f\big)\frac{S_x}{N} - \beta\left(1-\frac{\varepsilon_i}{2}\right)\left(\left(1-\frac{\varepsilon_o}{2}\right)I_x + \left(1-\frac{\varepsilon_o}{2}\right)\eta A_x\right)\frac{S_x}{N}$$

The inward mask efficiency (ε_i) is considered 50% against the masked, unmasked, and improperly masked sub-classes. The ε_i is considered zero against fomite-contact sub-class as the spread in this sub-class is independent of mask.

$$\frac{dE_x}{dt} = \beta\left(1-\frac{\varepsilon_i}{2}\right)(I_u+\eta A_u)\frac{S_x}{N} + \beta\left(1-\frac{\varepsilon_i}{2}\right)\left((1-\varepsilon_o)I_m+(1-\varepsilon_o)\eta A_m\right)\frac{S_x}{N}$$
$$+\beta\left(I_f+\eta A_f\right)\frac{S_x}{N} + \beta\left(1-\frac{\varepsilon_i}{2}\right)\left(\left(1-\frac{\varepsilon_o}{2}\right)I_x+\left(1-\frac{\varepsilon_o}{2}\right)\eta A_x\right)\frac{S_x}{N} - \sigma E_x$$

$$\frac{dI_x}{dt} = \alpha\sigma E_x - \varphi I_x - \gamma_I I_x$$

$$\frac{dA_x}{dt} = (1-\alpha)\sigma E_x - \gamma_A A_x$$

$$\frac{dH_x}{dt} = \varphi I_x - \delta H_x - \gamma_H H_x$$

$$\frac{dR_x}{dt} = \gamma_I I_x - \gamma_A A_x - \gamma_H H_x$$

$$\frac{\delta D_x}{dt} = \delta H_x$$

Baseline epidemiological parameters

The epidemiological parameters have been taken from the literature. The following tables describes all epidemiological parameters considered and their respective source.

Table 1: Basic epidemiological parameters

Parameter	Value	Source	Method
Incubation period (1/σ)	5.1 days	Lauer et al. [34]	Pooled analysis of confirmed COVID-19 cases reported between 4 January 2020 and 24 February 2020.
Effective transmission rate (β_0)	0.5 – 1.5	Eikenberry et al. [33]	Free parameter in the fits
Relative infectiousness of	0.5	Fergusson et al. [35]	Assumption

Parameter	Value	Source	Method
asymptomatic carriers (η)	0.42-0.55	Li et al. [36]	
Fraction of symptomatic cases (α)	0.5	Eikenberry et al. [33]	Assumption
Rate of hospitalization of symptomatic individuals (ψ)	0.025 day^{-1}	Eikenberry et al. [33]	Anecdotal midpoint
Disease-induced death rate (δ)	0.015 day^{-1}	Fergusson et al. [35]	Clinical opinion
Recovery rate, asymptomatic (γ_A)	1/7 day^{-1}	Eikenberry et al. [33]	Mean of historical data
Recovery rate, symptomatic (γ_I)	1/7 day^{-1}	Eikenberry et al. [33]	Mean of historical data
Recovery rate, hospitalized (γ_H)	1/14 day^{-1}	Eikenberry et al. [33]	Mean of historical data

CHAPTER 4. RESULTS

Population class curves

Model with no mask use

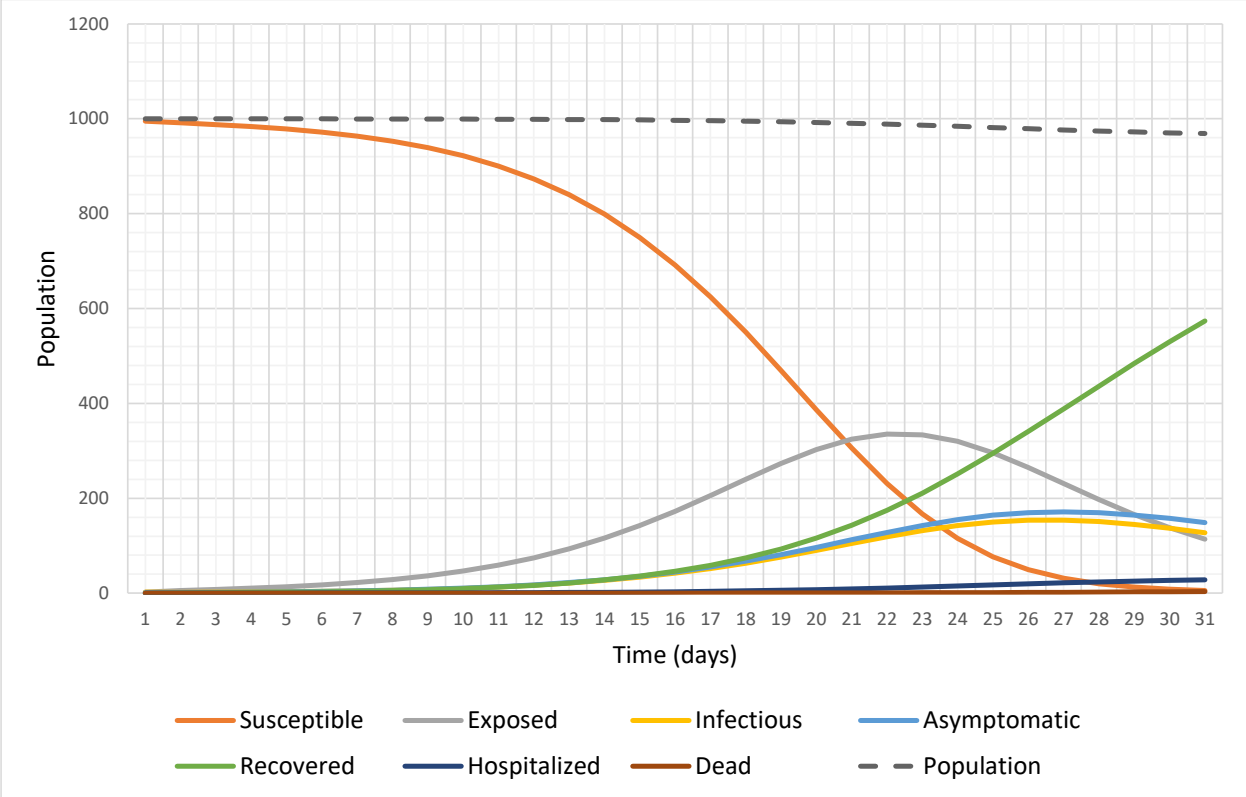

Figure 1: Model with no mask use

The above chart shows the progression of various population classes without any mask use. The population is exposed steadily with an equal fraction of symptomatic and asymptomatic cases as per our assumption. The curve starts flattening at day 22. The recovery overtakes infections on day 24. Based on our assumed hospitalization rate, very few cases need to be hospitalized and reach disease-induced death stage. The susceptible population number decreases sharply and moves lower than infected class much earlier on day 21. This goes in line with the notion that letting a pandemic run its course helps in attending herd immunity the fastest, but of

