

# The attainment of herd immunity with reduced risk of contact infection in modelling

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## Research Article

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# **The attainment of herd immunity with reduced risk of contact infection in modelling**

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## **Abstract**

The risk of contact infection among susceptible individuals in a randomly mixed population can be reduced by the presence of immune individuals and this concept is referred to as herd immunity<sup>1-3</sup>. The conventional susceptible-infectious-recovered (SIR) model does not feature a reduced risk of susceptible individuals in the transmission dynamics of infectious disease, therefore violates the fundamental of herd immunity<sup>4</sup>. Here we show that the reduced risk of contact infection among susceptible individuals in the SIR model can be attained by incorporating the proportion of susceptible individuals (model A) or the inverse of proportion of recovered individuals (model B) in the force of infection of the SIR model. We simulated the conventional SIR model and both new SIR models under the exact condition with a basic reproduction ratio of 3.0 and an expected herd immunity threshold of 0.667 (66.7%). All three models performed likewise at the initial stage of an epidemic. In the conventional SIR model, the epidemic continued until 94.0 % of the population had been infected and recovered, way above the threshold for eradication and control of the epidemic. Both models A and B simulated the epidemic waning when 66.7% and 75.6% of the population had been infected, as a result of the herd effect. As a result, model A provides a better framework for modelling vaccine-induced herd or population immunity, while model B provides a better framework for modelling infection-induced herd or population immunity. Our results demonstrate how the new modelling framework overcomes the drawback of the conventional SIR model and attain the effect of herd immunity in modelling outputs, which is important for modelling infectious disease in a randomly mixed population, especially for the COVID-19 pandemic.

## **Introduction**

Herd immunity became a fixture of epidemiology in 1930s, and took on fresh prominence in 1950s and 1960s as new vaccines raised crucial questions for public health policy on the proportion of the vaccinated population for the eradication of infectious disease<sup>2</sup>. The fundamental of herd immunity is that the risk of contact infection among susceptible individuals can be reduced by the presence and proximity of immune individuals in a randomly mixed population<sup>1</sup>. Although the impact of herd immunity has been observed in the vaccinated population of many periodical childhood epidemics, such as measles, mumps, rubella, pertussis, chickenpox, and polio for decades, it has never been truly attained in mathematical models.

Mathematical models such as deterministic models are widely used to study infection dynamics, especially in the COVID-19 pandemic. These models use a flexible compartmental

framework and robust assumptions for a wide range of indications<sup>5-8</sup>. The susceptible-infectious-recovered (SIR) model classifies individuals by their epidemiological status, based on their ability to host and transmit a pathogen<sup>9</sup>. This approach simplifies the manifestation of infection dynamics for most infectious diseases. Besides compartmentalization, the SIR model also assumes complete immunity is conferred by a single infection, hence encompassing the epidemiological notion of herd immunity<sup>1,3,4</sup>. However, the failure to incorporate the reduced risk of contact infection in the deterministic framework may lead to inaccurate estimation of disease dynamics and parameters during simulation.

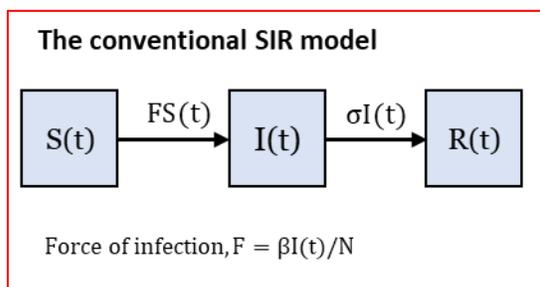
Based on declining transmissibility, the herd immunity threshold (HIT) can be estimated from the basic reproduction ratio ( $r_0$ ) of the SIR model to guide the eradication program for a target epidemic through vaccination<sup>1</sup>. However, the conventional SIR model simulates an epidemic with  $r_0$  of 3.0 waning when 94% of a population have been infected and recovered with immunity, far beyond the HIT of 67% required for control or eradication of an epidemic. The disconnection between model forecast and HIT exposes the fundamental drawback of using the conventional SIR model in modelling any infectious diseases in a randomly mixed population, especially for infectious diseases with high transmissibility like the COVID-19. As many models have been configured based on the conventional SIR framework during the COVID-19 pandemic, the vagueness of the conventional SIR model in modelling transmission dynamics of infectious disease in a randomly mixed population of practical and exceptional concern now<sup>10-14</sup>.

This study introduces two SIR frameworks that overcome the limitation of the conventional SIR model in attaining herd or population immunity, either through infection-induced immunity or vaccine-induced immunity in a randomly mixed population.

## Methods

### The Kermack & McKendrick's SIR model.

Kermack & McKendrick postulated the first SIR deterministic model for infectious disease in 1927, on the basis that an infectious disease spreads from affected to unaffected by contact infection<sup>4</sup>. The SIR model then became the fundamental of most infectious disease models developed since. In the SIR model, contact infection refers to the direct or indirect act of contracting pathogen, with the risk of contact infection given by the proportion of infectious individuals in a population. Besides, the SIR model also assumes that infectious disease would spread exponentially at the beginning and eventually diminishes due to the exhausting number of susceptible individuals<sup>4,9</sup>.



**Figure 1:** The conventional SIR model developed by Kermack & McKendrick (1927). Individuals move from class S(t) to I(t) at a rate proportional to  $\beta, \frac{I(t)}{N}$ , and S(t). The proportion of infectious individuals signifies the risk of contact infection among susceptible individuals.

The conventional SIR model divides a homogenous population (N) into three distinct classes: susceptible denoted by S(t), infectious denoted by I(t), and recovered denoted by R(t). Susceptible are individuals at equal risk of contracting an infection. Infectious are infected individuals who have developed infectivity and can transmit pathogens like viruses when coming into contact with susceptible individuals. Recovered are individuals who have recovered from infection with immunity. As the SIR model assumes recovered individuals are no longer susceptible to infection, hence, recovered individuals are also regarded as “removed” from the infection dynamics (Fig. 1). In brief, the conventional SIR model features a partially mixed population, in which the rate of disease transmission can only be affected by the proportion of infectious individuals in the population.

