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A Delay Differential Equation Model for the Spread of COVID-19

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ABSTRACT
In this work we propose the retarded logistic equation as a dynamic model for the spread of COVID-19 all over the world. This equation accounts for asymptomatic transmission, pre-symptomatic or latent transmission as well as contact tracing and isolation, and leads to a transparent definition of the instantaneous reproduction number*R*. For different parameter values, the model equation admits different classes of solutions.These solution classes correspond to, inter alia, containment of the outbreak via public health interventions, exponential growth despite interventions, containment despite reopening and second wave following reopening. We believe that the spread of COVID in every localized area such as a city, district or county can be accounted for by one of our solution classes. In regions where $R>1$ initially despiteaggressive epidemic managementefforts, we find that if the mitigation measures are sustained,then it is still possible for *R*to dip below unity when far less than the region's entire population is affected, and from that point onwards the outbreak can be driven to extinction in time. We call this phenomenonpartial herd immunity. Our analysis indicates that the trajectory of COVID-19 in any region is extremely sensitive to small changes in the parameter values.

Keywords: Retarded logistic equation Asymptomatic carriers Latent transmission Contact tracing Reproduction number calculation Partial herd immunity

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I. INTRODUCTION

The novel Coronavirus infectious disease (COVID-19) first appeared in a certain Oriental city in late 2019and spread all across the world over a matter of weeks. As the epidemic rages on,several thousand studies have emerged giving mathematical models of its spread. These models can be divided into three broad classes. The first is lumped parameter or compartmental models, which are ordinary differential equations (ODE) in one or several variables. This was the first ever approach adopted towards modeling an epidemic, and, though it requires assumptions of population mixing etc, it remains the model of greatest conceptual clarity and least computational time and effort. The second class of model is agent-based stochastic model where every person is treated as a lattice site on a network which can change its state from susceptible to infected to recovered etc. Sites change from susceptible to infected depending on whether their neighbours are infected. This is the most realistic model and can have very high predictive accuracy; the flipside is that the structure of the underlying network leaves scope for a lot of variability and the computational requirements are huge. The third class of model is stochastic ODE which combines features of the lumped parameter and agent-based models – sometimes at least, the features being synergized happen to be the most inconvenient ones, namely sweeping assumptions and massive computational effort.

Among the lumped parameter models, the most basic is the S-I-R (susceptible-infected-recovered) model[1], invented by WILLIAM KERMACK and ALEXANDER MCKENDRICK in 1927, and used to analyse an outbreak of plague. This model has been used very frequently for describing COVID-19 as well. Here however, it is less effective since it does not take into account the presence of asymptomatic carriers and the phenomenon of pre-

symptomatic or latent transmission. The first addition which is done is the introduction of a new compartment E (for exposed), which takes into account the incubation period – more complicated variants often have eight or more basic variables. While an elaborate, parameter-rich model may be very realistic, it also has the disadvantage of conceptual complexity, large number of unknowns and consequently non-unique data fits, which latter can occasionally be troublesome for the authors[2].

In this work we use delay differential equations (DDE) to propose a simple, single-variable, lumped parameter model – the retarded logistic equation – which we show is nevertheless capable of admitting a wide variety of solution classes observed in the real world.The advantage of simple models relative to elaborate ones has been highlighted by Jahedi and Yorke [3]. DDE has been used in Literature for modelingCOVID-19, for example in Refs. [4]–[6],but in our work we also account for sophisticated features such as contact tracing, asymptomatic carriers and latent transmission, and consequently our results too are more varied.The technical content of this Article is based on three preprints by our group [7]–[9].

§1 MODEL DERIVATION

The philosophyhere is sufficiently novel as to warrant a detailed treatment. We measure time t in days and introduce a single variable $y(t)$– we use y rather than x as a carryover from our original multivariate model [7] – which is the **cumulative**number of corona cases in the region of interest.This number includes active cases, recovered cases aswell as deaths (it is of course at the front and centre of every news article about the virus). Our philosophy is summarized by the following "word-equation"

$$\begin{pmatrix}\text{Rate of emergence}\\ \text{of new cases}\end{pmatrix}=\begin{pmatrix}\text{Spreading rate of}\\ \text{each existing case}\end{pmatrix}\times\begin{pmatrix}\text{Probability of}\\ \text{susceptibility}\end{pmatrix}\times\begin{pmatrix}\text{Number of}\\ \text{existing cases}\end{pmatrix}\quad . \qquad (0)$$

This word-equation has a transparent interpretation, which we now describe.The left hand side (LHS) of (0) is dy/dt, simple as that. The right hand side (RHS) is of course a little more involved.

The basic premiseunderlying (0)is that the disease is transmitted by sick people to healthy and susceptible people via interaction, which can be either inter-personal or via objects (this might appear obvious but it rules out situations such as where the virus is "floating in air" and infects everyone who passes). Because many patients are asymptomatic, and the symptomatic ones also turn transmissible prior to manifesting symptoms, at any time there will always be cases at large in society who are unaware of their infectious nature. Everysuch case has some rate of interaction with other people.For example,a working-from-home software engineerwho has the disease might go out once every three days and interact with one person on each trip, giving an interaction rate of 1/3 person/day, while an unknowingly infectiousbanker might go out once every day and interact with five customers every day, giving an interaction rate of 5 persons/day. For a lumped parameter model, the effectiveper-case interaction rate must be the average over all the atlarge cases – we call this rateq_0. For now, we assume that all the people whom the case is interacting with are susceptible – this holds true for example near the start of the epidemic when the susceptibles outnumber the recoveries by orders of magnitude. Every interaction however will not necessarily result in a transmission – rather, there will be some probability P_0 between 0 and 1 that the interaction will actually causethe virus to jump from the case to the susceptible person. This probability will depend on the level of precaution such as mask, handwashing and sanitization being adopted by both parties. Once again, in a lumped parameter model, the probability must be averaged over all at large cases. This average probability P_0multiplied by the average interaction rate $q_0$gives the per-case spreading rate, which we denote by m_0. In a nutshell,m_0 is the rate at which an at large case transmits the disease to other people, assuming they are all susceptible.This is the first term on the RHS of (0), and we take it as a fundamental parameter of our model.As the derivation makes clear, $m_0$is governed primarily by the strength of intervention measures such as social mobility restrictions, maintenance of six-foot separation minima and mask wearing.

