A Vaccination-Socialization SIR Model for Covid-19: Lessons Learned from the Pandemic

ABSTRACT

Background: Modified SIR models have been effective in simulating the spread of Covid-19 and predicting various phenomena associated with the pandemic.
Aims: This paper presents a vaccination-socialization model to determine the relative effects of vaccination and increased socialization among the vaccinated. The objective is to determine whether the disease is spread more so by the presence of the unvaccinated or by increased social activity among the vaccinated.
Methodology: To accomplish this, the SIR model is modified to include 9 compartments and 3 sub-populations. The resulting system of differential equations is solved inMatlab's programming language using the 4^{th} order Runge-Kutta method.
Results: The results show that the concept of a safe-zone in which vaccinated persons can increase their social activity only applies if the vaccine effectiveness is very high.

Keywords: Covid-19, SIR model, compartmental model, vaccination, herd immunity

1. INTRODUCTION

The susceptible-infected-recovered (SIR) model has been used extensively to model disease propagation. SIR is a compartmental model that employs a system of differential equations to simulate the transfer of persons between compartments. Its applications have included the Hong Kong Flu, measles, mumps and rubella, varicella, cholera and HIV [1]. More recently, the SIR model has been used to model the Covid-19 pandemic. Quite a few papers have been published using the basic SIR model to estimate disease parameters and to predict the spread of the virus in various geographic regions [1].

However, there are many aspects of Covid-19 that cannot be captured using the basic SIR model. In order to obtain a better understanding of transmissible diseases, the model must be modified by adding compartments, altering the interaction between compartments, and adjusting the system of governing differential equations. One such modification is the addition of a compartment to account for deaths [2]. Some advanced models are adaptive in that they consider variable rates of transmission and death, so as to model successive waves of the pandemic [3,4]. Cooper et al [5] added a variable to adjust the susceptible population so as to account for surges. Other models considered those who were asymptomatic [6] together with the effect of under-reporting [7]. As the pandemic progressed, social distancing and other lockdown measures were implemented, which affected the spread of the virus. The SIR model has been adapted to include the effect of quarantine [8,9], social distancing [10], and also to study the efficiency of lockdown measures [11,12]. Vaccination models have also been implemented to study the effectiveness of vaccination campaigns [13].

This paper presents a modified SIR model to study the relative effects of vaccination and social activity. It specifically aims to investigate which are the dominant factors in the spread of the disease – the presence of the unvaccinated, increased social activity among the vaccinated, and/or segregation between vaccinated and unvaccinated. To put it loosely, was the spread of Covid-19 mainly the result of the unvaccinated, or carelessness on the part of the vaccinated? To avoid fueling conspiracy theories, it is necessary to rephrase that question in a more mathematical way. For a given vaccine effectiveness and coverage rate, by how much must vaccinated individuals increase their social activity to result in a spread of the virus comparable to that in which no one was vaccinated? How much additional socialization is permissible to still maintain herd immunity? These questions will be answered with the aid of a mathematical model.

2. MODEL DEVELOPMENT

Typically the SIR model consists of 3 compartments that interact with one another – susceptible (S), infected (I) and recovered (R). The model presented in this paper aims to investigate the effect of partial segregation between the vaccinated and unvaccinated groups. It proposes 3 subpopulations / groups (1 = vaccinated; 2 and 3 = unvaccinated), each group with its own S, I and R. Thus there are 9 compartments, as shown in Figure 1.

Group 1 members interact with groups 1 and 2, but not with group 3. Consequently, group 3 members interact with groups 2 and 3, but not with group 1. This way there is partial segregation between the vaccinated and unvaccinated. For a fully mixed population, group 3 is empty, and for a totally segregated population, group 2 is empty.

The mixing model presented in this paper is derived differently from that of Fisman et al [14], who used an assortativity factor to determine the social interaction between vaccinated and unvaccinated groups. The present model divides the unvaccinated into two separate groups – one that mixes with the vaccinated and one that does not. Another advantage of the method used in this paper is that it allows for increased social activity among the vaccinated group separately from the other groups.

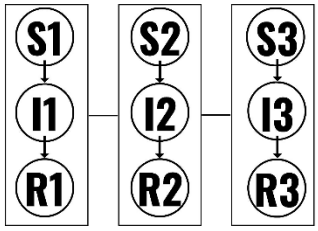

FIGURE 1. Model Schematic – flow of persons between compartments and interaction between the sub-populations

2.1 Assumptions

The present model makes the following assumptions:

1) The total population is fixed for the period of study i.e. the model ignores births, deaths, migration. Deaths are not considered because they are not relevant to this study. The emphasis is on the spread of the disease, not the severity of the infections. The model does not address whether unvaccinated individuals suffer more severe symptoms.

2) Those recovered cannot become reinfected – at least for the duration of the analysis.

3) The population is assumed to be partially vaccinated prior to the inception of the disease, but no further vaccination occurs afterward. The reason for this approach is to control as many variables as possible so as to isolate cause and effect.