In the SIR model, the transition of individuals between classes occurs at a rate proportional to the number of individuals in the respective classes, and constant coefficients of transmission and recovery. For instance, individuals move from class S(t) to I(t) at a transition rate proportional to the number of susceptible individuals, S(t), the transmission coefficient ( $\beta$ ), and the risk of contact infection, given by the proportion of infectious individuals  $\frac{I(t)}{N}$ . The product of  $\beta$  and  $\frac{I(t)}{N}$  denotes the force of infection,  $F$  of an infectious disease. After being infected, individuals would start to recover and move from class I(t) to R(t) at a transition rate proportional to the number of infectious individuals, I(t), and recovery coefficient ( $\sigma$ ), given by the reciprocal of infection duration.

Mathematically, the conventional SIR model functions in a set of coupled nonlinear ordinary differential equations (ODEs) that deterministically describes the slopes or flows between classes over time as follows:

$$\frac{dS(t)}{dt} = -\frac{\beta I(t)S(t)}{N}, \quad (1-1)$$

$$\frac{dI(t)}{dt} = \frac{\beta I(t)S(t)}{N} - \sigma I(t), \quad (1-2)$$

$$\frac{dR(t)}{dt} = \sigma I(t). \quad (1-3)$$

Without vital dynamics, the population size is constant and can be given by:

$$N = S(t) + I(t) + R(t) \quad (2)$$

Equation (2) can be converted into prevalence or proportion by dividing each notation with N:

$$1 = \frac{S(t)}{N} + \frac{I(t)}{N} + \frac{R(t)}{N}. \quad (3)$$

The inverse of proportion of recovered individuals is then given by:

$$1 - \frac{R(t)}{N} = \frac{S(t)}{N} + \frac{I(t)}{N}. \quad (4)$$

At the initial stage of an epidemic, both the number of infectious and recovered individuals are very small as compared to the population size, therefore,  $\frac{I(t)}{N} \approx 0$ ,  $\frac{R(t)}{N} \approx 0$ , and  $\frac{S(t)}{N} \approx 1$ . At the end stage of an epidemic, the number of infectious individuals becomes very small again, therefore,  $1 - \frac{R(t)}{N} \approx \frac{S(t)}{N}$ . Besides  $\frac{I(t)}{N}$ , both  $\frac{S(t)}{N}$  and  $1 - \frac{R(t)}{N}$  can be further utilized as the risk determinant for contact infection among susceptible individuals in a randomly mixed population.

### The basic reproduction ratio, $r_0$

According to Equation (1-2) of the conventional SIR model. At the initial stage of an epidemic,  $\frac{S(t)}{N} \approx 1$ . Then, we would obtain the following equation:

$$\frac{dI(t)}{dt} = (\beta - \sigma)I(t). \quad (5)$$

The integral of Equation (5) is an exponential function as follow:

$$I(t) = I_0 e^{(\beta - \sigma)t}. \quad (6)$$

Equation (6) indicates a crucial state that determines the spread of an infectious disease at its initial stage. An infectious disease would spread exponentially if:

$$\beta - \sigma > 0, \quad (7)$$

$$\frac{\beta}{\sigma} - 1 > 0, \quad (8)$$

$$\frac{\beta}{\sigma} > 1. \quad (9)$$

The ratio between  $\beta$  and  $\sigma$  denotes the basic reproduction ratio ( $r_0$ ) of an infectious disease, which is also defined as the number of secondary cases caused by a single primary case in a wholly susceptible population<sup>15,16</sup>. An infectious disease would become epidemic when its  $r_0$  is bigger than 1. Otherwise, it would diminish. The herd immunity threshold (HIT) of an infectious disease can be derived from its  $r_0$  with the following equation:

$$\text{HIT} = 1 - \frac{1}{r_0}. \quad (10)$$

The HIT marks the level at which the proportion of susceptible individuals has to be vaccinated to control or eradicate infectious disease.<sup>17</sup>

### The risk of contact infection in a randomly mixed population

In a randomly mixed population, the proportion of each epidemiological class changes during the process of infection, and the risk of contact infection could also differ as a result of those proportional changes. For instance, the rising proportion of infectious individuals would

increase the risk of contact infection. At the same time, the declining proportion of susceptible and the rising proportion of recovered individuals decrease the risk of contact infection as well. To overcome the limitation of the conventional SIR framework in modelling infectious disease in a randomly mixed population, two models were proposed and developed based on two assumptions below:

**Assumption A:** The risk of contact infection among susceptible individuals would reduce as soon as they are infected and moved from class  $S(t)$  to class  $I(t)$ . In this circumstance, the risk of contact infection is given by proportion of both susceptible individuals,  $\frac{S(t)}{N}$  and infectious individuals,  $\frac{I(t)}{N}$ . By incorporating both proportions into Equation (1-1) to (1-3), we obtain the first set of ODEs as follow:

$$\frac{dS(t)}{dt} = -\frac{\beta I(t)[S(t)]^2}{N^2}, \quad (11-1)$$

$$\frac{dI(t)}{dt} = \frac{\beta I(t)[S(t)]^2}{N^2} - \sigma I(t), \quad (11-2)$$

$$\frac{dR(t)}{dt} = \sigma I(t). \quad (11-3)$$

The product of  $\beta$ ,  $\frac{I(t)}{N}$  and  $\frac{S(t)}{N}$  denotes the new force of infection,  $F_A$  of an infectious disease in the new SIR model A.