The second term on the RHS corrects an obvious limitationof the previous reasoning – the assumption that everyone is susceptible.In reality, as the epidemic proceeds, there will be greater and greater number of people who are recovered and hence insusceptible. Thus a random person, such as the one with whom the at large case interacts, will actually have some probability $P_1\leq 1$ of being susceptible, and P_1 will vary over the course of the epidemic. The effective per-case spreading rate will then become$q_0P_0P_1$, or m_0P_1. In (0) we have shown P_1separately instead of clubbing it with m_0 since it depends on a person's immune response to COVID-19 rather than on public health interventions. In this Articlewe assume that onebout of infection renders a

person permanently insusceptible.This assumption remains valid ifthe immunity period is significantly longer than the duration of the epidemic. So far, the evidence for re-infection is at best scant and poorly documented[10]–[12]; on the other hand, a very recent study [13]based on monitoring of a huge patient cohort has found demonstrable evidence of long-lasting and effective antibodies. Another study [14]on benign coronaviruses (NOT COVID-19 virus) has found an immunity period approximating one year. If N be the initial number of susceptible people (and $y$is of course the cumulative number of cases), then the probability that a random person is a recovered case is approximately y/N and the probability that s/he is susceptible is (approximately) $1-y/N$. The word "approximately" arises because a case takes a finite time to recover – if y is the case count today then the actual recovery count today is less than y. Nevertheless, in this Article we shall stick to the expression $1-y/N$ for the second term on the RHS of (0); we shall explain our reasons after presenting our model equation. Note that $1-y/N$ is a logistic term; since it arises as a result of people acquiring immunity against the disease, it is a herd immunity effect.

The first two terms on the RHS of (0) account for the rate at which each case at large spreads the disease; the third term counts the number of such cases. Since y is a cumulative counter, this number will not be just y; all corona cases which have occurred since the beginning of time are surely not transmissible today. For ease of understanding, let us assume for nowthat all cases remain at large and that they stay transmissible for a duration of τ days before they recover. In reality, the recovery duration shows a spread around a mean value; for a lumped parameter model we must ignore the former and retain the latter. Then, the disease spreaders today will be exactly all those who have contracted the disease at any time between now and τ days back. Anyone else, who has contracted the virus more than τ days ago, has recovered by now and is no longer capable of spreading.The number of people who have contracted the disease within the last τ days is given by the total count now minus the total count τ days back, i.e., it is $y(t)-y(t-\tau)$. Here is the delay term which is at the heart of our proposed equation.

Having obtained the $y-y_d$ (d standing for delay) structure, we now relax the assumption which we had made for pedagogical purposes. Let a fraction μ_1 (between 0 and 1) of all cases be asymptomatic, who remain infectious for τ_1 days where τ_1 is the asymptomatic infection period. The fraction $1-\mu_1$of cases are symptomatic. Let τ_2 be the latency period i.e. the time for which these cases remain transmissible before manifesting symptoms, at which point they isolate themselves from society (we ignore the ones who don't). In this Article, we treat the incubation period to be equal to the latency period i.e. we treat as zerothe interval between exposure and the start of transmissibility (justification shortly). Finally, we incorporate the fact that the authorities conduct contact tracing drive. We assume here that this driveis instantaneous and goes forward starting from newly reportingsymptomatic cases. Itcapturespatients who were exposed to the new case right when s/heturned transmissibleτ_2 days ago, as well as patients who were exposed to the new case just before s/he reported for quarantine; the average duration for which these secondary patients have remained at largeis $\tau_2/2$, irrespective of whether they are symptomatic or asymptomatic.We note that the assumption of instantaneous contact tracing introduces an error opposite to that of zero non-transmissible incubation period – the former assumption reduces the time for which the contacts remain at large while the latter increases this time. Here we let these two effects just cancel, instead of introducing two additional parameters. The non-zero incubation period also shifts the overall epidemiological curve by a few days [7], which is an ignorable effect on the time-scale of the epidemic as a whole.Let a fraction μ_3 (between 0 and 1) of all cases escape from contact tracing drives while the fraction $1-\mu_3$get caught. Thus, we have threeclasses of at large cases : (a)fraction $1-\mu_3$ are contact-traced cases who remain at large for half the latency period$\tau_2/2$,(b) fraction $\mu_3(1-\mu_1)$ are untraced symptomatic cases who remain at large for the entire latency periodτ_2, and (c) fraction $\mu_3\mu_1$are untraced asymptomatic cases who remain at large for the infection periodτ_1. Using the $y-y_d$ argument for each class and adding them up, the total number of at large cases works out to

$$n=(1-\mu_3)\left(y-y(t-\tau_2/2)\right)+\left((1-\mu_1)\mu_3\right)\left(y-y(t-\tau_2)\right)+\mu_1\mu_3\left(y-y(t-\tau_1)\right) \quad . \qquad (1)$$

This is the mathematical form of the third term on the RHS of (0).
Multiplying all three terms on the RHS, simplifyingthe third one algebraically and equating LHS to RHS gives us the retarded logistic equation

$$\frac{\mathrm{d}y}{\mathrm{d}t} = m_0\left[1-\frac{y}{N}\right]\left[y(t)-(1-\mu_3)y(t-\tau_2/2)-(1-\mu_1)\mu_3 y(t-\tau_2)-\mu_1\mu_3 y(t-\tau_1)\right] \quad (2)$$

.

This equation is the model we propose for the spread of COVID-19. We now return to the issue of the approximation in the 1-y/N term. Ideally, we should have taken an average recovery time τ_3 and used $y(t-\tau_3)$ in the numerator instead of y. We did not do that for two reasons. The main reason is that (2) has the very important property that $\dot{y}=0$ if $y=N$, i.e. the epidemic identically stops the moment everyone has been infected. Preserving this property while using a delayed y would have forced us to introduce a separate category of quarantined cases etc merely to take care of the end behaviour. The second reason for our choice is that, in a long-haul epidemic which COVID-19 is turning out to be, the recovery period is significantly shorter than the overall course of the disease, so the error involved in replacing $y(t-\tau_3)$ by y will be rather small. Lumped parameter models (or any other model for that matter) are approximations of reality anyway, and this small effect did not induce us to either bring ina whole new variable or (worse) sacrifice the highly desirable property of mathematically stopping the disease when everyone has caught it.

II. DATA FITS FOR HOTSPOT REGIONS

In this Section we perform data fits for Italy and New York State, USA to our model, to demonstrate its credibility. The details of the fitting process can become very boring reading even though they are very necessary, so we first give a quick summary. The recorded case histories in these regionsespecially in the pre-peak regime feature gross undercounting on account of the scarcity of testing facilities and the high numbers of asymptomatic cases which go undetected. Hence, we use the much more reliable death histories to extrapolate the actual case histories and then attempt a fit of the data to (2) using suitable parameter values. For both regions, we consider only the period of full lockdown since m_0 is expected to be approximately constant then. We find good fits in both regions for the following approximate parameter values : m_0 slightly less than 1/4,μ_1 approximately 75 percent, μ_3 approximately 80 percent, and N approximately 8millions which corresponds to a sizeable fraction of the population of boththe New York Metropolitan area and Lombardy, Italy, where the maximum cases are concentrated.In the absence of detailed data from local medical and government authorities, the parameter space is over-rich which makes the fits non-unique; neverthelesswe believe that good fits for physically plausible values of the individual parameterslend credence to our model. The rest of this Section is just amplification of these points and can be skipped without loss of continuity.

