

A simple SIR model with a large set of asymptomatic infectives

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1 Introduction

There is increasing evidence that one of the most difficult problems in trying to control the ongoing COVID-19 epidemic is the presence of a large cohort of *asymptomatic infectives*. The initial estimates were that registered infectives would be between $1/3$ and $1/4$ of the actual infectives [1]; there have been claims (in the famous speech by the British Government scientific advisers) that this ratio could be as little as $1/10$. In a recent contribution [2] Li *et al.* estimate that 86% of infections remain undetected; in other words only about $1/7$ of the infections are detected and can thus be isolated.

In the first part of this note we develop a SIR-type model taking into account the presence of asymptomatic, or however undetected, infective, and the substantially long time these spend being infective and not isolated; this is called A-SIR, the A standing indeed for asymptomatic. We also discuss how, in such a model, the parameters can be estimated having only data from the first part of the epidemic dynamics.

Our main interest is in understanding how relevant it can be to uncover asymptomatic infectives and promptly isolate them; we then study (numerically) how the dynamics is affected by a reduction of the infective time-span for this class.

In the second part of the note, we apply our model to the COVID-19 epidemics in Northern Italy, i.e. we estimate the model parameters from a fit of data in the first decade of March, and see how the model performs in reproducing the data of the following days March 10 through 17 (in which the further restrictive measures by the Italian Government taken on March 8 could not yet display their effect). The A-SIR model outperforms the standard SIR model in this respect.

We also run full time simulations of the model; these are not so significant, as the effective parameters of the model are and will continue to be changing due to containment measures and public awareness, but this allows us to get an estimate on what would be the epidemic peak and the time span of the epidemic if no actions were taken. These estimates are – for the parameters which best fit the present situation – about one third of those for the SIR model as far as the height of the epidemic peak is concerned, and about half for what concerns the time scale of the epidemics. Moreover, the model suggests that an overwhelming part of the population will have been in contact with the virus – most of them with no or very little symptoms – so that if permanent immunity is gained by the immune system of infected, and the virus does not mutate too quickly, one would be confident that there will be no second run of COVID in next year.

Finally, we also consider the situation in which the restrictive measures are taken into account by a “reduction factor”, and discuss on the one hand how a prompt isolation of asymptomatic infectives would change the dynamics in this framework, and on the other what the time-scale could be in this context.

We start by recalling some basic facts about the (well known) SIR model, and discuss how this can be fitted against the data available in the first phase of an epidemic. We then discuss the new A-SIR model, and repeat the same type of discussion in that context (we will find that parameters present in the two models are fitted in the same way from available data). In the last part of the note we will apply our discussion to the COVID-19 epidemics in Northern Italy.

2 The SIR model

The SIR model for the dynamics of an infective epidemic providing permanent immunity to those who have already been infected and recovered [?, ?, 5] describes a homogeneous and isolated population of N individuals by partitioning them into three classes: each individual can be either susceptible (S), infected and infective (I), or removed (R) from the epidemic dynamics, i.e. either recovered, dead, or isolated. We denote by $S(t)$, $I(t)$ and $R(t)$ the populations of these classes at time t ; by assumption, $S(t) + I(t) + R(t) = N$ for all t .

The model is described by the equations

$$dS/dt = -\alpha S I \tag{1}$$

$$dI/dt = \alpha S I - \beta I \tag{2}$$

$$dR/dt = \beta I . \tag{3}$$

In the following, the parameter

$$\gamma = \beta/\alpha \tag{4}$$

will have a special relevance.

2.1 Epidemic dynamics, epidemic peak and total number of infections

It is immediately apparent that in the SIR model the number of infected will grow as long as

$$S > \gamma ; \tag{5}$$

thus γ is also known as the *epidemic threshold*. The epidemic can develop only if the population is above the epidemic threshold.

The parameters α and β describe the contact rate and the removal rate; they depend both on the characteristics of the pathogen and on social behavior. For example, a prompt isolation of infected individuals is reflected in raising β , a reduction of social contacts is reflected in lowering α , and both these actions raise the epidemic threshold γ . If this is raised above the level of the total population N , the epidemic stops (which means the number of infected individuals starts to decrease, albeit new individuals will still be infected). The same effect can be obtained by reducing the population N (keeping α and β constant), i.e. by partitioning it into non-communicating compartments, each of them with a population below the epidemic threshold.¹

One can easily obtain the relation between I and S by considering (1) and (2), eliminating dt ; these provide

$$dI/dS = -1 + \frac{\gamma}{S} . \tag{6}$$

Upon elementary integration this yields

$$I = I_0 + (S_0 - S) + \gamma \log(S/S_0) ; \tag{7}$$

with I_0, S_0 the initial data for $I(t)$ and $S(t)$; in ordinary circumstances, i.e. unless there are naturally immune individuals, $S_0 = N - I_0 \simeq N$. (Note that we always write “log” for the natural logarithm.)

As we know (see above) that the maximum I_* of I will be reached when $S = \gamma$, we obtain from this an estimate of this maximum (note that we do *not* have an analytical estimate of the time needed to reach this maximum); writing $\gamma = \sigma N$ (with $\sigma < 1$) this reads

$$I_* = (1 - \sigma) N - \sigma N \log(1/\sigma) = [1 - \sigma - \sigma \log(1/\sigma)] N . \tag{8}$$

It follows from (8) that increasing γ , even if we do not manage to take it above the population N , leads to a reduction of the epidemic peak; if we are sufficiently near to the epidemic threshold, this reduction can be rather relevant also for a relatively moderate reduction of α and thus increase of γ .

The formula (7) also allows to obtain an estimate for another parameter describing the severity of the epidemics, i.e. the total number of individuals

$$J_\infty = \int_0^{T_0} I(t) dt$$

¹Albeit strictly speaking these predictions only hold within the SIR model, and surely the exact value of the threshold refers to this model only, the mechanism at play is rather general, and similar behaviors are met in all kind of epidemic models.

which are infected over the whole span of the epidemics². In fact, the epidemic is extinct (at an unknown time $t = T_0$) when $I = 0$; the number of susceptibles S_∞ at this stage is provided there by the (lower) root of the equation

$$I_0 + (S_0 - S) + \gamma \log(S/S_0) = 0 ;$$

as I_0 is in general rather small, and – unless some individuals are either vaccinated for or naturally immune to the disease – $S_0 = N$, we can simply look at

$$(N - S_\infty) + \gamma \log(S_\infty/N) = 0 . \tag{9}$$

This is a transcendental equation, but it is easily solved numerically if γ is known. The sought for number of overall infected individuals I_∞ is of course provided by

$$I_\infty = N - S_\infty . \tag{10}$$

Remark 1. Another key quantity is the speed at which the epidemic dynamics develops, and in particular the time t_* at which I reaches its maximum value I_* , and the time t_∞ needed for I to get to zero (and $S(t_\infty) = S_\infty$, of course). In this case one can not get an analytical estimate, but it is possible to describe how this depends on the values of α and β for a given population level N and initial conditions $\{S_0, I_0, R_0\}$. In fact, the equations (1), (2), (3) are invariant under the scaling

$$\alpha \rightarrow \lambda \alpha , \quad \beta \rightarrow \lambda \beta , \quad t \rightarrow \lambda^{-1} t . \tag{11}$$

Note that the inverse scaling of β and t is enforced by the very physical meaning of β , which is the inverse of the characteristic time for the remotion of infectives.

The meaning of (11) is that if we manage to reduce α by a factor λ , even in the case β is also reduced and thus γ remains unchanged, the speed of the epidemic dynamics is also reduced by a factor λ . On the other hand, it is clear from (1) that reducing α reduces the speed at which new infective appear; if the removal rate β is unchanged, this will make that I grows slower and reaches a lower level. See Figure 2 in this regard. \odot

2.2 Early dynamics

The SIR equations are nonlinear, and an analytical solution of them turns out to be impossible; they can of course be numerically integrated with any desired precision *if* the initial conditions *and* the value of the parameters are known. In the case of well known infective agents (e.g. for the flu virus) the parameters are known with good precision, and indeed Health Agencies are able to forecast the development of seasonal epidemics with good precision. Unfortunately this is not the case when we face a new virus, as for COVID-19.

