A Hawkes process model for the propagation of COVID-19: simple analytical results

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Abstract – We present a model for the COVID-19 epidemic that offers analytical expressions for the newly registered and latent cases. This model is based on an epidemic branching process with latency that is greatly simplified when the bare memory kernel is given by an exponential function as observed in this pandemic. We expose the futility of the concept of "reaching the peak" of the epidemic as long as the number of latent cases is not depleted. Our model offers the possibility of laying out different scenarios for the evolution of the epidemic in different countries based on the most recent observations and in terms of only two constants obtained from clinical trials. Furthermore, by analyzing the number of registered new deaths, our model suggests that the recent surge in new COVID-19 cases in the USA is a consequence of an increase in testing, but only up to the second week of June of 2020.

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Introduction. – For the past six months the COVID-19 pandemic has ravaged the entire world bringing with it devastating human and economic losses. Enormous efforts have been undertaken to predict the evolution of this disease, in particular to quantify how different degrees of social distancing and travel restrictions may be affecting it [1-3]. However, partly because it is extraordinarily difficult to predict people's behavior, many models that can excel at *explaining* what has happened, tend to have a harder time *predicting* the evolution of the epidemic [4] except over short times. For example, SIR-type models [5]—that compartmentalize a population and lets its components evolve though a group of coupled differential equations— are highly sensitive to both initial conditions and parameters which makes them unreliable as tools to predict, for instance, when the peak of the pandemic will occur [6]. Failing to predict this turning point has become a common source of frustration because public opinion in some countries seems to have equated reaching the peak of the epidemic to having "tamed" it. This evidences a general lack of feeling of how safe it is to ease social distancing restrictions necessary to reopen the economy given the information available to the public, which in most countries consists of the daily number of new cases and new deaths as well as the number tests being performed. Besides SIR-type models, other valuable and effective approaches consist of agent-based simulations that incorporate census and mobility data [7, 1]. These models lay out different scenarios depending on the degree of compliance of social distancing measures. But in either case, the exact data being used as well as the assumptions

and algorithms are not always available to the public or straightforward to understand, let alone reproduce [4].

In this work we develop a simple epidemiologic model for the propagation of the current COVID-19 pandemic that is amenable to analytical solutions easy to implement. Our model evidences that reaching the peak is not as relevant as depleting the number of latent cases, while providing simple expressions for this quantity from publicly available data. We also apply our model to decouple the effects of a higher rate of testing and lower death-rates (brought about by new medical treatments) from the number of registered new cases.

The model. – Figure 1 shows the time series of the weekly new cases (red dots) for four different countries that have handled the epidemic very differently along with the number of latent cases (solid blue traces) calculated with our model. To construct it, we make use of the fact that in those countries in which the peak of a first outbreak has been reached, the relaxation of this dynamics is well described by a decaying exponential (insets of Figs. 1a and 1b). Epidemic processes for which the analytic form of the relaxation dynamics is known can be conveniently modeled by the so-called self-excited Hawkes conditional Poisson processes [8]. These processes are composed of two main ingredients: the first one is the "branching ratio" that accounts for contagion.
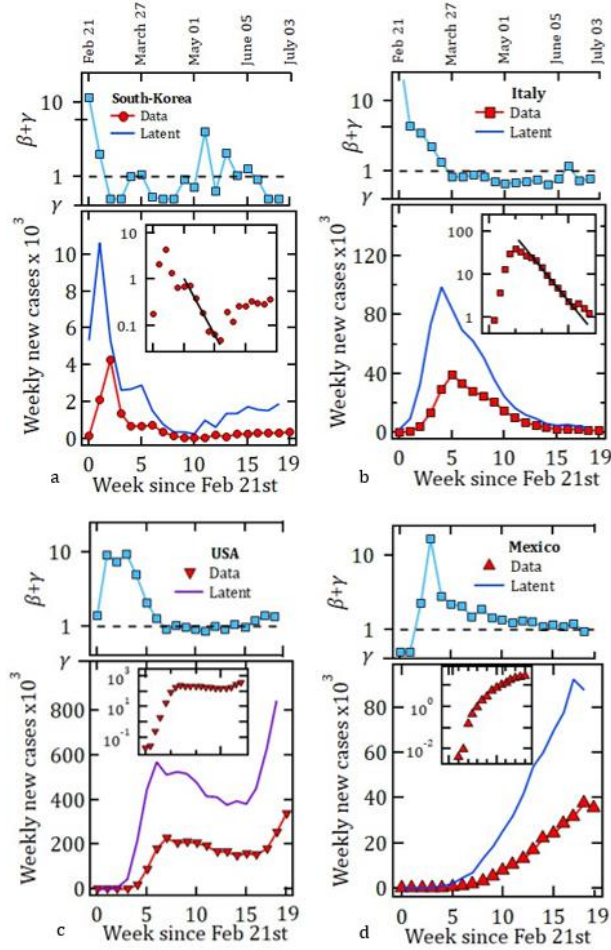
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Fig. 1: Weekly registered new cases (dots) and calculated latent ones (Eq. 3), along with the corresponding reproduction numbers $R_i=\beta_i+\gamma$ (Eq. 4) for (a) South Korea, (b) Italy, (c) USA and (d) Mexico. See main text for details. Insets show the weekly new cases in log-linear scale. The solid black line in (a) depicts an exponentially decaying activity (Eq. 5 with $\beta=0$) setting $\gamma = 0.5$ from clinical studies (see Methods in Supp. Mat.). In the inset of b), the solid line is the best fit to Eq. 5 ($\beta=0.194 \pm.004$) assuming $\gamma =0.5$ as before.

