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Modeling the COVID-19 Pandemic

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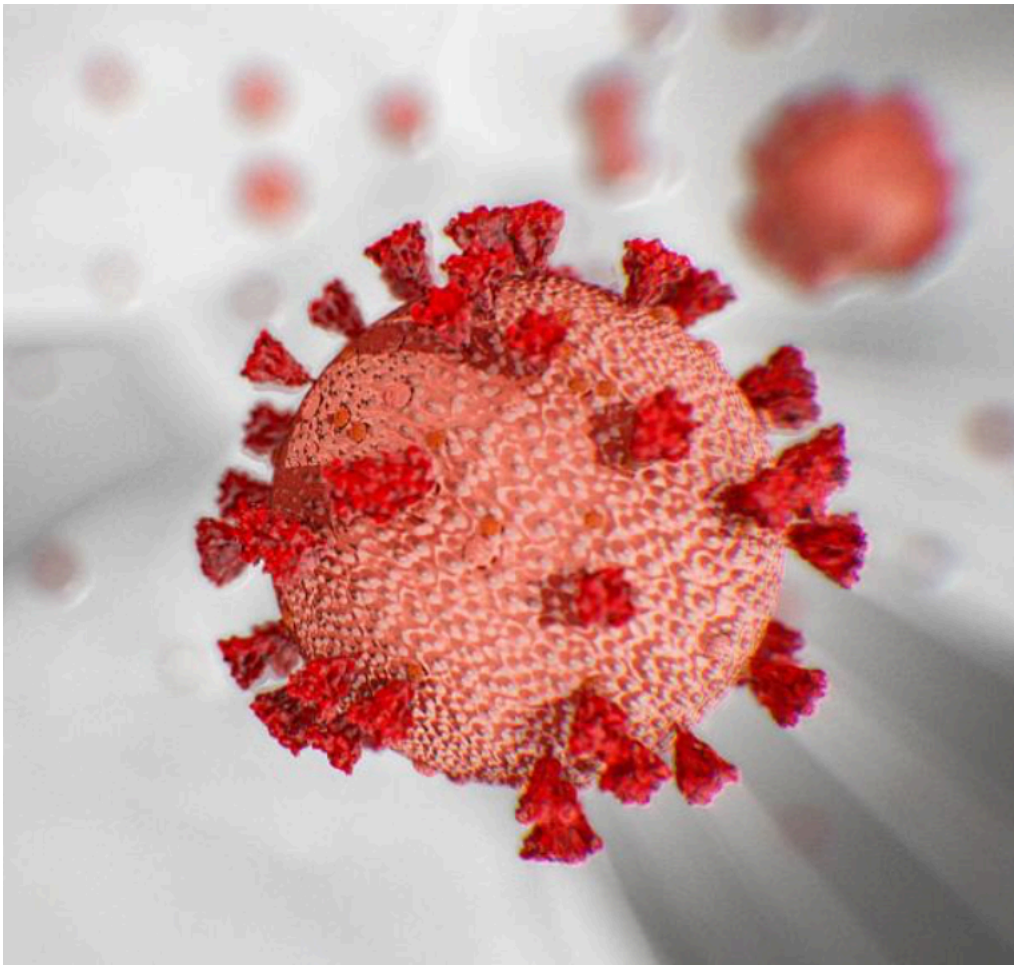
This paper is divided into three sections. The first section focuses on modeling the dynamics of a pandemic. The model embeds several parameters, which we estimate in order to explore the nature of the dynamics of a pandemic. We explore the timing of a generic shelter-in-place order to control growth of infection. In section 2 we use real data from Santa Clara County, CA to identify system parameters and validate the model. In section 3 we use the validated model for Santa Clara County to examine strategies for scaling back the shelter-in-place order, predicting the future of the pandemic.



1. Modeling a Pandemic

This paper describes the dynamics of a pandemic like COVID-19. The model targets a group of individuals in which the mechanism of contagion is community spread [1]. In this work we do not consider cases brought to the community from the outside. There is no attempt to segment the group by age or geographic distribution. On the second issue, think of this as the spread among individuals in their normal course of interaction within their communities.

Consider three groups within the population.

S	The number of persons susceptible to become infected
I	The number of persons infected
R	The number of persons recovered from infection

In the diagram to the right we represent the number of individuals in each category (S, I & R). If each infected individual interacts with (on the average) say m individuals during a time period T of contagion, what is the probability that one of those individuals will be a susceptible person? If the distribution at the right is random, and interactions randomly occur, then the likelihood of an infected person meeting a susceptible person is proportional to the density of susceptible persons in the population.

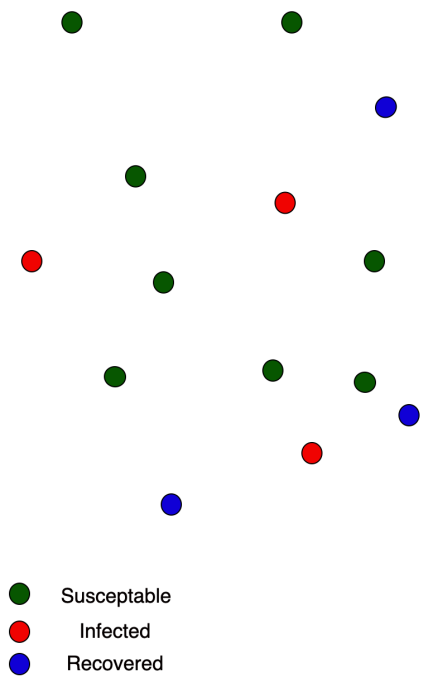


Fig 1: Random encounters among S, I, and R

$$(1) \quad p = \frac{\gamma S}{S + I + R} = \gamma \frac{S}{N}$$

If we include the chance of infection when there is an encounter, then the rate of new infections during that

time period T is given by:

(2) $\text{rate} = \frac{\rho IS}{N}$

Here, the multiplier ρ depends upon several factors, namely the number of interactions m that an infected individual makes as well as the probability of transmission of the pathogen when a contact is made. It is difficult to calculate this a-priori, but we may be able to infer the value from data describing the spread of the infection.

There is a mortality rate among infected individuals. The rate of death of these individuals is given by the following:

(3) rate of deaths = $-\mu I$

There is also a rate at which infected cases (who do not die) are cured—that is they move into the recovered class R. We will assume that this rate of conversion is given by the following:

(3) rate of recovery = $-\alpha I$

Here α is the cure rate.

Adding these rates together gives the rate of change of the infected class.

(4) $\frac{dI}{dt} = \frac{\rho IS}{N} - \mu I - \alpha I$

The equation for the susceptible group has a similar structure, but the sign of the $\frac{\rho IS}{N}$ term is opposite; while an infection increases the infected number, it decreases the susceptible cohort. Hence:

(5) $\frac{dS}{dt} = -\frac{\rho IS}{N}$

.

While it is true that there will be mortality in the S group it is significantly smaller than the I group, and we will ignore this term. There is, of course, no cure rate for the S group.

Finally, we need to model the recovered group R. Here the rate of change shares the cure rate from equation (4).

$$(6) \qquad \frac{dR}{dt} = \alpha I$$

Finally, we can write an expression for the number of deaths M(t) and the total number of cases Y(t).

$$(7) \qquad \frac{dM}{dt} = \mu I \qquad \frac{dY}{dt} = \rho I \frac{S}{N}$$

Stating it another way, M is the integral of μI over some time interval.

Model 1 Covid-19 Epidemic

$$(8) \qquad \begin{aligned} \frac{dS}{dt} &= -\frac{\rho IS}{N} \\ \frac{dI}{dt} &= \frac{\rho IS}{N} - \mu I - \alpha I \\ \frac{dR}{dt} &= \alpha I \\ \frac{dM}{dt} &= \mu I \\ \frac{dY}{dt} &= \rho I \frac{S}{N} \\ N &= S + I + R \end{aligned}$$

Initial Estimation of System Parameters

In a recent interview with CNN, Dr. Tom Frieden and Dr. Cyrus Shahpar[1] summarized the current understanding of COCID-19. One important dimension is the basic reproductive rate of the virus. According to these experts, "the best current estimate is that that this rate is between 2-2.5, roughly twice that of seasonal influenza. Some studies have reported higher estimates."[2] The next question to ask is: *How long is an infected individual contagious?* A recent article in the journal Lancet, presents statistics from a study of patients in Wuhan, China. I will assume that the period of contagion for an infected individual is 3 weeks. If that individual has severe symptoms and requires admission to the hospital or self quarantine, the effect will be to isolate that individual from the social network. How can we use this data to estimate the contagion parameter in our model? The expression below from (1.8b) shows the contribution to infection growth due to spread of the virus. Using $\Delta t = 3$ weeks, $\Delta I = 2.3$ for I=1 persons and $\frac{S}{N} \approx 1$ (near the start of the epidemic the bulk of the population is susceptible). We can estimate ρ.

$$\frac{dI}{dt} = \frac{\rho IS}{N} \qquad \frac{\Delta I}{\Delta t} = \frac{\rho IS}{N} \qquad \rho = \frac{2.3}{3} = .77$$

The next important parameter is the death rate. The World Health Organization reports that 3.8% of reported COVID-19 cases die. Now we have a problem. How many COVID-19 cases are NOT confirmed? Estimates are that 80% of infections present themselves as asymptomatic or mildly symptomatic. But, recall that the 3.8% is based on confirmed cases. This would imply that of 1000 reported cases there

[1] Dr. Tom Frieden is former director of the US Centers for Disease Control and Prevention and former commissioner of the New York City Health Department. Dr. Frieden is also Senior Fellow for Global Health at the Council on Foreign Relations.