**Assumption B:** The risk of contact infection among susceptible individuals would reduce after infected individuals have recovered and moved from class  $I(t)$  to class  $R(t)$ . In this circumstance, the risk of contact infection is given by the proportion of infectious individuals,  $\frac{I(t)}{N}$  and the inverse of proportion of recovered individuals,  $1 - \frac{R(t)}{N}$  or  $\frac{N-R(t)}{N}$ . By incorporating both proportion and inverse of proportion into Equation (1-1) to (1-3), we obtain second set of ODEs as follow:

$$\frac{dS(t)}{dt} = -\frac{\beta I(t)[N-R(t)]S(t)}{N^2}, \quad (12-1)$$

$$\frac{dI(t)}{dt} = \frac{\beta I(t)[N-R(t)]S(t)}{N^2} - \sigma I(t), \quad (12-2)$$

$$\frac{dR(t)}{dt} = \sigma I(t). \quad (12-3)$$

The product of  $\beta$ ,  $\frac{I(t)}{N}$  and  $\frac{N-R(t)}{N}$  denotes the new force of infection,  $F_B$  of an infectious disease in the new SIR model B. The inverse of proportion of recovered individuals was used to signify the reduced risk of contact infection after recovery.

According to Equation (11-2) and (12-2), at the initial stage of an epidemic when proportions of infectious and recovered individuals approach zero, both models A and B would generate the same  $r_0$  and HIT values as of the conventional SIR model. Hence, all three models can accurately describe the initial dynamics of transmission at the initial stage of infection when proportions of both infectious and recovered individuals approach zero. However, both models

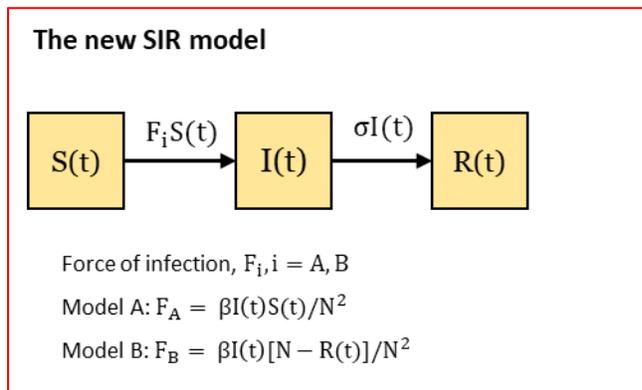
A and B would surpass the conventional SIR model at the later part of the epidemic when proportions of both infectious and recovered individuals can no longer be ignored.

### Model simulation and sensitivity analyses

First, we applied numerical simulations to obtain approximate solutions for all three models under the exact and arbitrary condition with  $\beta = 0.3$  and  $\sigma = 0.1$  to mimic infection dynamics with  $r_0 = 3.0$  and  $HIT = 0.667$ . In the arbitrary condition, 66.7% of population immunity level was required for herd immunity to take effect for control or eradication of epidemic. At the end of the epidemic, the proportion that remained susceptible in the population was expected to be 33.3%, given by the inverse of HIT. Next, we calculated differences between the conventional and new SIR models for susceptible, infectious, total infection (I Total), new infection (I New), and recovered over time  $t$ . In sensitivity analyses, we evaluated the total infections and recovered individuals achieved across  $r_0$  ranging from 1.1 to 4.0 in all three models. Numerical simulations were performed in R version 3.6.3 with “deSolve” package. Graphics were organized in Microsoft Excel 2019. Modelling outputs were presented in graphs with the proportion of population (%) as the y-axis and arbitrary time  $t$  as the x-axis.

### Results:

The new SIR model retains most of the underlying assumptions except for one, that is the rate of transmission can be reduced by the declining prevalence of susceptible individuals right after infection (model A) or the declining prevalence of susceptible individuals after recovery (model B) in a randomly mixed population. The above assumptions can be achieved by incorporating the proportion of susceptible individuals in model A or the inverse of proportion of recovered individuals in model B into the force of infection of the SIR model (Fig. 2).



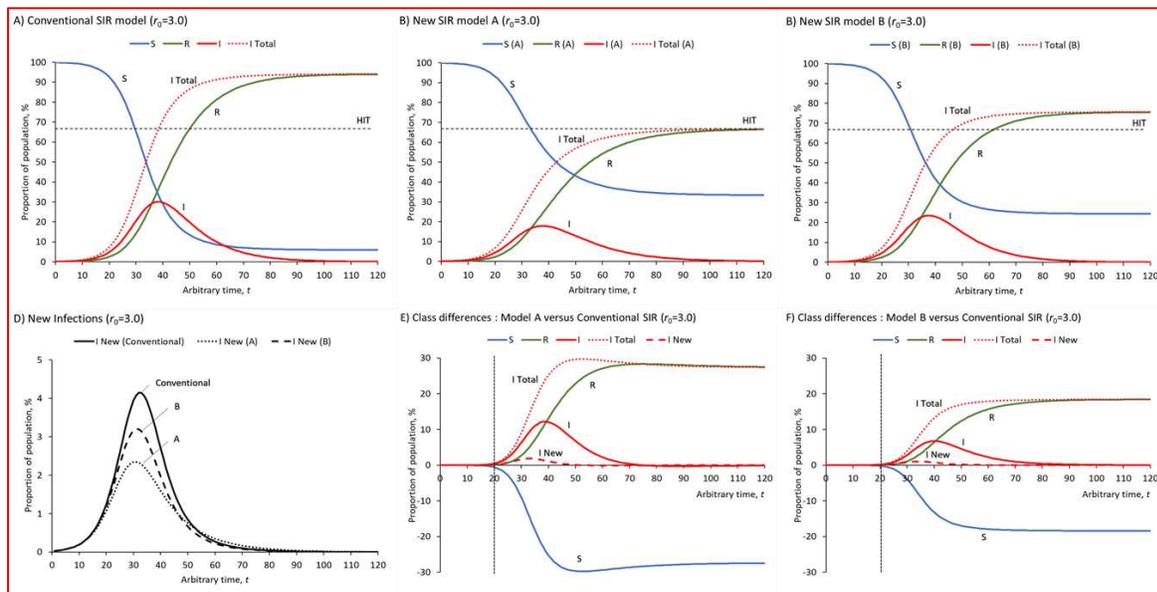
**Fig. 2:** The compartmental structure of new SIR model A and model B for infectious diseases in a randomly mixed population