4) The model is not interested in the type of vaccine or a comparison of vaccines. It simply characterizes vaccines by an effectiveness value.

5) Vaccine effectiveness is interpreted to mean a reduced probability of a vaccinated person contracting the disease. This is in slight contrast to mixing model reported by Fisman et al [14], who modeled vaccination by creating 2 additional compartments for those who are vaccinated and 100% immune, and those who are vaccinated and 100% susceptible. In this paper, everyone who is vaccinated has the same probability of infection. In the event of infection, vaccination status does not alter the probability of an individual transmitting the disease.

6) Covid-19 variants are not relevant to this study. The model parameters can simply be adjusted accordingly. The objective is to determine the relative effects of vaccination and social behavior.

2.1 Differential Equations

The governing differential equations are derived as follows. V is the vaccination coverage rate or fraction of the population that belongs to group 1. V_i is defined as the fraction of the total population that belongs to group i. A mixing variable, m is introduced to quantify the level of social interaction between the vaccinated and unvaccinated. m is essentially the fraction of unvaccinated persons that mixes with vaccinated persons. m = 0 means complete segregation between vaccinated and

unvaccinated, while m = 1 means complete and random mixing. This variable is important because it can be used to measure the effect of vaccinated persons reducing contact with unvaccinated persons.

$$V = V_1 \tag{1}$$

$$V_2 = m\ (1-V) \tag{2}$$

$$V_3 = (1-m)(1-V) \tag{3}$$

Suppose an average member of group i has k total contacts per day. If group i randomly mixes with all other groups, then we can expect this person to have kV_j total contacts with persons from group j, of which I_j/V_j is the fraction that is infected. Thus the average group i person has kI_j daily interactions with infected person from group j. If pi is the probability of such an interaction resulting in an infection, and there are Si total susceptible members in group i, then we can expect $p_iS_ikI_j$ total infections in group i per day from interactions with group j. We write β instead of p_ik.

To accomplish the objectives in this paper, there are some additional modifications that are required. Group 1-1 interactions are modified by a factor of 1+α to allow for additional socialization among the vaccinated. All i=1 contacts are further modified by 1-ε because vaccination decreases the probability of group 1 members becoming infected. We also need to ensure that groups 1 and 3 do not mix without reducing their total social contacts.

For the specific case of i = 1 (the vaccinated group), normally there would have been kV_3 interactions with group 3, however the present model requires zero interaction between groups 1 and 3. But if vaccinated persons simply reduce their contacts with unvaccinated persons, then this would result in segregation as well as reduced socialization, either of which could affect the results, thereby making it impossible to isolate the effective cause. We must achieve segregation without reduced socialization in order to properly control the variable. Thus in this model, if vaccinated persons cut contacts with unvaccinated persons, then these contacts must be replaced by increased contacts with other vaccinated persons. This way, segregation is achieved without decreasing the level of socialization. Thus a group 1 member has $k(V_1+V_3)$ interactions with other group 1 members. Group 3 members similarly have no interactions with group 1 members, so these kV1 interactions must be redistributed among group 2 and 3 in the ratio m:1-m.

Initially, a fractional calculus method similar to Jan et al [15-20] was considered. However, a simpler state-space method was eventually selected.The final system of differential equations, shown below, is solved in the Matlab programming language using the Runge-Kutta 4th order method.

$$\frac{d}{dt}\begin{bmatrix} S_1 \\ S_2 \\ S_3 \end{bmatrix} = -\beta \begin{bmatrix} (1-\varepsilon)S_1\left(m+\alpha+\frac{1-m}{V}\right) & (1-\varepsilon)S_1 & 0 \\ S_2 & S_2 & S_2 \\ 0 & \frac{1}{1-V}S_3 & \frac{1}{1-V}S_3 \end{bmatrix} \begin{bmatrix} I_1 \\ I_2 \\ I_3 \end{bmatrix} \tag{4}$$

$$\frac{d}{dt}\begin{bmatrix} R_1 \\ R_2 \\ R_3 \end{bmatrix} = \gamma \begin{bmatrix} I_1 \\ I_2 \\ I_3 \end{bmatrix} \tag{5}$$

$$\frac{d}{dt}\begin{bmatrix} I_1 \\ I_2 \\ I_3 \end{bmatrix} = -\frac{d}{dt}\begin{bmatrix} S_1 \\ S_2 \\ S_3 \end{bmatrix} - \frac{d}{dt}\begin{bmatrix} R_1 \\ R_2 \\ R_3 \end{bmatrix} \tag{6}$$

3. RESULTS AND DISCUSSION

3.1 Numerical Values

This paper uses a base R-naught value of 2.0 [21] and an average recovery time of 7 days [22]. This translates to $\beta = 2/7$ and $\gamma = 1/7$. Note that 7 days need not correspond to the actual duration of the disease, but rather the period of time during which a person is likely to spread the disease to others.