course, at the cost of increased hospitalizations and deaths. Analyzing this chart in singularity does not provide enough context, neither gives any recommendations to better prepare for the pandemic. We need to compare this with other scenarios to get the basic efficacy of mask guidelines, and then build a model with very specific data to get the timeline of progression. The pandemic can be best battled by improvising the guidelines in accordance with the progression of disease and consequently, by reacting ahead of time to prevent fatalities.

Model with perfect general population mask use

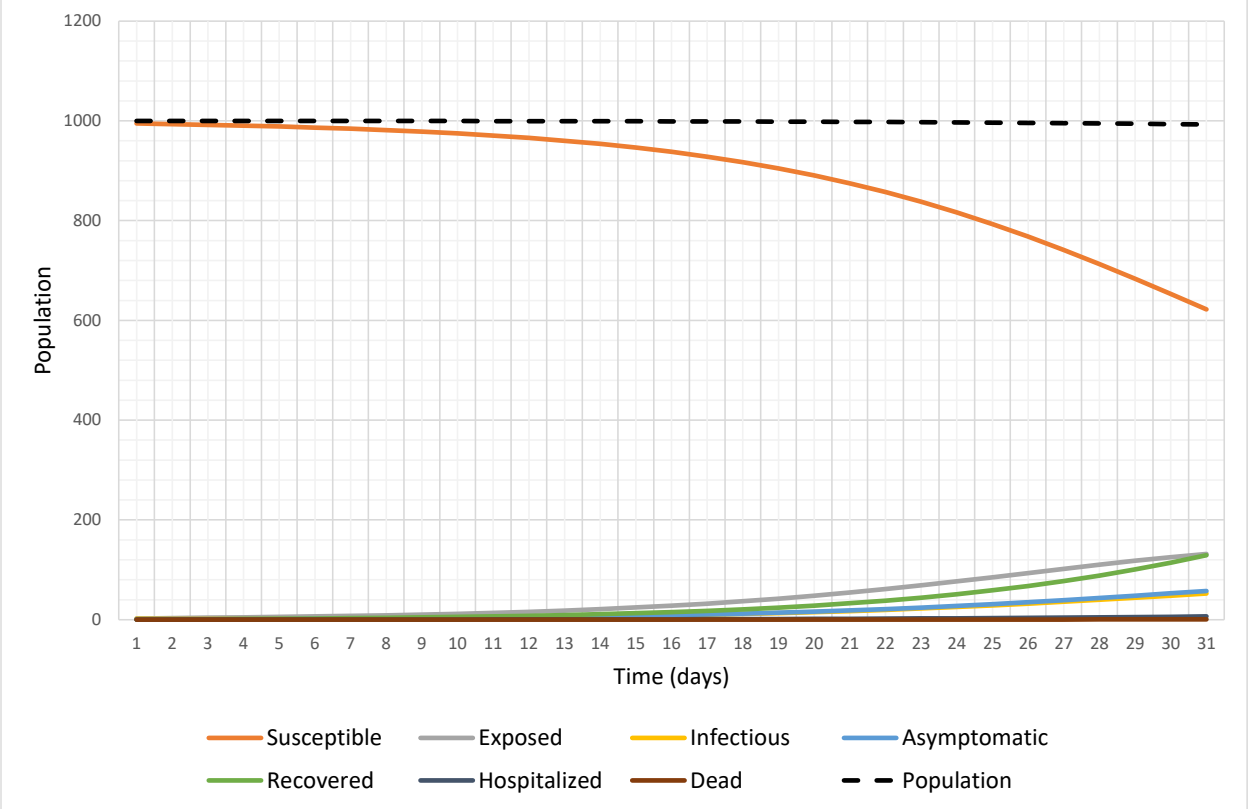

Figure 2: Model with perfect general population mask use

In our current assumption state, this model provides no significant information. Only thing to note here is with perfect general population mask use, the pandemic can be controlled to a significant extent and the curve of infectiousness stays flat with minimum hospitalizations and

deaths. Thus, it can be inferred that, with perfect mask use, we can avoid burdening the healthcare systems and give them breathing space to operate. The trade-off here is lesser herd immunity. In contrast, it means much of the population is still susceptible to the disease, and a change in underlying parameters can increase the risk greatly. Perhaps, a new strain of the virus, or a long-term side-effect can change things quickly.

Model with imperfect general population mask use

In models with general population mask use, beta (transmission rate) becomes a crucial parameter. In this scenario, we assume the default inward and outward efficiency for masks but take into account the spread through surface contacts and fomites. The surface contact depends on a number of factors like design of the building, high frequency areas, and intervention methods like hand-wash. These factors can be represented with a changing transmission rate and hence, it will be interesting to see the model play out with different values of beta.