At the initial stage of an epidemic, when the number of infectious and recovered individuals was very small compared to the population size, the proportion of infectious and recovered individuals would approach zero. Therefore, the conventional SIR model and both new models (model A and model B) would perform likewise. At the initial stage of an epidemic, the transmission dynamics of infectious disease can be accurately described by its basic reproduction number ( $r_0$ ) in all three models. Moreover, the herd immunity threshold (HIT) derived from the  $r_0$  value would be similar for infectious disease fitted using all three models. Figure 3 presents results of numerical simulations under the exact condition with transmission

coefficient ( $\beta$ )=0.3 and recovery coefficient ( $\sigma$ )=0.1 to mimic the transmission dynamics at  $r_0$  of 3.0 and HIT of 0.667 (66.7%) for all three models, respectively.

As a result of not incorporating the reduced risk of contact infection, the conventional SIR model simulated the epidemic waning at a level way above its HIT. According to Fig. 3A, the infectious disease transmitted and infected almost 94.0% of the population, then only diminished due to the exhausted susceptible stock for disease transmission. The proportion of total infections and recovered individuals was way above the HIT required for control and eradication of an infectious disease. Hence, the conventional SIR model did not display transmission dynamics controlled and eradicated at the expected immunity level. Expectedly, both new models presented transmission dynamics controlled consequent to the reduced risk of contact infection, as a result of incorporating the declining proportion of susceptible individuals in model A and the inverse of proportion of recovered individuals in model B. Model A simulated the epidemic waning when both total infections and recovered individuals reach 66.7%, in line with its HIT (Fig. 3B), while model B simulated the epidemic waning when 75.6% of the population had been infected and recovered with immunity, 8.9% above its HIT (Fig. 3C).

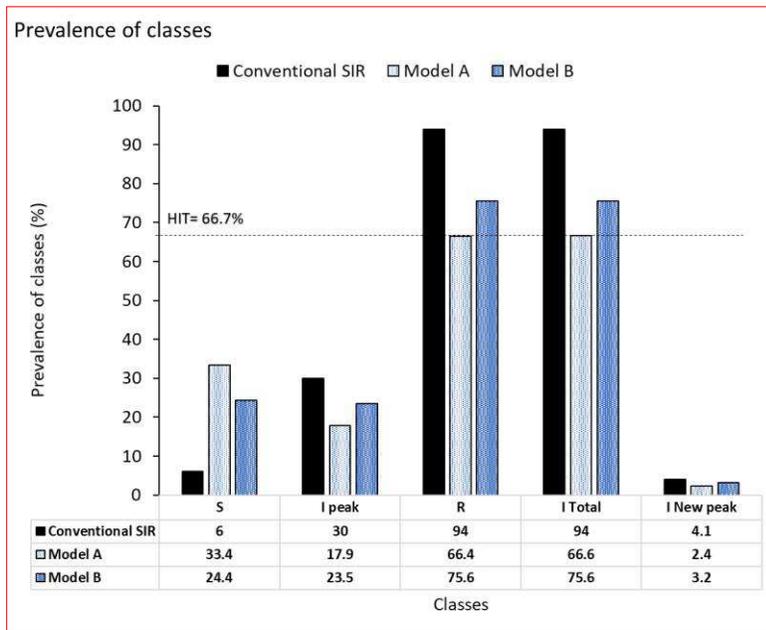
Besides, both new models generated fewer new infections than the conventional SIR model over time (Fig. 3D). The fundamental of herd or population immunity stipulates indirect protection given by the presence and proximity of immune individuals. This forms a self-limiting mechanism within the system dynamics of infectious disease and limits the number of total infections and new infections. Notably, the transmission dynamics reduced tremendously after  $t=20$  with the proportion of total infected individuals reach 6 to 7% and the proportion of total recovered individuals reach 2 to 3% in the population in both new models (Fig. 3E and 3F). The result implies that the protective effect of herd immunity could have been formed even at the early stage of an epidemic.



**Figure 3: The infection dynamics of both conventional and new SIR models with different risk frameworks of contact infection.**

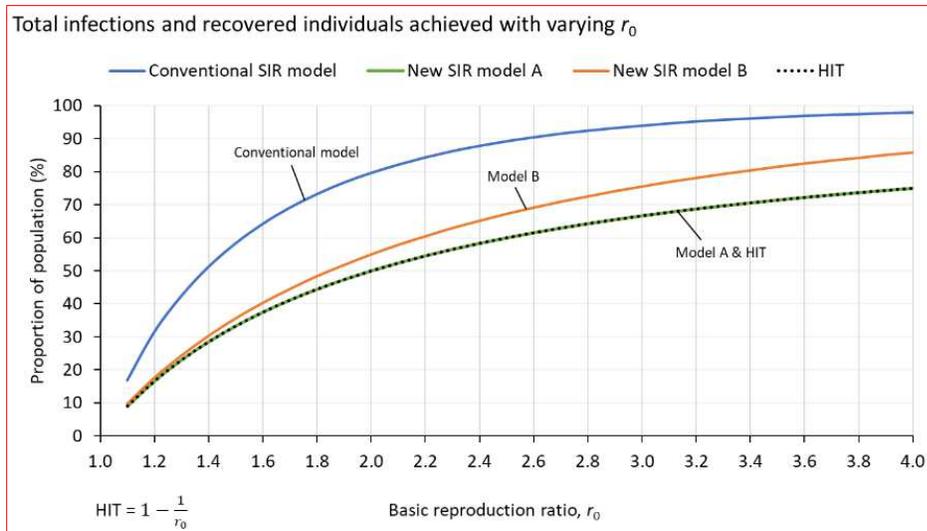
Part A, B and C illustrate the infection dynamics simulated by the conventional SIR model, new SIR model A and B. The HIT is marked by the horizontal dotted black line in part A, B and C. Part D presents new infections generated by the conventional SIR model, and new SIR models A and B. Part E presents class differences between the conventional SIR model and the new SIR model A. Part F presents class differences between conventional SIR model and new SIR model B. The time point where herd immunity starts to take effect is marked by the vertical dotted black line in parts E and F.