Further, according to the equation 4, the number of infections is supposed to peak when S drops to 1/R-naught (50%). However, the curves for most geographic areas peaked long before that. The most likely explanation for this is that the public began to change its social behavior once they realized the infections were increasing. To model that change of behavior, the value of β is changed from 0.286 to 0.118 around day 30.

3.2Validation

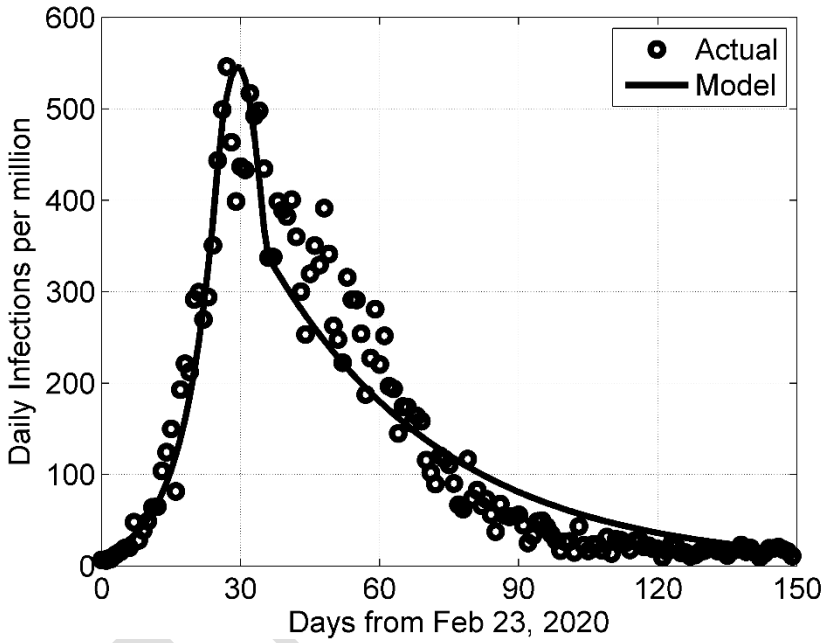

FIGURE 2. Model Validation – comparison between simulated results and the actual number of cases during Italy's first wave (ε=0, α=0, V=0, m=1)

The model is validated by comparing its predictions with the daily infections during Italy's first wave in 2020. This data is chosen because it represents a society that was not expecting an outbreak and thus conducting normal social activity during a pre-vaccination period. The main drawback is that testing was not as widespread back then, thus the actual number of cases was most likely higher than those reported – 4 to 20 times higher according to Noh and Danuser [23]. For the validation, this paper multiplies the daily infections at Worldometer [24] by a factor of 5. Figure 2 shows the model validation, comparing model predictions with data from Italy's first wave. It shows a very good fit during the increasing phase, and a good fit during the declining phase.

Having been calibrated and validated, the model will now be used to make further predictions. For the sections to follow, the nominal values are for a fully mixed population (no segregation) that is exhibiting normal social behavior with a 50% effective vaccine and a vaccination coverage rate of 50% ($\alpha = 0$, $m = 1$, $\varepsilon = 0.5$, $V = 0.5$). Unless otherwise stated, those are the values being used.

3.3Effect of Varying Vaccination Parameters (ε and V)

Figure 3 shows the daily infections of both the vaccinated and unvaccinated groups for different values of V and ε for a fully mixed population ($m = 1$). It should be noted that in Figure 3, the daily infections are normalized for each sub-population, e.g. 5 daily infections per million unvaccinated persons, etc. For each scenario, the normalized number of

infections was lower for the vaccinated group than the unvaccinated, as expected. For the nominal case (ε = 0.5, V = 0.5), the infections followed the typical bell curve for both the vaccinated and unvaccinated groups. For a population that is highly vaccinated with a very effective vaccine (ε = 0.8, V = 0.8), the daily infections began to decrease immediately indicating that herd immunity was achieved. For this scenario, the presence of the vaccinated group reduces the spread of the virus even among the unvaccinated. This is because for this scenario, each person interacts with 4 times as many vaccinated persons as unvaccinated persons. This increases the rate of spread among the vaccinated, but decreases the rate of spread among the unvaccinated at a faster rate.