[2] The article cites a report from the World Health Organization.

would be 5,000 cases (confirmed and unconfirmed). With an average duration of illness of 3 weeks, the number of deaths 38 per a total of 5000 infected individuals. Assuming an average time to of three weeks, and the weekly death rate would be

$$\mu = \frac{38}{5000 * 3} \approx .001$$

We can use similar reasoning to express the weekly cure rate as

$$\alpha = \frac{(1.0 - .001)^3}{3} = .33$$

$\rho = .77$
$\mu = .001$
$\alpha = .33$

Model Parameters: Covid-19 Epidemic

The simulation studies that follow, are intended to illustrate the dynamics of a pandemic. They are not predictors of any specific population.

Because there is uncertainty in the value of the parameters, the Figures 2 and 3 show variation of the infections (I) and deaths (M) using three different values of ρ = [.77, .70, .60].
The results depicted in figures 2 and 3 are for a population of 2,000,000 persons (roughly the size of Santa Clara County, CA). The time scale of the dynamic system appears to be reasonable.

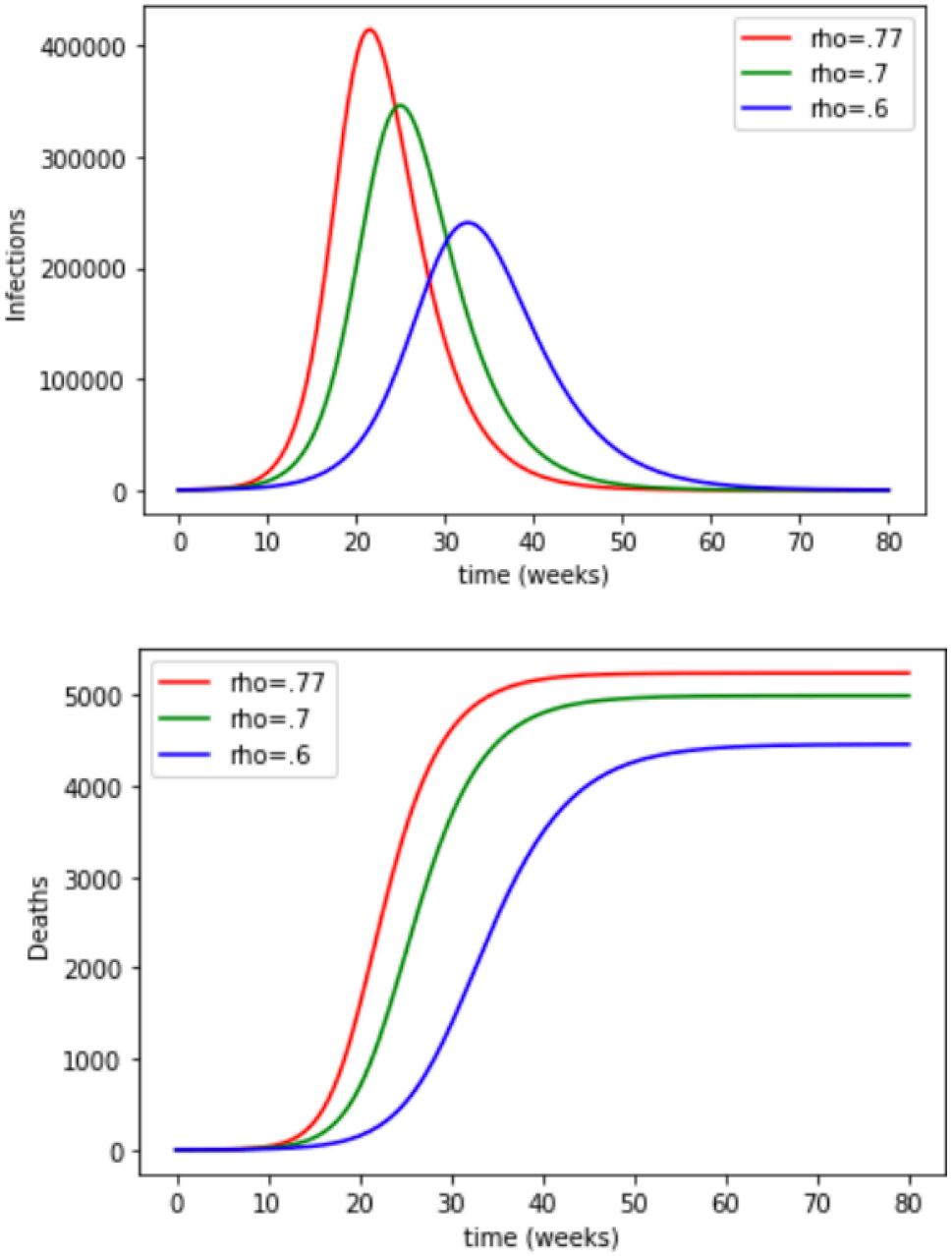


Fig 3: Deaths (M) with parametric change of ρ.

The contagion parameter ρ has a marked influence on the progress of the outbreak. The smaller the value of ρ, the lower the apex of infections and the more it is pushed into the future. When we implement social distancing, we reduce the frequency of contacts, and thus reduce ρ. Figure 3 demonstrates that lower values of ρ lead to fewer total deaths. The total number of deaths is proportional to the

integral of the infections over time. While decreasing ρ reduces the apex of the curve, to also broadens the time over which the epidemic is active. The result is that the total number of deaths does decrease, but not as markedly as one might hope.

There is no universal value for ρ. In a densely populated area like New York City where people crowd onto public transit, ρ is likely considerably larger than in a rural community. Without knowing the exact value of the contagion parameter, we can infer the general character of the epidemic, but we cannot predict things like maximum infections and time to apex.

In each of the cases illustrated in Figure 2, the pandemic has been allowed to die a natural death. If we look at the red curve (ρ=.77) we can plot how S (susceptible), I (infected), and R (recovered) populations behave.

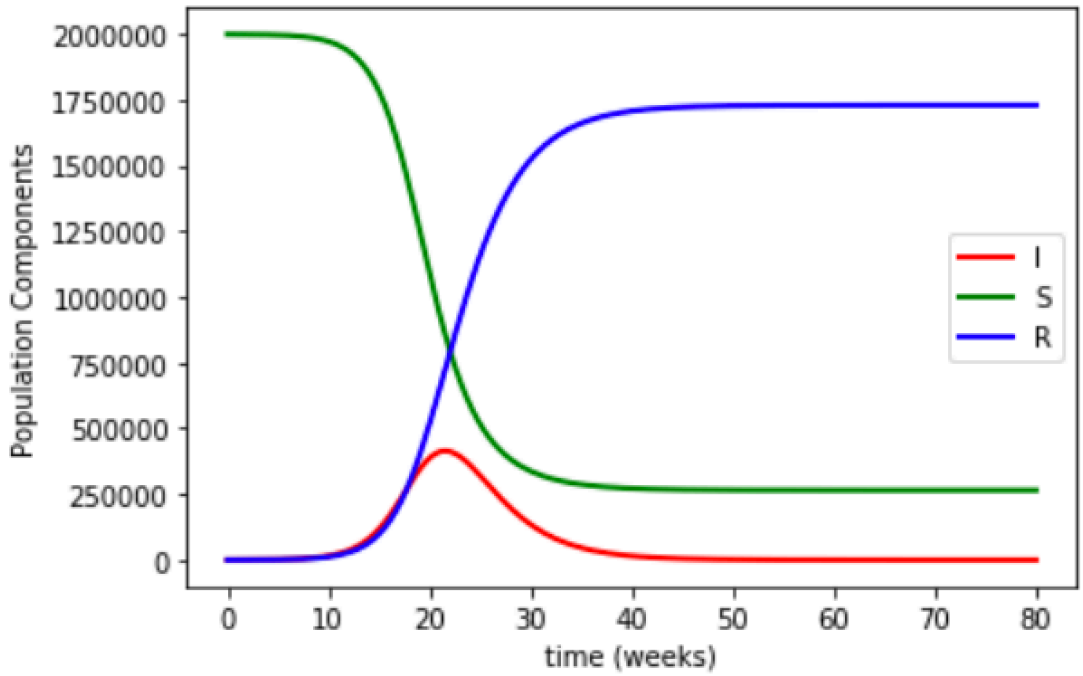


Fig 4: S,I and R as a function of time with ρ=.77

The infection has died out, but there still are susceptible members (S) of the population which could ignite a second round of infection. It should be noted that we have made an assumption that the recovered class R is not susceptible to another infection. At the present time, this is suspected, but not a proven fact.