Moreover, when we first face a new virus we only know, by definition, the early phase of the dynamics, so parameters should be extracted from such data.

²At least when this is short enough, i.e. if one can disregard deaths and new births; in particular the latter provide new fuel to the susceptible class and thus if the epidemic goes on for a long time related terms should be included in the model.

We will thus concentrate on this initial phase, with $I(t)$ and $R(t)$ rather small, and try to obtain approximate analytical expressions for the dynamics; the purpose will be to estimate the parameters α and β – and thus also the epidemic threshold γ – in this case.

Albeit we do *not* expect, for various reasons, the SIR model to provide a good description of the dynamics when the infection produces a large number of asymptomatic carriers, having an estimate of these parameters will be needed to compare the predictions which one would extract from the standard SIR model in such circumstances with those obtained by the modified model we will consider later on.

2.3 KMK approximate equations and their exact solution

In the case of “small epidemics” there is a way to obtain an analytical expression for the solutions to the SIR equations; this is associated to the names of Kermack and McKendrick [6], and we will therefore refer to it as the KMK method.

What matters more here, the expression obtained in this way is also an analytical expression holding *in the initial phase* of any epidemics, small or large, i.e. – as we will discuss in a moment – until $R(t) \ll \gamma$. Thus such an analytical expression can be compared to epidemiological data and used to estimate the unknown parameters α and β , and hence the fundamental parameter γ . Once this is done, the model can be studied numerically (or, if we are – as has to be hoped – in the favorable situation where $N \simeq \gamma$, one can set predictions on the basis of the “small SIR epidemic” model) – recalling of course that the SIR model itself is far too simple to be reliable in a situation where the actions undertaken have heavy consequences on public health – in order to have some kind of estimate of the length of the epidemics and of other relevant outcomes, such as the numbers I_* and J_∞ considered above.

It should be noted that we do not have certain knowledge about the number of infective people at each time; the best we can have is the number of people who are hospitalized or however registered by the health system. Assuming that infective people are immediately isolated, this provides an estimate (actually from below) of $R(t)$. Thus we should be able to compare the predictions for the removed class with epidemiological data, and in order to do this we should focus on $R(t)$. We stress that this problem was already clear to Kermack and McKendrick [6], see e.g. the discussion in Murray [5], and that we will basically follow their idea albeit with a relevant difference, which will allow for a simpler fit of the data.

Putting together (1) and (3), we have

$$\frac{dS}{dR} = -\frac{S}{\gamma}, \quad (12)$$

which of course provides

$$S(R) = S_0 e^{-(R-R_0)/\gamma}. \quad (13)$$

We can proceed similarly with (2) and (3), getting

$$\frac{dI}{dR} = -1 + \frac{S}{\gamma}, \quad (14)$$

where now S should be thought of as a function of R through (13). Solving this equation we get

$$I(R) = I_0 + S_0 (1 - \exp[-(R - R_0)/\gamma]) - (R - R_0). \quad (15)$$

We are however interested in the temporal dynamics of the model. In order to do this, we can substitute for $I = I(R)$ using (15) in (3); moreover we will look at the variable

$$P(t) := R(t) - R_0, \quad (16)$$

which of course satisfies $P(0) = 0$ and $dP/dt = dR/dt$. In this way we have

$$I(P) = I_0 + S_0 (1 - e^{-P/\gamma}) - P. \quad (17)$$

Plugging now this into (3), we finally get

$$\frac{dP}{dt} = b \left[I_0 + S_0 (1 - e^{-P/\gamma}) - P \right]. \quad (18)$$

This is a transcendental equation and can *not* be solved exactly. However, as long as $P/\gamma \ll 1$, i.e. as long as $R(t)$ is well below the epidemic threshold, we can replace the exponential by (a suitable truncation of) its Taylor series expansion.

Remark 2. In textbook discussions, it is usually required to consider a *second order* Taylor expansion; this guarantees that counter-terms preventing the exponential explosion of $R(t)$ (and thus the violation of the condition $R(t) \ll \gamma$) are present, and allows to obtain an analytical expression for $R(t)$ valid at *all times*. This is, more precisely, in the form

$$R(t) = \frac{\alpha^2}{S_0} \left[\phi + k_1 \tanh \left[\frac{k_1 \beta}{2} t - k_2 \right] \right], \quad (19)$$

where we have written $\phi := (S_0/\gamma - 1)$ and k_1 and k_2 are explicitly given by

$$k_1 = \sqrt{\phi^2 + 2(S_0/\gamma^2)(N - S_0)}; \quad k_2 = k_1^{-1} \operatorname{arctanh}(\phi). \quad (20)$$

As we assume there is natural immunity, we can take $S_0 \approx N$, obtaining $k_1 \approx \phi$ and hence slightly simpler complete expressions. \odot

Remark 3. In particular, in this case the maximum of $R'(t)$ – and hence of $I(t)$, see (3) – is obtained at time

$$t_* = \frac{2 \operatorname{arctanh}(\phi)}{\beta \phi};$$

as our result holds for the “small epidemics”, ϕ is small and we can write

$$\tilde{t}_* \simeq \frac{2}{\beta \phi} + \frac{2 \phi}{3 \beta}.$$

Note that t_* is therefore rapidly decreasing with ϕ (for small ϕ). On the other hand, looking back at (8), and noticing that in terms of the notation used there $\sigma = 1/(1+\phi)$, we obtain immediately that I_* grows with ϕ .³ \odot

2.4 Small time solution of the KMK equations

However, here we are less ambitious: we can in any case only fit the initial phase of the epidemic, which shows an exponential increase of $R(t)$, and correspondingly we can expand the exponential in (18) at *first* order in P/γ . This yields the equation

$$\frac{dP}{dt} = \beta \left[I_0 + \left(\frac{S_0}{\gamma} - 1 \right) P \right]. \quad (21)$$

which is immediately solved to give, with initial condition $P(0) = 0$,

$$P(t) = I_0 \frac{\exp[\beta(S_0/\gamma - 1)t] - 1}{(S_0/\gamma - 1)}. \quad (22)$$

Introducing the parameter, which we stress is *not* assumed to be small,

$$\phi := \frac{S_0}{\gamma} - 1, \quad (23)$$

the above is more simply written as

$$P(t) = \frac{I_0}{\phi} [e^{\beta\phi t} - 1], \quad (24)$$

and finally we get

$$R(t) = R_0 + \frac{I_0}{\phi} [e^{\beta\phi t} - 1]. \quad (25)$$

As expected this – at difference with (19) – is not saturating but just expanding exponentially, and thus cannot be valid for all times, but only for t sufficiently small.

The expression (25) can then be expanded in series to give the small t expression of the solution, which can be fitted against experimental data thus determining (some of) the parameters.

Remark 4. It is relevant – for the following of our discussion – to note that the solution (25) can be obtained also in a different way, i.e. noticing that in the initial phase of the epidemic the number of susceptibles vary very little and can thus be

³This also means that if one would be able to tune the parameters α and β (and hence ϕ) there would be a contrast between trying to have a low I_* and hence a small ϕ , and trying not to have the epidemic running for too long – which can be devastating on social and economic grounds. If, on the other way, the priority from the temporal point of view is on slowing down the epidemic, e.g. to have the time to prepare the health system facing the peak, a small ϕ should be pursued.

considered as constant, $S(t) \simeq S_0$. Within his approximation, and writing again $\phi = (S_0/\gamma - 1)$, the SIR equations reduce to

$$\begin{cases} dI/dt = \beta \phi I \\ dR/dt = \beta I \end{cases} ; \quad (26)$$

this is a linear system, and it is promptly solved to yield indeed (25).