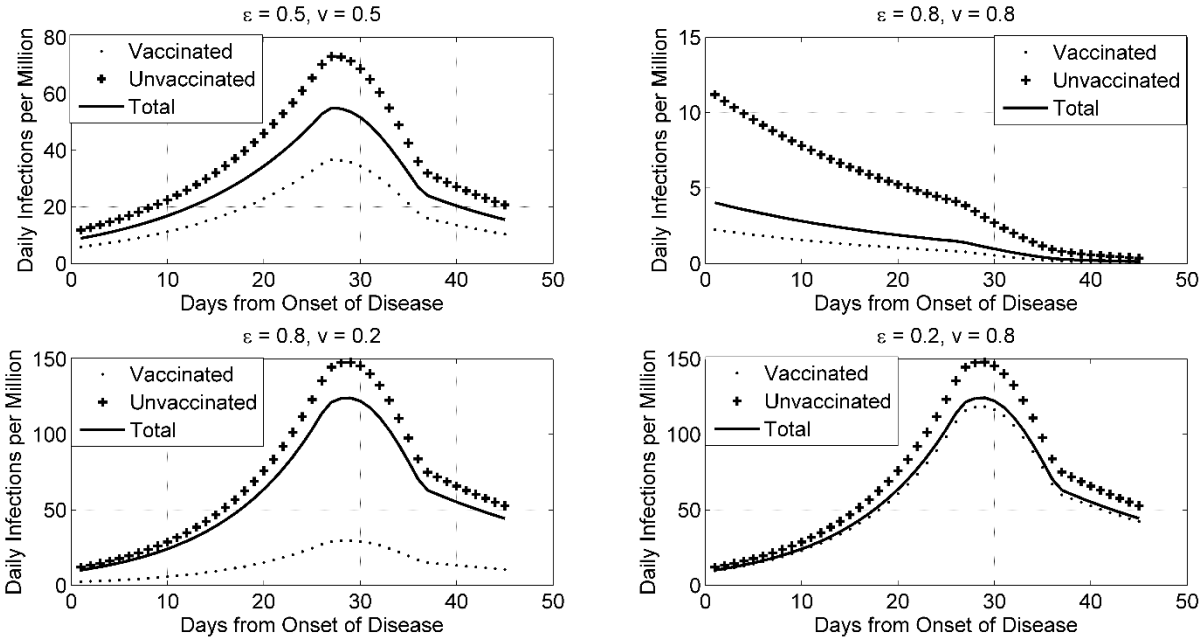

FIGURE 3. Effect of Vaccine Parameters on Each Sub-Population – how vaccine effectiveness and vaccination coverage affect the spread of the virus (α=0, m=1)

We can then compare the sensitivity of the rate of infection to ε and V. Figure 3 also shows the results for ε = 0.8, V = 0.2 and ε = 0.2, V = 0.8. Remarkably, the graphs for the total and the unvaccinated are almost identical in both cases. The significant difference is that the vaccinated infections are much less when the vaccine effectiveness is high. This is true even if the vaccination coverage is low. In the case of ε = 0.8, V = 0.2, each person interacts with vaccinated and unvaccinated persons in a 1:4 ratio, which explains why the vaccinated infections increase. However, the peak vaccinated infections are approximately 5 times less than the peak unvaccinated infections.

Figure 4 shows the total infections (vaccinated + unvaccinated for a period of one year) vs vaccination coverage for various values of vaccine effectiveness. As expected, all graphs show an inverse exponential decrease, with the rate of decay increasing with ε. If the vaccine effectiveness is 50%, then 43% of the population needs to be vaccinated to decrease the total infections by 90% (compared to an unvaccinated population). Similarly 99% needs to be vaccinated to decrease infections by 99%. If the vaccine is more effective (80%), then the corresponding vaccination percentages are 27% and 62% to achieve a 90% and 99% reduction respectively. If the actual vaccine effectiveness is only 20%, then it is impossible to achieve a 90% reduction in infections even with a fully vaccinated population. Thus it is possible to control the spread of the virus with a vaccine that is only 50% effective, however this is not the case if the effectiveness is less than 20%.

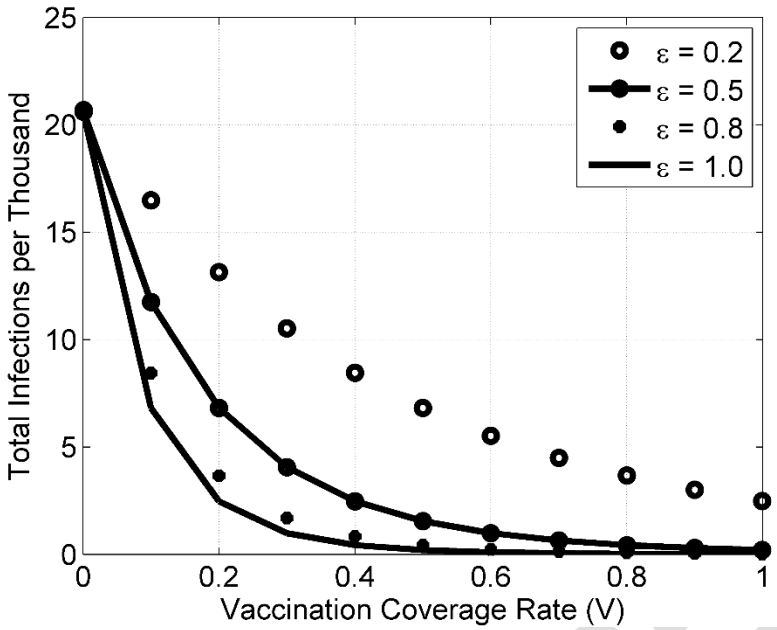

FIGURE 4. Effect of Vaccination Coverage Rate – how vaccination coverage affect the spread of the virus for various values of vaccine effectiveness (α=0, m=1)

