Social distancing versus early detection and contacts tracing in epidemic management

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Different countries – and sometimes different regions within the same countries – have adopted different strategies in trying to contain the ongoing COVID-19 epidemic; these mix in variable parts social confinement, early detection and contact tracing. In this paper we discuss the different effects of these ingredients on the epidemic dynamics; the discussion is conducted with the help of two simple models, i.e. the classical SIR model and the recently introduced variant (A-SIR, arXiv:2003.08720) which takes into account the presence of a large set of asymptomatic infectives.

I. INTRODUCTION

Different countries are tackling the ongoing COVID-19 epidemics with different strategies. Awaiting for a vaccine to be available, the three tools at our disposal are *contact tracing*, *early detection* and *social distancing*. These are not mutually exclusive, and in fact they are used together, but the accent may be more on one or the other.

Within the framework of classical SIR [1–5] and SIR-type models, one could say (see below for details) that these strategies aim at changing one or the other of the basic parameters in the model.

In this note we want to study – within this class of models – what are the consequences of acting in these different ways. We are interested not only in the peak of the epidemics, but also in its duration.

In fact, it is everybody's experience in these days that social distancing – with its consequence of stopping all kind of economic activities – has a deep impact on our life, and in the long run is producing impoverishment and thus a decline in living conditions of a large part of population. We all want to survive to COVID, but if we are successful in this it would be better not to die of hunger or of some related illness in the next year.

In the present study we will not specially focus on COVID, but discuss the matter in general terms and by means of general-purpose models.

Our Examples and numerical computations will however use data and parameters applying to the current COVID epidemic in Northern Italy, in order to have realistic examples and figures; we will thus use data and parameters arising from our analysis of epidemiological data in the early phase of this epidemic [6]. Unavoidably, we will also here and there refer to the COVID case.

This work was triggered by the work of M. Cadoni [7] on time scaling in the SIR model; the credit for pointing out the relevance of acting on one or the other of the classical SIR model parameters should go to him.

II. THE SIR MODEL

In the SIR model [1–5], a population of constant size (this means the analysis is valid over a relatively short time-span, or we should consider new births and also deaths not due to the epidemic) is subdivided in three classes: Susceptibles, Infected (and by this also Infectives), and Removed. The infected are supposed to be immediately infective (if this is not the case, one considers so called SEIR model to take into account the delay), and removed may be recovered, or dead, or isolated from contact with susceptibles.

The equations governing the SIR dynamics are nonlinear, and explicitly written as

$$\begin{aligned} dS/dt &= -\alpha\, S\, I \\ dI/dt &= \alpha\, S\, I \;-\; \beta\, I \\ dR/dt &= \beta\, I\ . \end{aligned} \tag{1}$$

These should be considered, in physicists' language, as mean field equations; they hold under the (surely not realistic) assumption that all individuals are equivalent, and that the numbers are sufficiently large to disregard fluctuations around mean quantities.

Note also that the last equation amounts to a simple integration, $R(t) = R_0 + \beta \int_{t_0}^{t} I(y)dy$; thus we will mostly look at the first two equations in (1).

We also stress, however, that epidemiological data can only collect time series for $R(t)$: so this is the quantity to be compared to experimental data [2].

With (1), $S(t)$ is always decreasing until there are infectives. The second equation in (1) immediately shows that the number of infectives grows if S is above the *epidemic threshold*

$$\gamma \;=\; \beta/\alpha\ . \tag{2}$$

Thus to stop an epidemic once the numbers are too large to isolate all the infectives, we have three (non mutually exclusive) choices within the SIR framework:

(a) Wait until $S(t)$ falls below the epidemic threshold;

(b) Raise the epidemic threshold above the present value of $S(t)$ by decreasing α;

(c) Raise the epidemic threshold above the present value of $S(t)$ by increasing β.

In practice, any State will try to both raise β and lower α, and if this is not sufficient await that S falls below the attained value of γ.

In order to understand how this is implemented, it is necessary to understand what α and β represent in concrete situations.

The parameter β represents the *removal rate* of infectives; its inverse β^{-1} is the average time the infectives spend being able to spread the contagion. In the context of COVID, raising β means lowering the time from infection to isolation, hence from infection to detection of the infected state.

The parameter α represents the *infection rate*, and as such it includes many thing. It depends both on the infection vector characteristics (how easily it spreads around, and how easily it infects a healthy individual who gets in contact with it), but is also depends on the occasions of contacts between individuals. So, roughly speaking, it is proportional to the number of close enough contacts an individual has with other ones per unit of time. It follows that – if properly implemented – social distancing results in reducing α.

Each of these two actions presents some problem. The mean time for the appearance of symptoms of COVID is a bit over five days [8], and the first symptoms are usually quite weak. So early detection is possible only by fast tracing and laboratory checking of all the contacts of those who are known to be infected. This has a moderate cost (especially if compared to the cost of an Intensive Care hospital stay) but requires an extensive organization.

On the other hand, social distancing is cheap in immediate terms, but produces a notable strain of the societal life, and in practice – as many of the contacts are actually work related – requires to stop as many production and economic activities as possible, i.e. has a formidable cost in the medium and long run. Moreover, it cannot be pushed too far, as a number of activities and services (e.g. those carrying food to people, urgent medical care, etc.) can not be stopped.

Let us come back to (1); using the first two equations, we can study I in terms of S, and find out that

$$I \;=\; I_0 \;+\; (S_0 - S) \;-\; \gamma\, \log(S_0/S)\;. \qquad (3)$$

As we know that the maximum I_* of I will be reached when $S = \gamma$, this allows immediately to determine the *epidemic peak*. In practice, I_0 is negligible and S_0 corresponds to the whole population, $S_0 = N$; thus

$$I_* \;=\; N \;-\; \gamma \;-\; \gamma\, \log(N/\gamma)\;. \qquad (4)$$

Note that only γ appears in this expression; that is, raising β or lowering α produces the same effect as long as we reach the same γ.

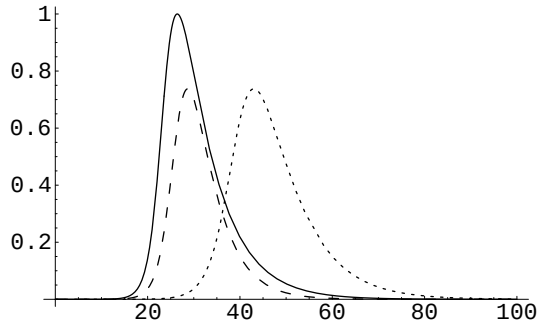

FIG. 1: Different effect of acting on the α or the β parameter. The SIR equations (1) are numerically integrated and $I(t)$ plotted in arbitrary units for given initial conditions and α, β parameters (solid), the maximum I_* being reached at $t = t_*$. Then they are integrated for the same initial condition but raising β by a factor $\vartheta = 3/2$ (dashed) with maximum $I_\beta = rI_*$ reached at time $t_\beta = \sigma_\beta t_*$; and lowering α by the same factor $\vartheta = 3/2$ (dotted) with maximum $I_\alpha = \mathcal{I}_\beta$ reached at time $t_\alpha = \sigma_\alpha t_*$. Time unit is one day, $\alpha = (4/3) * 10^{-8}$, $\beta = 1/7$; these parameters arise from our fitting of data from the early phase of COVID epidemics in Northern Italy [6, 9]; the population of the most affected area in the initial phase is about 20 million, that of the whole Italy is about 60 million. The numerical simulation is ran with $N = 6 * 10^7$; it results $r = 0.74$, $\sigma_\alpha = 1.63$, $\sigma_\beta = 1.09$, $I_* = 3.08 * 10^7$, $t_* = 26.4$; note that $\sigma_\alpha/\sigma_\beta = 3/2 = \vartheta$.