One of the critical issues that must be considered is: can our medical infrastructure support the number of severely impacted individuals? At the apex of the infection, there are 400,000 infected individuals (about 20% of the population). Earlier we stated that 80% of this group was asymptomatic or mildly symptomatic. Using the 20% who are symptomatic, suppose that 10% required hospitalization. there might be as many as 80,000 with some symptoms. If 10% of this group requires hospitalization, that means that we will require 8,000 beds at the apex of the epidemic. If the load on the medical infrastructure exceeds capacity, the mortality rate will escalate and the total number of deaths will increase.

Implementing Social Distancing

Let's try a few experiments with our model along with the parameters specified on page 5. Suppose we can reduce the level of social interaction dropping the contagion parameter from .77 to 0.5 . The three trial runs in the following figure are for no implementation of social distancing, implementation at 10 weeks and finally delay in implementation until 15 weeks.
It is clear that the earlier implementation of social distancing reduces the apex event and delays it's occurrence. It is clear that the earlier we implement social distancing, the better off we are. The blue and green curves in the diagram above follow the uncontrolled curve until the implementation of the strategy. The early implementation of the strategy reduces the apex event by about 40% and shifts the timing of this maximum about 10 weeks. This is precisely the result that social distancing is designed to accomplish. It will greatly reduce the stress on medical infrastructure and provide more time to put together assets to address the apex.

Fig 5: Impact of timing of social distancing on the infections.

Clearly, social distancing has a devastating impact on the economy. We will run some tests to explore when it may be appropriate to lift the restrictions.

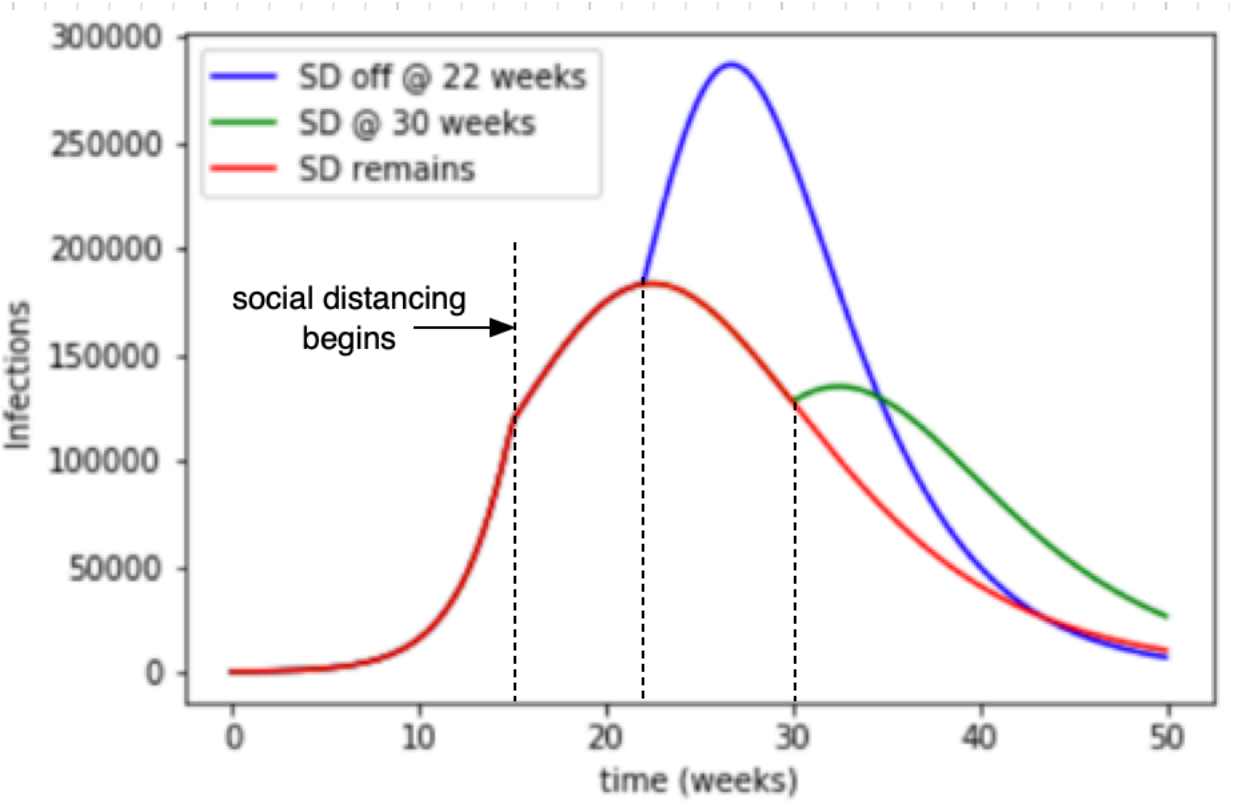


Fig 6: Impact of removing social distancing

In figure 6, we show (in red) applying social distancing at 15 weeks and keeping it in place. The red curve shows a discontinuity in slope at the point of application of SD. We next allow social distancing to be in place until we reach the apex of the red curve; we then revert to the original value of $\rho = .77$. The number of infections begins to grow rapidly and approaches three quarters of the infections if we never implemented social distancing at all (compare the apex of the blue curve in Fig 6 with the apex of the red curve in Fig. 5).

Delaying the removal of social distancing until 30 weeks is shown by the green curve. Here, infections do grow upon removal, but it is far less severe.

Why does this non-intuitive behavior happen? Let's look at equation (7b) describing the rate of increase in infections.

$$\frac{dI}{dt} = \frac{\rho IS}{N} - \mu I - \alpha I = I\left(\frac{\rho S}{N} - \mu - \alpha\right)$$

For fixed $(\mu + \alpha)$ the when ρ increases from .5 back to .77, S/N must have fallen sufficiently to have a small increase in dI/dt. For the parameters in this run (after social distancing is removed) $\frac{S}{N} < .43$

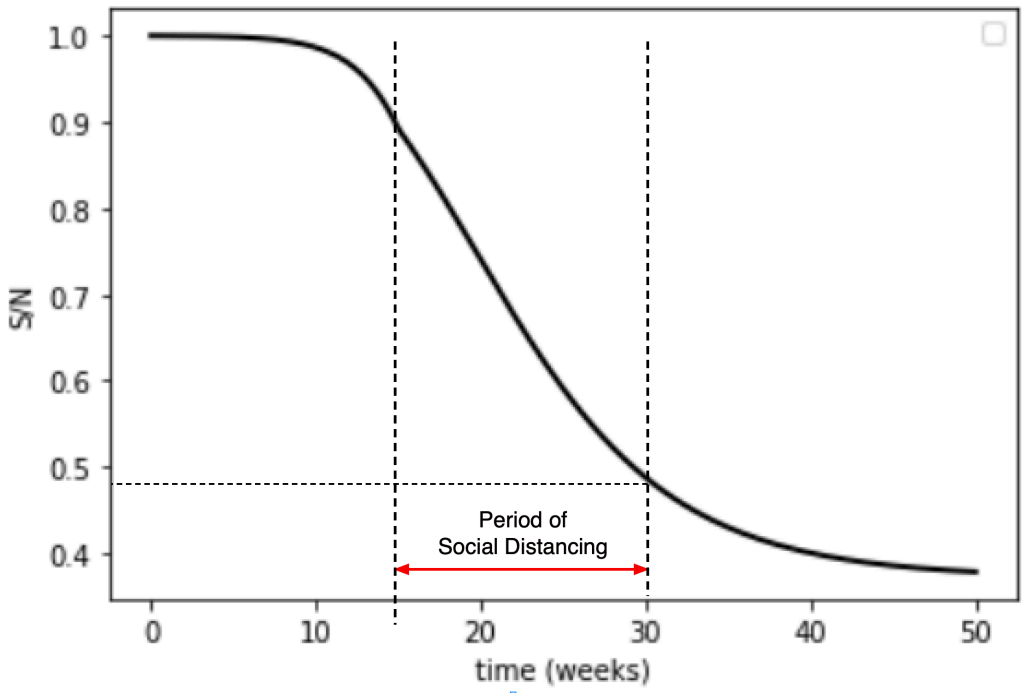


Fig 7: S/N with social distancing applied between 15 and 30 weeks

The small positive slope of the green curve in figure 6 can be seen here; at 30 weeks the value of S/N is still a little above the critical value of .43.

2. Fitting Model Parameters to Santa Clara County Data

There is a data file on github.com which contains COVID-19 data for many counties in the United States. The data for Santa Clara County begins with the first detected case on 31 JAN 2020 (day 0) and as this paper was written ends on 12 MAY 2020 (day 890. The county was one of the first to order a shelter-in-place directive on 31 MAR 2020 (day 61). The following figure plots the cases (cumulative) over time (from this data set) in days starting 31 JAN 2020.