The transmission dynamics of infectious disease could be self-limiting due to the reduced risk of contact infection as shown in models A and B. This explains why vaccinating a population to the level given by the HIT could help in control and eradicating an infectious disease. As model A assumes a reduction of risk of contact infection right after susceptible individuals are infected, hence, generating the least total and new infections in simulation. However, model A did not conceptualize the fundamental of herd immunity through infection correctly as the risk of contact infection should be reduced after recovery, not immediately right after infection. By incorporating the inverse of proportion of recovered individuals into the force of infection of SIR model, model B conceptualized the fundamental of herd immunity through infection correctly. As the recovery process and gaining of immunity through infection take time, we should expect more infections than the calculated HIT based on the  $r_0$  value (Fig. 4).



**Figure 4:** Comparison of proportions of classes achieved in the conventional SIR model and new SIR models. The HIT is marked by the dotted black line.

Sensitivity analyses were conducted for all three models to estimate the threshold of infectious disease waning across a range of  $r_0$  values from 1.1 to 4.0 (Fig. 5). The threshold of infectious disease waning through infection-induced herd immunity in the conventional SIR model was consistently way above the calculated HIT based on its  $r_0$ , ranging from 16.9% to 98.0% for  $r_0$  values from 1.1 to 4.0. The threshold of infectious disease waning through infection for model A was given by its calculated HIT based on the  $r_0$  values, ranging from 9.1% to 75.0%. Finally, the threshold of infectious disease waning through infection for model B increased as  $r_0$  became larger and above the calculated HIT, ranging from 9.7% to 85.9%.



**Figure 5:** Total infections and recovered individuals projected by the conventional SIR model, new SIR model A and B.

Both new and conventional SIR models differed in many aspects due to different risk determinants of contact infection. Table 1 summarizes similarities and differences between the conventional SIR model and new SIR models.

**Table 1:** Similarities and differences between the conventional SIR model and new SIR models.

Similarities		
<ul style="list-style-type: none"> <li>• Compartmental structure.</li> <li>• Homogenous population.</li> <li>• Complete immunity is conferred by a single infection.</li> <li>• Transition rate proportional to the number of individuals in the respective classes, constant coefficients of transmission and recovery, and risk of contact infection.</li> <li>• The <math>r_0</math> can be estimated at the initial stage of infection.</li> </ul>		
Differences		
Conventional SIR model	New SIR model A	New SIR model B
<ul style="list-style-type: none"> <li>• Partially mixed population</li> <li>• The risk determinant of contact infection is the proportion of infectious individuals.</li> <li>• The risk of contact infection changes according to proportion of infectious individuals.</li> <li>• Epidemic vanishes as a result of the exhausted number of susceptible.</li> </ul>	<ul style="list-style-type: none"> <li>• Randomly mixed population</li> <li>• The risk determinant of contact infection are proportions of infectious and susceptible individuals.</li> <li>• The risk of contact infection declined as soon as susceptible individuals are infected.</li> <li>• Epidemic vanishes as a result of the exhausted number of susceptible and declined risk of contact infection right after infection</li> </ul>	<ul style="list-style-type: none"> <li>• Randomly mixed population</li> <li>• The risk determinant of contact infection are proportion of infectious individuals and inverse of proportion of recovered individuals.</li> <li>• The risk of contact infection declined after infectious individuals have recovered from infection.</li> <li>• Epidemic vanishes as a result of the exhausted number of susceptible and declined risk of contact infection after recovery</li> </ul>

<ul style="list-style-type: none"> <li>• Epidemic wanes at the threshold way above the HIT calculated based on <math>r_0</math>.</li> <li>• <math>F = \frac{\beta I(t)}{N}</math></li> <li>• More total and new infections, and less susceptible at the end of epidemic.</li> <li>• Herd immunity is not attained in modelling output.</li> <li>• Not in line with the fundamental of herd immunity.</li> </ul>	<ul style="list-style-type: none"> <li>• Epidemic wanes precisely at the HIT calculated based on its <math>r_0</math>.</li> <li>• <math>F_A = \frac{\beta I(t)S(t)}{N^2}</math></li> <li>• Less total and new infections, and more susceptible at the end of epidemic.</li> <li>• Herd immunity is attained in modelling output.</li> <li>• In line with the fundamental of vaccine-induced herd or population immunity.</li> </ul>	<ul style="list-style-type: none"> <li>• Epidemic wanes at the threshold close to the HIT calculated based on its <math>r_0</math> when the <math>r_0</math> is small, and deviate from the HIT when the <math>r_0</math> becomes bigger</li> <li>• <math>F_B = \frac{\beta I(t)[N-R(t)]}{N^2}</math></li> <li>• Less total and new infections, and more susceptible at the end of epidemic, depending on the transmissibility of infectious disease.</li> <li>• Herd immunity is attained in modelling output.</li> <li>• In line with the fundamental of infection-induced herd or population immunity.</li> </ul>
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## Discussion

Although the conventional SIR model assumes immunity is conferred by a single infection, the model fails to realize the impact of herd immunity in its modelling outputs due to violation of the fundamental of the herd or population immunity. This violation has resulted in more total infections and new infections in projection. The over-projected transmission dynamics rendered the conventional SIR model inaccurate in modelling any infectious diseases in a randomly mixed population, especially toward the end of the epidemic. Our simulation confirmed that the conventional SIR model would overestimate the total infections by 10 to 30% above the HIT calculated based on its  $r_0$  values.