Following the theoretical approach of Kermack and McKendrick [25], the virus is considered to be contained if the rate of recovery exceeds the rate of infection resulting in a negative net rate of infections from t = 0. From equations 4-6, it can be shown that the required condition for herd immunity is,

$$1-\varepsilon V + \left(1-\varepsilon\right)\alpha V^2 < \frac{\gamma}{\beta} = \frac{1}{R_0} \qquad (7)$$

It is noteworthy that this expression is independent of the mixing fraction, m. For the case where α = 0, we obtain the required vaccination coverage rate for herd immunity.

$$V_c = \frac{1}{\varepsilon}\left(1-\frac{1}{R_0}\right) \qquad (8)$$

For R0 = 2, the theoretically required coverage rates for herd immunity using vaccines that are 50% and 80% effective, are respectively 100% and 62.5%. This matches very closely with the corresponding values predicted by the present model – 99% and 62% respectively.

3.4Effect of Varying the Mixing Fraction

In this section, the value of the mixing variable, m is altered to investigate its effect on the rate of infection. It is important to recall that decreasing m means that vaccinated persons have decreased contact with unvaccinated persons, but at the same time have replaced those contacts with other vaccinated persons, as discussed earlier.

Figure 5 shows the effect of varying the mix variable, m. In all of the cases shown, the total infections increase as m→0. For most cases, the total infections reach a minimum when m → 1. The exception is when a population is highly vaccinated with a low effectiveness vaccine (ε = 0.2, V = 0.8), in which case there is a slight minimum around m = 0.56, however, this minimum is only 5% lower than the value for m = 1. Generally, we see that total infections are better controlled when the population is mixed rather than segregated.

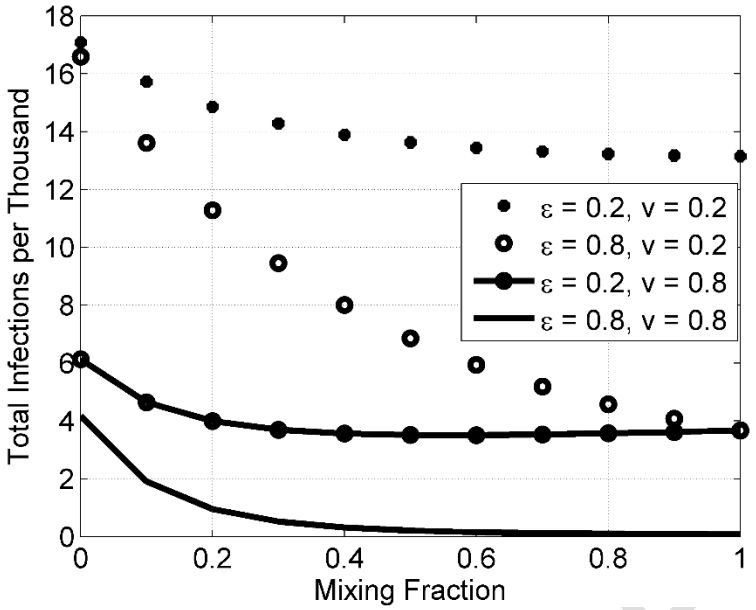

FIGURE 5. Effect of Mixing Fraction – how much segregation between the vaccinated and unvaccinated affects the spread of the virus in the overall population (α=0)

This result may seem counter-intuitive, but there is a logical explanation for it. For a population where the vaccinated and unvaccinated are completely segregated (m = 0), the virus will spread in each sub-population independently. It will be quickly contained within the vaccinated group, while the spread in the unvaccinated group will be the same as an unvaccinated population (Figure 1). Essentially the total cases will predominantly consist of the unvaccinated. Thus segregation works out better for the vaccinated group, however the unvaccinated infections will increase at a significantly greater rate, and thus increase indirect cross-contamination of vaccinated persons. It is better for the overall population if there is no segregation between the vaccinated and unvaccinated. This is because segregation hurts the unvaccinated more than it helps the vaccinated.

We can also use Figure 5 to compare the low effectiveness / high coverage (ε = 0.2, V = 0.8) and the high effectiveness / low coverage (ε = 0.8, V = 0.2) curves. For m = 1, there is negligible difference between them. However, as m decreases (increased segregation between vaccinated and unvaccinated), the low effectiveness / high coverage option results in lower total infections. Thus if there is to be some level of social segregation, it is better to have a high coverage of a low effectiveness vaccine, than a low coverage of a high effectiveness vaccine. Having said that, if the coverage rate is high, then segregation becomes meaningless, since only a minority would be unvaccinated.

3.5Effect of Increased Socialization Among the Vaccinated

Table 1 shows the effect of increasing the social activity among the vaccinated by 50% for various scenarios. In every case, increasing α has a direct effect of increasing the number of vaccinated infections, as well as an indirect effect of increasing unvaccinated infections. Note that both groups are indirectly affected by cross contacts between the groups, while the vaccinated group is further affected by increased social activity. This would explain why in each case, increasing α results in vaccinated infections increasing at a greater rate than unvaccinated infections.