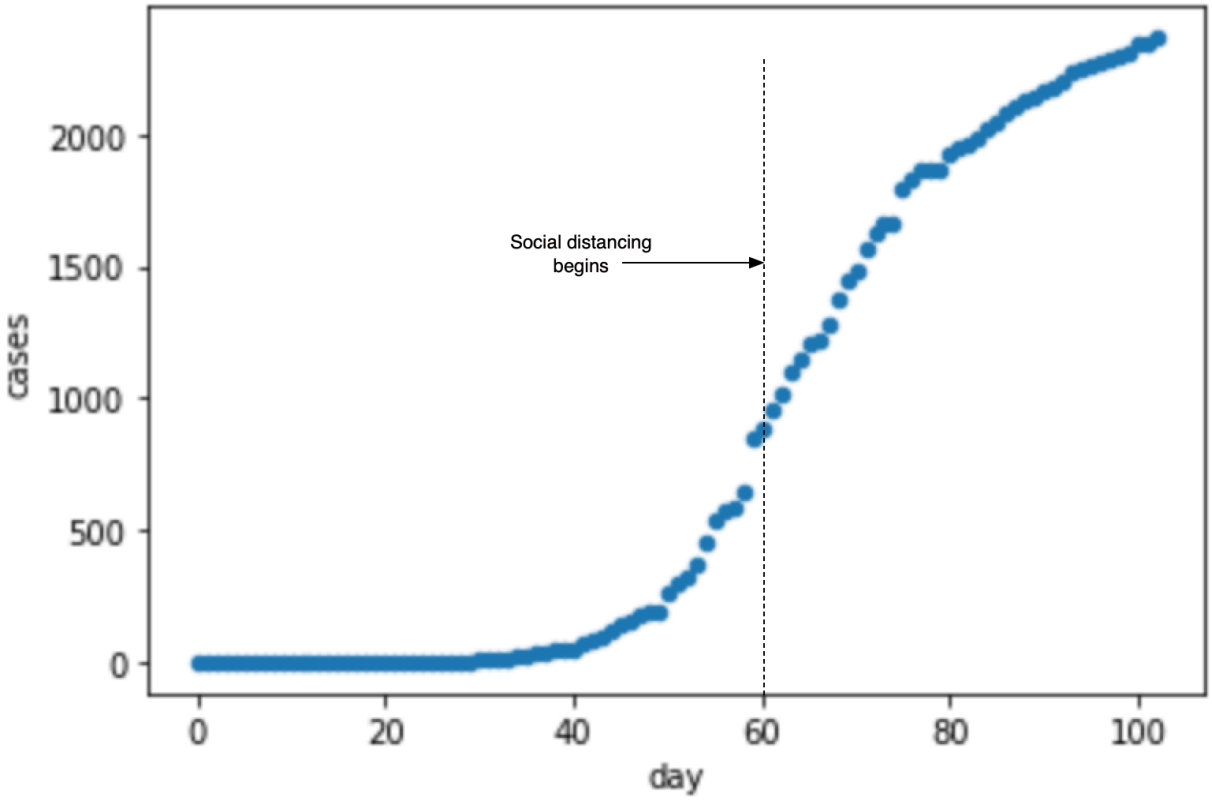


Fig. 8: Cumulative cases of COVID-19 reported by Santa Clara County, CA.

It is important to realize that these are confirmed cases. A study by Stanford University[3] revealed that the actual cases are on the order of 50 times as large as the cases confirmed by testing. There is a reason for this. First, many cases of COVD-19 present as asymptomatic or mildly symptomatic. Testing has not been done randomly throughout the population. Only those individuals who have severe cases and enter the hospital, medical staff and first responders, and individuals with symptoms whose medical doctor has written an order for testing. I will assume that the actual cases and the confirmed cases are related by the following.

$$I^c(t) = kI(t) \tag{9}$$

Equations (8) can then be rewritten in terms of the confirmed cases. If we multiply equations (8) by k and define $I^c(t) = kI(t)$, $S^c(t) = kIS(t)$, $R^c(t) = kR(t)$, and $N^c = kN$, then we obtain equations very similar to (8) except these are written in terms of scaled population parameters.

$$\frac{dS^c}{dt} = -\frac{\rho I^c S^c}{N^c}$$

$$\frac{dI^c}{dt} = \frac{\rho I^c S^c}{N^c} - \mu I^c - \alpha I^c$$

(9)

$$\frac{dR^c}{dt} = \alpha I^c$$

$$\frac{dM^c}{dt} = \frac{dM}{dt} = \mu I^c \,, \qquad \frac{dY^c}{dt} = \rho \frac{S^c}{N^c} I^c$$

$$\frac{dN^c}{dt} = \mu I^c$$

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[3] A pre publication report by faculty of Stanford University and others indicates the true number of infections in Santa Clara County, CA is at least 50 times higher than the confirmed cases suggest. The research group administered blood serum tests to a random sample of residents of Santa Clara County to detect antibodies to COVID-19, which is an indicator that an individual had been infected. The report has been criticized because, at present, we do not know the reliability of the serum test: does it give false positive or negative results? A summary of the report was printed in the Mercury News.

There is a reason why we did not define $M^c = kM$. The deaths which have occurred are countable; they are not sampled. While there have been deaths for individuals who were not tested, autopsies have concluded that the death was the result of COVID-19. There is likely some under count in M(t), but I have assumed that this is negligible.

If you compare equations (9) to equations (8) they are very similar with I, S, R replaced by I^c, S^c, R^c . However equations (9) are written for a scaled population $N^c = kN$. In the subsequent work in this paper we will be working with this set of scaled equations because that is the data we have. We can make an estimate of what is happening in the real population by dividing each of the "confirmed values" by the constant k.

We now have 4 unknown parameters ρ, μ, α, k embedded in the model. We will use data from Santa Clara County to identify these parameters.

The data in figure 8 is not $I^c(t)$ but $Y^c(t) = \int_0^t \rho \frac{S^c}{N^c} I^c(\tau) d\tau.$

If we assume that the susceptible fraction of the population $\frac{S^c}{N^c} \approx 1$ (a condition near the beginning of the epidemic) then equation (9b) can be approximated by

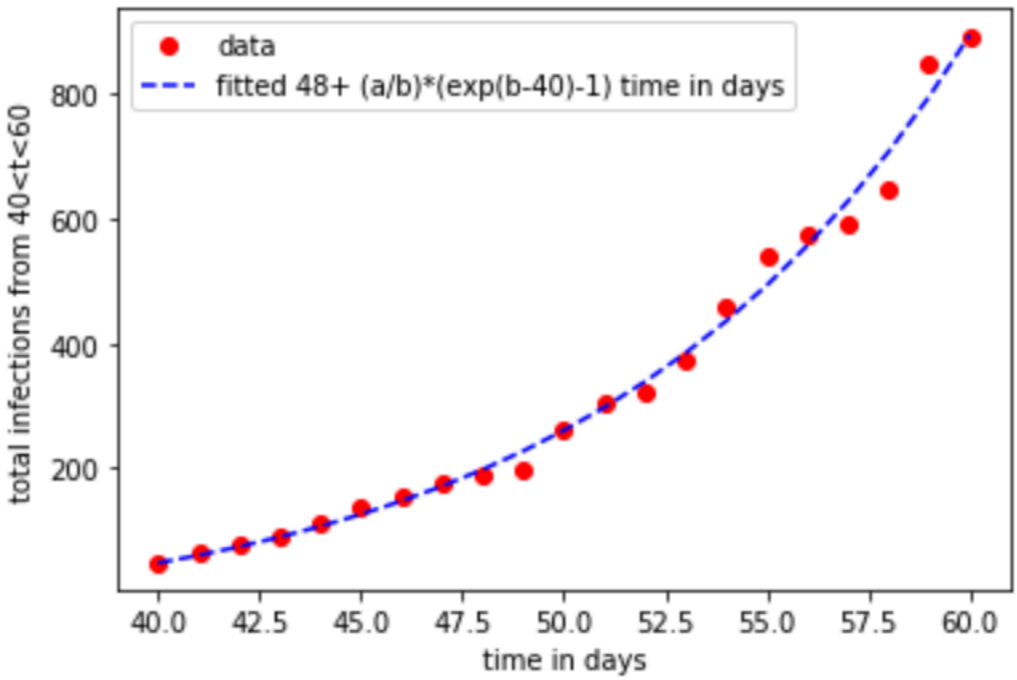
(10) $$\frac{dI^c}{dt} = (\rho - \mu - \alpha) I^c = \gamma_1 I^c \quad \text{where } \gamma_1 = \rho - \mu - \alpha$$

We will solve this equation with the initial condition at t=40days $I^c(40)$

(11) $$I^c(t) = I^c(40) e^{\gamma_1 (t-40)}$$

Since Multiplying equation (11) by ρ_1 and integrating over the time span $40 < \tau < t$

(12) $$Y^c_{40,t}(t) = Y^c_{40,40} + \int_{40}^{t} \rho_1 I^c(\tau) d\tau = \frac{\rho_1 I^c(40)}{\gamma_1} \left[e^{\gamma_1 (t-40)} - 1 \right]$$




```
Y(60)= 894.9260076479738
b= 0.10915462981266467
a= 11.741089532474133
```

Fig 9: Data fit to total infections 40<t<60

In fitting the data we have forced $Y_{40,40} = 48$; this is from the Santa Clara data. We next use the Santa Clara data to do a least squares fit to determine the constants a and b.