On the other hand, this simple formula does not tell us *when* the epidemic peak is reached, but only that it is reached when S has the value γ. But if measures are taken, these should be effective for the whole duration of the epidemic, and it is not irrelevant – in particular if the social and economic life of a nation is stopped – to be able to evaluate how long this will be for.

Acting on α or on β to get the same γ will produce different timescales for the dynamics; see Figure 1, in which we have used values of the parameters resulting from our fit of early data for the Northern Italy COVID-19 epidemic [6, 9].

This observation can be made more precise considering the scaling properties of (1). In fact, consider the scaling

$$\alpha \to \lambda\, \alpha\;, \quad \beta \to \lambda\, \beta\;, \quad t \to \lambda^{-1}\, t\;. \qquad (5)$$

It is clear that under this scaling γ remains unchanged, and also the equations are not affected; thus the dynamics is the same but with a different time-scale.

The same property can be looked at in a slightly different way. First of all, we note that one can write $\alpha = \beta/\gamma$; moreover, α appears in (1) only in connection with S, and it is more convenient to introduce the variable

$$\vartheta \;:=\; S/\gamma\;. \qquad (6)$$

Now, let us consider two SIR systems with the same initial data but different sets of parameters, and let us for ease of notation just consider the first two equations of

each. Thus we have the two systems

$$\gamma^{-1}\, d\vartheta/dt \;=\; -\beta\,\vartheta\, I\;,\quad dI/dt \;=\; \beta\,(\vartheta-1)\, I\;; \quad (7)$$
$$\widetilde{\gamma}^{-1}\, d\widetilde{\vartheta}/dt \;=\; -\widetilde{\beta}\,\widetilde{\vartheta}\,\widetilde{I}\;,\quad d\widetilde{I}/dt \;=\; \widetilde{\beta}\,(\widetilde{\vartheta}-1)\,\widetilde{I}\;. \quad (8)$$

We can consider the change of variables ($\lambda > 0$)

$$\widetilde{\beta} = \lambda\,\widetilde{\beta} := \widehat{\beta}\;,\quad t \to \lambda^{-1}\, t := \tau\;. \quad (9)$$

With this, (8) becomes

$$\lambda\widetilde{\gamma}^{-1}\,(d\widetilde{\vartheta}/d\tau) \;=\; -\lambda\widehat{\beta}\,\widetilde{\vartheta}\,\widetilde{I}\;,\quad \lambda(d\widetilde{I}/d\tau) \;=\; \lambda\widehat{\beta}\,(\widetilde{\vartheta}-1)\,\widetilde{I}\;.$$

We can thus eliminate the factor λ in both equations. However, if we had chosen $\lambda = \widetilde{\beta}/\beta$, we get $\widehat{\beta} = \beta$; if moreover $\widetilde{\gamma} = \gamma$, the resulting equation is just

$$\gamma^{-1}\, d\widetilde{\vartheta}/d\tau \;=\; -\beta\,\widetilde{\vartheta}\,\widetilde{I}\;,\quad d\widetilde{I}/d\tau \;=\; \beta\,(\widetilde{\vartheta}-1)\,\widetilde{I}\;. \quad (10)$$

But we had supposed the initial data for $\{S, I\}$ and for $\{\widetilde{S}, \widetilde{I}\}$ (and hence also for ϑ and $\widetilde{\vartheta}$) to be the same. We can thus directly compare (10) with (7).

We observe that $\{\widetilde{\vartheta}, \widetilde{I}\}$ have thus exactly the same dynamics – but in terms of the rescaled time τ – as $\{\vartheta, I\}$ in terms of the original time t. In particular, if the maximum of I is reached at time t_*, the maximum of $\widetilde{I}$ is reached at $\tau_* = t_*$, and hence at

$$\widetilde{t}_* \;=\; \lambda\,\tau_* \;=\; \lambda\, t_*\;. \quad (11)$$

More precise analytical results on the timescale change induced by a rescaling of the α and β parameters have recently been obtained by M. Cadoni (personal communication) [7].

III. A-SIR MODEL

One of the striking aspects of the ongoing COVID-19 epidemic is the presence of a large fraction of *asymptomatic infected*, and hence infectives; note that here we will always use "asymptomatic" as a shorthand for "asymptomatic or paucisymptomatic", as also people with very light symptoms will most likely escape to clinical detection of COVID – and actually most frequently will not even think of consulting a physician.[23]

A. The model and its parameters

In order to take this aspect into account, we have recently formulated a variant of the SIR model [9] in which together with known infectives $I(t)$, and hence known removed $R(t)$, there are unregistered infectives $J(t)$ and unregistered removed $U(t)$. Note that in this case removal amounts to healing; so while the removal time β^{-1} for known infected corresponds to the time from infection to isolation (thus in general slightly over the incubation time $T_i \simeq 5.1$ days for COVID), the removal time η^{-1} for unrecognized infects will correspond to incubation time plus healing time.

In the model, it is supposed that symptomatic and asymptomatic infectives are infective in the same way. This is not fully realistic, as one may expect that somebody having the first symptoms will however be more retired, or at east other people will be more careful in contacts; but this assumption simplifies the analysis,and is not completely unreasonable considering that for most of the infection-to-isolation time β^{-1} the symptoms do not show up.

The equations for the A-SIR model [9] are

$$\begin{aligned} dS/dt &= -\,\alpha\; S\,(I+J) \\ dI/dt &= \alpha\,\xi\; S\,(I+J) \;-\; \beta\, I \\ dJ/dt &= \alpha\,(1-\xi)\; S\,(I+J) \;-\; \eta\, J \\ dR/dt &= \beta\, I \\ dU/dt &= \eta\, J\;. \end{aligned} \quad (12)$$

Note that here too we have a "master" system of three equations (the first three) while the last two equations amount to direct integrations, $R(t) = R_0 + \beta\int_{t_0}^{t} I(y)dy$, $U(t) = U_0 + \eta\int_{t_0}^{t} J(y)dy$.

The parameter $\xi \in [0, 1]$ represents the probability that an infected individual is detected as such, i.e. falls in the class I. In the absence of epidemiological investigations to trace the contacts of known infectives, this corresponds to the probability of developing significant symptoms.

In our previous work [9], some confusion about the identification of the class J was present, as this was sometimes considered to be the class of asymptomatic infectives, and sometimes that of not registered ones[24]. While this is not too much of a problem considering the "natural" situation, it becomes so when we think of action on this situation.