Beta = 1

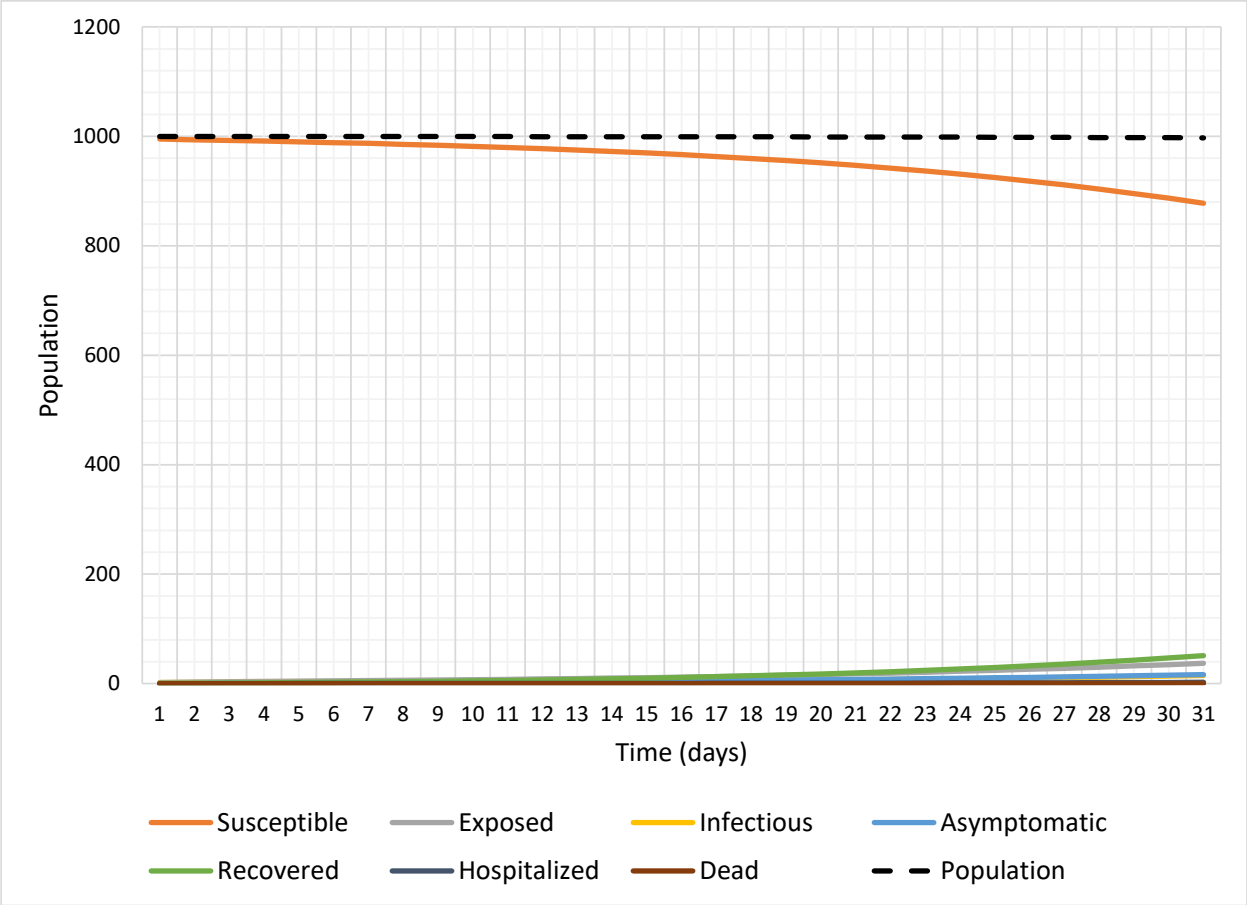

Figure 3: Model with imperfect general population mask use (beta = 1)