Note this approach is actually simpler than the one followed above (so the reader may wonder why we have not taken this immediately), but on the one hand the procedure given above is along the lines of the tradition of SIR analysis, and on the other hand having seen that derivation gives us more confidence that a rough approach as this one provides the same results as a more refined one; this will be of use dealing with more complex models, where the Kermack-McKendrick approach can not be followed, see Section 3.2 below. \odot

2.5 Fitting the SIR parameters

Note that the solution (25) depends on three parameters, i.e. β , I_0 and ϕ , which in turn depends on the known number $S_0 \simeq N$ and γ . None of the parameters $\{\beta, \phi, I_0\}$ is known, but β can somehow be estimated as it corresponds to the inverse of the typical removal time (for trivial infections, this corresponds to the time of healing; in the case of COVID it is the time from infection to isolation), and similarly once we fix a time $t = 0$ the number I_0 can be estimated *a posteriori* looking at epidemiological data for the next few days and depending on our estimate of β .

In order to estimate the parameters on the basis of the measurements of R , we can work either on R itself, or on its logarithm. That is, we have two alternative ways to proceed.

(1) *Working on the time series for $R(t)$.*

We fit the time series around t_0 by

$$R(t) = r_0 + r_1 t + \frac{1}{2} r_2 t^2 ; \quad (27)$$

Having these coefficients r_k , we can compare with the series expansion for $R(t)$ given by (25), which is just

$$R(t) = R_0 + (\beta I_0) t + \frac{1}{2} (\beta^2 I_0 \phi) t^2 . \quad (28)$$

We obtain easily that – using also the definition of ϕ (23) – our parameters and the associated parameter γ are given by

$$R_0 = r_0 , \quad I_0 = \frac{r_1}{\beta} , \quad \phi = \frac{r_2}{r_1 \beta} ; \quad \gamma = \frac{\beta S_0 r_1}{\beta r_1 + r_2} . \quad (29)$$

(2) *Working on the time series for $\log[R(t)]$.*

As $R(t)$ grows – in the early phase – in a substantially exponential way, one usually deals with data in logarithmic form; that is one has a fit for $\log[R(t)]$, say of the form⁴

$$\log[R(t)] = A + Bt + \frac{1}{2}Ct^2. \quad (30)$$

Comparing with the series expansion of $\log[R(t)]$ for R as in (25), i.e.

$$\log[R(t)] = \log(R_0) + \beta \frac{I_0}{R_0} t - \frac{1}{2} \left(\frac{\beta I_0}{R_0} \right)^2 \left(1 - \phi \frac{R_0}{I_0} \right) t^2, \quad (31)$$

we obtain that the I_0 , ϕ and γ parameters can be estimated as

$$R_0 = e^A, \quad I_0 = \frac{B}{\beta} e^A, \quad \phi = \frac{B^2 + 2C}{\beta B}; \quad \gamma = \frac{\beta S_0 B}{\beta B + B^2 + 2C}. \quad (32)$$

3 A model with asymptomatic infectives

It may happen to have an epidemic such that a rather large fraction of infected people are actually asymptomatic, but still fully infective, as it appears to be the case for COVID-19.⁵

A little reflection shows that the presence of a large population of asymptomatic infectives, or however of infectives which show only very mild symptoms, easily thought not to be related with the concerned infective agent, changes the dynamics in two – contrasting – ways:

1. On the one hand, they are a formidable vehicle of contagion, as they have no reason to take special precautions, and get in contact with a number of people which themselves do not take the due precautions (which would be taken in the case of an individual with evident symptoms);
2. On the other hand, assuming once the infection is ceased they have acquired permanent immunity, they contribute to group immunity reached once the population of susceptibles falls below the epidemic threshold.

We are thus going to study how the SIR dynamics is altered by the presence of a large class of asymptomatic infectives.

Remark 5. An obvious but important Remark is in order here. If we find out that known infectives are only a fraction $\xi < 1$ of the total infectives, this means that on the one hand the mortality rate (number of deceased over number of infected) is actually smaller by the same factor. On the other hand, the total number of infected persons

⁴Obviously all series expansions could be performed at higher orders as well, but we believe this would have little sense, as in the early phase of an epidemic one is by definition dealing with a limited set of data, and already including second order fitting is questionable.

⁵Actually in this case the exact meaning of this “fully” is not completely clear. While there is a generalized consensus on the fact that infection can be transmitted by asymptomatic people, and a fortiori by people with very weak symptoms, it is not certain if they are as infective as people having more serious symptoms.

is increased by a factor ξ^{-1} , so that it looks more difficult to stop the spread of the epidemics, and the final number of infected will be quite large. In this respect, one should however remember that the total number of casualties does not depend only on the total number of individuals with symptoms (in China this ratio was around 2% [7]) but also on the number of patients needing Intensive Care (in China this was estimated at 20 % of hospitalized patients [7]) and on the availability of such care; from this point of view, slowing down the pace of the epidemics can substantially lower the death toll. \odot

3.1 The A-SIR model

We will formulate a very simplified model, where infective people are either symptomatic or asymptomatic. A more refined subdivision of their state would be more realistic, but the discussion of this simple case will suffice to show how to proceed in a more general setting.

In our model we still assume permanent immunity of individuals who have been infected and recovered, and constant population. We will have susceptibles $S(t)$ in a unique class, but two classes of infected and infective people: symptomatic $I(t)$ and asymptomatic $J(t)$; and similarly two classes of removed people: registered removed $R(t)$ and unregistered removed (those who were passing unnoticed through the infection) $U(t)$. Symptomatic infectives are removed by the epidemic dynamics through isolation (in hospital or at home) at a removal rate β (thus with typical delay β^{-1} , while asymptomatic people are removed from the epidemic dynamics through spontaneous recovery, at a recovery rate $\eta \ll \beta$, thus after a typical time $\eta^{-1} \gg \beta^{-1}$).

We assume that both classes of infected people are infective in the same way, and that an individual who gets infected passes with probability ξ to the class I and with probability $(1 - \xi)$ to the class J .⁶

Our model, which we will call A-SIR (Asymptomatic-SIR) will then be

$$\begin{aligned}
 dS/dt &= -\alpha S(I + J) \\
 dI/dt &= \alpha \xi S(I + J) - \beta I \\
 dJ/dt &= \alpha(1 - \xi) S(I + J) - \eta J \\
 dR/dt &= \beta I \\
 dU/dt &= \eta J .
 \end{aligned} \tag{33}$$

Note that the last two equations amount to an integral, i.e. are solved by

$$R(t) = R_0 + \beta \int_0^t I(\tau) d\tau , \quad U(t) = U_0 + \eta \int_0^t J(\tau) d\tau . \tag{34}$$

⁶In the case of COVID-19, it is known that the incubation time is about 5.1 days; assuming that symptomatic infection is promptly recognized and swiftly treated, epidemiological and clinical data suggest the approximate values (note that asymptomatic removal time η^{-1} includes both the incubation time and the healing time) $\beta^{-1} \simeq 5 - 7$, $\eta^{-1} \simeq 14 - 21$ for the removal and recovery rates; the value of ξ is more controversial, as mentioned in the Introduction.

Moreover, the total population $N = S + I + J + R + U$ is constant.

Some general considerations can be done immediately. First of all, we note that $I(t)$ will increase as far as the condition

$$\alpha \xi S (I + J) > \beta I$$

is satisfied; that is, as far as

$$S > \gamma_1 := \frac{1}{\xi} \frac{\beta}{\alpha} \frac{I}{I + J} . \quad (35)$$

Thus the epidemic threshold (for symptomatic patients) γ_1 depends both on the fixed parameters ξ, α, β and on the variable ratio $x(t)$ of known infective over total infective,

$$x(t) := \frac{I(t)}{I(t) + J(t)} . \quad (36)$$

Similarly, the number of asymptomatic infectives $J(t)$ will grow as far as

$$\alpha (1 - \xi) S (I + J) > \eta J$$

is satisfied, i.e. as far as

$$S > \gamma_2 := \frac{1}{1 - \xi} \frac{\eta}{\alpha} \frac{J}{I + J} . \quad (37)$$

Again the epidemic threshold (for asymptomatic patients) γ_2 depends both on the fixed parameters ξ, α, η and on the variable ratio $y(t) = 1 - x(t)$ of asymptomatic – and thus “hidden” – infectives over total infectives,

$$y(t) := \frac{J(t)}{I(t) + J(t)} . \quad (38)$$

Note that

$$\frac{\gamma_1}{\gamma_2} = \left(\frac{1 - \xi}{\xi} \right) \left(\frac{\beta}{\eta} \right) \left(\frac{I}{J} \right) .$$

As we expect on the one hand to have $\xi < 1/2$ and $\beta > \eta$, but on the other hand $I < J$, we cannot claim there is a definite ordering between γ_1 and γ_2 ; this means that we will have situations where I declines and J is still growing, but the opposite is also possible.