Actually, and unfortunately, this confusion has a consequence exactly on one of the points we want to discuss here, i.e. the effect of a campaign of chasing the infectives, e.g. among patients with light symptoms or within social contacts of known infectives; let us thus discuss briefly this point.

If J is considered to be the set of asymptomatic virus carriers, then a raise in the fraction of these who are known to be infective, and thus isolated, means that the average time for which asymptomatic infectives are not isolated is decreasing. In other words, we are lowering η^{-1} and thus raising η. On the other hand, in this description ξ is the probability that a new infective is asymptomatic, and this depends only on the nature of the virus and its interactions with the immune system of the infected people; thus in this interpretation ξ should be considered as a constant of nature, and it cannot be changed.[25]

On the other hand, if J is the class of unknown infectives, things are slightly different. In fact, to be in this class it is needed (a) that the individual has no or very

light symptoms; but also (b) that he/she is not traced and analyzed by some epidemiological campaign, e.g. due to contacts with known infected or because belonging to some special risk category (e.g. hospital workers). In this description, η is a constant of nature, depending on the nature of the virus and on the response of the "average" immune system of (asymptomatic) infected people, while our efforts to trace asymptomatic infectives will act on raising the probability ξ.

We want to discuss the effect of early detection of infectives, or tracing their contacts, within the second mentioned framework. Note that a campaign of tracing contacts of infectives is useful not only to uncover infectives with no symptoms, but if accompanied by effective isolation of contacts with known infectives, and thus of those who are most likely to be infective, it will also reduce the removal time of "standard" (i.e. symptomatic) infectives, possibly to a time smaller than the incubation time itself.

B. A glimpse at COVID matters in this respect

This approach, indeed, was taken in one of the areas of early explosion of the contagion in Northern Italy, i.e. in Vò Euganeo; this had the advantage of being a small community (about 3,000 residents), and all of them have been tested twice while embargo was in operation. In fact, this was the first systematic study showing that the number of asymptomatic carriers was very high, quite above the expectations [10]. Apart from its scientific interest, the approach proved very effective in practical terms, as new infectives were quickly traced and in that specific area the contagion was stopped in a short time.

While testing everybody is not feasible in larger communities, the "follow the contacts" approach could be used on a larger scale, especially with the appearance of new very quick kits for ascertaining positivity to COVID.

The model will thus react to a raising of ξ by raising the fraction of I within the class of infectives, i.e. in $K = I + J$; but at the same time, as critical patients are always the same, i.e. represents always the same fraction of K, we should pay attention to the fact they will now represent a lower fraction of I. The Chinese experience shows that critical patients are about 10 % of hospitalized patients (i.e. of those with symptoms serious enough to require hospitalization); and hospitalized patients represented about half of known infected, the other being cured and isolated at home. Similar percentages were observed in the early phase of the COVID epidemic in Italy; the fraction of infectives isolated at home has afterwards diminished, but it is believed that this was due to a different policy for lab exams, i.e. checking prioritarily patients with multiple symptoms suggesting the presence of COVID rather than following the contacts. Actually this policy was followed in most of Italy, but in one region (Veneto) the tracking of contacts and lab exams for them was pursued, and in there the percentages were much more similar to those known to hold for China.

C. Numerical simulations protocol. Parameters and initial data

In our previous work [9] we have considered data for the early phase of COVID epidemics in Italy, and found that $\beta^{-1} \simeq 7$ best fits them while the estimate $\eta^{-1} \simeq 21$ was considered as a working hypothesis. This same work found as value of the contact rate in the initial phase $\alpha \simeq 1.13 * 10^{-8}$, and we will use this in our numerical simulations.

It should be stressed that the extraction of the parameter α from epidemiological data is based on the number $S_0 \simeq N$ of susceptibles at the beginning of the epidemic, thus α and hence γ depend on the total population. The value given above was obtained considering $N = 2 * 10^7$, i.e. the overall population of the three regions (Lombardia, Veneto and Emilia-Romagna) which were mostly affected in the initial phase.

Our forthcoming discussion, however, does *not* want to provide a forecast on the development of the COVID epidemic in Northern Italy; we want instead to discuss – with realistic parameters and framework – what would be the differences if acting with different strategies in an epidemic with the general characteristics of the COVID one. Thus we will adopt the aforementioned parameters as "bare" ones (different strategies consisting indeed on acting on one or the other of these) but will apply these on a case study initial condition; this will be given by

$$I_0 \; = \; 10 \; , \quad J_0 \; = \; 90 \; ; \quad R_0 \; = \; U_0 \; = \; 0 \; . \tag{13}$$

One important parameter is missing from this list, i.e. the detection probability ξ. Following Li *et al.* [11] we assumed in previous work that ξ is between 1/10 and 1/7. Later works (and a general public interview by the Head of the Government agency handling the epidemic [12]) suggested that the lower bound is nearer to the truth; moreover a lower ξ will give us greater opportunity to improve things by acting on it (we will see this is not the best strategy, so it makes sense to consider the setting more favorable to it). We will thus run our simulation starting from a "bare" value $\xi = 1/10$.[26]

As for the total size of the population, we set $N = 2 * 10^7$. With these choices we get

$$\gamma \; = \; 1.26 \; * \; 10^7 \; , \quad \frac{S_0}{\gamma} \; \simeq \; 1.58 \; . \tag{14}$$

We would like to stress once again that we will work with constant parameters, while in reality the parameters are changing all the time due to the continuing efforts to contain the epidemic. So our discussion is valid for what concerns the effect of different actions, but the absolute values of infected etc are by no means a forecast of what will happen; rather they should be seen – in particular,

those relating to the "bare" parameters – as a projection of what could have happened if no action was undertaken.

D. Balance between registered and unregistered infectives

A look at eqs.12 shows that I will grow provided

$$\frac{\xi\, S}{\gamma} \;>\; \frac{I}{I+J} \;=\; \frac{I}{K} \;:=\; x\;, \tag{15}$$

where again $\gamma = \beta/\alpha$, and we have introduced the ratio $x(t)$ of known infectives over total infectives. In other words, now the epidemic threshold

$$\gamma_I \;=\; \left(\frac{x}{\xi}\right)\;\gamma \tag{16}$$

depends on the distribution of infectives in the classes I and J. Note that if $x = \xi$ (as one would expect to happen in early stagesof the epidemic), then $\gamma_I = \gamma$.

Needless to say, we have a similar result for J, i.e. J will grow as far as

$$(1-\xi)\, S\, \frac{\alpha}{\eta} \;>\; \frac{J}{I+J} \;=\; \frac{J}{K} \;:=\; y \;=\; 1-x\;; \tag{17}$$

thus the epidemic threshold for unregistered infectives is

$$\gamma_J \;=\; \left(\frac{1-x}{1-\xi}\right)\;\frac{\eta}{\alpha}\;. \tag{18}$$

For $x = \xi$ (see above) we would have $\gamma_J = (\eta/\beta)\gamma < \gamma$.