Beta = 2

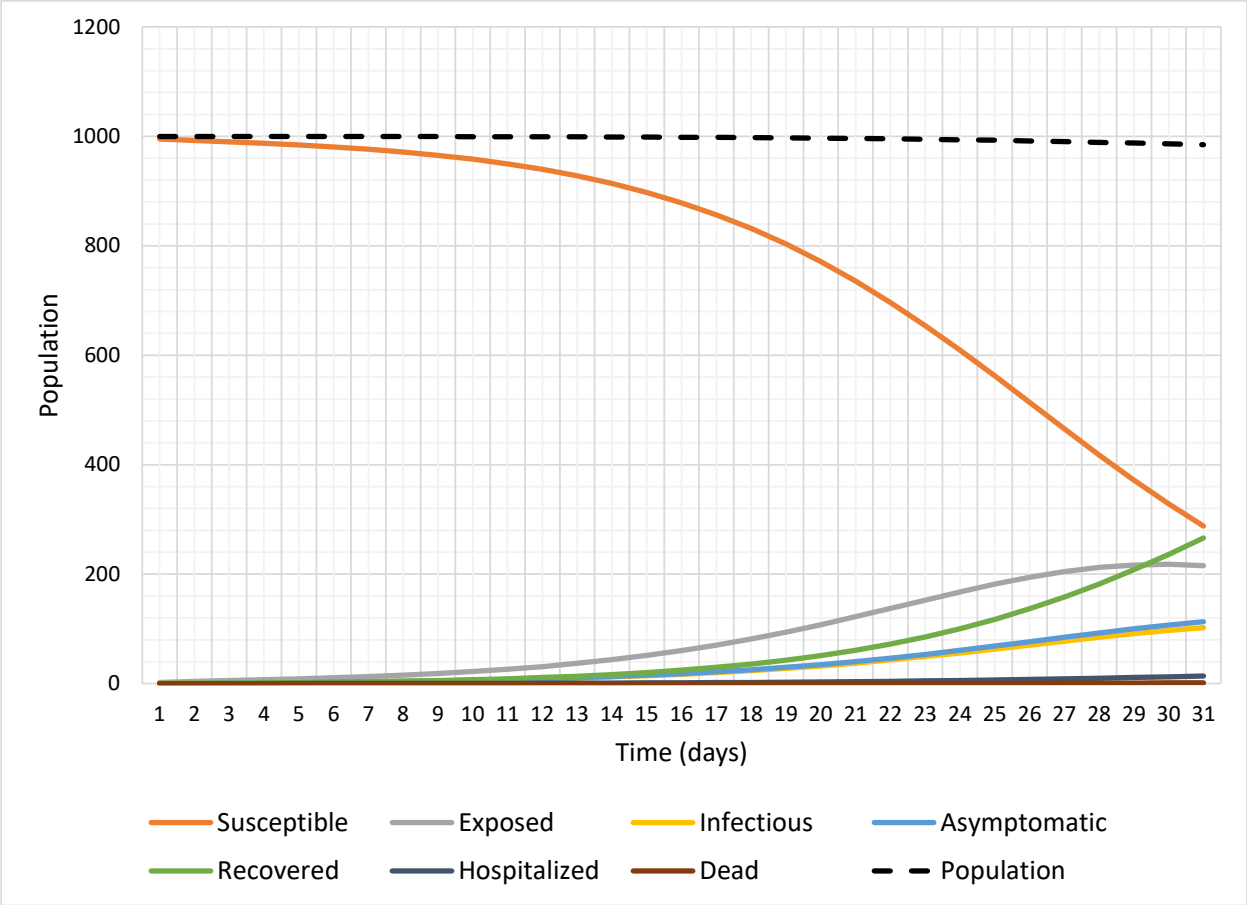

Figure 4: Model with imperfect general population mask use (beta = 2)

Beta = 3

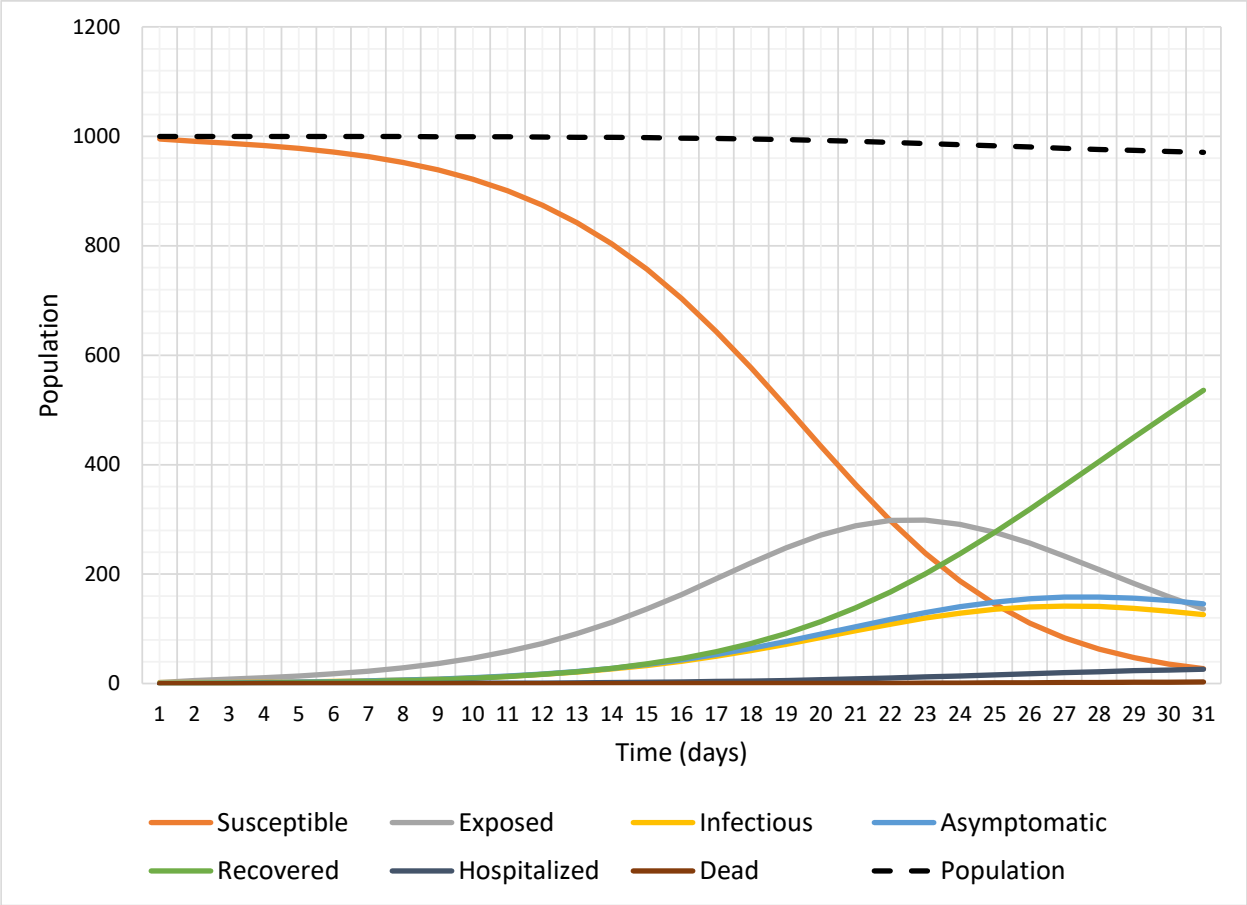

Figure 5: Model with imperfect general population mask use (beta =3)

It is interesting to see that the numbers play out like those in the case of perfect mask use when beta is 1. It is obvious as the underlying transmission rate does not change, and the inward and outward mask efficiencies stay the same. The marginal difference is due to a sub-class coming into contact with infected fomites which is controlled by a tight transmission rate parameter. As the beta increases, the numbers play out similar to those in the case of no mask use. This concludes that for mask mandates to be effective, surface contact must be minimized first. In various closed settings with mask mandates, the effective transmission rate is high owing to the high contact rate in high-frequency areas.

Comparison of model with default baseline parameters

Table 2: Comparison of model with default baseline parameters

Event	Model with no mask use	Model with perfect general population mask use	Model with imperfect general population mask use		
			Beta = 1`	Beta = 2	Beta = 3
Total infections at peak (I + A)	325	110	32	215	271
Total hospitalized	28	7	3	14	26
Total deaths	3	1	0	1	3
Susceptible population lower than infected population on day –	23	-	-	-	25
Recoveries overtake exposed on day –	27	-	-	-	25
Exposed curve flattens on day –	24	-	-	30	23

The above table represents the comparison for different scenarios we calculated. It is interesting to note that with imperfect mask use, the infections are comparatively lesser, but hospitalizations and deaths are equal to the model with no mask use. In imperfect mask use model, the model fails to give any worthwhile information for beta = 1, as the population is divided into four sub-classes. This leaves us with too few individuals in the sub-class for an

epidemic, which progresses exponentially, to show any effect. It would be a better approach to adjust the beta or consider a large enough population for the model to give complete picture.