We expect that in the very first phase –when the different removal times have not yet shown their effects – we have

$$J \simeq \frac{1 - \xi}{\xi} I ;$$

under this condition, we get

$$\frac{\gamma_1}{\gamma_2} \simeq \frac{\beta}{\eta} > 1 .$$

The evolution law for the quantities x and y can be obtained through simple computations; using the equations (33) and $y = 1 - x$, we get

$$dx/dt = \alpha\xi S - (\alpha S + \beta - \eta)x + (\beta - \eta)x^2 ; \quad (39)$$

and similarly for $y(t)$.

Note that – as we assume $b > \eta$ – for a given S the x dynamics has an attractive fixed point in

$$x_0 = \frac{(\alpha S + \beta + \eta)}{2(\beta - \eta)} \left(1 - \sqrt{1 - \frac{4\alpha S \xi (\beta - \eta)}{(\alpha S + \beta + \eta)^2}} \right) .$$

3.2 Early dynamics

It is quite clear that we can not go through the Kermack-McKendrick procedure to obtain approximate equations valid in the case of “small epidemics”, not even through the simplified (first rather than second order) procedure valid only for the initial times we have used above.

We can however go through the even simpler approach mentioned in Remark 4 (and which we have seen there produces the same results as the KMK procedure). With $S(t) \simeq S_0$, the above equations reduce to a *linear* system of four equations with constant coefficients, or more precisely to a “master” system of two equations

$$\frac{dI}{dt} = (\alpha \xi S_0 - \beta)I + (\alpha \xi S_0)J \quad (40)$$

$$\frac{dJ}{dt} = [\alpha(1 - \xi)S_0]I + [\alpha(1 - \xi)S_0 - \eta]J \quad (41)$$

plus two auxiliary equations amounting to a direct integration, which are just (34).

As for the two equations, (40) and (41), we can get their solution in explicit form by means of some standard algebra; they are slightly involved and we do not report them here.

With these, we can compute $R(t)$ and $U(t)$; their explicit expressions are also quite involved, and we do not report them here.

3.3 Fitting the parameters

We can now proceed as in Section 2.5, i.e. series expand $R(t)$ in order to fit the parameters⁷. From the explicit expression of $R(t)$ we get

$$R(t) \simeq R_0 + \beta I_0 t + \frac{1}{2} \beta [\alpha(I_0 + J_0)S_0\xi - \beta I_0] t^2 ; \quad (42)$$

$$\log[R(t)] \simeq \log(R_0) + \beta \frac{I_0}{R_0} t$$

⁷It should be stressed that, by definition, we only have access to the $R(t)$ time series. So we can only estimate the parameters by using this.

$$+ \frac{1}{2} \frac{\beta[\alpha(I_0 + J_0)R_0S_0\xi - \beta I_0(I_0 + R_0)]}{R_0^2} t^2 . \quad (43)$$

Comparing these with the generic form of the fits (27) and (30), which we repeat here for convenience of the reader,

$$\begin{aligned} R(t) &\simeq r_0 + r_1 t + \frac{1}{2} r_2 t^2 , \\ \log[R(t)] &\simeq A + B t + \frac{1}{2} C t^2 , \end{aligned}$$

we can express the parameters I_0 and $\gamma = \beta/\alpha$. Note that we can not express both γ and J_0 with the same fitting, as both of them only appear in the coefficient of the quadratic term⁸. Note also that in this context γ is *not* any more the epidemic threshold, as discussed in Section 3.1; the time-varying epidemic threshold $\gamma_1 = (\gamma/\xi)[I/(I + J)] = (\gamma/\xi)x(t)$ is however expressed in terms of γ , so that it makes sense to fit it.

Actually, since new infected are with probability ξ in the class I and with probability $(1 - \xi)$ in the class J , it is natural to set as initial conditions

$$J_0 = \left(\frac{1 - \xi}{\xi} \right) I_0 ; \quad (44)$$

with this assumption, we have

$$\gamma_1 = \gamma . \quad (45)$$

It should be noted that actually if we want to fit γ we need to have some estimate on J_0 (while I_0 can be fitted from first order coefficient in the series for $R(t)$ or $\log[R(t)]$); to this aim we will use consistently (44).

In particular, using the fit of R we get (through this assumption)

$$R_0 = r_0 , \quad I_0 = \frac{r_1}{\beta} , \quad \gamma = \frac{\beta r_1 S_0}{(\beta r_1 + r_2)} . \quad (46)$$

Using instead the fit of $\log[R(t)]$, and again the assumption (??), we get

$$R_0 = e^A , \quad I_0 = \frac{B e^A}{\beta} , \quad \gamma = \frac{\beta S_0 B}{\beta B + B^2 + 2C} . \quad (47)$$

It is immediate to check that these expressions – which we recall were obtained under the assumption (44) for J_0 – are exactly the same as for the SIR model; see (29) and (32).

Once the parameters are estimated, the nonlinear equations (33) can be solved numerically.

⁸One could try to fit higher order truncation of the Taylor series, but –as already remarked – this would not be reliable in the presence of a short time series.

4 Comparing SIR and A-SIR dynamics. The COVID-19 epidemics in Northern Italy

As discussed above, we are not able to extract relevant analytical predictions from the nonlinear A-SIR equations; thus the only way to compare the predictions of this model with those of a standard SIR model (or actually variations on it, such as the SEIR model [5]) – and thus see how the presence of a large class of asymptomatic infectives affects the dynamics – is at present by running numerical simulations, i.e. numerically integrate the SIR and the A-SIR equations for *coherent* sets of parameters. By coherent here we mean “extracted from the same time series for $R(t)$ in the early phase of the epidemics”.

We thus need a concrete given set of data to be used for the comparison. We will use those for the ongoing COVID-19 epidemics in Northern Italy.

4.1 Epidemiological data

The data for the cumulative number of registered infected communicated by the Italian Health System is reported in Table I for the first part of March. One should note, in this respect, that the first cases in Italy (apart from sporadic cases) were discovered on February 21. The public awareness campaign started immediately, the first local mild restrictive measures were taken a few days later (February 24), and more restrictive measures involving the most affected areas were taken on March 1⁹. A more stringent set of measures went into effect for the whole nation on March 8.

Thus the epidemics developed with varying parameters. Moreover, as the incubation time for COVID ranges from 2 to 10 days, with a mean time of 5.1 days [1], there is a notable delay in the effect of any measure. In this sense, our fits cannot give any kind of prediction on the future development of the actual epidemic dynamics, and should rather be seen as a case study for the comparison between SIR and A-SIR model. On the other hand, we will explore several possibilities concerning the main control parameter, and see how these would change the dynamics starting from the parameters resulting from the fit with the real data.

day	Mar 1	Mar 2	Mar 3	Mar 4	Mar 5	Mar 6
R	1694	1835	2502	3089	3858	4636
day	Mar 7	Mar 8	Mar 9	Mar 10	Mar 11	Mar 12
R	5883	7375	9172	10149	12462	15113
day	Mar 13	Mar 14	Mar 15	Mar 16	Mar 17	Mar 18
R	17660	21157	24747	27980	31506	35713

Table I. Cumulative number of COVID-19 registered infect in Italy in the first part

⁹Due to a leak of information, a number of people fled from the most affected area before the prohibition to do so went into effect; this has most probably pushed the spreading of the infection in different regions.

of March [8, 9]. In our fits, $t = 0$ corresponds to March 5 and (t_i, t_f) to the period March 1 through March 10.