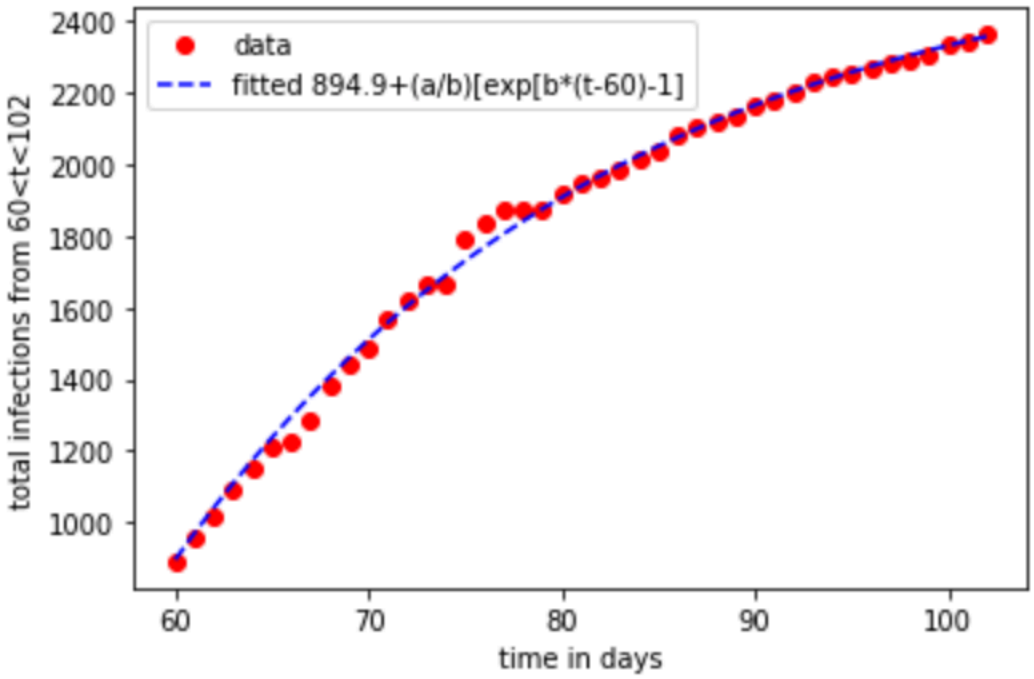
(13) $$Y^c_{40,t}(t) = Y^c_{40,40} + \frac{a}{b}\left[e^{b(t-40)} - 1\right] \qquad a = \rho_1 I^c(40)$$