Case study – population class curves for Iowa

Model with no mask use

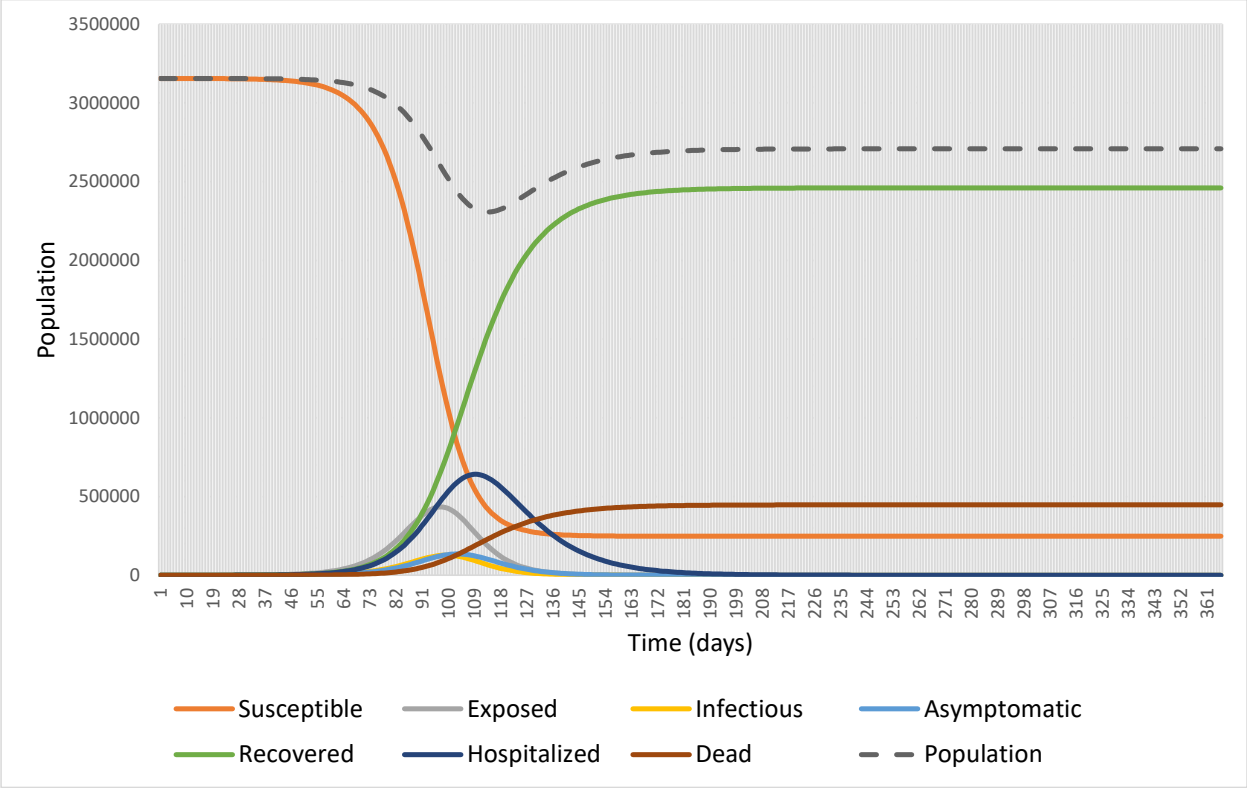

Figure 6: Iowa: Model with no mask use

Model with no mask use (simulated for 175 days)

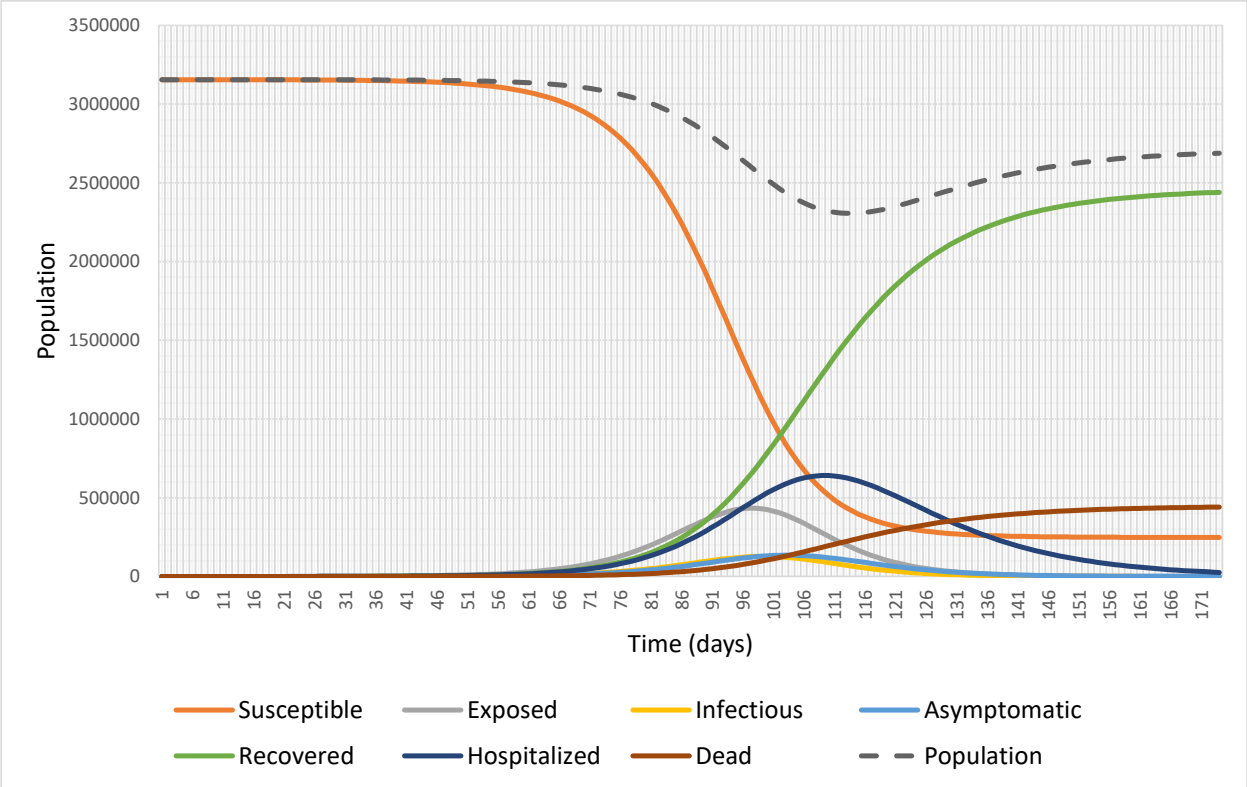

Figure 7: Iowa - Model with no mask use (175 days)