The second one is a latency function—also known as the bare memory kernel— that incorporates the fact that there is a statistical lag between contagion and the specific action whose aggregated dynamics is recorded. We propose that the weekly new observed or "registered" COVID-19 cases at time t— henceforth called the "activity" and denoted by λ_t— obeys the dynamics from a discrete time Hawkes processes which in general takes the form:

$$\lambda_t = S_t + \sum_{i,t_i \leq t}^{t} \beta_i \varphi_{t-t_i} \qquad (1)$$

where φ_t is the latency function, β_i is the number of people infected of first generation (i.e. directly infected by individual i at time t_i) that will become registered at any future time, and the term S_t represents the exogenous sources of new cases. In the case at hand, these sources are the number of people already infected arriving in some new city [7]. A key feature characterizing Hawkes processes is that when the latency function is given by either a power law —as in the case of many human activities [9-11] and earthquakes [12] — or by a decaying exponential [13], the activity during relaxation is slowed down, but it remains described by the same function. In other words, contagion renormalizes the function that describes the activity in the absence of exogenous sources plus small constant contagion rates. Thus, if the form of the bare memory kernel is known, the relaxation dynamics can give information about the magnitude of the contagion taking place during the relaxation period. In general, the activity derived from Hawkes processes is non-Markovian [14], but in the special case when the bare kernel is given by a decaying exponential, as is the case of the COVID-19 pandemic, the activity at time t depends only on the activity at time t-1 making it amenable to simple analytic solutions that help shed light into the relevant mechanisms at play [13].

The discrete time-step process of Eq. 1 can be understood more clearly with the schematic diagram shown in Fig. 2. We consider an initial number of "patients-zero" λ_{-1} infectious at time $t = -1$ that are exogenously introduced to the population. At time $t = 0$, these individuals will follow one of three paths: they will either 1) be registered with probability α, 2) not be registered but continue being contagious with probability γ, or recover with probability $\rho = 1 - \alpha + \gamma$. The λ_{-1} patients-zero will also infect a number $\beta_0 \lambda_{-1}$ of new individuals at time $t = 0$. In SIR-type models, the factor β is proportional to the so-called "susceptible" portion of the population that gets depleted as the epidemic advances. In the form displayed in Fig. 2, the model we propose here is valid only for the initial propagation of the pandemic since an effectively infinite source of susceptible individuals is considered, but it could be modified to incorporate a finite-sized population. A second important assumption we make is that individuals that have been registered will either self-isolate or be hospitalized but in either case will no longer be a source of new infections. In contrast, those that have not been registered yet can continue infecting others until they finally either recover or fall ill after which they will become registered. This contemplates the fact that asymptomatic individuals have been shown to be an important source of new COVID-19 infections [15] (see, for example, the dashed path in Fig. 2).