For New York State, we have collected the daily reported case and death data over the period 01 March to 27 May 2020. We now assume that the mortality rate is constant and that the time to death is constant at 20 days[15]. In both regions, we find a mere 4 to 5 day interval between the peaks in reported cases and deaths, implying that the case histories are unreliable. So, weassume that the true case counts are the death counts multiplied by some number α (the inverse of the mortality rate), andshifted back in time by 20 days. We treat α as unknown, to be determined during fitting. We then assume that, when the reported peak in case counts is reached, all symptomatic cases are being accounted for. This is because, by our hypothesis, the climb in cases prior to the peak is only an artefact of the limited but increasing testing capacity, and in such a situation, symptomatic cases will get precedence in testing. The reported peak will be attained just when all new symptomatic cases can be accounted for, and thereafter the increased testing capacity can be used to start detecting asymptomatics also. Given α, this assumption enables us to determine μ_1.

Full lockdown was imposed in New York State on 22 March 2020, which corresponds to the 22nd day of the data. Since $m_0$will be fluctuating wildly prior to the lockdown, we start running (2) after the lockdown. The DDE needs to be seeded with an initial function running for the maximum delay involved in the problem, which is 7 days. Accordingly, we treat the assumed actual case counts of the 22nd to the 29th day as the seeding function, joining the discrete available data points by straight lines to create a continuous function. We then adjust the parameters of (2) so that the evolution of y in the subsequent period gives a good fit to the assumed actual case counts. Since these are derived from the death counts, they are available only upto a time of 20 days before the last day of data i.e. upto07 May.

We find a good fit for the values α=100 (mortality rate 1 percent), μ_1=0.7334 (which follows

from the prescription above), μ_3=0.75 (when a region is overwhelmed with cases, contact tracing will not capture too many of them),m_0=0.23 (pretty low, as appropriate for a full lockdown) and N=8×10^6.The plots are given below where the blue lines are derived from the model while the green bars denote the assumed actual case histories.

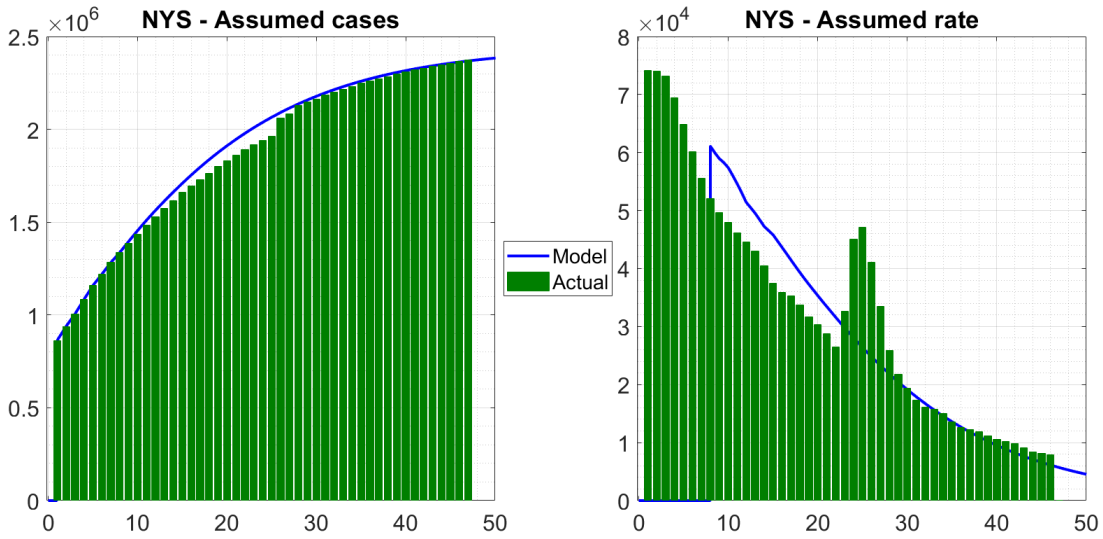

Figure 1 :*Case count and case rate predictions for New York State obtained from (2) compared with assumed actual case histories. Note that the assumed actual counts are derived by extrapolation from the reported death data.*

For Italy, the data starts from 20 February 2020. The lockdown was imposed on 09 March so we again start seeding from the 21st day. The process is identical to what we did for New York.We again fix α=100, which leads to μ_1=0.8183. We assume μ_3=0.9 since Italy was the first country to be hit by this virus so there was no warning and no procedure in place for what should be done. Thereafter, m_0=0.22 and N=8.6×10^{6}produce the fits we give below.

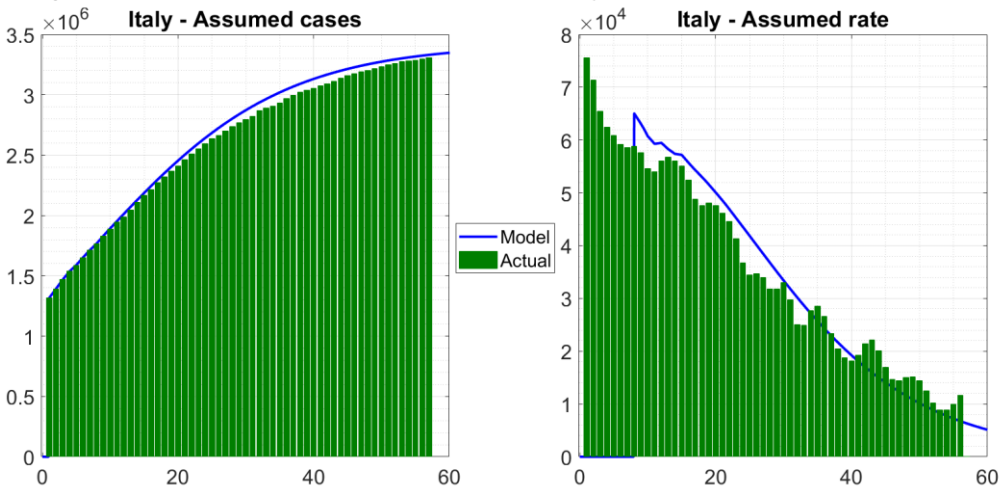

Figure 2 : *Case count and case rate predictions for Italy obtained from (2) compared with assumed actual case histories. Note that the assumed actual counts are derived by extrapolation from the reported death data.*

As we have already mentioned in the summary, the parameter space is multi-dimensional and without insider knowledge (for example of how many patients are falling sick after being detected from a contact tracing drive)there will very likely be excellent fits in an entire region of that space contiguous to the point where we have achieved ours. Hence, the fits should not be taken as proof that the mortality rate is indeed 1 percent or that m_0 during a lockdown is exactly 0.22. However, they should be indicative of the fact that the parameter values are in the right ballpark. Moreover, we get encouragement from two facts. One is that the well-fitting values of $m_0$naturally came out very close in the two regions – we have not doctored anything to make this work. The second is that the values of N came out to 8 million in New York State and 8.6 millionin Italy. In New York State, almost all the cases are concentrated in New York City and the adjoining area. The population of this area is approximately20 million, among which 8 millionseems a reasonable estimate of the susceptible and atlarge fraction. In Italy, about one third of the cases are coming from Lombardy so this part might be given an effective N of about 2.9million. This is again a reasonable fraction of the total 10million population of Lombardy.