$$b = \gamma_1$$

The result of the least squares fit isγ_1

$$\gamma_1 = .1092\ [days]^{-1}$$

(13)




```
Y(60)= 894.9
Y(90)= 2165.965503612215
b= -0.043459710406265534
a= 75.82728389253418
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Fig 9: Data fit to total infections 60 < t

$$a_1 = \rho_1 I^c(40) = 11.741 \ \ [days]^{-1}$$

We next move to fit data to the cumulative cases after the shelter-in-place order on day 61. The second set of data ranges from 60days<t. Again we begin with the equation[4] for $I^c(t)$

(14) $$I^c(t) = I^c(60)e^{\gamma_2(t-60)} \qquad 60 < t$$

In order to insure no discontinuity in the fitting function, we will use $Y^c_{40,60} = Y^c_{60,60} = 894.9$. Let us write an expression for $Y^c_{60,t}$

[4] There is an assumption that S^c/N^c is still nearly 1. This will be checked after the model is completed.

$$I^c(60) = I^c(40)e^{\gamma_1(60-40)} = 8.882I^c(40)$$

$$Y^c_{60,t}(t) = Y^c_{60,60} + \int_{60}^{t} \rho_1 I^c(\tau)d\tau = \frac{\rho_2 I^c(60)}{\gamma_2}\left[e^{\gamma_2(t-60)} - 1\right] \tag{15}$$

Incorporating these into (15)

$$Y^c_{60,t}(t) = 894.9 + \int_{60}^{t} \rho_2 I^c(\tau)d\tau = \frac{8.882\rho_2 I^c(40)}{\gamma_2}\left[e^{\gamma_2(t-60)} - 1\right] \tag{16}$$

We will do a least squares fit to this equation

$$Y^c_{60,t}(t) = 894.9 + \frac{a}{b}\left[e^{b(t-60)} - 1\right] \tag{17}$$

The fit to this second group of data is shown in Figure 9.

$$b_2 = -.04346[days]^{-1}$$

$$a_2 = 8.588\rho_2 I^c(40) = 75.827[days]^{-1} \tag{18}$$

Dividing (18b) by (13b) we get

$$\frac{a_2}{a_1} = \frac{75.827}{11.741} = 8.588\frac{\rho_2}{\rho_1} = 6.458$$

$$\frac{\rho_2}{\rho_1} = .7520 \tag{19}$$

From equation (10) we can write

$$\gamma_2 - \gamma_1 = \rho_2 - \rho_1 = -.04346 - .1092 = -.1527 \tag{20}$$

.

Solving (19) and (20) yields the following. The dimension of these contagion rates from the least squares fit are in infections/day. We must convert these to infections/week to use in our simulation.

$$\rho_1 = .6157[days]^{-1} = 4.31[weeks]^{-1}$$

$$\rho_2 = .4208[days]^{-1} = 2.9456[weeks]^{-1} \tag{21}$$

In the least squares fit to the data, the growth coefficients γ have units of $\frac{1}{days}$. Later when we do a simulation of the pandemic in Santa Clara County, we will use the time parameter as weeks, so equation (21) expresses these growth coefficients in the units of the simulation environment $\frac{1}{weeks}$.

We have data on the total number of deaths in the Santa Clara data. One important thing to realize is that when we measure deaths, there is no difference between confirmed deaths and real deaths. We will assume that all individuals who die of the disease would have been tested[5]. Let us begin to estimate of μ from equation (9d). Figure 8 displays the total number of infections plotted against time (in days). Integrating (9b) from time 0 to time t yields the following. But the data does not tabulate I(t), rather it tabulates Y(t) .

$$M(t) - M(40) = \frac{\mu}{\rho_1}\int_{40}^{t} \rho_1 I^c(\tau)d\tau = \frac{\mu}{\rho_1} Y^c_{40,t} \qquad 40 < t < 60$$

$$M(t) - M(60) = \frac{\mu}{\rho_2}\int_{60}^{t} \rho_2 I^c(\tau)d\tau = \frac{\mu}{\rho_2} Y^c_{60,t} \qquad 60 < t < 90 \tag{22}$$

[5] Either because they have had severe symptoms or post mortem revealed they had the disease.

We will fit the following straight line to the data on the left hand pane of Figure 10. At t=40 days, $Y_{40,40} = 45$ and M(40) = 1

$$M(t) = m(Y_{40,t} - 45) + 1$$

The least squares fit gives $m = \dfrac{\mu}{\rho_1} = .0383$

$$\mu_1 = .0236[days]^{-1} = .1651[weeks]^{-1}$$

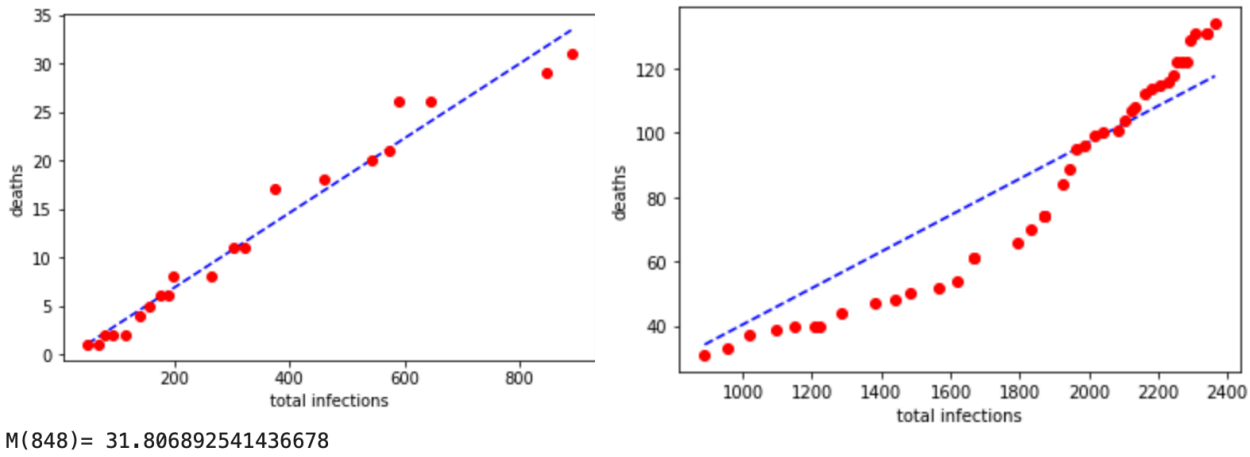


Fig 10: Deaths vs. cumulative cases (left)40<t<60 (right) 60<t<90

The fitting curve for the second pane (60 < t)

$$M(t) = m(Y_{60,t} - 894.9) + 31.776$$

M(60) corresponds to $Y_{40,60} = 894.9$ and M(60)=31.776

This assures that the two fitting curves do not have a discontinuity of value at t=60. They do have a discontinuity of slope, however.

There is a visual distortion in the least squares fit. In the diagram above (right) the data points are more dense in the final segment of the

data, so it "appears" that there is an error in the least squares fit. This is not true.

From the least squares fit $m = \frac{\mu}{\rho_2} = .0592$

$$\mu_2 = .0249[days]^{-1} = .1744[weeks]^{-1}$$

There is no direct data in the Santa Clara records which addresses the cure rate α. However, Using our data fit shown in figures 8 and 9 we found that:

$$\gamma_1 = .1092 = \rho_1 - \mu - \alpha = .6157 - .0233 - \alpha$$

$$\alpha_1 = .4832[days]^{-1} = 3.382[weeks]^{-1}$$

$$\gamma_2 = -.04357 = \rho_2 - \mu - \alpha_2 = .4208 - .0233 - \alpha_2$$