Our model can be considered as a simplified version of other more complex epidemiological models that take into account social mobility to be able to relate the reproduction number with confinement measures [2].
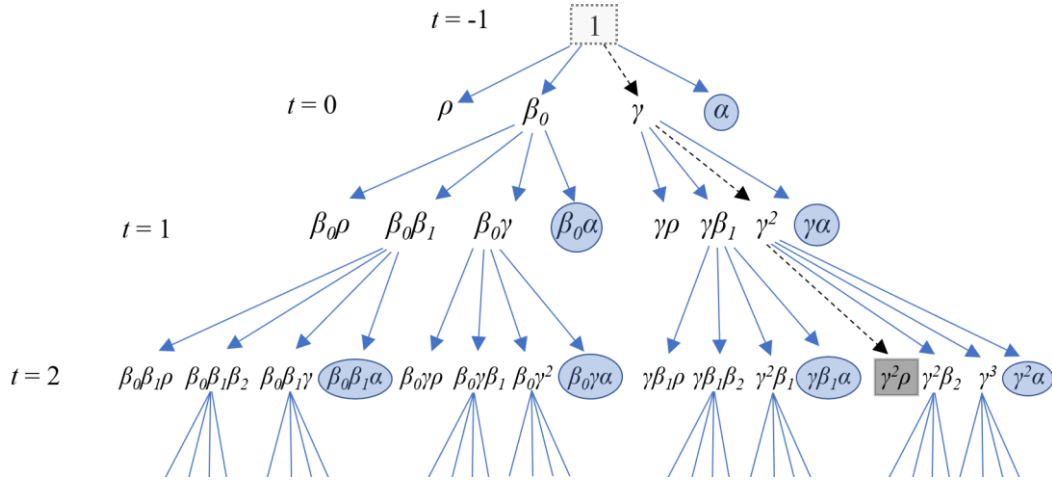
Fig. 2: Hawkes model with an exponential bare memory kernel. A normalized initial population of "patients-zero" on week -1 follows three possible outcomes on the following week given by the fractions: ρ (recovered), γ (still infectious but not registered) and α (registered and isolated). A new generation of infectious individuals is given by β_t times the population that originated it at time t. The sum of elements containing an α (elements inside circles) at a given time gives the total activity (Eq. 1), while the latent cases are the sum of the remaining terms after the recovered ones (elements with a ρ) have been subtracted. A tree is formed by replicating the four possible trajectories for each element that is still infectious at subsequent times. The dashed black arrows ending at the grey square depict the trajectory followed by the portion of the patients-zero that were asymptomatic and recovered at time $t = 2$. Note this symptomatic population infected other individuals before recovering.

The solution to Eq. 1 for the registered new cases in the absence of exogenous sources is

$$\lambda_t = \lambda_{-1}(1-\gamma-\rho)\prod_{k=0}^{t-1}(\gamma+\beta_k) = \lambda_{t-1}(\gamma+\beta_{t-1}). \quad (2)$$

This simple expression for the activity is the consequence of the fact that, for an exponential memory kernel, both contagion and latency effects can be represented as a fraction of previous activity. This property is not shared by processes involving power-law bare memory kernels. The activities at times t and $t+1$ yields the relation:

$$\beta_t = (1-\gamma) + \frac{\lambda_{t-1}-\lambda_t}{\lambda_t} \quad (3)$$

From this equation we can establish the condition for the contagion to either grow ($\beta_t + \gamma > 1$) or die out exponentially ($\beta_t + \gamma < 1$). Thus, we associate $(\beta_t + \gamma)$ with the effective reproduction number R_t at time t. While the collection of reproduction numbers $\{(\beta_0+\gamma),(\beta_1+\gamma),...,(\beta_t+\gamma)\}$ merely constitutes a parametrization of the activity in terms of exponential changes, from it we can construct the time series of the latent cases. From Fig. 2, these latent cases are given by

$$\lambda_{L,t} = \lambda_{-1}\prod_{k=0}^{t}(\gamma+\beta_k) = \lambda_{L,t-1}(\gamma+\beta_t). \quad (4)$$