Due to all the variables involved in fitting, we have desisted from attempting or presenting fits of more regions. Rather, armed with confidence from these two fits, we now illustrate some general properties of our model (2).

III. COMPARISON WITH CLASSICAL MODELS

Here we present some of the differences between the classical S-I-R style ordinary differential equation (ODE) models and the DDE model (2). We start from the fixed points of (2). As already discussed, $y=N$ is a solution. Inspection reveals that$y$=const. for any constant is a solution as well, since it makes the big box bracket on the RHS vanish.This means that the epidemic can terminate at any level of the disease. This is a significant difference from the ordinary differential equation (ODE) models where the system typically has onlya single non-trivial fixed point, called the endemic equilibrium, where the disease can end. One situation where this feature of ODE models becomes a limitation is when an intervention such as a lockdown is applied for a finite period. Lockdowns etc are modelled through changes in the equation parameter values. In many ODE formulations, the moment the lockdown is lifted and the parameters revert to their original pre-lockdown values, the epidemic gravitates towards its asymptotic pre-lockdown state. This has been pointed out by Raju [16]in a discussion of S-E-I-R model and the effectivity of the lockdown in India. In our DDE approach however, we find (more details in the next Section) that the system retains the memory of a temporary intervention, and such an action can significantly alter the trajectory of the epidemic in the long run.

To find the stability of a constant solution $y=y^*$, we first linearize about this point i.e. we assume that the logistic term is a constant and then carry through the procedure detailed in Ref.[8]. In summary, this procedure consists of positing that the fixed point must be stable for very small delays, postulating that as the delays are increased, the loss of stability must occur through either a Hopfbifuraction or a saddle node bifurcation, showing that the former is impossible, and setting $y=t$as a solution to obtain the stability condition. This yieldsan expression containing y^*; since y^* is arbitrary, we can replace it by $y$and write the stability condition as

$$m_0\left(1-\frac{y}{N}\right)\left(\frac{1+\mu_3-2\mu_1\mu_3}{2}\tau_2+\mu_1\mu_3\tau_1\right)<1 \quad .$$

In other words, the solution $y=y^*$is stable if the above condition holds and unstable otherwise. It is natural to identify the LHS of this condition as the reproduction number R.

We would like to highlight that the expression for the reproduction number, and not just the starting value R_0, comes out naturally from our DDE model. The extraction of R_0 from an S-I-R styleODE model has been demonstrated by Diekmann et. al.[17]. These authorsfirst linearize the system about the disease-free equilibrium and then isolate the transmission and transition matrices to obtain R_0. In the ODE formulation, $R_0<1$ corresponds to stability of the disease-free equilibrium while$R_0>1$ corresponds to instability of the said equilibrium. We note that in reality, even when $R_0<1$, introduction of the disease into society does lead to an increase of cases before the epidemic dies out – the system does not return to the disease-free state. This fact is captured by the DDE model, as we will demonstrate shortly. Moreover, the calculation of the instantaneous reproduction number at an arbitrary point of the disease evolution appears to be impossible from the ODE formalism since it requires linearization about a fixed point,and every disease state is not a fixed point of the system.

This direct route to R arises, we believe, on account of our formulating the governing equation

(2) in terms of the number of cases and their rate of spread – concepts which are central to*R*. In the classical ODE formalism, the statement "asymptomatic cases remain transmissible for τ_1 days" gets expressed as $\dot{x} = -x / \tau_1$ (*x*being the number of asymptomatic cases); while this formulation does retain an average transmissibility duration of τ_1 for each case, it also admits many cases who recover much faster and some who recover much slower. This introduces an inherent complication into the issue of exactly how many persons are infected by one case, andmakes the calculation of *R* somewhat indirect.

IV. SOLUTION CLASSES OF (2)

We now demonstrate various classes of solutions of the DDE (2). Although analytical approach using perturbative methods would have been a fascinating exercise, (2) looks quite complicated, so we leave this for a future study. Right now, we use numerical integration to solve (2). The routine is the same as in Refs.[7], [8], which is second order Runge Kutta with a time step of 1/1000 day.To demonstrate the solution classes, we consider a Notional City having an initial susceptible population of 3,00,000, fraction of asymptomatic carriers 80 percent(this is the maximum value we found in the Literature[18], [19]), τ_1=7 days and τ_2=3 days[15].The initial condition is zero cases to start with and 100 cases/ day for the first seven days (which is the maximum delay involved in the problem and hence the length of the initial function).

We first consider Notional City A having the parameter values m_0=0.23 and μ_3=1/2, corresponding to moderately effective contact tracing in a hard lockdown. This gives a starting *R* of 0.886.Predictably enough, the epidemic burns itself out[8], as shown below.In this and subsequent plots, we show three things in the same plot – the cumulative case count *y*(*t*) as a blue line, its derivative $\dot{y}(t)$ as a green line and the weekly increments in cases, or the "epi-curve", as a grey bar chart. For better visibility we have scaled down the weekly increments by a factor of 7. We report the rates on the left hand side *y*-axis and the cumulative cases on the right hand side *y*-axis.

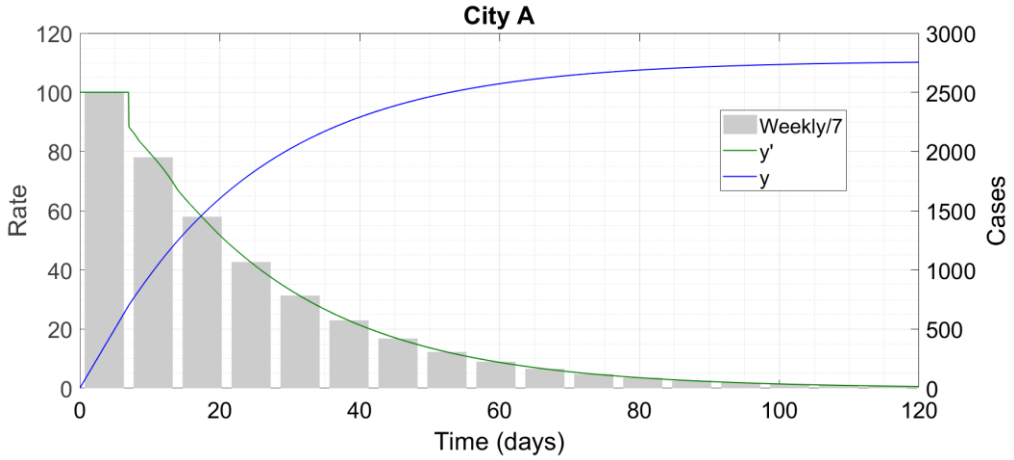

Figure 3 : *City A proceeds to self-burnout right from the start.*

We can see that the rate decreases monotonically; the maximum rate is 100 cases/day, during the seeding period. The country of New Zealand has indeed demonstrated this behaviour in practice, showing it to be composed entirely of cities of Type A. It is noteworthy that this solution does not exist in the S-E-I-R model, as highlighted by Raju op. cit.[16]. It is also qualitatively and quantitatively different from the return to the disease-free equilibrium (*S*=*N*, *E*=*I*=*R*=0)which happens in an S-E-I-R model when R_0<1.