It is important to note that x is evolving in time. More precisely, by the equations for I and J we get

$$\begin{aligned}\frac{dx}{dt} \;&=\; \alpha\,\xi\, S \;-\; (\alpha S + \beta - \eta)\; x \;+\; (\beta-\eta)\, x^2 \\ &=\; \alpha\, S\;(\xi - x) \;+\; (\beta-\eta)\,(x^2 - x)\;.\end{aligned} \tag{19}$$

The behavior observed in Fig.2 can be easily understood intuitively. In the first phase of the epidemic, there is an exponential growth of both I and J; due to the structure of the equations, they grow with the same rate, so their ratio remains constant; on the other hand, once the dynamics get near to the epidemic peak, the difference in the permanence time of the two (that is, the time individuals remain in the infect class) becomes relevant, and we see (plots (a2) and (b2) of Fig.2) that not only the peak for J is higher than the one for I, but it occurs at a slightly later time. Moreover, descending off the peak is also faster for I, as $\beta^{-1} < \eta^{-1}$, and thus x further decreases, until it reaches a new equilibrium while both classes I and J go exponentially to zero.

If we look at (19) we see that for fixed S the variable x would have two equilibria (one stable with $0 < x < 1$ and one unstable with $x > 1$,stability following from $\beta - \eta > 0$), easily determined solving $dx/dt = 0$. Numerical simulations show that – apart from an initial transient – actually $x(t)$ stays near, but in general does not really sticks to, the stable fixed point determined in this way.

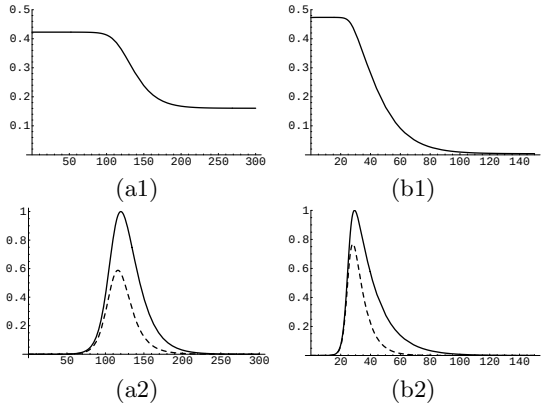

FIG. 2: Dynamics of $x(t)$ in the A-SIR model. We plot $x(t)$ in upper plots (a1) and (b1); and $I(t)$ (dashed curve) and $J(t)$ (solid curve) in lower plots (a2) and (b2). These are considered along the solutions of the A-SIR model for $\alpha = 1.13 * 10^{-8}$, $\beta = 1/7$, $\eta = 1/14$, and $\xi = 1/10$; in the left column, i.e. in plots (a1) and (a2), with $S_0 = 2 * 10^7$ and $I_0 + J_0 = 10$; in the right column, i.e. in plots (b1) and (b2), for $S_0 = 6 * 10^7$ and $I_0 + J_0 = 30$. The scale of plots (a2) and (b2) is chosen so that the maximum of $J(t)$ is at level one.

E. The basic reproduction number

A relevant point should be noted here. If we consider the sum

$$K(t) \;:=\; I(t) \;+\; J(t) \tag{20}$$

of all infectives, the A-SIR model can be cast as a SIR model in terms of S, K, and $Q = R + U$ as

$$\begin{aligned} dS/dt \;&=\; -\,\alpha\, S\, K \\ dK/dt \;&=\; \alpha\, S\, K \;-\; B\, K \\ dQ/dt \;&=\; B\, K \end{aligned} \tag{21}$$

where B is the *average* removal rate, i.e.

$$B \;=\; x\,\beta \;+\; (1-x)\,\eta\;.$$

As x varies in time, this average removal rate is also changing. On the other hand, the *basic reproduction number* (BRN)[27] ρ_0 for this model will be

$$\rho_0 \;=\; \frac{\alpha}{B}\; S \;>\; \frac{\alpha}{\beta}\; S\;. \tag{22}$$

In other words, not taking the asymptomatic infectives into account leads to an *underestimation* of the BRN. If the standard SIR model predicts a BRN of ρ_0, the A-SIR model yields a BRN $\widehat{\rho}_0$ given by

$$\widehat{\rho}_0 \;=\; \frac{\beta}{B}\;\rho_0 \;=\; \frac{\beta}{x\beta + (1-x)\eta}\;\rho_0 \;>\; \rho_0\;. \tag{23}$$

This means that the epidemic will develop faster, and possibly much faster, than what one would expect on the basis of an estimate of ρ_0 based only on registered cases,

which in the initial phase are a subset of symptomatic cases (as the symptoms may easily be leading to a wrong diagnosis; in the case of COVID they lead to a diagnosis of standard flu).

With our COVID-related values $\beta = 1/7$, $\eta = 1/21$, and assuming that in the early phase $x = \xi = 1/10$, we get

$$\widehat{\rho}_0 \;=\; \frac{5}{2}\,\rho_0\;; \tag{24}$$

there is thus a good reason for being surprised by the fast development of the epidemic: *the actual BRN is substantially higher than the one estimated by symptomatic infections* [16].

IV. HIDDEN INFECTIVES AND EPIDEMIC DYNAMICS

More generally, one would wonder what is the effect of the "hidden" infectives $J(t)$ on the dynamics of the known infectives $I(t)$ – which, we recall, include the relevant class of seriously affected infectives – and it appears that there are at least two, contrasting, effects:

1. On the one hand, the hidden infectives speed up the contagion spread and hence the raise of $I(t)$;
2. On the other hand, they contribute to group immunity, so the larger this class the faster (and the lower the I level at which) the group immunity will be reached.

The discussion above shows that the balance of these two factors leads to a much lower epidemic peak, and a shorter epidemic time, than those expected on the basis of the standard SIR model (albeit in the case of COVID these are still awful numbers).

On the other hand, we would like to understand if uncovering a larger number of cases (thus having prompt isolation of a larger fraction of the infectives) by *early detection*, i.e. raising ξ, would alter the time-span of the epidemic. It appears that this effect can be only marginal, as it appears only past the epidemic peak.

We stress that this statement refers to "after incubation" analysis; if we were able to isolate cases *before* they test positive – i.e. to substantially reduce β^{-1} – the effect could be different. We will discuss this point, related to *contact tracing*, later on.

A. Observable and "clean" observable data

An ongoing epidemic is not a laboratory experiment, and apart from not having controlled external conditions, i.e. constant parameters, the very collection of data is of course not the top priority of doctors fighting to save human lives.

There has been considerable debate on what would be the most reliable indicator to overcome at least the second of these problems. One suggestion is to focus on the number of deaths; but this is itself not reliable, as in many cases COVID is lethal on individuals which already had some medical problem, and registering these deaths as due to COVID or to some other cause depends on the protocol adopted, and in some case also on political choices, e.g in order to reassure citizens (or on the other extreme, to stress great care must be taken to avoid contagion).

Another proposed indicator, possibly the most reliable in order to monitor the development of the epidemic, is that of patients in Intensive Care Units. This appears to be sufficiently stable over different countries, and e.g. the Italian data tend to reproduce in this respect - at least in Regions where the sanitary system is not overstretched – the Chinese ones.[28]

In this case, IC patients are about 20 % of the total number of hospitalized cases; in China and for a long time also in Italy (when protocols for choosing would-be cases to be subject to laboratory analysis have been stable), hospitalized cases have been about half of the known infection cases, the other having shown only minor symptoms and been cured (and isolated) in their home.