Knowing $\lambda_{L,t}$ allows us to calculate the number of cases that can still be expected to be registered in the extreme optimistic scenario where one could assure no more contagion events will take place. The number of such individuals is readily calculated as $(1-A)\lambda_{L,t}$, where A is the fraction of

asymptomatic individuals (see Methods section in the Supp. Mat.). This scenario is a particular case of $\beta_i = \beta$ for all i's. In this case, the activity λ_t can be written as

$$\lambda_t = \lambda_{-1}(1-\gamma-\rho)(\beta+\gamma)^t \propto e^{-\frac{t}{\tau}(1-\theta)}, \quad (5)$$

where $\theta \equiv Ln\left(1+\beta\left\{e^{1/\tau}-1\right\}\right)$ and $1/\tau \equiv Ln(\gamma)$. The form of this equation shows explicitly how contagion renormalizes the activity from a decaying exponential with constant $(1/\tau)$ in the absence of contagion, to one with constant $(1/\tau)(1-\theta)$ otherwise. Among other benefits, being able to approximate the number of latent cases with Eq. 5 may help plan for hospital occupancy levels. Furthermore, this information may also help estimate the impact of relaxing measures on new cases as we show ahead.

Results and discussion. – To compare our model with data, we aggregate the registered, publicly available daily new cases of all the regions (or states) of a given country and during a whole week into the observed activity. This means that the step-size of our model is equal to one week, thus smoothing out the daily fluctuations observed in most countries. We are aware that in general the epidemic tends to evolve differently in each region of a given country since states may follow particular social distancing laws. Therefore, in its present formulation, ours can be considered as a mean field model that also ignores the topology of the social network [16] and any mobility factors. According to clinical studies, the average time from contagion to attending a hospital is ≈ 2 weeks [17] from which we obtain $\gamma = 0.5$, or, in terms of the decay constant, $1/\tau \simeq -0.693$. (see Methods section in the Supp. Mat.). The inset of Fig. 1a shows that an exponentially decaying activity

with constant $1/\tau \equiv Ln(1/2) \simeq -0.59$ is in apparent agreement with the trend observed by the activity of South-Korea, suggesting the intense cluster tracing, mass testing, and the imposition of quarantines on people travelling from abroad [18,19] brought the number of new infections close to zero in this country during that period. Similarly, we calculate $\rho=0.1$ after assuming a conservative estimate of 20% of asymptomatic cases [20] (see Methods section in the Supp. Mat.). Fig. 1 shows the latent cases *vs. t* using Eq. 4 and the values for γ and ρ calculated above for four countries that have responded in very different ways to the pandemic. This figure shows how South-Korea (Fig. 1a) managed to stop contagion by week #6 since Feb. 21st (April 3rd/2020), a fact reflected in the reproduction number being closer to γ. Consequently, the number of latent cases also decreased sharply reaching almost the same number of registered cases by week #8 (mid. April/2020). By then, our model shows there were practically no unregistered cases propagating the infection. In the case of Italy (Fig. 1b), where it took longer for the government to impose confinement measures, the number of latent cases reached almost 140,000 by week #4 (March 20th/2020) according to our model. It then took almost 10 weeks since the peak of activity was reached for these latent cases to decay, despite a strict lockdown was enforced around week #4. Using Eq. 5, we estimate the reproduction number in Italy in that period to be $(\beta+\gamma) = 0.694 \pm 0.004$ (see inset of Fig. 1b). After reaching this stage, relaxing some social distance measures necessary to reopen their respective economies has been done with some confidence. Having just a few latent cases is why a second wave of infection has been successfully contained in South-Korea (from week #14, see inset of Fig. 1a). In contrast, for both the USA and Mexico, the number of latent cases as of July 3rd is enormous. For the USA, latent cases simply were not given enough time to decay before a surge of new cases emerged in mid-June/2020. In the case of Mexico, a plateau for the activity has not even been reached yet. This analysis shows that reaching the peak of the epidemy and even having a downwards trend is meaningless in terms of public safety unless such trend lasts long enough as to allow for most of the latent cases to decay. Furthermore, given that the dynamics of Eq. 2 and 4 is Markovian, a sudden change in confinement practices will have strong effects regardless of any apparent trend. It is worth pointing out that trends reflect social behavior and not any kind of inertia or "weakening" of the pathogen itself.