In Notional City B, everything remains the same except that the contact tracing is less effective :μ_3(the escape fraction) is 75 percent.This gives a starting *R* of 1.16, which reduces to unity at *y*=40500 cases.

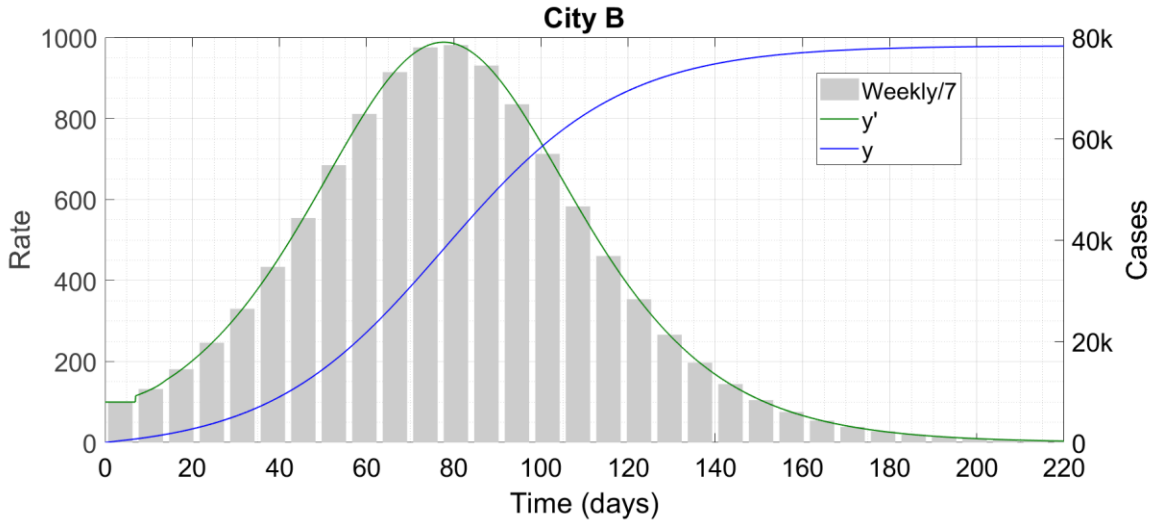

Figure 5 : *City B grows at first before reaching burnout.The symbol 'k' denotes thousand.*

We can see that the outbreak takes off quickly, entering exponential growth right after being released. As *y*increases, *R*gradually reduces so the growth slows down until it peaks when the case count is about 39,000. This is very close to the point where our analytical *R* crosses the unity threshold, and from that point onwards, the epidemic proceeds along a decelerating path to self-burnout. The overall progression of the disease is extremely protracted with the time to the end being almost double of the previous case. However, the small size of the peak should prevent overwhelming of City B's healthcare facilities and thus avoid unnecessary deaths.Cities such as Delhi and Mumbai in Indiaand Los Angeles in USA, where the disease slowly proliferated despite hard lockdowns, very likely belong to this category.

We nowconsider Notional City C which has the same parameters as City B except that $m_0$is 0.5. This means that the state of lockdown is replaced by a state of much milderrestriction on mobility. The starting *R* is above 2.5 and it comes down to unity only at 1,80,000 infections.

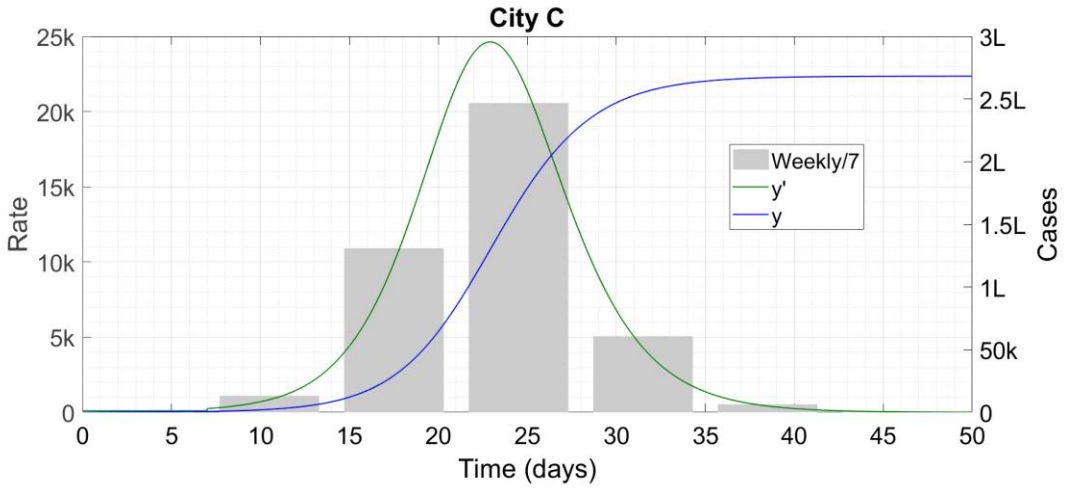

Figure 6 : *City C proceeds all the way to herd immunity.The symbol 'k' denotes thousand and 'L' hundred thousand.*

This time, the disease explodes like an atomic bomb, tearing through almost the entire population in a very short period and presumably leaving behind a trail of dead bodies in its wake.

Notional City D is a combination of B and C. This city starts off with m_0=0.5 like City C but then senses trouble and cuts down instantaneously to m_0=0.23 like City B when the number of cases becomes 40,000.

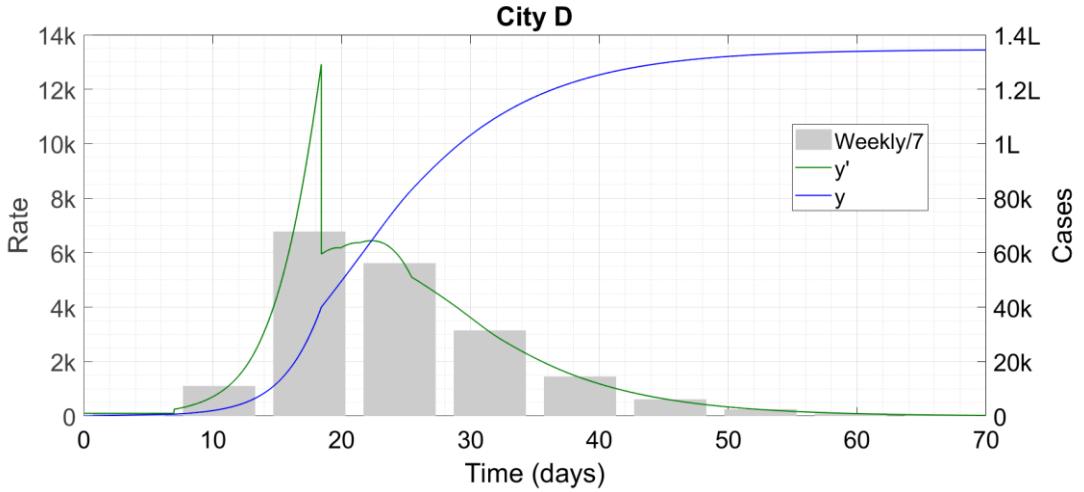

Figure 7 : *City D is a mixture of B and C.The symbol 'k' denotes thousand and 'L' hundred thousand.*

The sudden lockdown throws boron onto the atom bomb before it can reach full detonation power. The ultimate upshot is about 70 percent more cases than City B but a significantly shorter time to the end of the outbreak, which can offset the higher case cost, depending on the urgency of the external economic and other conditions. However, the peak rate of 12,920 cases/day is still very high and likely to overstress all but the world's most advanced healthcare systems.New York City very likely belongs to this class.