The other, more widely used, indicator is simply the total number of known cases of infection. In view of the presence of a large class of asymptomatic infectives, this itself is strongly depending on the protocols for chasing infectives. On the other hand, this is the most available indicator: e.g., the W.H.O. situation reports [19] provide these data.

Each of these indicators, thus, has advantages and disadvantages. We will just use the WHO data on known infected.

In particular, in the case of COVID we expect that with ξ_0 the "bare" constant describing the probability that an infection is detected[29], out of the class $I(t)$ we will have a 50% of infected with little or no symptoms (I_L), a 40% of standard care hospitalized infected (I_H), and a 10% of IC hospitalized infected (I_{IC}). Needless to say, this class is the most critical one, also in terms of strain on the health system.

More generally, we say that with ξ_0 the "bare" constant describing the probability that the infection under study is detected, there is a fraction χ_0 (of the detected infections) belonging to the I_{IC} class; that is, $I_{IC}(t) = \chi_0 I(t)$.

V. MODIFYING THE PARAMETERS

We are now ready to discuss how modification of one or the other of the different parameters (α, β, ξ) on which we can act by various means will affect the A-SIR dynamics. As it should be expected, this will give results similar to those holding for the SIR model, but now we have one more parameter to be consideredand thus a more rich set of possible actions.

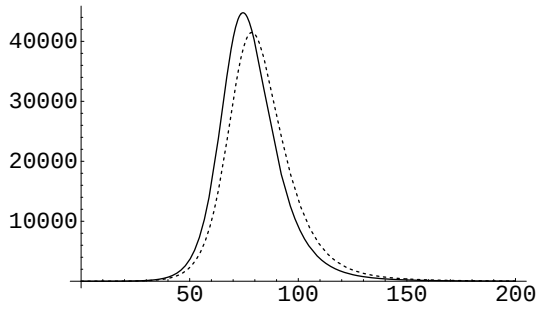

FIG. 3: The effect of a change in ξ on the I_{IC} class. We have used $\beta = 1/7$, $\eta = 1/21$, and $\alpha = 1.13 * 10^{-8}$ as in Fig.2, with a total population of $N = 2 * 10^7$, and ran simulations with $\xi = 1/10$ (solid curve) and with $\xi = 1/4$ (dashed curve). The substantial increase in ξ produces a reduction in the epidemic peak and a general slowing down of the dynamics, but both these effects are rather small.

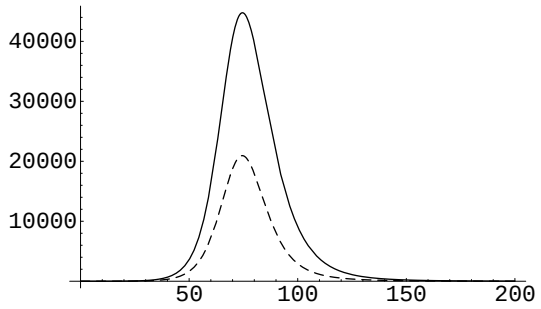

FIG. 4: The effect of a change in β on the I_{IC} class. We have used $\xi = 1/10$, $\eta = 1/21$, and $\alpha = 1.13 * 10^{-8}$ as in Fig.2, with a total population of $N = 2 * 10^7$, and ran simulations with $\beta = 1/7$ (solid curve) and with $\beta = 1/3$ (dashed curve). The substantial increase in β produces a marked reduction in the epidemic peak and a very slightly faster pace in the dynamics.

A. Raising the detected fraction

A more extensive test campaign will raise ξ, say from ξ_0 to ξ_1; but of course this will not change the number of the most serious cases, as these are anyway getting to hospital and detected as being due to the infection in question. Thus the new fraction χ_1 of detected infections which need special care will be such that $\chi_1 \xi_1 = \chi_0 \xi_0$, i.e. we have

$$\chi_1 \ = \ \frac{\xi_0}{\xi_1} \, \chi_0 \ . \tag{25}$$

In order to describe the result of raising ξ, we should thus compare plots of

$$I_{IC}(t) \ = \ \chi \, I(t) \ . \tag{26}$$

This is what we do, indeed, in Fig.3.

B. Running ahead of the epidemic wave

Raising ξ corresponds to having more infective detected, and has some advantages from the point of view of the epidemic dynamics. In practical terms, this means extending tests to a larger class of subjects, and be able to isolate a larger fraction of asymptomatic infectives with the same speed and effectiveness as symptomatic ones.

A different strategy for rapid action is also possible, and it consists of rapid isolations of subjects who had contacts with people known to have been infected, or who have themselves been in contact with known infectives (and so on). In other words, the strategy would be to isolate would-be infection carriers *before* any symptom could show up. This means that β^{-1} could be even smaller than the usual infection-to-isolation time (about seven days for COVID) for symptomatic infectives, and even shorter than the incubation time (about five days for COVID).[30]

We have thus ran a simulation in which ξ is not changed, but β raises from $\beta_0 = 1/7$ to $\beta = 1/3$; the result of this is shown in Fig.4. In this case we have a marked diminution of the epidemic peak, and a very slight acceleration of the dynamics.

C. Social distancing

We have so far not discussed the most basic tool in epidemic containment, i.e. social distancing. This means acting on the parameter α by reducing it.[31]

This is a basic action to be undertaken, and in fact it is being taken by all Nations. It is also the simplest one to be organized (albeit with high economic and social costs in the long run) and an action which can be taken together with other ones. No doubt this should be immediately taken when an epidemic is starting, and accompanied by other measures – such as those discussed above. But here we want to continue our study of what it means by itself in terms of modification of the epidemic dynamics.

It is not clear what can be achieved in terms of reduction of social contacts. In fact, once the epidemic starts most of the dangerous contacts are the unavoidable ones, such as those arising from essential services and production activity (e.g. production and distribution of food or pharmaceutical goods), contacts at home, and above all contacts in Hospitals. Thus, after a first big leap downward corresponding to closing of schools and Universities on the one side, and a number of unessential commercial activities on the other, and restrictions on travels, it is difficult to further reduce social contacts[32], not to say that this would have huge economic and social costs, and also a large impact on the general health in terms of sedentariness-related illness (and possibly mental health).