Since the recent second outbreaks experienced by South-Korea, Germany and Australia, it has that become clear that ending the lockdown in countries that have brought the epidemic under control will not be an easy task [21]. Information about changes in β_k for countries that have relaxed their confinement measures can be plugged in directly into Eq. 2 to lay out scenarios for the possible evolution of the epidemic in other countries. For example, Italy brought down its reproduction number to below 1 for 11 consecutive weeks (Fig. 1a) even after the lock down had ended. But on the week #16 (June 26th), this number grew to $\simeq 1.2$, despite reverting to

below 1 on the following two weeks. Over a whole month, $R = 1.2$ brings about a two-fold increase in the number of new cases ($1.2^4 \simeq 2.1$). For countries like Italy, Spain or Portugal that have brought the number of weekly infections to a couple of thousands per week, this would represent a manageable increase. But for the UK, the same scenario translates into 10,000 weekly new cases. In this same line, we can also answer the following question: what could happen if the USA continues to have the same average reproduction number it has been displaying for the last three weeks, $(\gamma+\beta) = (0.5+0.78) \simeq 1.31$ (from June 19th to July 3rd/2020) ? Over a one-month period (by July 31st), this would translate into an increase by a factor of $(1.3)^4 \simeq 2.94$ for the reported new cases, amounting to just shy of 100,000 new cases per day, in agreement with recent warnings. The case of Mexico is just as alarming: we calculate the average reproduction number in the last month (from June 5th to July 3rd/2020) to be 1.1. At this rate, we can expect 53,000 weekly new cases within a month, which is a higher per-capita count than the scenario explored for the USA. Given that in Mexico some restrictions have been recently lifted, this is actually a best-case scenario.

Weekly New Deaths. It has been argued that rather than the number of new registered COVID-19 cases, the number of the new deaths should be used to study the evolution of the epidemic, since the latter tends to be a more reliable indicator of the real number of cases. In this respect, it is worth pointing out that there exist serious concerns regarding undercounting of COVID-19 related deaths as well. However, as we did before for the weekly new cases, we will take the publicly available data for the death-count at face value. As we now show, it is possible to reproduce the number of observed deaths directly from λ_t, and from it obtain clues about the origin of the surge in the number of cases as the one the USA is currently experiencing.

The simplest possible assumption one can make is that the number of new weekly deaths are proportional to the weekly new cases, and allow for a lag of θ weeks,

$$\lambda_{D,(t+\theta)} = c\lambda_t. \qquad (6)$$

Figure 3 shows that Eq. 6 reproduces well the observed new weekly deaths in each of the six examples presented. There, the whole dataset for Italy (Fig. 3a), Mexico (Fig. 3b), Japan (Fig. 3f) and most of the time series for Germany (Fig. 3e), is well reproduced by $\lambda_{D,(t+\theta)}$. However, note that Eq. 6 systematically overestimates the observed new deaths for the USA (Fig. 3c) and Brazil (Fig. 3d), after the weeks ending on May 22nd and May 8th, respectively (vertically dashed lines on the corresponding plots). This may be explained by either a sudden decrease in the death-rate, or by an increase in the number of tests performed. As mentioned above, there exists a current dispute over the origin a surge in new cases after a period of relative stability had been achieved in the USA. One side argues that this is the result of an increase in the rate of infections. Meanwhile, the other side argues this is simply the result of more tests being performed nationwide.
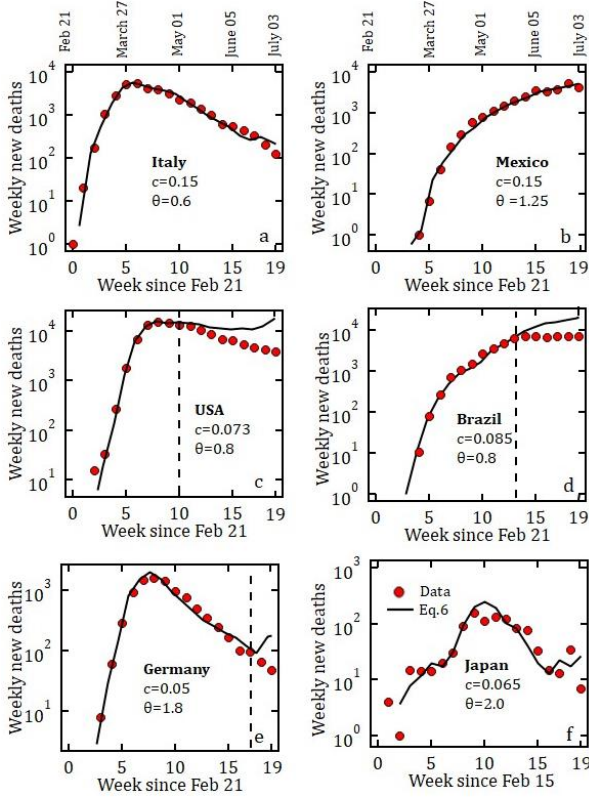
Fig. 3: Weekly new deaths from weekly new cases. Weekly new deaths registered (dots) and the result of Eq. 6 (black trace) for (a) Italy (c=0.15, θ=0.6), (b) Mexico (c=0.15, θ=1.25), (c) the USA (c=0.073, θ=0.8), (d) the Brazil (c=0.085, θ=0.8), (e) Germany (c=0.05, θ=1.8) and (f) Japan (c=0.65, θ=2.0). Dashed lines in c), d) and e) mark the week after which Eq. 6 no longer reproduces the data.