Now we consider two cities which are trying to reopen after a lockdown. In both these cities, we start things off with the parameter values of the self-burnout City A. Then, the city reopens on the 80th day by increasing m_0 from 0.23 to 0.5, and simultaneously decreasing μ_3. In City E, μ_3=0.1 after reopening i.e. 90 percent of all cases get detected and isolated through contact tracing when the reopening is achieved. This generates an R of 0.985 on the 80th day; here is the time trace of evolution.

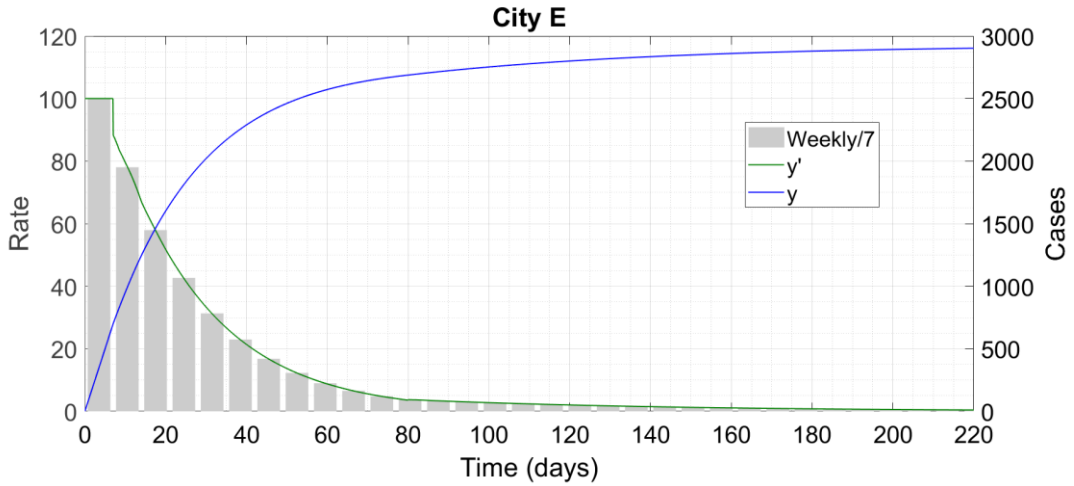

Figure 8 : *Like City A, City E also burns the epidemic out.*

The sudden increase in R at the reopening increases the time to burnout.Indeed, the time taken is double that of City A. However, this excess time comes as the flip side of much higher social mobility, and thereis very little extra case cost. Thus, the lockdown has been extremely effective, allowing retardation of the disease while the contact tracing capacity was being built up. Another important feature of the plot is that, since R<1 throughout, there is no increase in the case rate at any stage.

City F is almost the same as City E, but for the fact that the post-reopening μ_3 is 0.2; 80 instead of 90 percent of cases are captured through contact tracing. This raises the R at reopening to 1.22, which comes down to unity only at about 53,000 infections. The time trace of evolution is below.

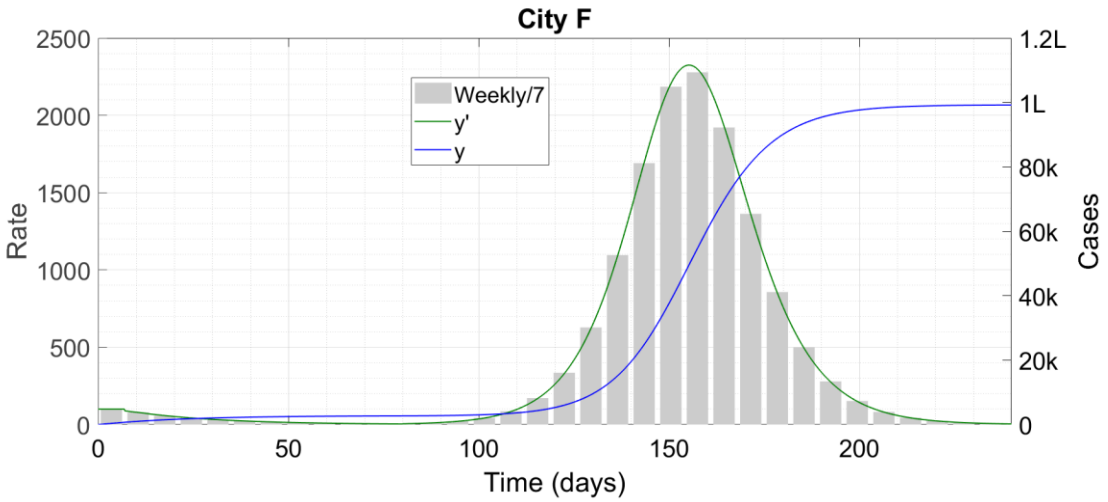

Figure 9 : *After reopening, City F becomes similar to City B. The symbol 'k' denotes thousand and 'L' hundred thousand.*

This is a quintessential reopening disaster – the contact tracing after the reopening was not sufficient to sustain the higher mobility level, and the cases ballooned in a second wave after the 80th day, just as in City B. The 80 days of full lockdown at the beginningended up being completely futile and it would have been better to go with the post-lockdown parameter values right from the start.In practice however, the incipient growth in case rate which occurs when the lockdown is released should at once indicate to the authorities that R has gone up; accordingly the reopening measures should immediately be reversed if such a move is at all compatible with economic and other external constraints.

Finally, we note that if the susceptible population N of each city is varied while keeping all other parameters constant, then the graph heights scale almost in exact proportion to N, and the epidemic durations vary by only minuscule amounts.

V. IMPLICATIONS OF THE SOLUTIONS

We first explain the concept of partial herd immunity. Notional City Bfrom the previous Section is the best demonstrator of this. Here, the epidemic was started off in unfavourable conditions and entered exponential growth as we would expect. However,as it grew, herd immunity started kicking in, to an extent that the disease entered a stable zone (R<1) when only 13.5 percent of the total susceptible population was infected. From this point onwards, it got extinguished in time like the self-burnout City A, infecting another approximately 13 percent people in the process. Thus, in this example, herd immunity worked in concert with public health measures to achieve a halt to the epidemic with only 26 percent of the population infected, which is significantly lower than the conventional herd immunity threshold of 70-90 percent [20]. This is what we call partial herd immunity.Britton et. al. [21]and Peterson et. al.[22]both find lower herd immunity thresholds than the conventional value; we believe that our work explains these findings.Note that with the social restrictions off, partial herd immunity does not work any longer, as in City C, where the disease goes all the way upto 90 percent infection level.