We point out that there is a further obstacle to reduc-

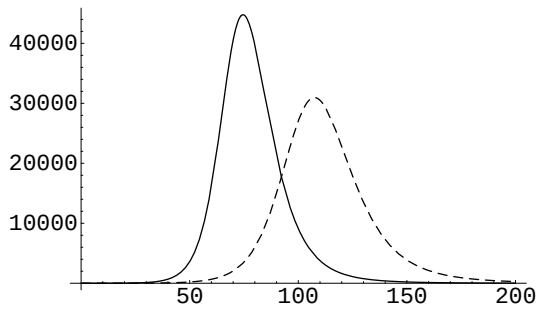

FIG. 5: The effect of a change in α on the I_{IC} class. We have used $\beta = 1/7$, $\xi = 1/10$, $\eta = 1/21$, with a total population of $N = 2 * 10^7$, and ran simulations with $\alpha = 1.13 * 10^{-8}$ (solid curve) and with $\alpha = 8.47 * 10^{-9}$ (dashed curve). The reduction in α produces a marked reduction in the epidemic peak and also a marked slowing down in the dynamics.

ing social contacts: as seen in the context of the simple SIR model, reducing α will lower the epidemic peak, but it will also slow down the whole dynamic. While this allows to gain precious time to prepare Hospitals to stand the big wave, there is some temporal limit to an extended lockdown, and thus this tool cannot be used to too large an extent.

We have thus ran a simulation in which β and ξ are not changed, while α is reduced by a factor 0.75 (smaller factors, i.e. smallerα, produce an untenable length of the critical phase); the result of this is shown in Fig.5. In this case we have a relevant diminution of the epidemic peak, and also a marked slowing down in the dynamics.

An important remark is needed here. It may seem, looking at this plot, that social distancing is less effective than other way of coping with the epidemic. But these simulation concern a SIR-type model; this means in particular that there is no spatial structure in our model [2]. The travel ban is the most effective way of avoiding the spreading of contagion from one region to the others; while the "local" measures of social distancing can (and should) be triggered to find a balance with other needs, travel ban is the simplest and most effective way of protecting the communities which have not yet been touched by the epidemic.

D. Comparing different strategies

We can thus compare the different strategies we have been considering. This is done in Fig.6 where we plot together $I_{IC}(t)$ for all our different simulations; and in Table I where we compare the height of the epidemic peak – again for $I_{IC}(t)$ – and the time at which it is reached.

In Fig.6 we have also drawn a line representing the hypothetical maximal capacity of IC units. This stresses that not only the different actions lower the epidemic peak, but they also – and to an even larger extent – reduce the number of patients which can not be conveniently treated.[33]

α	β	ξ	max	time
$1.13 * 10^{-8}$	1/7	1/10	44768	75
$1.13 * 10^{-8}$	1/7	1/4	41482	79
$1.13 * 10^{-8}$	1/3	1/10	20943	74
$8.47 * 10^{-9}$	1/7	1/10	30956	107

TABLE I: Epidemic peak (for I_{IC}) and time for reaching it (in days) as observed in our numerical simulations. All simulation were ran with $N = 2 * 10^7$ and $\eta = 1/21$.

It should be stressed that the strategies of contacts tracing and early detection are usually played together; but as confusion could arise on this point, let us briefly discuss it. We have tried to stress that these two actions are *not* equivalent: one could conduct random testing, so uncovering a number of asymptomatic infectives, and just promptly isolate them without tracing their contacts;or on the other extreme one could just isolate everybody who had a (direct or indirect) contact with a known infective, without bothering to ascertain if they are themselves infective or not. This strategy would be as effective in containing the contagion (and less costly in terms of laboratory tests) than that of tracking contacts, test them (after a suitable time for the infection to develop and test give positive if this happens), and isolate only those who really turn infective. The difference is that if we isolate everybody this would involve a huge number of people (e.g. all those who have been in the same supermarket the same day as an infective; and their families and contacts etc etc); so in this context early detection actually should be intended as early detection *of non-infectives*, so that cautionary quarantine can be kept reasonably short in all the cases where it is not really needed.

Finally we recall that it is a triviality, and it was already mentioned in the Introduction, that in real situations one has not to choose between acting on one or the other of the parameters, and all kind of actions should be pursued simultaneously.

VI. DISCUSSION AND CONCLUSIONS

We have considered epidemic dynamics as described by "mean field" models of the SIR type; more specifically, we have first considered the classical Kermack-McKendrick SIR model [1–5] and then a recently introduced modified version of it [9] taking into account the presence of a large set of asymptomatic – and thus most frequently not detected – infectives. These models depend on several parameters, and different types of measures can to some extent change these parameters and thus the epidemic dynamics. In particular, this action can effect two basic characteristics of it, i.e. the height of the epidemic peak and the time-span of the epidemic.

While it is clear that in facing a real lethal epidemics

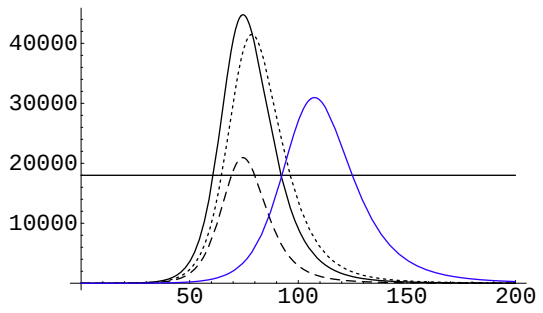

FIG. 6: The effect of different strategies. We plot $I_{IC}(t)$ for $N = 2 * 10^7$ in the "bare" case, i.e. for $\alpha = 1.13 * 10^{-8}$, $\beta = 1/7$, $\xi = 1/10$, $\eta = 1/21$, and in cases where (only) one of the parameters is changed. In particular we have the bare case (solid line), the case where ξ is changed into $\xi = 1/4$ (dotted), the case where β is changed to $\beta = 1/3$ (dashed), and that where α is changed to $\alpha = 8.47 * 10^{-9}$ (solid, blue). We also plot a horizontal line representing a hypothetical maximal capacity of IC units.

(such as the ongoing COVID epidemic) all actions which can contrast it should be developed at the same time, in this paper we have considered the result – within these models – of different tools at our disposal, i.e. (generalized) *social distancing*, *early detection* (of asymptomatic infectives) and *contacts tracing* (of symptomatic and asymptomatic infectives).

It turns out that – both in the classical SIR model and in the modified A-SIR one – social distancing is effective in reducing the epidemic peak, and moreover it slows down the epidemic dynamics. On the other hand, *early detection* of asymptomatic infectives seems to have only a moderate effect in the reduction of the epidemic peak for what concerns critical cases, and also a very little effect on the temporal development of the epidemic. In contrast, *contact tracing* has a strong impact on the epidemic peak – also in terms of critical cases – and does not substantially alter the temporal development of the epidemic.[34]

Slowing down the epidemic dynamic can be a positive or negative feature depending on the concrete situation and on the desired effects. It is surely positive in what concerns getting ready to face the epidemic peak, in particular in the presence of a faltering Health System. On the other hand, it may be negative in that maintaining a generalized lockdown for a long time can have extremely serious economic and social consequences. Balancing these two aspects is not a matter for the mathematician or the scientist, but for the decision maker; so we will not comment any further about this.

It should also be recalled that our analysis was conducted in terms of very simple SIR-type models, with all their limitations. In particular, we have considered no age or geographical or social structure, and only considered a population of "equivalent" individuals. In particular, as we have noted above, in the early stage of an epidemic, which presumably develops in very populated areas, a generalized travel ban can simply stop the contagion to propagate to other (possibly less well equipped in medical terms) areas; moreover, social distancing measures can be implemented very simply – basically, by a Government order[35] – and are thus the first action to be taken. In fact, in relation with the ongoing COVID epidemics, one of the reproaches made to many Governments is usually to have been too slow or too soft in stopping crowd gatherings (and, in Europe, football matches), surely not the contrary.