In order to address this issue, we first note that in the construction of the expression for λ_D we have tacitly assumed that the rate of registered cases ending up in death is constant throughout time. This certainly seems to be the case of Italy, Mexico, Japan, and for most of the time series for Germany, but not for the USA and Brazil. This rate can be related to the percentage P_t of COVID-19 tests performed that were positive, as follows. The top panel of Figure 4 shows that after week 5 (end of March/2020) the number of weekly performed tests in the USA increased substantially while the P_t decreased. Presumably, on any given week when P_t =1 (late February 2020) all tests came out positive because they were performed only on people who arrived at the hospital with severe symptoms. Thus, a lower P_t plus a higher testing rate implies that a larger portion of the population is being tested which may now include individuals testing negative. But it may also include asymptomatic cases that will not end up engrossing the death-toll. While the exact dependency of the percentage of new cases that with develop symptoms on P_t is not known, for simplicity we assume it is to be proportional to $P_t{}^a$, where the power a is a positive constant. We incorporate this factor into a similar branching process for the evolution of deceased individuals from the registered ones (see Supp. Mat.). Given a number of individuals who were registered on week t, let ε and δ be the factions that will recover, and continue to be ill on week t+1, respectively. Then, the fraction that on average will die on week t+1 is $1-\delta-\varepsilon \equiv \Delta$, and the number of new deaths at time t from this model is (see Supp. Mat.)

$$\lambda_{D,t} = \lambda_{-1}P_t^a\left(1-\gamma-\rho\right)\Delta\sum_{i=0}^{t-1}\delta^i\prod_{k=0}^{t-i-2}\left(\gamma+\beta_k\right),\qquad(7)$$