A feature which (3) shares with the ODE models is that it does not have a solution y which is linear in time, i.e. where the case rate is constant, except unless R=1 identically which is a marginal condition. Yet, we have been seeing linear case profile for extended periods in multiple countries and regions such as Canada, Sweden, California in USA and Maharashtra in India – still more examples are given in Verma et. al.[23]. One factor which can contribute to explaining these growths is limitations in testing capacity. A second – and completely different – explanation has been given by Thurner et. al. [24]. They have used an agent-based model andfor some properties of the network they have found solutions which grow linearly until they stop abruptly. This is an example of the potentially higher accuracy of agent-based models relative to differential equation models. We hasten to add however that on the whole,Thurner et. al.'s solutions are in excellent agreement with what we have got for Cities A to D – where they find progression to herd immunity, so do we; where they have linear growth till an abrupt stop, we have a gentle S-shape to a smooth stop. After a point, a reality of this nature (unlike a law of physics) is not governed by

mathematics – any model is at best a representation and a simplification.

We believe that equations having the basic structure (2) with different parameter values to account for the different lockdown and contact tracing situations, combined with anomalies arising from testing capacity limitations, can explain the case histories of Coronavirus all over the world.Naturally, the model (2) can also be used for predictive purposes. Once a case history is obtained, auxiliary parameters such as required hospitalization capacity, death count, PPE requirements etc can also be calculated without too much difficulty. The simple structure of (2), relative to models such as SIDHARTE[25]and extended SEIR[26],makes it easier to work with while producing equally,ormore,detailed results. It is also interesting that the governing equation is in some sense a small variation on the original logistic equation, which has frequently been used for modelingcorona dynamics[27], [28].Whereas these works often appear overly simplified, the addition of the delay enables us to encapsulate a lot of the realistic phenomena associated with the transmission of this virus.

Of course, (2) does have scope for refinement, for example the following :

- Accounting for the latency and incubation period separately instead of in one lumped delay τ_2
- Accounting for bi-directional contact tracing and not just forward from symptomatic patients
- Accounting for the time taken in contact tracing and for intentional violations of quarantine by irresponsible citizens
- Accounting for temporary quarantine of healthy people on grounds of suspected exposure
- Accounting for multiple population categories with varying degrees of susceptibility and risk

Here we have not attempted these refinements since they will destroy the simple structure of (2) and introduce a plethora of additional parameters of whose values we are currently ignorant. But,whatever the modifications made, the basic structure of a logistic term multiplying some$y\square_{\square}y_d$ terms should remain intact, and this can help us to understand and forecast the dynamics of the virus all over the world.

An interesting issue we have observed is that the case trajectories in a region are extremely sensitive to the parameter values in the logistic DDE (2). This adds to the unpredictability of what is already an emerging infectious disease with almost everything unknown. Parameter sensitivity is best illustrated by comparisons between the Notional Cities of the previous Section. For example, to get from City A to B, all we did was increase by 50 percent the fraction of people who escaped the contact-tracers' net. The result was a 30 times (not 30 percent!) increase in the total number of cases. Similarly, the difference between Cities B and D is an 11-day delay (recall that the first seven days in the plots are the seeding period, so they don't count) in imposing the lockdown in D. 11 days out of a 200-plus-day run might appear insignificant. But, that was enough to create tens of thousands of additional cases, risk overstressing healthcare systems and at the same time shorten the epidemic duration by a factor of three. To take yet another example, the difference between Cities E and F is 10 percent more cases failing to get quarantined in the latter city after reopening. That alone has caused 40 times more cases.

Not all equations are so sensitive to changes in parameter values. For example, if the spread of Coronavirus had been governed by the (non-retarded) logistic equation $\dot{y} = ay - by^2$ with a and b positive, then the stable fixed point would have been $y^*=a/b$. The asymptotic case counts would have been linear in the parameters instead of depending on them in such a complex manner. Since the parameter values in any region are determined primarilyby the public health intervention measures, this extreme sensitivity implies that two regions differing only marginally in terms of interventions can differ drastically in terms of case histories. Value judgements made on the basis ofregional case counts alone are thus not scientifically grounded.

Further uncertainty comes from the fact that the parameter values are changing constantly. For example, the reported fraction of asymptomatic carriers has increased continuously over the last three months or so[29]–[32]. Considering the sensitivity of this or any other model to parameter values, such changes can completely invalidate the results of a model as well as any decision which was made on their basis. The contact tracing parameter μ_3, on which so much rests, is also extremely variable. Identifying potential exposures is much easier in a smaller city than a large or densely populated one.Factors such as race also influence a person's susceptibility and/or transmissibility – the details are completelyunknown[9], [33]. Another grey zone is the mutations which this new and vicious virus are undergoing and what effect they might have on the spreading dynamics. Some reports also reflect that the change in genetic composition due to mutation might be the reason behind huge differences in the crude infection rate between countries [34], [35].What this discussion shows more than anything else is the extreme

unpredictability of the case trajectories and the need for solidarity and unity in the fight against corona.

In conclusion, we have proposed the retarded logistic equation (2) as a universal model governing the spread of COVID-19. We have presented some advantages of this model relative to conventional models and have demonstrateda wide variety of solutions which the model can exhibit. We hope that our work proves useful to scientists who are trying to forecast the spread of the disease.

REFERENCES

[1]. W. O. Kermack and A. G. McKendrick, "A Contribution to the Mathematical Theory of Epidemics," *Proc. R. Soc. A Math. Phys. Eng. Sci.*, vol. 115, no. 772, pp. 700–721, 1927.

[2]. A. Dhar, "A critique of the Covid-19 analysis for India by Singh and Adhikari," pp. 1–9, 2020, [Online]. Available: http://arxiv.org/abs/2004.05373.

[3]. S. Jahedi and J. A. Yorke, "When the best pandemic models are the simplest .," *medRxiv*, pp. 1–22, 2020, doi: https://doi.org/10.1101/2020.06.23.20132522.

[4]. L. Dell'Anna, "Solvable delay model for epidemic spreading: the case of Covid-19 in Italy," 2020, [Online]. Available: http://arxiv.org/abs/2003.13571.

[5]. J. Mendenez, "Elementary time-delay dynamics of COVID-19 disease," *Medrxiv*, pp. 1–4, 2020, doi: https://doi.org/10.1101/2020.03.27.20045328.

[6]. A. K. Gupta, N. Sharma, and A. K. Verma, "Spatial Network based model forecasting transmission and control of COVID-19," *medRxiv*, p. 2020.05.06.20092858, 2020, doi: 10.1101/2020.05.06.20092858.

[7]. B. Shayak, M. M. Sharma, R. H. Rand, A. K. Singh, and A. Misra, "Transmission Dynamics of COVID-19 and Impact on Public Health Policy," *medRxiv*, p. 2020.03.29.20047035, 2020, doi: 10.1101/2020.03.29.20047035.