The effect of increasing α depends on how effective the vaccine is. If the effectiveness is low (ε = 0.2), even with a highly vaccinated population, increasing social activity among the vaccinated proves to be catastrophic. For moderate effectiveness (ε = 0.5), increasing social activity by 50% produces a two-fold to three-fold increase in total infections, even if a high fraction of the population is vaccinated. However, for very effective vaccines (ε = 0.8), there is only a small increase in infections when α increases. This is the case even if the population is not highly vaccinated. Thus the concept of a “safe zone” for the vaccinated only applies if the vaccine has a high effectiveness. This is true even if the coverage rate is low.

TABLE 1. Effect of Increased Socialization on the Spread of the Virus

ε	V	α	Vaccinated infections per thousand	Unvaccinated infections per thousand	Total infections per thousand
0.2	0.8	0	3.50	4.37	3.67
0.2	0.8	0.5	68.2	60.8	66.7
0.5	0.5	0	1.03	2.06	1.55
0.5	0.5	0.5	1.83	3.07	2.45
0.5	0.8	0	0.360	0.720	0.432
0.5	0.8	0.5	1.40	2.045	1.53
0.8	0.2	0	0.875	4.37	3.67
0.8	0.2	0.5	0.908	4.42	3.72
0.8	0.8	0	0.0431	0.216	0.0776
0.8	0.8	0.5	0.0688	0.265	0.108

To determine the maximum amount of additional socialization permissible among the vaccinated, the following theoretical approach can be adopted. The objective is to determine the amount of additional socialization among the vaccinated in order to produce the same results as if no one was vaccinated. Substituting V = 0 in equation 7, the left hand side simply becomes 1. For a non-zero vaccine effectiveness and coverage rate, the critical amount of additional socialization that produces the same left hand side is,

$$\alpha_c = \frac{\varepsilon}{1-\varepsilon}\ \frac{1}{V} \tag{9}$$

For example, if the vaccine is 50% effective and half the population is vaccinated, and if each vaccinated person doubles their social interactions with other vaccinated persons, then this would exactly negate the benefits of vaccination. If the vaccine effectiveness is high, this would give vaccinated persons a high amount of leeway to increase their socialization. We can also see that increasing the coverage rate does not have the same effect. This may seem counter-intuitive, but this is because for low values of V, both the vaccinated and unvaccinated groups would suffer from a high degree of cross-contamination. Thus, vaccine effectiveness is a much more critical factor than vaccination coverage in determining how much additional socialization is permissible. The concept of a safe-zone only applies if the vaccine effectiveness is very high.

We can also determine the critical amount of additional socialization required to maintain herd immunity. Assuming that the coverage rate is above the herd immunity value required by equation 8, the value of α that maintains the inequality of equation 7 is,

$$\alpha < \frac{1/R_0 - (1-\varepsilon V)}{(1-\varepsilon)\ V^2} \tag{10}$$

Consider the example of a population that has a coverage rate of 80% with an 80% effective vaccine. This coverage rate exceeds the minimum required value of 62.5%. The critical socialization values according to equations 9 and 10 are α=5 to produce the same transmission rate as if no one was vaccinated, and α=1.09 to keep the virus contained. Figure 6 illustrates these results. For ε = 0.8 and V = 0.8, the net rate of infections is always negative for α=0 and α=1, but initially positive for α=2.

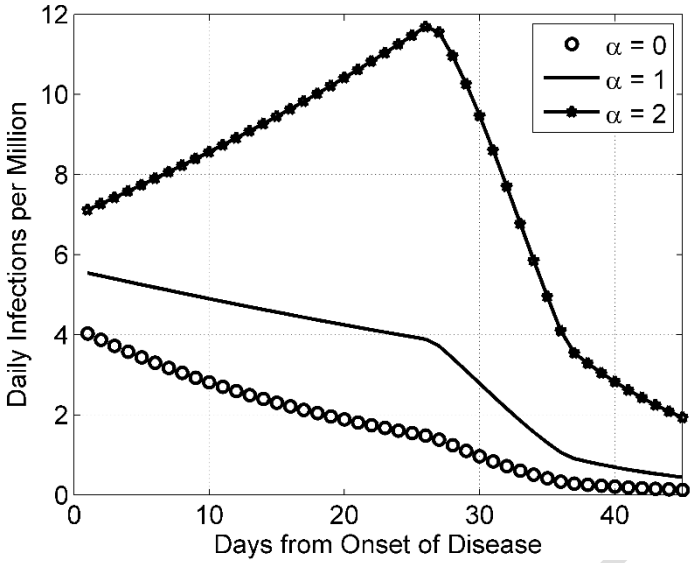

FIGURE 6. Effect of Increased Socialization – the effect of increased socialization on herd immunity (m=1, ε = 0.8, V=0.8)

3.6Effect on a Highly Vaccinated Population

Figure 7 shows the daily infections for various vaccine effectiveness values for a population with a high coverage rate (V = 0.9). A vaccine that is 50% effective is sufficient to contain infections in a 90% vaccinated population. Even a vaccine that is only 10% effective can reduce infections to less than half of those in a totally unvaccinated population – Figure 2 vs Figure 7.