On the other hand, we hope that this study makes clear what are the consequences of different options. In particular, our study shows that *contacts tracing*, followed by prompt isolation of would-be infected people – is the only way to reduce the impact of the epidemic without having to live with it for an exceedingly long time. The Veneto experience shows that this strategy can be effectively implemented without hurting privacy or personal freedom.

Acknowledgements

I thank L.Peliti (SMRI), M. Cadoni (Cagliari) and E. Franco (Roma) for useful discussions and for pointing out shortcomings in my work. Special thanks are due to M. Cadoni for insisting on the relevance of time scalings. The (mathematical and non-mathematical) opinions expressed in this work belong solely to the author. The work was carried out at SMRI. I am also a member of GNFM-INdAM.

Appendix A: The COVID-19 epidemics in Northern Italy

As stated above, our discussion is of general nature, but was triggered by the ongoing COVID-19 epidemics in Northern Italy; this in turn provides some concrete arena for testing our conclusions. In fact, in Italy the competence of the Health System pertains to the regions, so that there were slightly different strategies followed by nearby regions.

We have considered in particular Lombardia, Veneto and Emilia Romagna, i.e. the regions which were first and more severely affected by the COVID epidemics. Some debate took place, in particular between health officers of Lombardia and Veneto, about the use of laboratory tests; in the end Veneto performed far more tests (per thousand inhabitants) than Lombardia, with Emilia in between. (Data about this can be extracted from official sites [20] or from general press [21].)

The evolution of (registered) infections in Italy and in these regions is depicted (in logarithmic scale) in Fig.7.

We have monitored the evolution of these by the following protocol (dates are based on governmental measures; the impact of these will be discussed elsewhere).

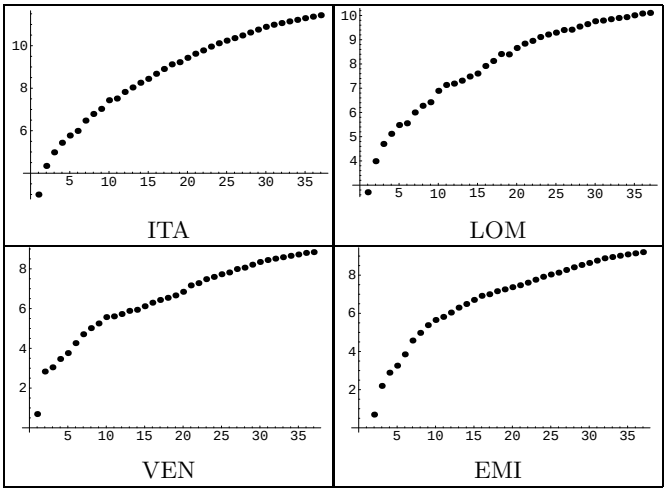

FIG. 7: Cumulative registered cases in Italy (ITA) and active registered cases in Lombardia (LOM), Veneto (VEN) and Emilia-Romagna (EMI); day 1 is February 21, data go until March 29.

	ITA	LOM	VEN	EMI
Feb 23 – Mar 1	0.34	0.29	0.37	0.51
Mar 1 – Mar 8	0.22	0.17	0.13	0.20
Mar 8 – Mar 15	0.17	0.15	0.18	0.13
Mar 15 – Mar 22	0.13	0.09	0.12	0.12
Mar 22 – Mar 29	0.08	0.05	0.07	0.08

TABLE II: The exponent A, see (A1), for Italy (ITA), Lombardia (LOM), Veneto (VEN) and Emilia-Romagna (EMI), in different weeks; see text.

The time-span of the epidemic has been divided into weeks. For each of these weeks we have fitted data on infectives[36] by a simple exponential

$$R(t) \;=\; \exp[A\,(t-t_0)]\;R(t_0)\;, \qquad \text{(A1)}$$

and measured A in different weeks and different regions.

This should allow to follow in a quantitative way the descent of the epidemic speed. The result of this exercise is summarized in Table II, and show that there is little difference between different regions.

But, these data concern *registered* positive and are thus greatly influenced by the testing strategy. In Table III we have collected some demographic data about the three considered regions and data about the number of tests carried out, together with the number of COVID-related deaths. Here, together with the first and most affected regions, we also consider Piemonte and Marche, which were struck later but also quite heavily.

	LOM	VEN	EMI	PIE	MAR
Population ($*10^6$)	10.088	4.906	4.459	4.356	1.523
Density (inhab/km^2)	423	267	199	172	162
tests (March 8)	20135	15956	4906	1681	1250
deaths (March 8)	333	20	70	13	10
tests (March 29)	107398	94784	56491	24058	10431
deaths (March 29)	6360	392	1443	684	386
tests/m (March 29)	10.65	19.32	12.67	5.52	6.85
deaths/M (March 29)	630.45	79.90	323.62	157.02	253.45
cases (cumulative)	41007	8358	13119	8206	3558
deaths/cases	0.155	0.047	0.110	0.083	0.108

TABLE III: Demographic data, number of tests and deaths at March 8 and March 29; together with tests per thousand inhabitants, deaths per million inhabitants and deaths per registered case at the latter date. Data for Lombardia (LOM), Veneto (VEN), Emilia-Romagna (EMI), Piemonte (PIE) and Marche (MAR).

Large differences are evident in the testing policy; it is also evident that the region performing more tests, i.e. Veneto, also has a specially good score in saving its COVID patients. The data for the mortality rate are of course largely influenced by the testing policy itself, i.e. by the fact there are more registered COVID patients. But the really relevant data are those concerning the ratio between COVID deaths and the total population: this is not affected by the testing policy, and actually performing more tests leads to recognizing COVID as the death cause in a larger set of cases.

In view of our discussion, we would not expect diffuse testing *by itself* to give a substantial advantage. Thus our interpretation of the specially low number of victims in Veneto (not only compared to Lombardia, which was probably also specially unlucky in this circumstance and has a higher population density, but also with respect to other regions[37] which were affected by COVID only later) is that the winning point in their strategy was not so much the high number of tests, but the fact *they were properly targeted*, tracing contacts and isolating potential new infectives *before* the infection showed up – and using tests to ascertain if they should remain isolated or be allowed to go back to usual life.[38]

This is even more remarkable as it was conducted without the help of any personal control technology (app) or other means interfering with privacy and personal freedom – and without the help of the many thousand people working at tracing contacts in Hubei – showing that effective epidemic control is not necessarily in contrast with personal freedom.