where λ_{-1} as well as the collection of β_k's were obtained from the time series for the weekly new cases through Eq. 3, γ=0.5 and ρ=1 as before, and δ=0.5 to make the model self-consistent with respect to the average time necessary for recovery (assuming symptomatic and symptomatic behave similarly in this respect, see Methods section in the Supp. Mat.). The lower panel of Fig. 4 shows that this model (cyan trace, with $\varepsilon = \varepsilon_0 = 0.36$ and $a = 2/3$, see Supp. Mat.) qualitatively reproduces the time series for the weekly new deaths in the USA up to June 5th/2020 (week #15). This supports the theory that the number of new deaths up to that date was the result of more testing and not of a larger number of infections. It also suggests that a new effect has been taking place since week #16 (June 12th/2020) that cannot be explained by an increase in the number of tests. There are several possible explanations for this relatively low number of new deaths, including 1) earlier infection identification, 2) better equipped hospitals, 3) younger average infected population (down from 65 to 35 years old), 4) and better knowledge about how to treat critically ill patients. In this last respect, a breakthrough was announced on June 16th/2020 [22]: a clinical evaluation showed that the mortality rate of COVID-19 hospitalized patients treated with the steroid Dexamethasone was reduced from 24.6% to 21.6% over a 4-week period [23]. This corresponds to an overall 12% decrease in the mortality rate, even though it was estimated to be closer to 33% for critically ill patients. From Eq. 7, the mortality rate for isolated infected individuals ($\gamma = 0$, $\beta_k = 0$) is $1-\left(\varepsilon/1-\delta\right)$ (see Supp. Mat.), from which an increment in ε by 4.6% and 13% from the original ε_0 yields, respectively, 12% and 33% increments in the mortality rate for our model. The black trace of Fig. 4 shows how progressively lowering the mortality rate by these percentages on weeks #16,17 and 18 (from June 12th to June 26th see Fig. 4 caption for details), qualitatively reproduces the data, suggesting that the use of Dexamethasone is what is responsible for the low mortality rates observed during those weeks. Unfortunately, by the same token, this means that the observed surge in cases after June 12th is likely the result of an increase in the number of infections. To be able to reproduce the number of deaths registered on week #19 (last data point, July 3rd/2020) it is necessary to assume a huge decrease in the relative mortality rate of 64% (ε=1.33ε_0) that cannot be explained with the use of this drug alone. Additional factors as the ones we have mentioned above may be behind this.
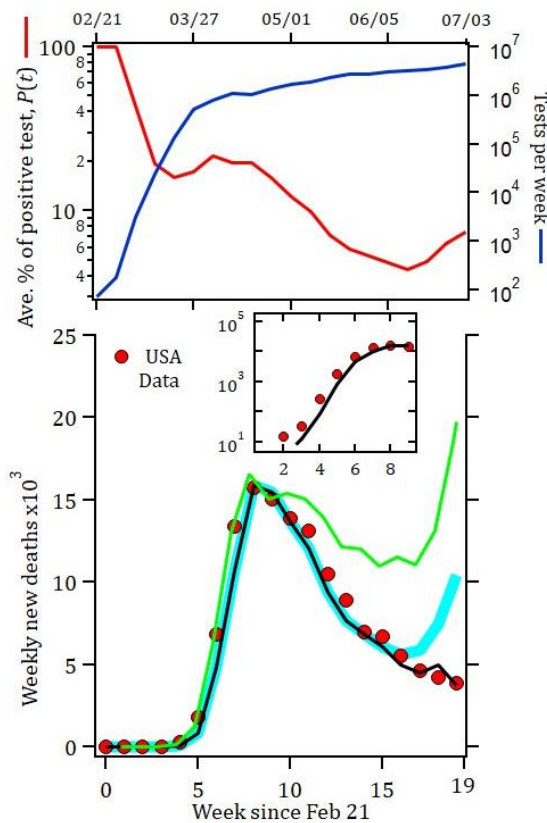
Fig. 4: Models for the number of new deaths in the USA. (Top) Weekly average % of positive tests, P_t (red trace, left axis) and total number of tests per week (blue trace, right axis) for the USA obtained from https://covidtracking.com/data/us-daily. (Bottom) Weekly new deaths (dots) for the USA and the approximation of this quantity assuming 1) a constant death rate (Eq. 6, green trace), 2) a death rate weighted by P_t (Eq. 7, cyan trace), 3) a death rate weighted by P_t with a variable ε for the last four weeks (Eq. 7, black trace) to account for the possible reduction of the death-rate with the use of Dexamethasone [23]. (Inset) Same data but in log-linear scale up to week #9. The values of ε used were $\varepsilon_0 = 0.36$ for the first 15 weeks, and 104%, 109%, 113% and 125% of ε_0 on weeks #16,17,18 and 19, respectively, corresponding to relative decreases in the mortality rate of 12%, 23%, 33% and 64%.

Outlook. – The analysis we have just carried out suggests that a better approximation for the reproduction number could be obtained in general by looking for an agreement between the new cases and the new deaths reported with a model like the one we have presented. This model could be improved further by incorporating the fact that hospitalized patients have been shown to be a source of new infections through contact with medical personnel [24].

We end by emphasizing that the model we have proposed here is only valid for the initial stages of the epidemic. To account for the finite size of a given population, the β_k factors could be made proportional to the product of the susceptible and infected populations as is done in SIR-type models. Also, more precise scenarios than the ones we have laid out in this work could be obtained by atomizing our mean field model to the state or county level if, for example, there exists local information about whether people are following social distancing measures.

The authors declare that there is no conflict of interest regarding the publication of this article

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