$$\alpha_2 = .4412[days]^{-1} = 3.088[weeks]^{-1}$$

After having fitted these parameters to the data, we will perform a simulation and plot the Santa Clara data with the simulated response.

The one parameter we have not estimated is the coefficient k. The Stanford study indicated that this was on the order of magnitude of 1/50. For lack of any other data, we will use this value.

The simulation of the Santa Clara pandemic using the following parameters:

k= .02

$$\alpha_1 = 3.382[weeks]^{-1} \quad \alpha_2 = 3.088[weeks]^{-1}$$

$$\mu_1 = .1651[weeks]^{-1} \quad \mu_2 = .1744[weeks]^{-1}$$

$\rho_1 = 4.31$ (before shelter-in-place)

$\rho_2 = 2.9456$ (after shelter-in-place)

I chose to use two different parameters for the cure rate and the death rate.

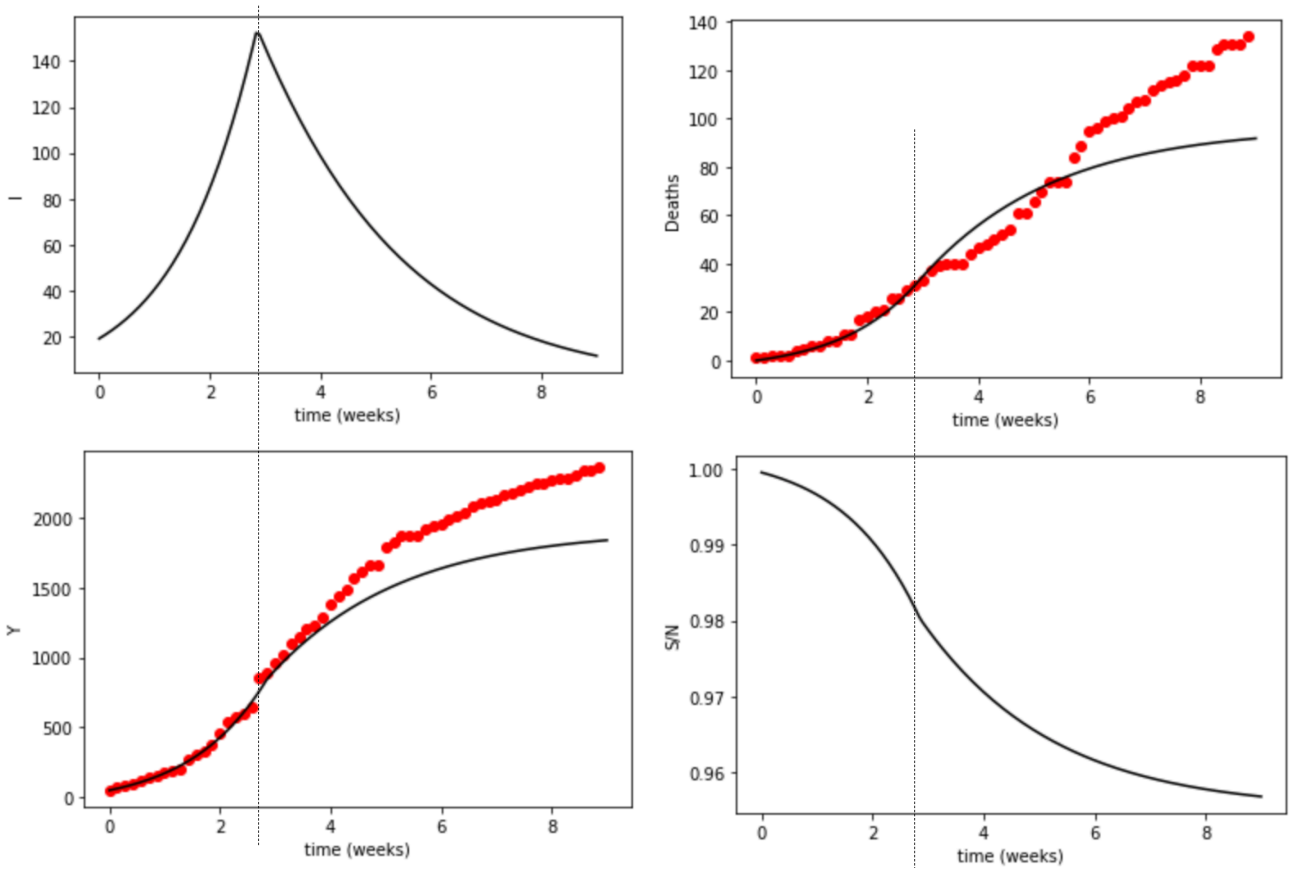


Fig. 11: Simulation results for the first test of the validated Santa Clara County Model.

We note that the total infections and the total deaths in the simulation under-predict the actual data. Note, however, that after the introduction of the shelter-in-place order (dotted line) that

$$.96 < S/N < .98$$

Remember when we estimated ρ_2 we assumed this was 1.0. Using an average value of .97, this would increase ρ_2 to a value above 3.0. In the simulation, I adjusted ρ_2 until I got a good match between the model and the Santa Clara data. This occurred when: $\rho_2 = 3.15$.

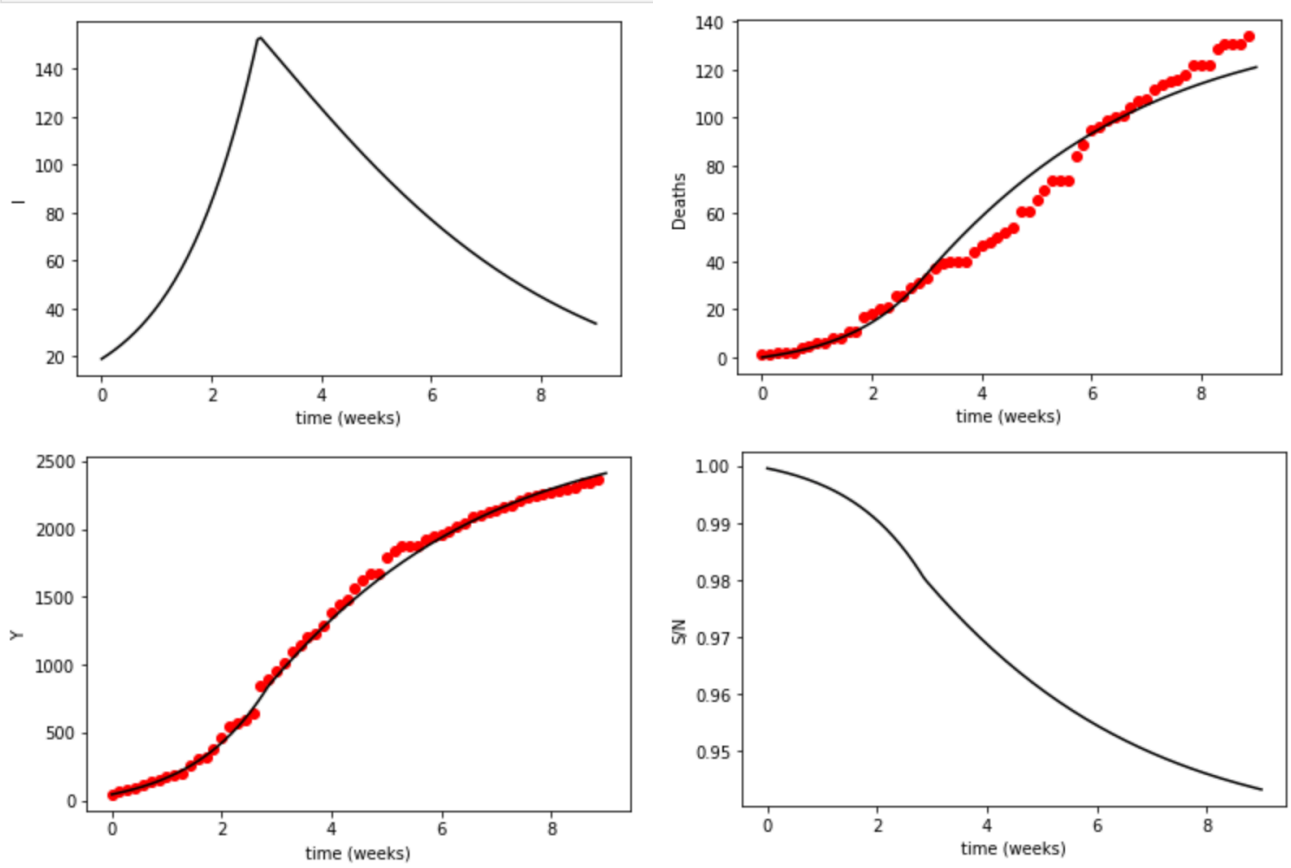


Fig 12: Final validation of the model against the Santa Clara data.

There is very good agreement between the Santa Clara data and the simulation of the total number of infections. The second graph in Figure 10 shows $I^c(t)$. Remember, there is not data for this variable. In the simulations in section 1 of this paper, I(t) continued to grow after social distancing was in place. Eventually, when S/N became small enough, I(t) reached a maximum, and the infections began to decline. In Santa Clara County, the data shows that the implementation of the shelter-in-place order was very effective, and there was an immediate decline in the number of infections. The total infections continued to increase. The slope of the total number of infections began to diminish as soon as the shelter-in-place order was effected.

One might ask why the cumulative deaths over estimates the data from week 3 to week 6. There is no dynamic in the equations that could create this variation. My guess is that there has been a delay in counting the deaths at any given time. Other than this small variation, the model does predict what is happening in Santa Clara County. Another explanation could arise from the fact that there is an additional lag-time as patients in their final stages of life are sustained in the ICU.

In the next section of this paper, I will use the model to predict some possible outcomes of changing the pandemic management stragtegy. When we used data to estimate the contagion parameters ρ_1 and ρ_2 we made an assumption that $\frac{S^c}{N} \approx 1$. The output of the simulation yields the fact that at the point of the shelter in place order this ration is 0.9996 .

3. Model Projections for Santa Clara County

One reasonable question to ask is: What would have happened if Santa Clara County did not implement Social Distancing? In figure 13 we superimpose two simulations: (1) Red: evolution of the pandemic if shelter-in-place had not been ordered, and Black: evolution of the pandemic with shelter-in-place order. One of the first things to see from the data is that the Santa Clara response did not just slow the growth by flattening the curve and shifting it toward the future, but

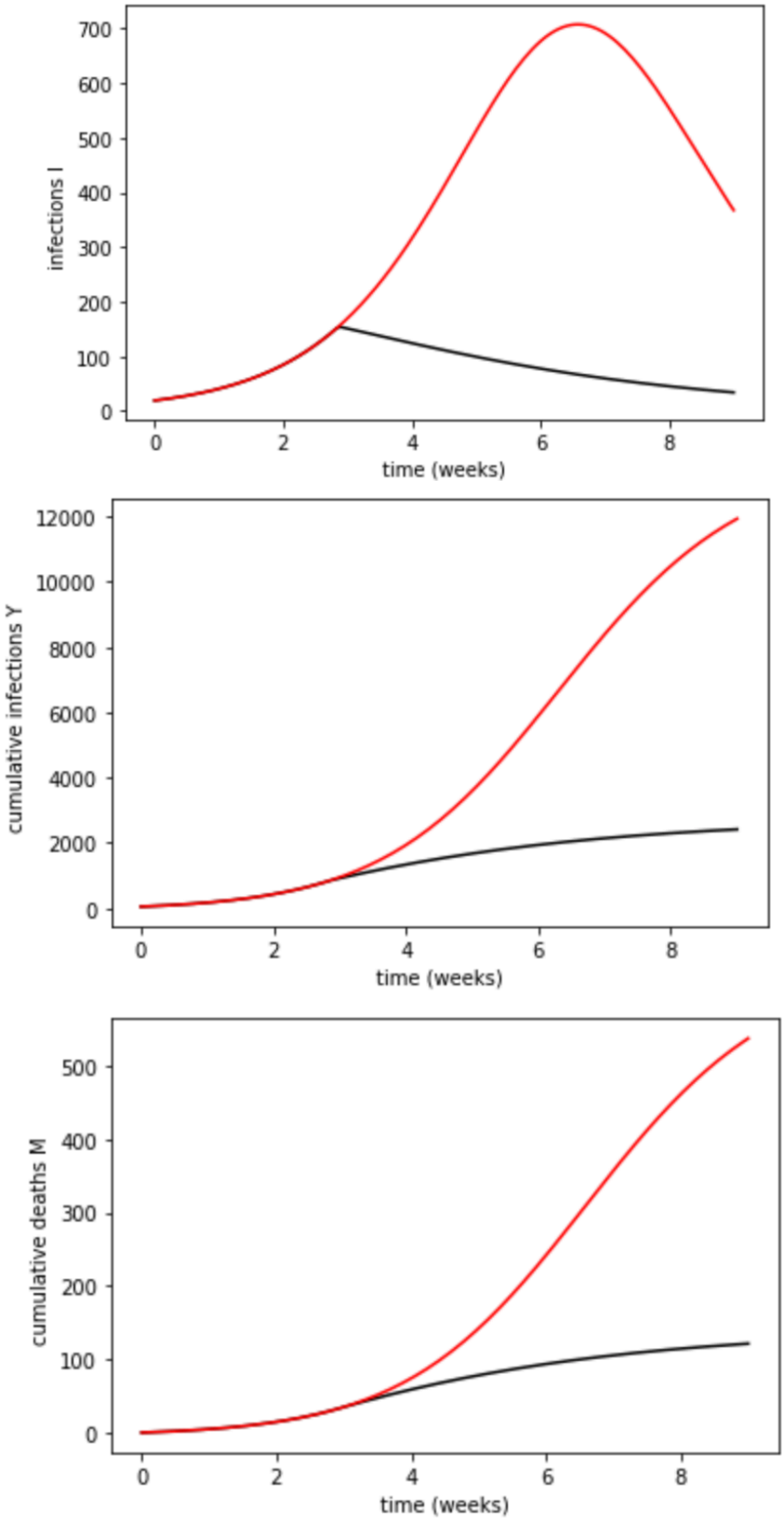


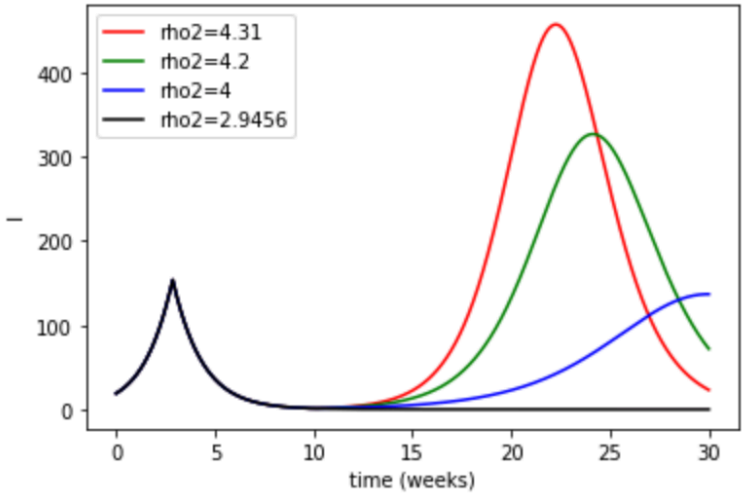
Fig. 13: The response (I, Y, and M) comparing Santa Clara shelter-in-place (black) to no directive (red)

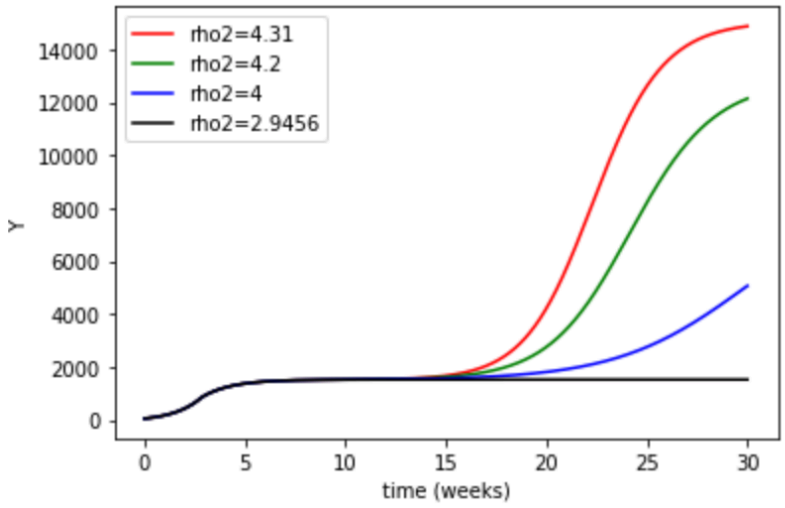
because of timely decision making, it actually created a strong decay in the infections. Comparing the non-managed response (red) to the managed response (black) the impact was a reduction in the maximum number of infected cases, from what would have been about 700 to about 200. Remember this is not the total number of cases of infection. More on that shortly. This max peak of the infections is critical to maintaining a working medical infrastructure—one which does not collapse under extreme loads.