Here we simulated a model for the state of Iowa with real parameters. The parameters were taken from an official government website [37]. It is interesting to see how quickly the pandemic progresses without an intervention, but it comes at the cost of increased hospitalizations and deaths. It must be noted that, the increase in population after the initial slump does not indicate addition of new population but decrease in number of hospitalizations. Remember we do not consider hospitalized persons in our calculation as they do not spread the infection in outdoor settings. As the pandemic progresses, less people are hospitalized and hence we see the increase in total population.

Model with perfect general population mask use

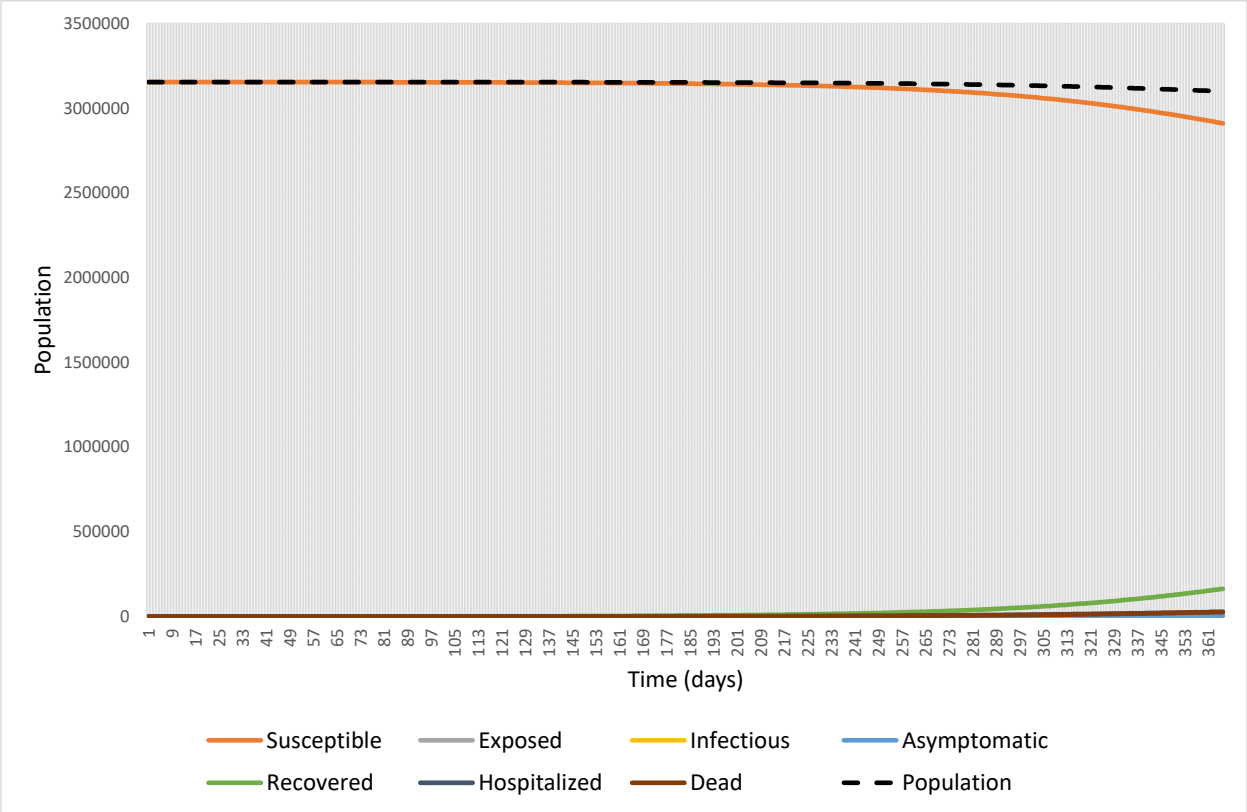

Figure 8: Iowa - model with perfect general population mask use

The above chart shows that with perfect mask use, a very small subset of population gets infected. This represents an idealistic assumption which is never the case in real world. There are no significant takeaways from this experiment except that at a high level, ideal use of face coverings helps in preventing the spread.