Yet, it is notable that with the omicron surge, populations that were heavily vaccinated reported infections far surpassing those of the first wave. The possible reasons for this are low actual vaccine effectiveness, increased social activity, and/or greater transmissibility of the variant compared to the original strain. These results may suggest that the Covid-19 vaccines were less than 10% effective against the omicron variant. In such a case, increasing social activity among the vaccinated in so-called safe zones even by as little as 11% can produce results comparable to a totally unvaccinated population.

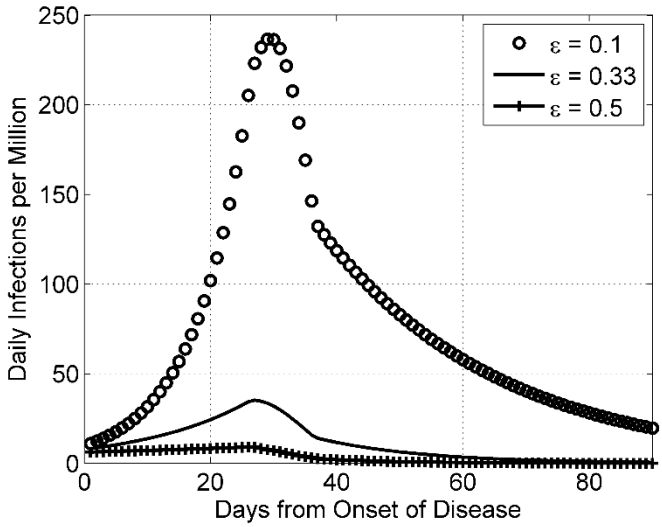

FIGURE 7. Effect of Vaccine Effectiveness on a Highly Vaccinated Population (α=0, V=0.9, m=1)

4. CONCLUSION

The modified vaccination-socialization SIR model presented in this paper was calibrated and validated using the data from Italy's first wave in 2020. The model was then used to determine the effect of varying the vaccine effectiveness, vaccination coverage, the level of mixing between vaccinated and unvaccinated individuals, and the level of socialization among the vaccinated. These are highly controversial issues that have spawned various conspiracy theories. However, this paper sought to remove the bias by phrasing the question in a mathematical way. By how much must vaccinated persons increase their social activity to achieve the same spread of the disease as if no one was vaccinated?

The results show that segregation of vaccinated and unvaccinated persons is always a bad thing for the overall population. Also, increased socialization among the vaccinated was only a good idea if the vaccine effectiveness was very high. For a population that is 80% vaccinated with an 80% effective vaccine, vaccinated persons can increase their social activity by 109% and still keep the virus contained. But if they increase it 5-fold, they would negate the benefits of vaccination.

Vaccine effectiveness was found to be more crucial than vaccination coverage rate. Further, the results generated in this model suggest that the actual effectiveness of the available vaccines might have been less than 10% against the omicron variant. Perhaps these results might inform policy decisions in any future pandemics.

Disclaimer

This paper is an extended version of a preprint document of the same author.

The preprint document is available in this link: https://papers.ssrn.com/sol3/papers.cfm?abstract_id=4545213

[As per journal policy, preprint article can be published as a journal article, provided it is not published in any other journal]

REFERENCES

[1] Padmanabhan R, Abed HS, Meskin N, Khattab T, Shraim M, Al-Hitm MA. A review of mathematical model-based scenario analysis and interventions for COVID-19. Computer Methods and Programs in Biomedicine 2021; 209:106301. https://doi.org/10.1016/j.cmpb.2021.106301

[2] Shams F, Abbas A, Khan W, Khan US, Nawaz R. A death, infection, and recovery (DIR) model to forecast the COVID-19 spread. Computer Methods and Programs in Biomedicine Update 2022;2:100047. https://doi.org/10.1016/j.cmpbup.2021.100047

[3] Muñoz-Fernández GA, Seoane JM, Seoane-Sepúlveda JB. A SIR-type model describing the successive waves of COVID-19. Chaos, Solitons and Fractals 2021;144:110682. https://doi.org/10.1016/j.chaos.2021.110682

[4] Marinov TT, Marinova RS. Inverse problem for adaptive SIR model: Application to COVID-19 in Latin America. Infectious Disease Modelling 2022;7:134-148. https://doi.org/10.1016/j.idm.2021.12.001

[5] Cooper I, Mondal A, Antonopoulos CG. A SIR model assumption for the spread of COVID-19 in different communities. Chaos, Solitons and Fractals 2020;139:110057. https://doi.org/10.1016/j.chaos.2020.110057

[6] Neves AGM, Guerrero G. Predicting the evolution of the COVID-19 epidemic with the A-SIR model: Lombardy, Italy and São Paulo state, Brazil. Physica D 2020;413:132693. https://doi.org/10.1016/j.physd.2020.132693