The sensitivity analysis suggests that model A provides a better framework for modelling vaccine-induced herd or population immunity for two reasons. First, vaccination often occurs at a rate much faster than the spread of infectious disease in a population and immunity can be acquired almost immediately right after receiving a vaccine. The sensitivity analysis also suggests that model B provides a better framework for modelling infection-induced herd or population immunity, with a higher threshold for control or eradication of infectious disease through an infection-induced herd or population immunity, depending on the  $r_0$  values.

Our simulation demonstrates that the HIT calculated based on the  $r_0$  value can only be followed if one assumes that the risk of contact infection among susceptible individuals reduces immediately as a result of infection, but not recovery. This implies that the simple threshold theorem proposed by Dietz in 1975 based on the conventional modelling framework could have underestimated the actual threshold required for control and eradication of infectious disease through infection-induced herd immunity in a randomly mixed population<sup>18</sup>.

Another noteworthy finding is that herd immunity may take effect even at the early stage of epidemic even with a small proportion of immune individuals. Both model A and model B may explain the sharp fall of COVID-19 cases in countries like the United Kingdom, Indonesia and Germany, shortly after the rollout of vaccine, and countries like India even before the rollout

of vaccine<sup>19</sup>. With the sharp fall of COVID-19 cases after the rollout of vaccines, United Kingdom has initiated the unlocking process guided by the COVID-19 wave<sup>20</sup>. While many countries were still struggling with the COVID-19 unrest, the sharp fall of COVID-19 cases in India since September 2020 without vaccines had caught global attention. Many researchers attributed the decline of COVID-19 cases without vaccination in India to its population herd immunity and younger demographic. A national serological survey conducted by the Indian Council of Medical Research (ICMR) revealed that up to 21% or 290 million of the adult population in India have been exposed to the COVID-19 virus and developed immunity against the virus<sup>21</sup>.

As the world is still under attack by the COVID-19 pandemic, the use of the right deterministic framework is critical in providing the right projection to support population control strategies, pandemic preparedness and decision-making. In the COVID-19 pandemic, a modelling framework that features a reduced risk of contact infection consequent to the rising prevalence of immune individuals, either through infection or vaccination is preferred. Having said that, the attainment of herd immunity phenomenon with the new modelling framework does not imply that herd immunity can be achieved in real-life without considering other critical factors, such as population heterogeneity, vaccination rate, vaccine uptake by the community, and the emergence of new viral strains.

As of this writing, more than 100 million individuals have been infected by the novel coronavirus with a death toll of approximately 2.5 million<sup>19</sup>. Due to exhausted healthcare resources and increasing asymptomatic infections, many infected individuals are not captured in the COVID-19 statistics. At the same time, many countries have started mass vaccination programs with the hope to end the COVID-19 nightmare through vaccine-induced population or herd immunity, with target HIT calculated based on the estimated  $r_0$  of COVID-19. Hence, applying the right vaccination threshold and deterministic modelling framework in studying COVID-19 and vaccine-induced herd immunity becomes exceptionally relevant and critical now.

## Conclusion

The study offers two new frameworks for the SIR model and its variant models in simulating any infectious diseases in a randomly mixed population. Model A which incorporates the proportion of susceptible individuals in its force of infection is more credible for simulating vaccine-induced herd or population immunity, while model B which incorporates the inverse of proportion of recovered individuals in its force of infection is more credible for simulating infection-induced herd or population immunity. Infection-induced herd or population immunity requires a threshold above the level derived based on the  $r_0$  of an infectious disease.

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### **Competing interests**

The authors declare no competing interests.

### Contributions

K.B.L and K.M.P conceived and planned the study.

K.B.L contributed to the design of the compartmental model and simulation.

K.M.P, H.S and N.H.A supervised the implementation of the study.

Data analysis and graphics were done by K.B.L.

All authors contributed to the interpretation of the findings and approved the final version for publication.

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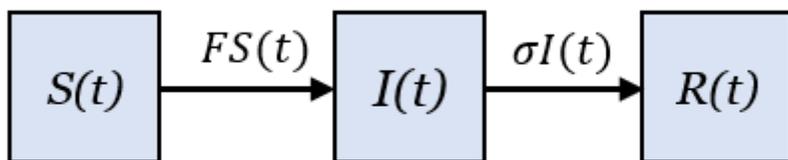
Correspondence to Kian Boon Law

### **Ethics requirement**

The study was registered with National Medical Research Register. No ethics approval was required.

# Figures

## The conventional SIR model

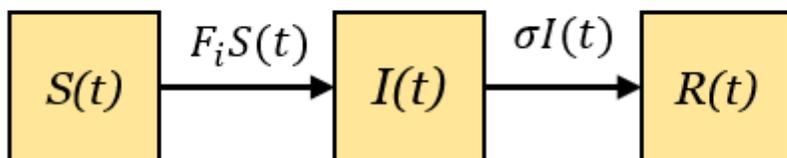


Force of infection,  $F = \beta I(t)/N$

Figure 1

The conventional SIR model developed by Kermack & McKendrick (1927). Individuals move from class  $S(t)$  to  $I(t)$  at a rate proportional to  $\beta$ ,  $(I(t))/N$ , and  $S(t)$ . The proportion of infectious individuals signifies the risk of contact infection among susceptible individuals.