Model with imperfect general population mask use

Beta = 2

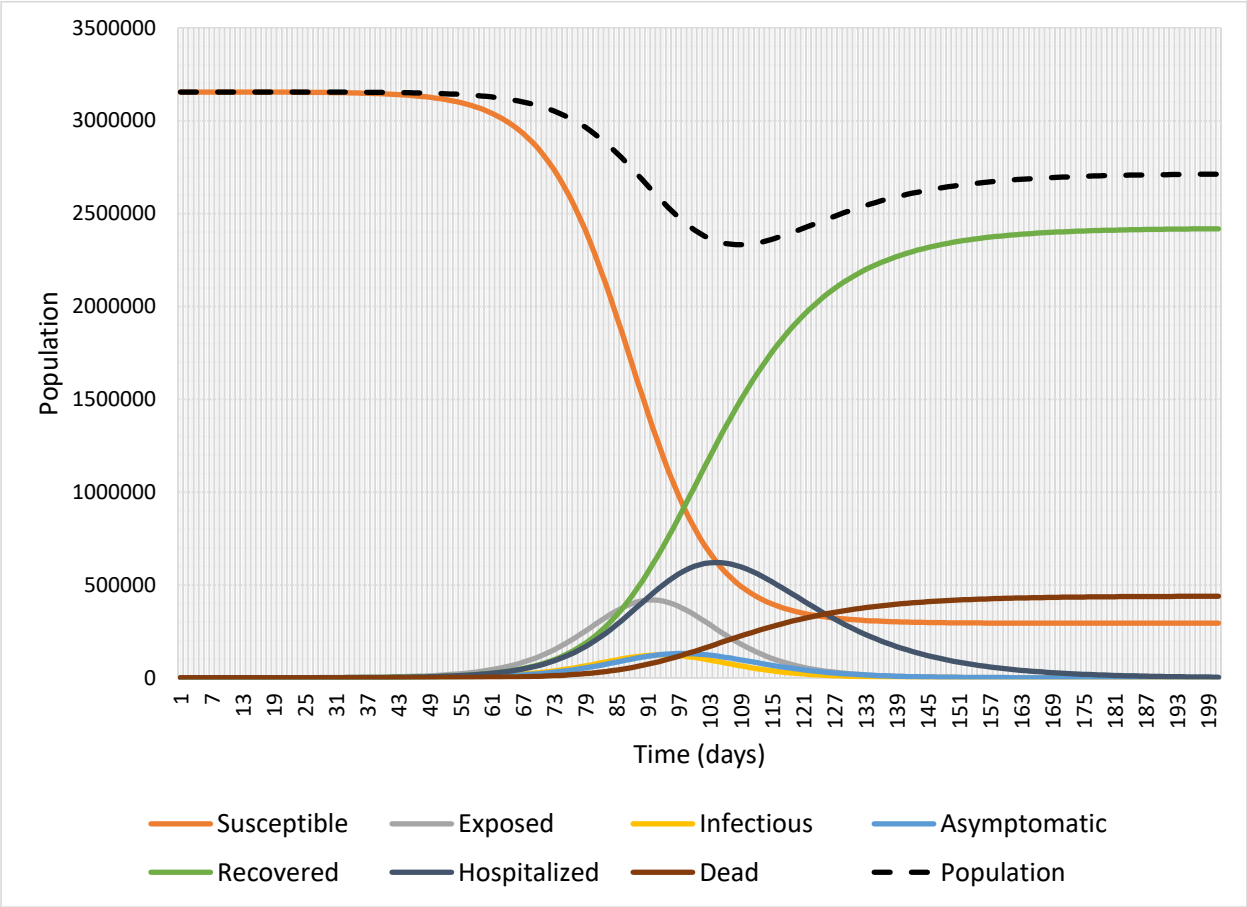

Figure 9: Iowa - model with imperfect general population mask use (beta =2)

Beta = 3

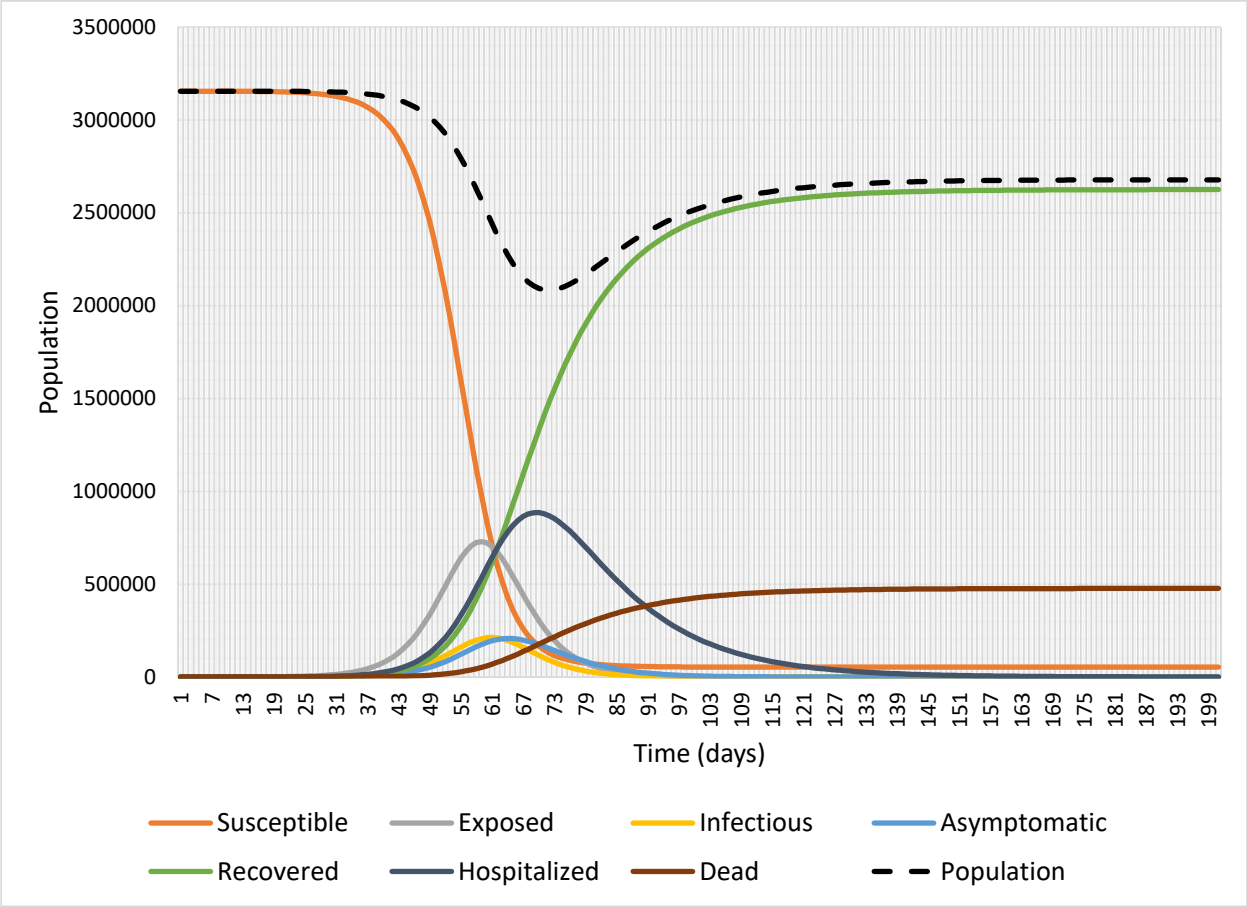

Figure 10: Iowa - model with imperfect general population mask use (beta =3)

We did not consider a model for beta =1 as it simulates an idealized scenario which is quite unrealistic and has no practical takeaways. For above scenarios with beta = 2 and beta = 3, a clear difference can be seen in progression of curves. There are significantly higher deaths in latter scenario, while the positive is almost entire population recovers at around 90-day mark. This does not hold true in real cases as the population is not a closed space and there is a possibility of reinfection. The most important takeaway here is that lower the beta, flatter the progression curve. This can be of great significance since, in most real-life cases the objective is to buy time to relieve healthcare resources. Thus, the conclusion can be drawn that the directives

should focus on tackling surface contacts in high frequency areas for any mask mandate to be effective.