4.2 Fit of the data

For our fitting, we will consider the data of the period March 1 through March 10, denoted in the following as t_i and t_f respectively; this leaves us some later days to compare the functions obtained through the fit with subsequent evolution.

The best direct fit of $R(t)$ through a quadratic function

$$R(t) \approx r_0 + r_1 t + \frac{1}{2} r_2 t^2 := f(t) \quad (48)$$

is obtained with the constants

$$r_0 = 3862.32, \quad r_1 = 966.54, \quad r_2 = 80.35. \quad (49)$$

The fit is reasonably good in the considered time interval (t_i, t_f) , but fails completely for $t < t_i$ (in Figure 1 we use data from February 24 on) and is rather poor for $t > t_f$. This is not surprising, as we know that $R(t)$ is, in this early phase, growing through a slightly corrected exponential law, see (25).¹⁰

Let us then look at the fit of $\log[R(t)]$ as

$$\log[R(t)] \approx A + B t + \frac{1}{2} C t^2 := F(t). \quad (50)$$

In this case the best fit is obtained with the constants

$$A = 8.26648, \quad B = 0.221083, \quad C = -0.00430354. \quad (51)$$

In this case the fit is very good not only within (t_i, t_f) but also outside it, at least for the time being. We will thus work only with this (exponential) fit. Note that in Figure 1 we consider data for $R(t)$, and correspondingly plot the function

$$\mathcal{F}(t) := \exp[F(t)]. \quad (52)$$

We will consider these numbers for the coefficients $\{r_0, r_1, r_2\}$ or for the coefficients $\{A, B, C\}$ as experimental measurements.

We can now use the formulas obtained before, both for the SIR and the A-SIR model, to estimate the parameters of these models in terms of these fits following the discussion in Sections 2.5 and 3.3.

¹⁰Note however that here the fit has not the goal to provide an analytical description of $R(t)$ for a larger interval of time, but only to estimate some parameters.

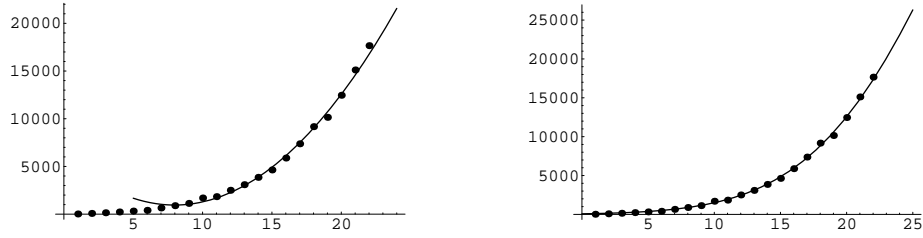


Figure 1: Data for $R(t)$ in the COVID epidemics in Northern Italy from February 24 to March 13, with fits obtained using data for March 1 through March 10. Left: polynomial (quadratic) fit (48); Right: corrected exponential fit (52).

4.3 SIR and A-SIR parameters for the COVID-19 in Northern Italy

We have remarked in Section 3.3 that the SIR and A-SIR models yield (under the (44) assumption for J_0) exactly the same values for the I_0 and γ parameters. Now we want to estimate these values for the data given in Section 4.2; this amounts to a direct application of formulas (46) and (47) (or equivalently (29) and (32), as already remarked).

The values obtained using the direct fit of R are tabulated for different values of β in Table II.a.

We can also proceed by using the fit of $\log[R(t)]$; the values obtained in this way are tabulated for different values of β in Table II.b.

We remind that the delay time $\delta = \beta^{-1}$ from infection to arise of symptoms is estimated to be around $\delta \simeq 5.2$ [1]; thus albeit we have tabulated several options for β , the two central columns are the relevant ones for our discussion. In all cases, S_0/γ is quite far from one and ϕ from zero, so one can not rely on the “small epidemic” formulas [5].

We will thus resort to numerical integration, see next Section.

β	1/3	1/4	1/5	1/6	1/7	1/8
I_0	2900	3866	4833	5799	6766	7732
ϕ	0.25	0.33	0.42	0.50	0.58	0.67
S_0/γ	1.25	1.33	1.42	1.50	1.58	1.67

Table II.a. Parameters for the SIR and A-SIR models obtained through the SIR quadratic local fit of $R(t)$; see (29), (46).

β	1/3	1/4	1/5	1/6	1/7	1/8
I_0	2581	3441	4301	5162	6022	6883
ϕ	0.55	0.73	0.91	1.09	1.28	1.46
S_0/γ	1.55	1.73	1.91	2.09	2.28	2.46

Table II.b. Parameters for the SIR and A-SIR models obtained through the SIR modified exponential local fit of $R(t)$; see (32), (47).

5 Numerical simulations. Timescale of the epidemic

We can now run numerical simulations with the SIR and the A-SIR equations and the parameters which have been determined in the previous Section, and which depend on the removal rate β . In all of our simulations, day one is February 21, so the fitting period (t_i, t_f) is centered around day 14.

5.1 General study

Note that in the A-SIR equation we also need to introduce the removal rate for asymptomatic individuals, i.e. η ; this is related to the time length $\delta = \eta^{-1}$ of their infective period, which is equal to the incubation time plus the spontaneous healing time. While the former is around $\beta^{-1} \simeq 5$, the latter is generally considered to be around 14 days, albeit we know that for hospitalized patients this may be longer. We ran a number of simulations, both for the SIR and the A-SIR dynamics, with varying β and with $\eta = 1/21$, see Figures 2 and 4. It should be stressed that the situation is quite different in the cases of SIR and of A-SIR dynamics.

It is natural to look at these simulations wondering how long the epidemic will last. This is not a well posed question, because there are restrictive measures being taken which will reduce the contact rate and thus the spread of the epidemic – and if these show to be not sufficient one would expect new measures are taken. So, these simulations can at their best what would be the behavior (of the system described by the SIR or A-SIR equations, which do not necessarily describe correctly the COVID epidemics) *with constant coefficients*. On the other hand, they can give an idea of what should be expected in case of no action.

It should be stressed in this context that the containment measures do not act on β , but on α ; albeit in general $\alpha = \beta/\gamma$, in studying the effect of restrictive measures it is more convenient to consider the reduction factor r . That is, if the fit of the initial phase of the epidemic yields $\alpha_0 = \beta_0/\gamma_0$ (where γ_0 is determined through the formulas of Sections 2.5 and 3.3), we consider in later phases a contact rate

$$\alpha = r \alpha_0, \quad 0 < r < 1 .$$

At the moment in Italy we get $r \simeq 0.5$, albeit in some regions the analysis of epidemic data yields $r = 0.25$; in these weeks Korea achieved a reduction factor of $r \simeq 0.1$ [10].

We thus run also a number of simulations at fixed β and varying r ; these can give an idea of the impact of containment measures on the development of the epidemic. See Figures 2, 4 and 6.

However, the real concrete interest of this study is in a different point. That is, there is considerable debate on the most appropriate way to use laboratory exams, and in particular if there should be a generalized COVID testing, at least of those having had contacts with known infects, or if only clinically suspect cases should be tested. We are of course aware that the real obstacle to a generalized testing (which should however be repeated over and over to be sure the individual has not been infected since the last test) is of practical nature, as testing a population of several tens of million people –not to say about China – is unfeasible, so that this alternative is a concrete one only in small communities (which could be isolated areas or also e.g. the community of people working in a Hospital).

In any case, we want to study what the impact of reducing δ , thus raising η , would be on the development of the A-SIR dynamics. This is illustrated in Figures 4 and 6.

The results of our numerical simulations, see in particular Figure 4, suggest that the epidemic in Northern Italy is (or more precisely, was before the latter restrictive measures went into operation) better described, in terms of our model, by the situation with $\xi \in (1/10, 7)$, $\beta = 1/7$ and $\eta = 1/21$. We will thus devote further analysis to this setting.

5.2 More detailed study with selected parameters

As mentioned in the previous subsection, we will devote a more detailed study to the case with $\beta = 1/7$ and $\xi = 1/7$ or $\xi = 1/10$. It should be stressed that these parameters cannot be altered: indeed, ξ depends on the interaction of the virus with human bodies and is thus fixed by Nature, while a removal time of β can hardly be compressed considering that typically the first symptoms arise after 5 days, but these are usually weak and thus receive attention (especially in a difficult situation like the present one) only after some time.