Turning next to the total number of infections. For the simulation with the shelter-in-place order, the total number of infections for the time period simulated was about 2,000. For the same region with no social-distancing requirements that number increases to around 12,000— a six-fold increase. This is even more concerning when one considers the factor k representing the ratio of actual to confirmed cases. The Stanford pre-print referred to earlier in this paper suggests that this is a number on the order of 1/50. that would imply that a total of some 600,000 persons in the county[6] would have been infected without the shelter-in-place order. Deaths also show about a six-fold increase without social distancing requirements.

We ask the question: What will happen if we remove the shelter-in-place order? We can explore this possibility by again simulating several scenarios. The simulation begins 10 MAR 2020 (this is t=0). The shelter in place order was effective 30 Mar 2020 (week 2.9), and the date of modification of the shelter-in-place directive in the simulation is 19 MAY 2020. In Figure 14, the black curve represents the evolution of the pandemic if we continue on the current shelter-in-place plan. The red curve represents the evolution if we return to interactions pre-SIP. The green and blue curves are intermediate strategies between these two extremes. It is most clear if we focus on the infections I(t). **In all cases, there is a second wave of infections**. It makes sense that the closer we stay to the current SIP state, the smaller the peak. However, worst case scenerio (red) still yields a peak greater than that when SIP was initiated, and about 70% of what would have occurred if we never had a shelter-in-place order in March 2020. This is a strong

[6] That would amount to about 30% of the county's population.





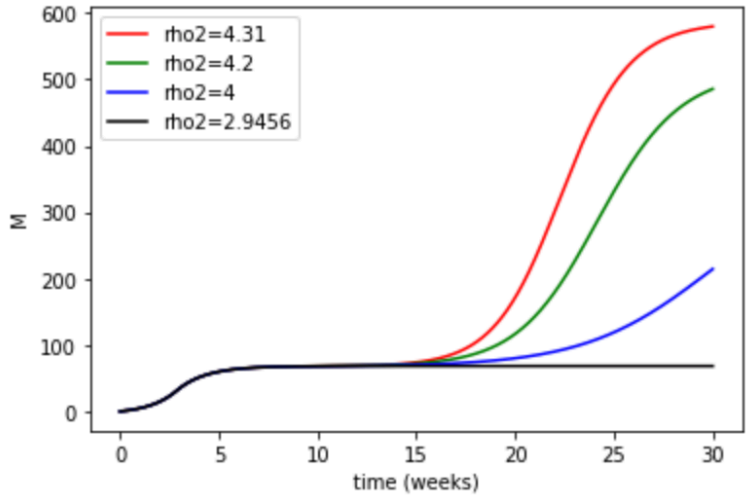


Fig 14: Santa Clara Projections with modification of shelter-in-place order at week 10. (black) continue shelter-in-place, (red) go back to interactions before directive, plus 2 intermediate strategies.

warning that the way we move out of managing interactions is absolutely critical! Why does this happen? Relaxing restrictions on social interaction increases ρ and the infection rate grows decreasing S/N. When S/N achieves a critical value, the infection will peak and then diminish. For any value of $\rho, \mu,$ and α The growth of the infection (equation 9b) will reach a maximum, when $\frac{dI^c}{dt} = 0$ in equation (23).

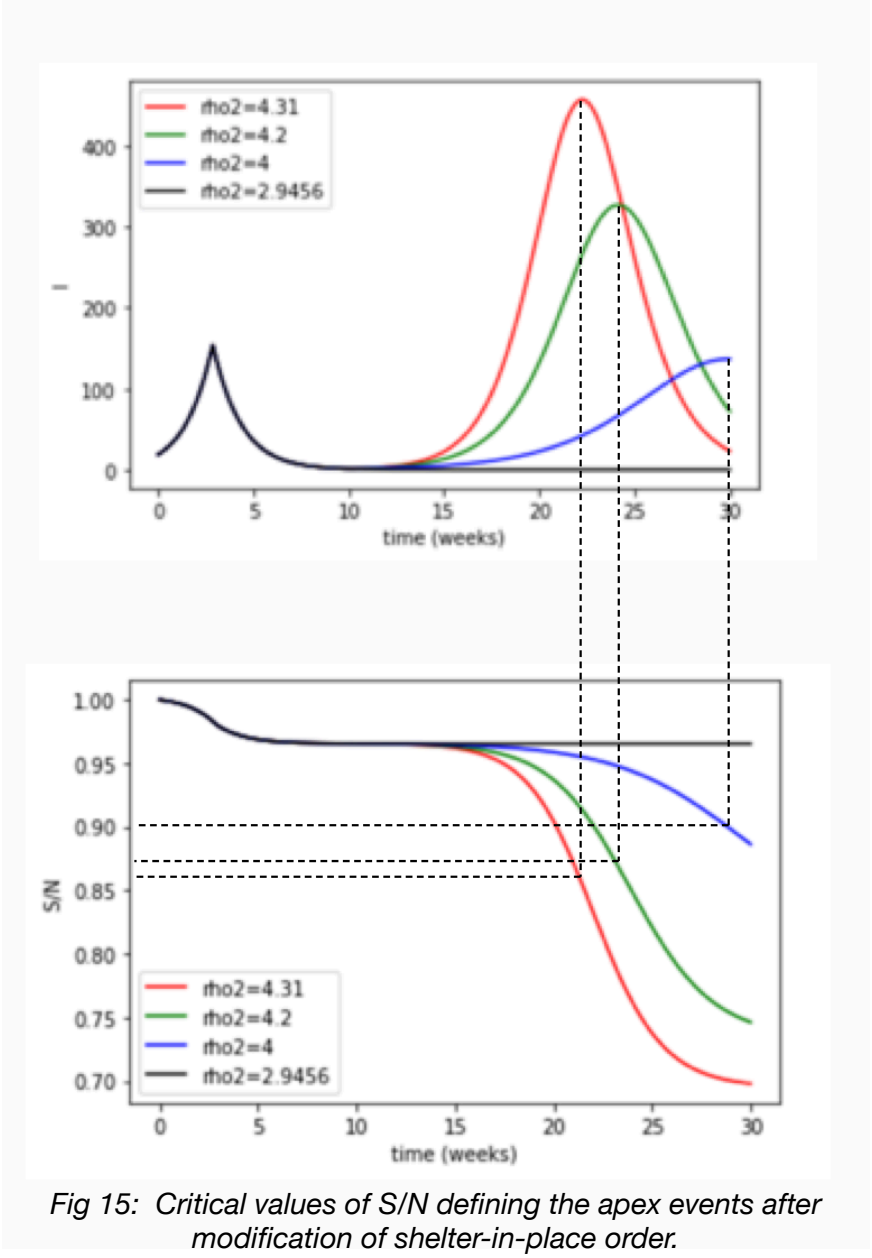


Fig 15: Critical values of S/N defining the apex events after modification of shelter-in-place order.

(23)

$$\frac{dI^c}{dt} = \rho I^c \frac{S^c}{N^c} - \mu I^c - \alpha I^c$$

$$\frac{dI^c}{dt} = 0 \qquad \text{when} \qquad \frac{S^c}{N^c} = \frac{\mu + \alpha}{\rho}$$

This is the critical value of $\frac{S^c}{N^c}$ which will cause the infections to decline.

Of course, these simulations cannot link back to key factors in reigniting the economy such as when to reopen restaurants, hair salons, etc. Rather, by tracking the resurgence of infections when change is made, we should be able to see what the trajectory to our future will be.

The only long term solution to the problem of the COVID-19 epidemic is the development and deployment of a vaccine. Remember, the strategy of requiring social distancing is a delaying factor, not the solution. Critical issues to relaxing the shelter-in-place orders are:

- New cases should be declining
- Medical infrastructure must be prepared for a "second wave"
- Testing must be in place and deployed
- Contact tracing must be implemented to track and isolate outbreaks

References

[1] Smith, David and Lang Moore, "The SIR Model for Spread of Disease - The Differential Equation Model", Mathematical Association of America: Convergence (teaching resources), 2004

[2] World Health Organization, Coronavirus Disease 2019 (COVID-19) Situation Report-46.

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Resources

The simulations described in this paper were written in Python using the Anaconda IDE. I used Jupyter Lab to document the development. If you have a Jupyter enabled environment, you can use the notebook below to explore my work. Failing that, you can use an internet browser to open the Notebook as an html file (although it will not be executable). The data for Santa Clara County is in the cvs file linked below.

Jupyter Notebook
Jupyter Notebook as html file
Santa Clara County csv file

Disclaimer

The author is not a medical doctor or a public health professional. He is a systems engineer with considerable experience in systems modeling and control. The material in this paper has not been peer reviewed and has not been vetted by medical or public health professionals.

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