Total affected population curve

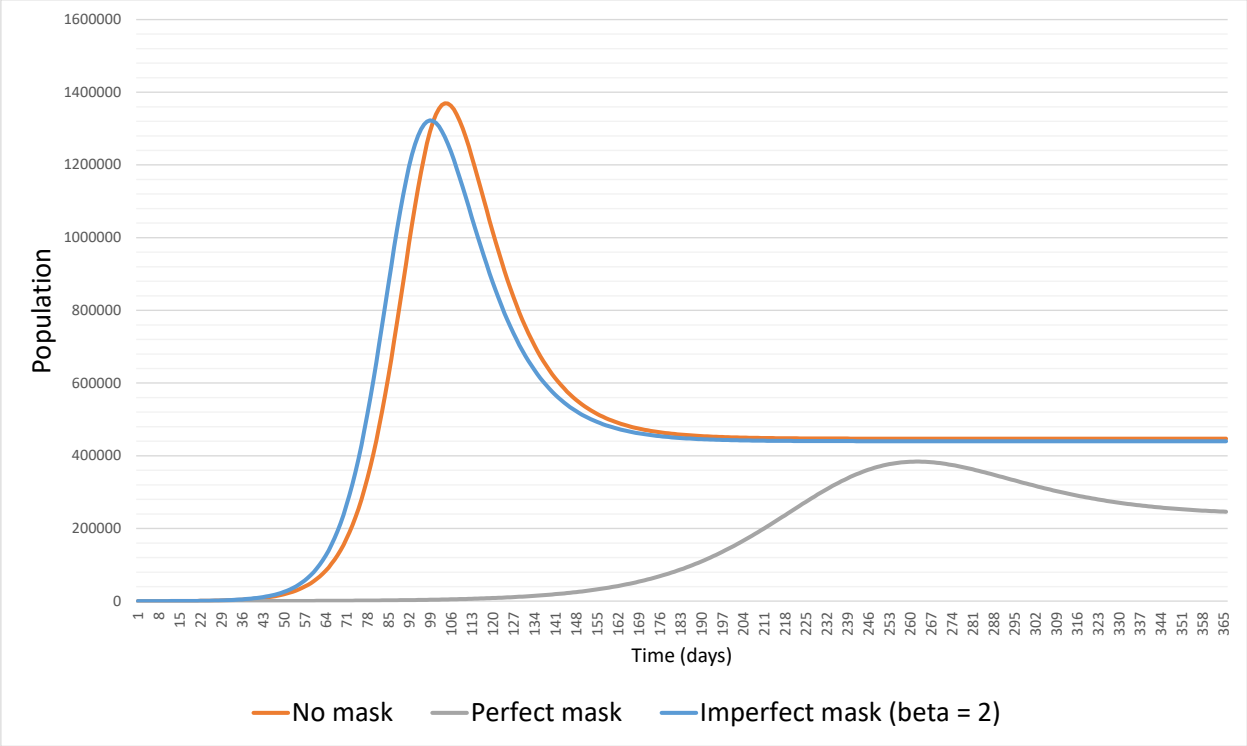

Figure 11: Iowa - total affected population curve

The above chart is a simple plot of affected population with respect to time. It visualizes the idea of flattening the curve. As expected, perfect mask use results in best possible progression, while no mask use results in worst. With no masks, the cases rise sharply, thus putting unreasonable stress on healthcare resources. The imperfect mask use models lie somewhere in the between with beta being the controlling parameter. The above chart represents beta = 2, the midpoint value, and thus lies closer to no mask model.

CHAPTER 5. CONCLUSION

We get crucial takeaways from the three scenarios modeled. While it is true that prevailing mask use proves to be an effective weapon against tackling the pandemic, the general mask population use depends significantly on the effective transmission rate. The effective transmission rate in outdoor setting increases with increasing high frequency areas like entrance doorknobs, purchase counters, billing stations, etc. For any mask mandate to be effective on a larger scale, high compliance and lower transmission rates are underlying necessities. The thesis contributes to showing this relation of practical mask mandates with mask mandate models with ideal assumptions.

Thus, a better approach to reducing the spread during a pandemic is to use masks and reduce surface contacts, place other interventions like sanitizer and hand soaps in high-frequency areas. Without these complementary efforts, mask mandates alone have little to no effect on spread. The same phenomenon can be seen validated anecdotally [38]. The states with no-mask mandate do not necessarily do worse than the states with mask-mandates. This holds true especially in the non-metro areas of United States where population density is relatively lower. The lower population density inadvertently ensures social distancing and curbs the spread through human contact. In such places, an added effort with reducing surface contact coupled with existing mask mandates will prove to be the most effective strategy.

CHAPTER 6. FUTURE SCOPE

Since, this is a compartmentalized model, the model can be extended by adding more compartments. It will be an interesting effort to see how policies affect various risk groups. The susceptible population can be further divided into different compartments based on risk parameter. Several studies have shown obesity as a risk factor for covid-19 severity [39]. By considering these risk factors, we can have a better plan for those at higher risk than the general population. Studies have also found that most high-risk patients showed an underlying vitamin-D deficiency [40]. These risk factors can be considered to further classify susceptible population.

The model can be customized for a particular setting. Since each outdoor setting is different in terms of design, floorplan, occupancy, and other behavioral factors, the parameters can be customized to calculate risk factors independently for those settings.

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