On the other hand, it is conceivable that η^{-1} could somehow be compressed if a general screening was conducted, or more simply if all individuals having even the lightest symptoms would more rigorously isolate themselves. At the same time, the contact rate α can be reduced by a more or less rigorous lockout; in our discussion, this reduction is encoded in the reduction parameter r , which yields the ratio of the achieved contact rate over the "natural" one – i.e. the one measured at the beginning of the epidemic.

In the case of Korea, which is similar in several respects¹¹ (total population and political system) to Italy, the reduction factor was measured to be $r = 0.2$ at the beginning of March, and $r = 0.03$ at mid-March. It is thus conceivable that similar results can be obtained in Italy; this would lead the population to be *below* the epidemic threshold, and thus lead effectively to a stop of it (with all due cautions concerning return infections from other countries, containment

¹¹But has a more than double population density, 507 against 201 inhabitants per square kilometer; both countries have wide fluctuations in local population density, due to the presence of substantial mountain ranges.

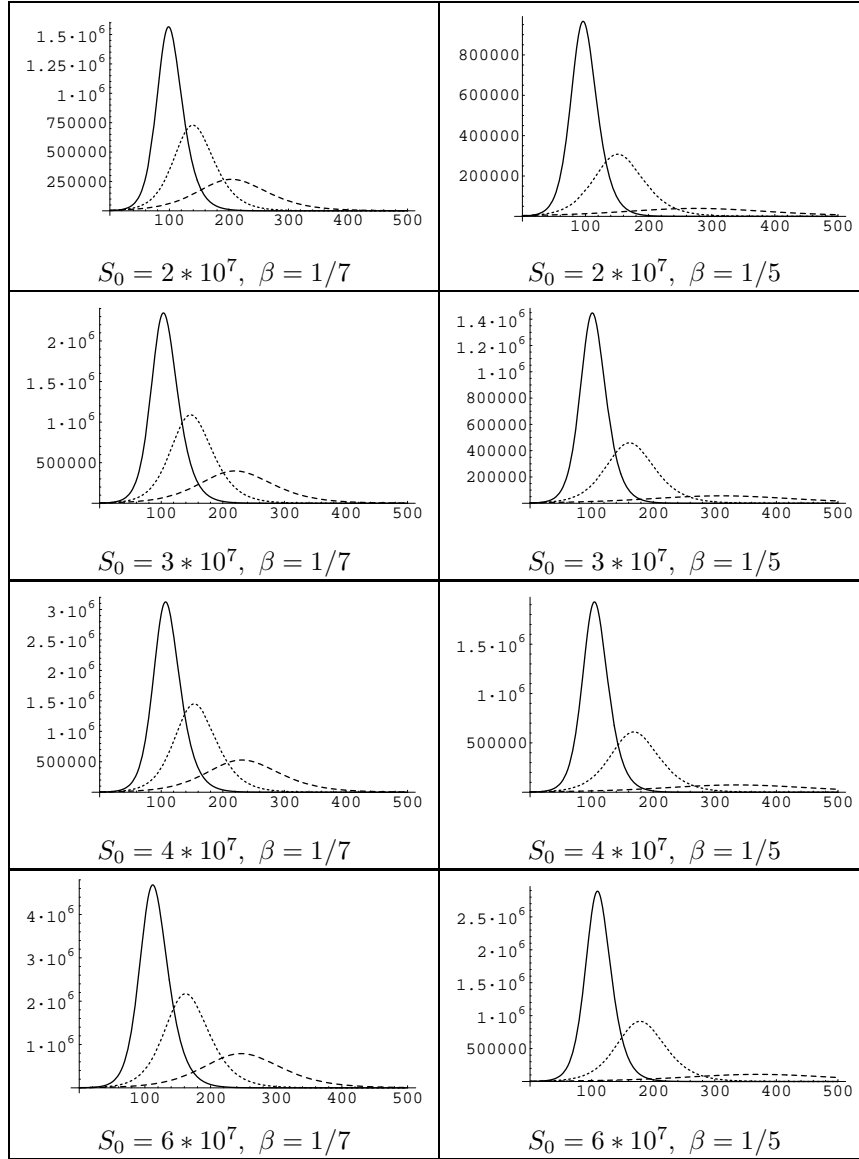


Figure 2: Numerical solution of the SIR equations for different values of total population S_0 , removal rate β , and reduction factor r , using for the parameters I_0 and γ the fit of eqs. (46), (47) on the basis of the data of Table I. In all cases, the plots of $I(t)$ – where t is measured in days – are shown for: $r = 1$ (solid curve), $r = 0.85$ (dotted curve) and $r = 0.75$ (dashed curve). In all cases, the curve for $R(t)$ outside the fitting region but within the presently available data does not fit at all the latter, so we are glad to say these are not predictions of the evolution of the real COVID epidemics in Northern Italy.

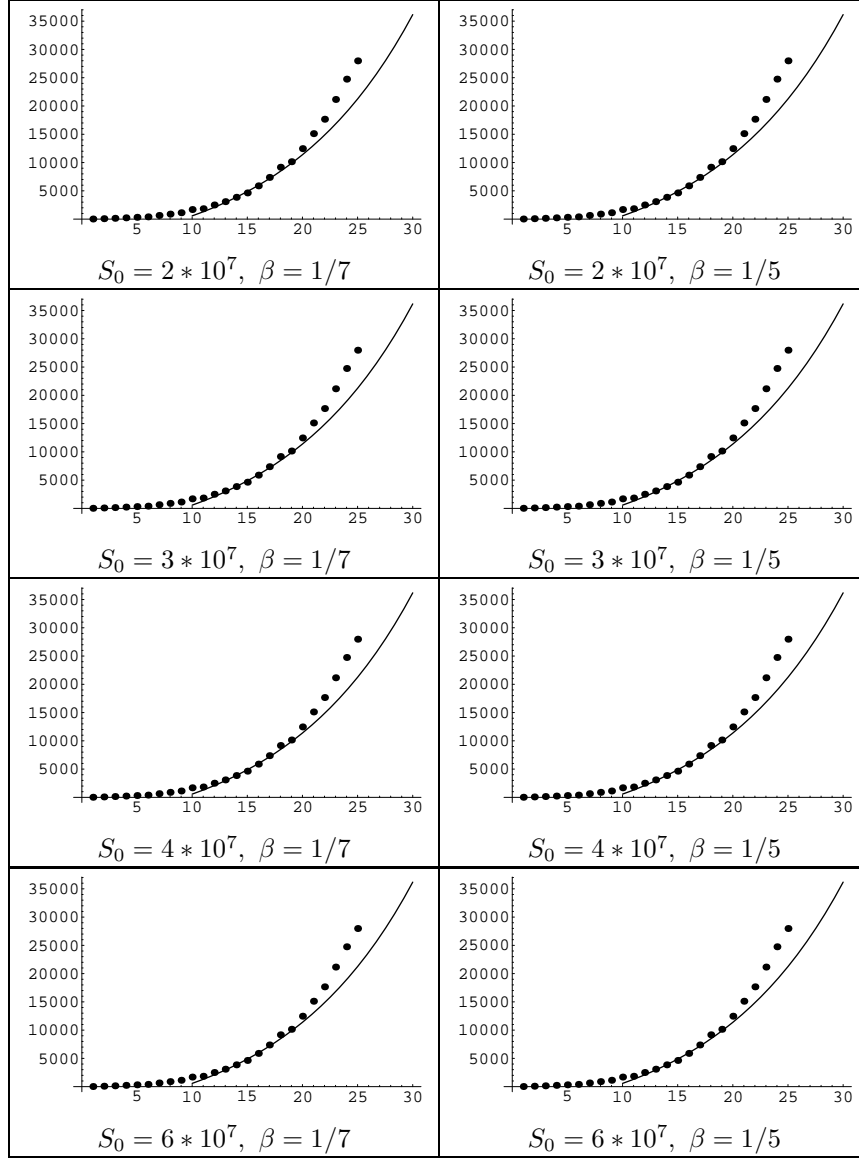


Figure 3: Plot of the data for the COVID epidemics in Italy versus the numerical integration of the SIR model (with $r = 1$) for different values of S and β . The parameters I_0 and γ were obtained through the fit of eqs. (46), (47) on the basis of the data of Table I for the period 1-10 March; plotted data go until March 15. In all cases the prediction of subsequent data is rather poor and the error has the wrong sign, as we expect the contact rate to be diminishing in time due to public awareness and restrictive measures