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[5] L. Edelstein-Keshet, *Mathematical Models in Biology*, SIAM 2005

[6] G. Gaeta, "Data analysis for the COVID-19 early dynamics in Northern Italy", arXiv:2003.02062; "Data Analysis for the COVID-19 early dynamics in Northern Italy. The effect of first restrictive measures" arXiv:2003.03775

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[10] E. Lavezzo *et al.*, "Suppression of COVID-19 outbreak in the municipality of Vò, Italy",submitted paper (2020)

[11] R. Li et al., Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV2), *Science* 10.1126/science.abb3221 (2020)

[12] A. Borrelli, interview to the newspaper "La Repubblica", March 24, 2020, page 3

[13] https://www.theguardian.com/commentisfree/2020/mar/26/virus-infection-data-coronavirus-modelling

[14] J. Lourenco *et al.*, "Fundamental principles of epidemic spread highlight the immediate need for large-scale seologicalsurvey to assess the sage of the SARS-CoV-2 epidemic", https://www.medrxiv.org/content/10.1101/2020.03.24.20042291v1

[15] http://www.eecs.qmul.ac.uk/ norman/papers/Coronavirus-death-rates-causal-model.pdf

[16] G. Gaeta, arXiv ***

[17] The Novel Coronavirus Pneumonia Emergency Response Epidemiology Team: "Vital Surveillances: The Epidemiological Characteristics of an Outbreak of 2019 Novel Coronavirus Diseases (COVID-19) – China, 2020", *China CDC Weekly 2020, 2(8): 113-122.*

[18] https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf

[19] https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports

[20] http://www.salute.gov.it/nuovocoronavirus

[21] https://lab.gedidigital.it/gedi-visual/2020/coronavirus-i-contagi-in-italia

[22] http://www.protezionecivile.gov.it/media-comunicazione/comunicati-stampa

[23] The problem is probably even worsened in countries – like Italy and to some extent also Spain – where media have transformed a medical problem into a public order one, putting the accent on social controlling – in some areas citizens are invited to signal people walking in the street, which in most cases results in controlling people going to work or to shop for food – and on sanctions rather than on prevention issues; in this social atmosphere, and with hospitals and general medicine doctors overworked, it is even more probable that people with light symptoms will just avoid to get in contact with the health system.

[24] I am grateful to Enrico Franco (Roma) for pointing out this shortcoming of his work.

[25] This is the point of view taken in [9]; however some of the assumptions made there were very reasonable only within the concurrent interpretation, described in a moment.

[26] A much discussed – also in general press [13] – note by an Oxford group [14] hinted that, in Italy and UK, this fraction could be as low as $\xi = 1/100$. We have ascertained that with this value of ξ, and assuming α ws not changed by the restrictive measures adopted in the meanwhile, the A-SIR model fits quite well the epidemiological data available to date. However, despite this, we do not trust this hypothesis – at least for Italy – for various reasons, such as (in order of increasing relevance): (*i*) A viral infection showing effects only in 1% of affected individuals would be rather exceptional; (*ii*) Albeit in our opinion the effect of social distancing measures adopted in Italy is sometimes overestimated, we trust that there has been some effect; (*iii*) if only 1% of infected people was detected,in some parts of Italy the infected population would be over 100%. On the other hand, the main point made by this report [14], i.e. that only a large scale serological study, checking if people have COVID antibodies, will be able to tell how diffuse the infection is – and should be performed as soon as possible – is by all means true and correct. See also [15].

[27] This is usually denoted as R_0, but we prefer to change this notation in order to avoid any confusion with initial data for the known removed $R(t)$.

[28] We tend to use only these two countries: in one case (Italy) the author has a direct knowledge of the epidemic and of different side conditions which may have affected data; in the other case (China) excellent reports by the Chinese CDC and by WHO are available [17, 18] and provide an established view. The situation in other European countries is still evolving too fast – and subject to many side conditions which I do not know – to be reliably analyzed.

[29] We stress this depends on the protocol used to trigger laboratory tests; in our general theoretical discussion, this is any such protocol and we want to discuss the consequences of changing this in the sense of more extensive tests.

[30] It should be stressed that as each of these "possible infected" might have a small probability of being actually infected (depending on the kind of contacts chain leading to him/her from known infectives), here "isolation" does not necessarily mean top grade isolation, but might amount to a very conservative lifestyle, also – and actually, especially – within home, where a large part of registered Chinese contagions took place.

[31] Direct measurement on the epidemiological data for Northern Italy show that this parameter can be reduced to about 20% of its initial value with relatively mild measures. In fact, albeit the media speak of a generalized lockdown in Italy, the measures have closed schools and a number of commercial activities, but for the rest are ac-

tually more pointing at limiting leisure walk and sports – no matter if this limitation is harmful to the immune system, so that open-air exercise was instead *recommended* in other countries, e.g. Belgium – and somewhat avoiding contacts in shops or in work environment than to a real lockdown as it was adopted in Wuhan. In fact, e.g. public transport are still working, albeit at a reduced intensity, and all kind of work which cannot be performed in remote mode is still allowed and actually an admitted reason to travel between different cities.

[32] A number of countries tried to further reduce these by forbidding citizens to get out of their home; this makes good sense in densely populated areas, but is useless in many other areas. Moreover, citizens are usually allowed to go to work if this cannot be made remotely. So the same citizen who cannot have a walk alone in the woods is then working side by side with others in chain production. More generally, the fortunate slogan "stay home" risks to hide to the general public that the problem is not to seclude oneself in self-punishment, but to *avoid contacts*; so that staying home is a tool, not the goal.

[33] In looking at this plot, one should remember that the model does not really discuss permanence in IC units, and that I_{IC} are the infected which *when detected* will require IC treatment; this may go on for a long time – which is the reason why IC units are saturated in treating COVID patients. So the plots are purely indicative, and a more detailed analysis (also with real parameters) would be needed to estimate the IC needs in the different scenarios.

[34] One point needs maybe further discussion. One may have the impression that a tight social distancing policy is, after all, keeping people from having contacts and is thus equivalent to isolate not only would-be infectives but everybody; thus this should be equally effective: actually, if people were isolated each in a different box for two or three weeks, the epidemic should stop. The point is that a "no contacts for everybody" policy is simply not feasible: ill people need help, people living in cities need to buy food, a number of essential services simply cannot be stopped.

[35] Albeit if we look at the goal of these measures, i.e. reducing the occasion of exchanging the virus, a substantial role would be played by individual protection devices, such as facial masks; or, in many European countries, these were simply not available to the general public – and in some cases neither to medical operators – thus substantially reducing the impact of these measures.

[36] Note that, following the format in which data are provided, nationwide data are cumulative, while regional data concern only "active"infections, i.e. do not include those who are dead or recovered

[37] In this respect, one should note that the data for Emilia-Romagna are also heavily depending on the Department of Piacenza, which is next to Lodi and Codogno, i.e. the first center of contagion in Italy, but for some reason was not included in the first "red area".

[38] It should be thought that also the different model of relations between Hospitals and general medicine doctors played a relevant role. By now, somebody with flu symptoms has a much higher probability of being infected with COVID than with flu, so that (as at some point in Wuhan) one can safely rely on *clinical* detection of COVID infection. The problem is how patients with this diagnosis and light symptoms are handled. This point is most relevant, but not the subject of a mathematical modeling paper, so we will not pursue it. On the other hand, I cannot avoid to express the opinion that the *real* winning point of this regional strategy was (possibly together with some luck, always needed in real life) to follow the indications of a most reliable team working on epidemics control at Padua University and Imperial College, and trust them.