[7] Deo V, Grover G. A new extension of state-space SIR model to account for Underreporting – An application to the COVID-19 transmission in California and Florida. Results in Physics 2021;24:104182. https://doi.org/10.1016/j.rinp.2021.104182

[8] Zhu WJ, Shen SF. An improved SIR model describing the epidemic dynamics of the COVID-19 in China. Results in Physics 2021;25:104289. https://doi.org/10.1016/j.rinp.2021.104289

[9] Prasad PSH. COVID-19 disease spread modeling by QSIR method: The parameter optimal control approach. Clinical Epidemiology and Global Health 2022;13:100934. https://doi.org/10.1016/j.cegh.2021.100934

[10] Lymperopoulos IN. #stayhome to contain Covid-19: Neuro-SIR – Neurodynamical epidemic modeling of infection patterns in social networks. Expert Systems With Applications 2021;165:113970. https://doi.org/10.1016/j.eswa.2020.113970

[11] Sharov KS. Creating and applying SIR modified compartmental model for calculation of COVID-19 lockdown efficiency. Chaos, Solitons and Fractals 2020;141:110295. https://doi.org/10.1016/j.chaos.2020.110295

[12] Fu Y, Xiang H, Jin H, Wang N. Mathematical Modelling of Lockdown Policy for COVID-19. Procedia Computer Science 2021;187:447–457.

[13] Angeli M, Neofotistos G, Mattheakis M, Kaxiras E. Modeling the effect of the vaccination campaign on the COVID-19 pandemic. Chaos, Solitons and Fractals 2022;154:111621. https://doi.org/10.1016/j.chaos.2021.111621

[14] Fisman DN, Afia Amoako A, Tuite AR. Impact of population mixing between vaccinated and unvaccinated subpopulations on infectious disease dynamics: implications for SARS-CoV-2 transmission. CMAJ 2022;194(16):E573-E580. https://doi.org/10.1101/2021.12.14.21267742

[15] Jan A, Srivastava HM, Khan A, Mohammed PO, Jan R, Hamed YS. In Vivo HIV Dynamics, Modeling the Interaction of HIV and Immune System via Non-Integer Derivatives. Fractal and Fractional. 2023;7(5):361.

[16] Jan A, Boulaaras S, Abdullah FA, Jan R. Dynamical analysis, infections in plants, and preventive policies utilizing the theory of fractional calculus. The European Physical Journal Special Topics. 2023;1-16.

[17] Jan R, Boulaaras S, Alyobi S, Jawad M. Transmission dynamics of Hand–Foot–Mouth Disease with partial immunity through non-integer derivative. International Journal of Biomathematics. 2023;16(6):2250115.

[18] Tang T-Q, Jan R, Khurshaid A, Shah Z, Vrinceanu N, Racheriu M. Analysis of the dynamics of a vector-borne infection with the effect of imperfect vaccination from a fractional perspective. Scientific Reports. 2023;13(1):14398.

[19] Jan R, Abdul Razak NN, Boulaaras S, Rehman ZU, Bahramand S. Mathematical analysis of the transmission dynamics of viral infection with effective control policies via fractional derivative. Nonlinear Engineering. 2023;12(1):20220342.

[20] Jan R, Abdul Razak NN, Boulaaras S, Rajagopal K, Khan Z, Almalki Y. Fractional perspective evaluation of chikungunya infection with saturated incidence functions. Alexandria Engineering Journal. 2023;83:35-42.

[21] Achaiah NC, Subbarajasetty SB, Shetty RM. R0 and Re of COVID-19: Can We Predict When the Pandemic Outbreak will be Contained? Indian J Crit Care Med 2020;24(11):1125–1127.

[22] Centers for Disease and Control. Ending Isolation and Precautions for People with COVID-19: Interim Guidance, https://www.cdc.gov/coronavirus/2019-ncov/hcp/duration-isolation.html; 2019 [accessed February 1, 2022].

[23] Noh J, Danuser G. Estimation of the fraction of COVID-19 infected people in U.S. states and countries worldwide. PLoS ONE 2021;16(2): e0246772. https://doi.org/10.1371/journal.pone.0246772

[24] Worldometer. Italy Coronavirus case, https://www.worldometers.info/coronavirus/country/italy/; 2022 [accessed February 1, 2022].

[25] Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society A 1927;115(772):700-721.

NOMENCLATURE

m	Mixing variable – fraction of unvaccinated that mixes with the vaccinated
S	Fraction of the population that is susceptible
I	Fraction of the population that is infected
R	Fraction of the population that has recovered
t	Time measured in days

R0	Reproduction number
V	Vaccination coverage rate or fraction of the population that is vaccinated
α	Socialization factor that measures how much vaccinated persons increase their social activity above normal
β	Coefficient related to the reproduction number
ε	Vaccine effectiveness or the reduced probability of a vaccinated person contracting the virus
γ	Coefficient related to the rate of recovery
1,2,3	Subscripts denoting sub-populations (1 = vaccinated, 2,3 = unvaccinated)