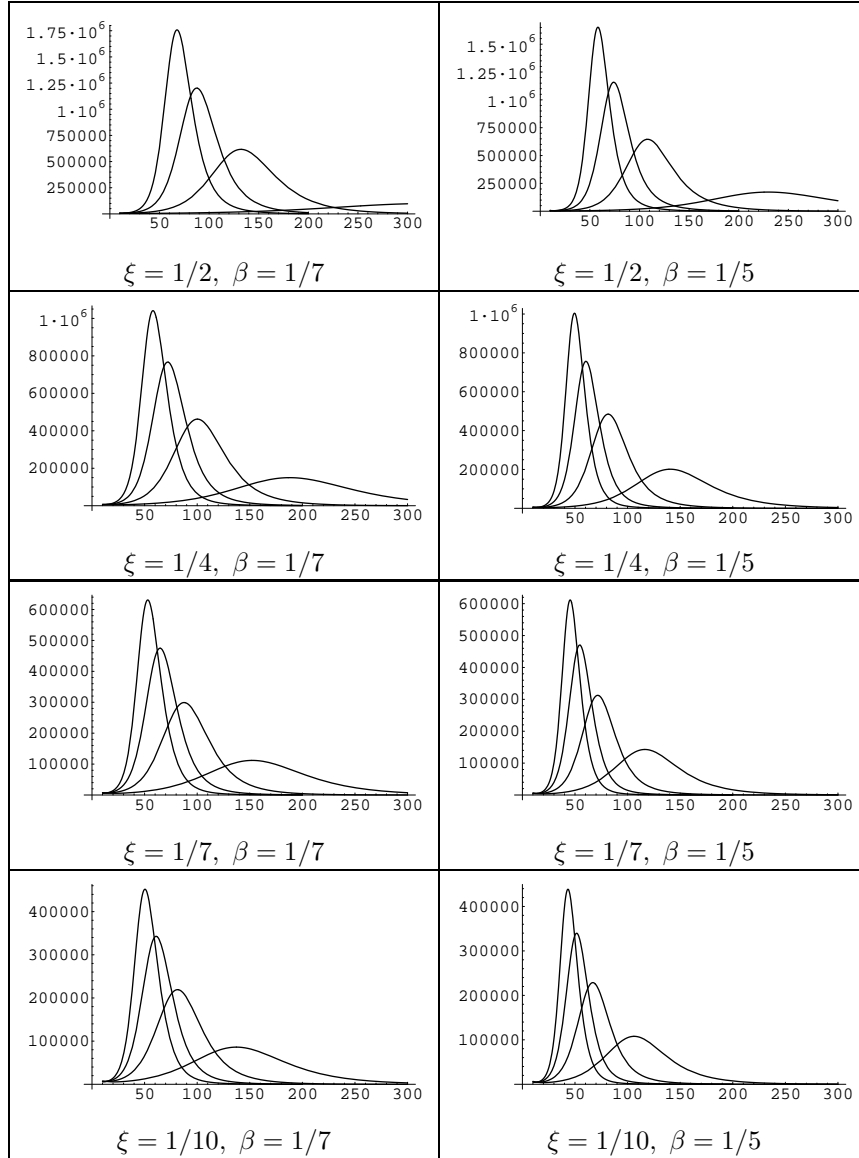


Figure 4: Numerical solution of the A-SIR equations for $S_0 = 2 * 10^7$, $\eta = 1/21$ and different values of removal rate β , symptomatic infection rate ξ , and reduction factor r , using for the parameters I_0 and γ the fit of eqs. (46), (47) on the basis of the data of Table I. In all cases, the plots of $I(t)$ – where t is measured in days – are shown for: $r = 1$, $r = 0.8$, $r = 0.6$ and $r = 0.4$; the curves for higher r are those with higher peak.

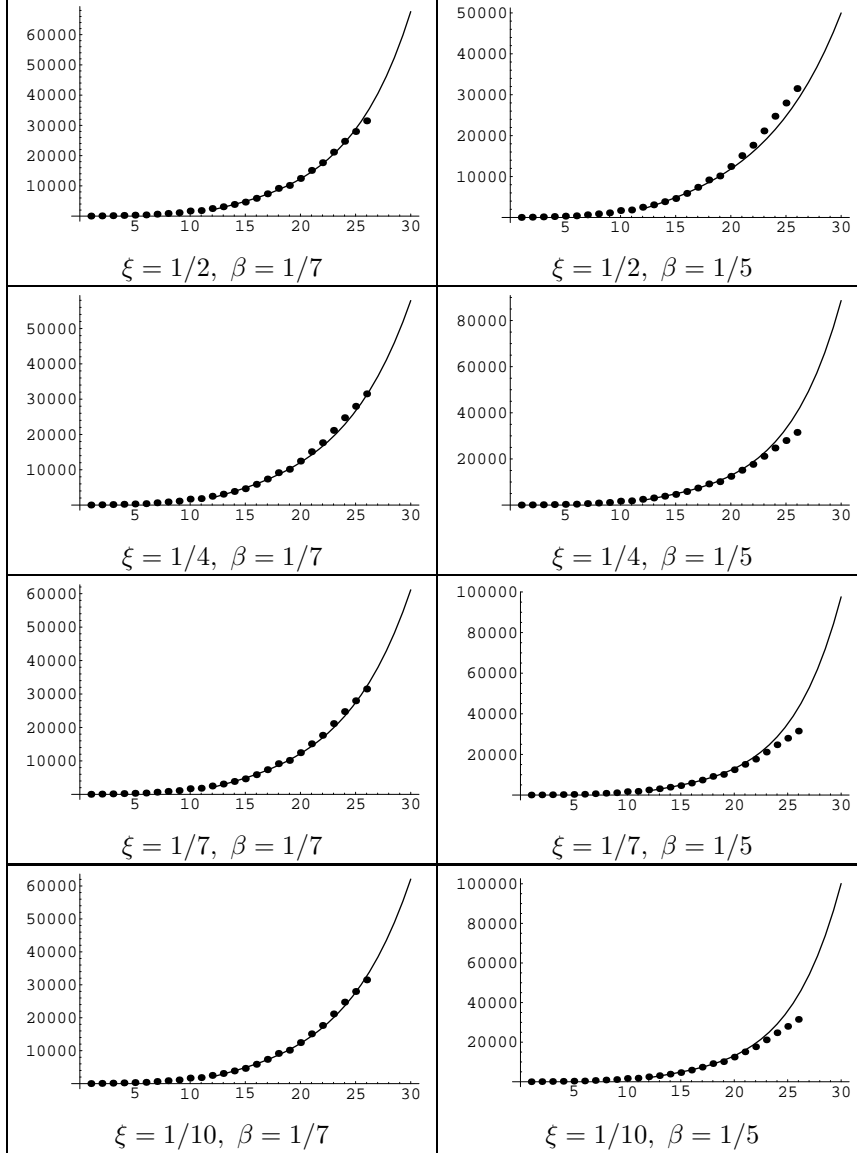


Figure 5: Plot of the data for the COVID epidemics in Italy versus the numerical integration of the A-SIR model (with $r = 1$) for $S_0 = 2 * 10^7$ (the total population of the three most affected regions), $\eta = 1/21$, and various values of the parameters β and ξ . The parameters I_0 and γ were obtained through the fit of eqs. (46), (47) on the basis of the data of Table I for the period 1-10 March; plotted data go until March 17.