## The new SIR model



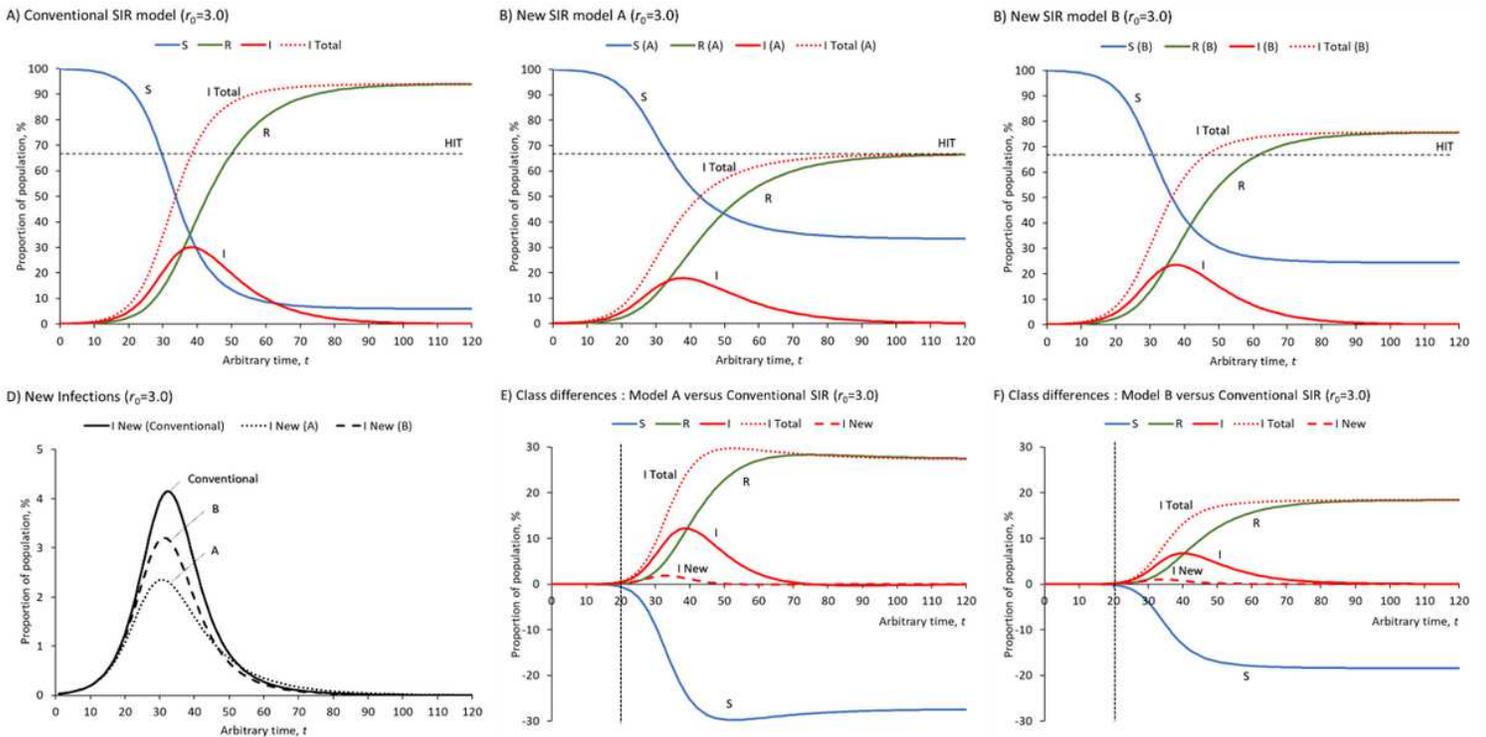
Force of infection,  $F_i, i = A, B$

Model A:  $F_A = \beta I(t)S(t)/N^2$

Model B:  $F_B = \beta I(t)[N - R(t)]/N^2$

Figure 2

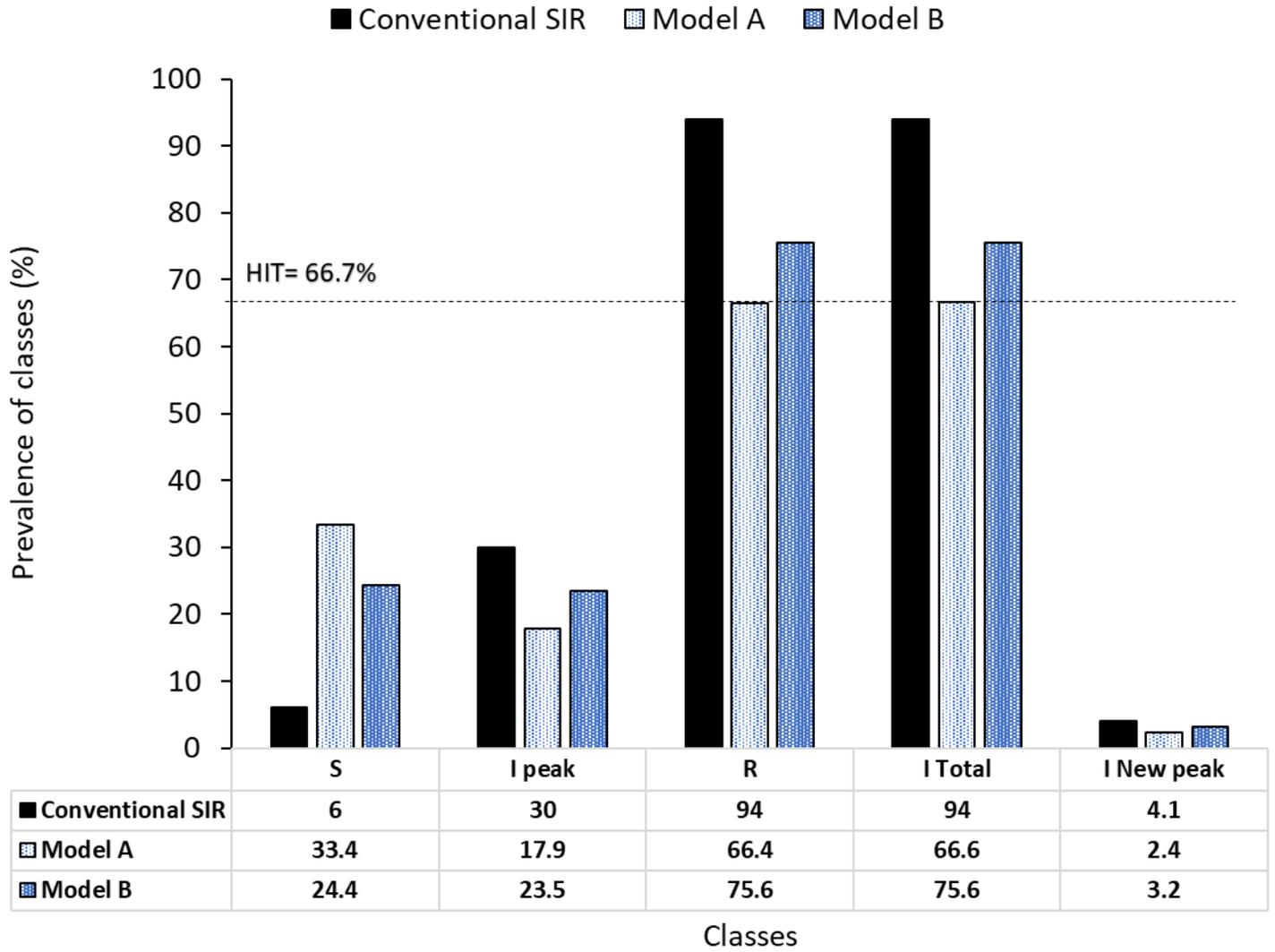
The compartmental structure of new SIR models and force of infections in a randomly mixed population