[8]. B. Shayak and R. H. Rand, "Self-burnout - A New Path to the End of COVID-19," *medRxiv*, pp. 1–14, 2020, doi: https://doi.org/10.1101/2020.04.17.20069443.

[9]. B. Shayak and M. M. Sharma, "Retarded Logistic Equation as a Universal Dynamic Model for the Spread of COVID-19," *medRxiv*, pp. 1–27, 2020, doi: 10.1101/2020.06.09.20126573.

[10]. S. McCamon, "13 USS Roosevelt Sailors Test Positive For COVID-19, Again."

[11]. Y. Saplakoglu, "coronavirus-reinfections-were-false-positives." [Online]. Available: https://www.livescience.com/coronavirus-reinfections-were-false-positives.html.

[12]. D. C. Ackerly, "Getting COVID-19 twice." VOX, [Online]. Available: https://www.vox.com/2020/7/12/21321653/getting-covid-19-twice-reinfection-antibody-herd-immunity.

[13]. A. Wajnberg *et al.*, "SARS-CoV-2 infection induces robust, neutralizing antibody responses that are stable for at least three months," *medRxiv*, 2020, doi: https://doi.org/10.1101/2020.07.14.20151126.

[14]. A. W. D. Edridge *et al.*, "Human coronavirus reinfection dynamics: lessons for SARS-CoV-2," *Medrxiv*, pp. 1–10, 2020, doi: 10.1101/2020.05.11.20086439.

[15]. M. L. Childs *et al.*, "The impact of long-term non-pharmaceutical interventions on COVID-19 epidemic dynamics and control," *medRxiv*, vol. 22, p. 2020.05.03.20089078, 2020, doi: 10.1101/2020.05.03.20089078.

[16]. S. Raju, "Has the Indian Lockdown Averted Deaths?," *SSRN Electron. J.*, pp. 0–22, 2020, doi: 10.2139/ssrn.3630409.

[17]. O. Diekmann, J. A. P. Heesterbeek, and M. G. Roberts, "The construction of next-generation matrices for compartmental epidemic models," *J. R. Soc. Interface*, vol. 7, no. 47, pp. 873–885, 2010, doi: 10.1098/rsif.2009.0386.

[18]. "Delhi CM says COVID-19 deaths very less." Times of India, [Online]. Available: https://timesofindia.indiatimes.com/city/delhi/delhi-cm-says-covid-19-deaths-very-less-but-75pc-cases-asymptomatic-or-showing-mild-symptoms/articleshow/75658636.cms.

[19]. "Taking over hospital beds, conducting survey." New Indain Express, [Online]. Available: https://www.newindianexpress.com/nation/2020/may/30/taking-over-hospital-beds-conducting-survey-uddhav-government-goes-after-covid-19-as-state-tally-c-2149989.html.

[20]. G. A. D'Souza and D. Dowdy, "What is herd immunity and how we can achieve it with COVID-19 ?" [Online]. Available: https://www.jhsph.edu/covid-19/articles/achieving-herd-immunity-with-covid19.html.

[21]. T. Britton, F. Ball, and P. Trapman, "The disease-induced herd immunity level for Covid-19 is substantially lower than the classical herd immunity level," pp. 1–15, 2020, [Online]. Available: http://arxiv.org/abs/2005.03085.

[22]. A. A. Peterson, C. F. Goldsmith, C. Rose, A. J. Medford, and T. Vegge, "Should the rate term in the basic epidemiology models be

second-order?," 2020, [Online]. Available: http://arxiv.org/abs/2005.04704.

[23]. M. K. Verma, A. Asad, and S. Chatterjee, "COVID-19 pandemic: Power law spread and flattening of the curve," *medRxiv*, doi: 10.1101/2020.04.02.20051680.

[24]. S. Thurner, P. Klimek, and R. Hanel, "Why are most COVID-19 infection curves linear?," vol. 1, pp. 1–9, 2020, [Online]. Available: http://arxiv.org/abs/2005.11302.

[25]. G. Giordano *et al.*, "A SIDARTHE Model of COVID-19 Epidemic in Italy," 2020, doi: 10.1038/s41591-020-0883-7.

[26]. A. Das, A. Dhar, S. Goyal, and A. Kundu, "Covid-19: an analysis of an extended SEIR model and a comparison of different intervention strategies," 2020, [Online]. Available: http://arxiv.org/abs/2005.11511.

[27]. G. Vattay, "Predicting the ultimate outcome of the COVID-19 outbreak in Italy," no. 1, pp. 1–4, 2020, [Online]. Available: http://arxiv.org/abs/2003.07912.

[28]. E. M. Koltsova, E. S. Kurkina, and A. M. Vasetsky, "Mathematical modeling of the spread of COVID-19 in Moscow and Russian regions."

[29]. K. Mizumoto, K. Kagaya, A. Zarebski, and G. Chowell, "Estimating the asymptomatic proportion of coronavirus disease 2019 (COVID-19) cases on board the Diamond Princess cruise ship, Yokohama, Japan, 2020," *Eurosurveillance*, vol. 25, no. 10, pp. 1–5, 2020, doi: 10.2807/1560-7917.ES.2020.25.10.2000180.

[30]. E. Lavezzo *et al.*, "Suppression of COVID-19 outbreak in the municipality of Vo, Italy," *medRxiv*, no. Ci, p. 2020.04.17.20053157, 2020, doi: 10.1101/2020.04.17.20053157.

[31]. "71 percent of patients in Maharashtra are asymptomatic." Mumbai Mirror, [Online]. Available: https://mumbaimirror.indiatimes.com/coronavirus/news/covid-19-71-of-patients-in-maharashtra-are-asymptomatic-mumbai-cases-at-16579/articleshow/75754328.cms.

[32]. A. Pal and J. K. Bhattacharjee, "The hidden variable in the dynamics of transmission of COVID-19 : a Henon map approach," *medRxiv*, 2020, doi: https://doi.org/10.1101/2020.06.02.20119859.

[33]. C. DiMaggio, M. Klein, C. Berry, and S. Frangos, "Blacks/African Americans are 5 Times More Likely to Develop COVID-19: Spatial Modeling of New York City ZIP Code-level Testing Results," *medRxiv*, p. 2020.05.14.20101691, 2020, doi: 10.1101/2020.05.14.20101691.

[34]. L. van Dorp *et al.*, "Emergence of genomic diversity and recurrent mutations in SARS-CoV-2," *Infect. Genet. Evol.*, vol. 83, no. April, p. 104351, 2020, doi: 10.1016/j.meegid.2020.104351.

[35]. H. Ellyatt, "Coronavirus no longer exists clinically - controversy." CNBC, [Online]. Available: https://www.cnbc.com/2020/06/02/claim-coronavirus-no-longer-exists-provokes-controversy.html.

B SHAYAK, et. al. "A Delay Differential Equation Model for the Spread of COVID-19." *International Journal of Engineering Research and Applications (IJERA)*, vol.10 (10), 2020, pp 01-13.