**Figure 3**

The infection dynamics of both conventional and new SIR models with different risk frameworks of contact infection. Part A, B and C illustrate the infection dynamics simulated by the conventional SIR model, new SIR model A and B. The HIT is marked by horizontal dotted black line in part A, B and C. Part D presents new infections generated by the conventional SIR model, and new SIR model A and B. Part E presents class differences between conventional SIR model and new SIR model A. Part F presents class differences between conventional SIR model and new SIR model B. The time point where herd immunity starts to take effect is marked by the vertical dotted black line in part E and F.

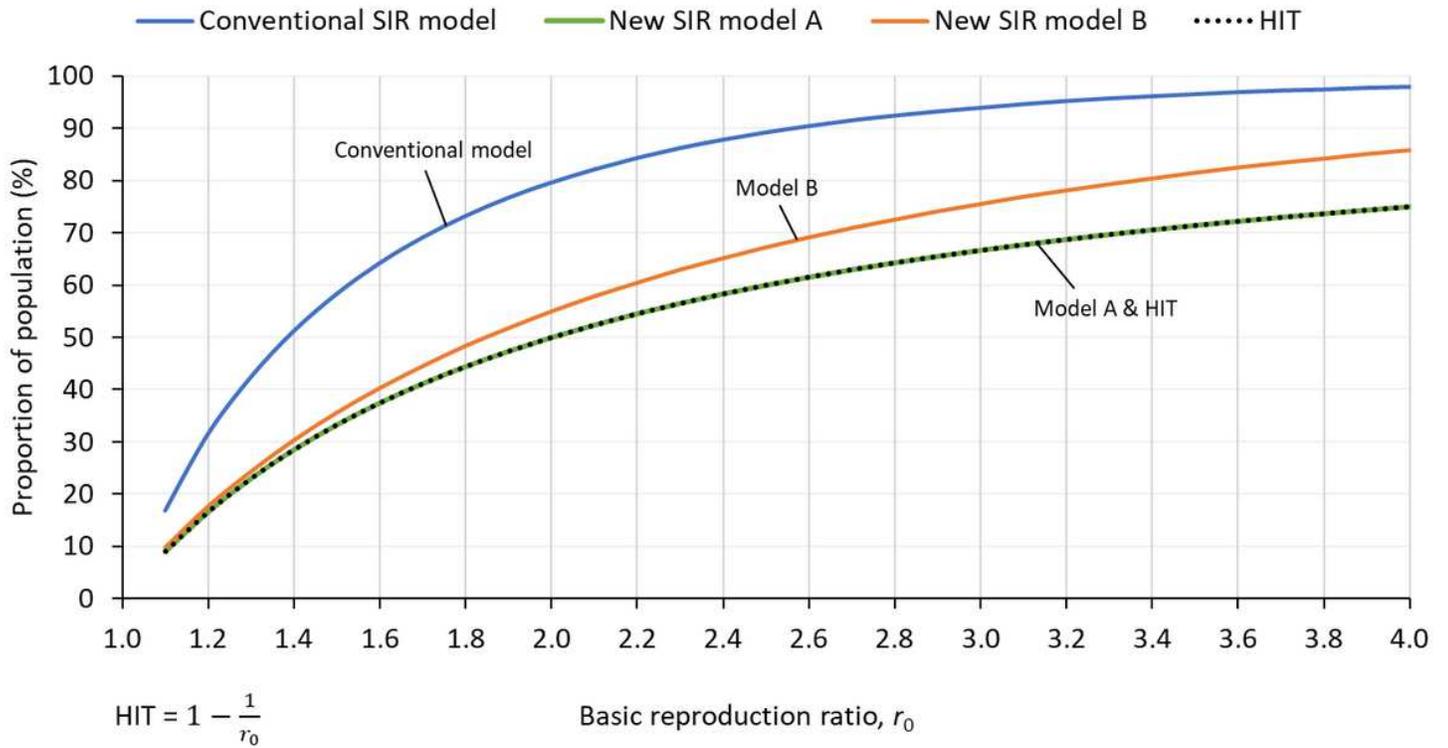
# Prevalence of classes



**Figure 4**

Comparison of proportions of classes achieved in the conventional SIR model and new SIR models. The HIT is marked by the dotted black line.

## Total infections and recovered individuals achieved with varying $r_0$



**Figure 5**

Total infections and recovered individuals simulated by the conventional SIR model, new SIR model A and B.