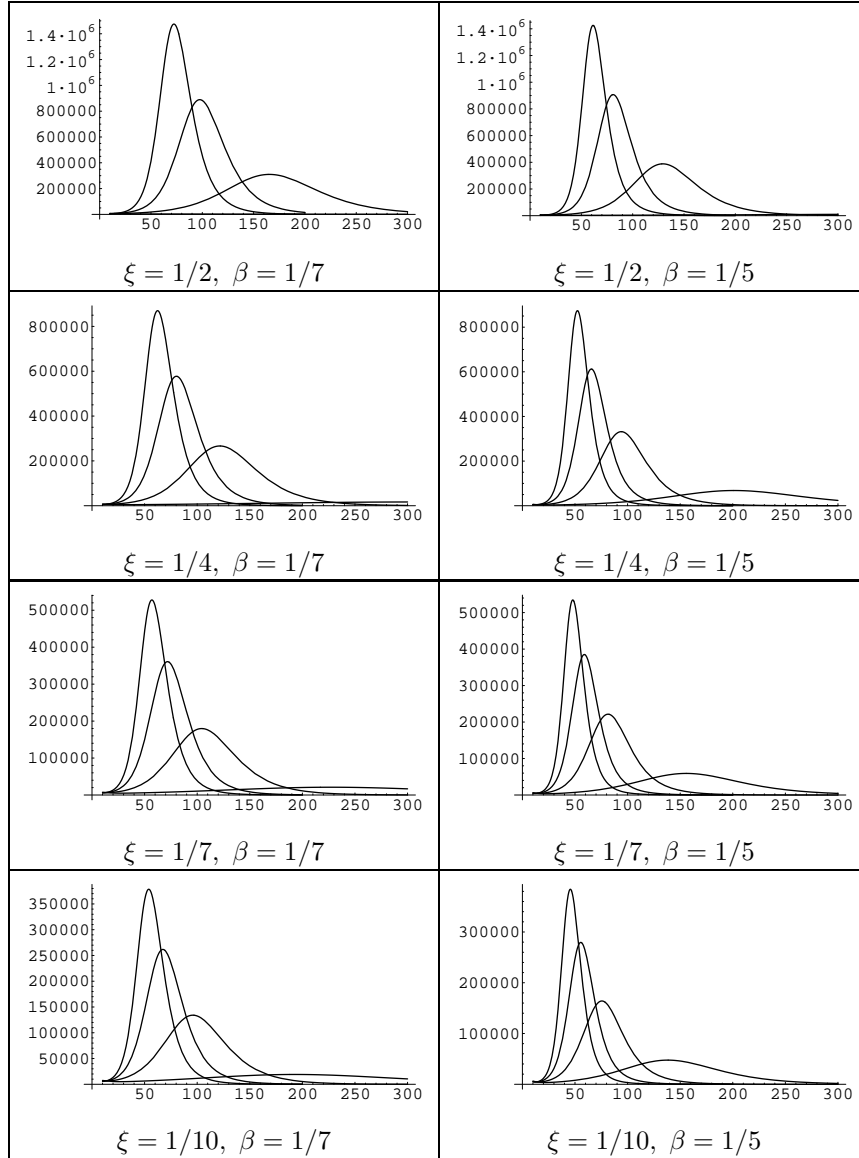


Figure 6: Numerical solution of the A-SIR equations for $S_0 = 2 * 10^7$, $\eta = 1/14$ and different values of removal rate β , symptomatic infection rate ξ , and reduction factor r , using for the parameters I_0 and γ the fit of eqs. (46), (47) on the basis of the data of Table I. In all cases, the plots of $I(t)$ – where t is measured in days – are shown for: $r = 1$, $r = 0.8$, $r = 0.6$ and $r = 0.4$; the curves for higher r are those with higher peak.

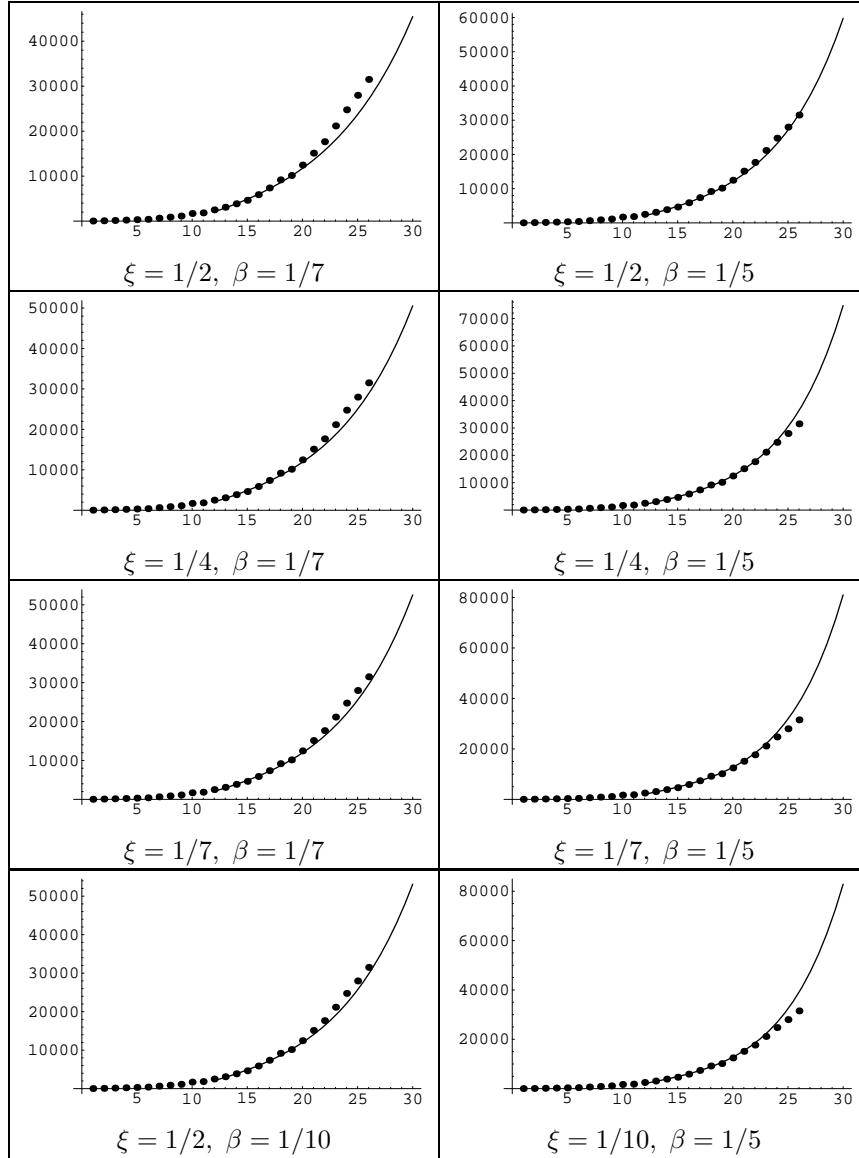


Figure 7: Plot of the data for the COVID epidemics in Italy versus the numerical integration of the A-SIR model (with $r = 1$) for $S_0 = 2 * 10^7$ (the total population of the three most affected regions), $\eta = 1/14$, and various values of the parameters β and ξ . The parameters I_0 and γ were obtained through the fit of eqs. (46), (47) on the basis of the data of Table I for the period 1-10 March; plotted data go until March 17.

of remaining cases, etc.).¹²

We have thus ran several numerical simulations for these values of β and ξ , both for a total population of $N = 2 * 10^7$ (the total population of the initially most affected regions) and for $N = 6 * 10^7$ (the total population of Italy). These give of course very similar results – if referred to the total population – as our estimates for the parameters, and in particular for the one leading the dynamics, i.e. γ , depend themselves on S_0 . Moreover, the questions discussed in this subsection do not concern the early phase of the epidemics (which was then limited to Northern Italy), but its future development. We will thus present the results directly for the case $N = S_0 = 6 * 10^7$.

We have investigated two questions:

- (A) How a reduction in the removal time for asymptomatic infectives, i.e. in η^{-1} , would affect – according to the A-SIR model – the dynamics and the basic epidemiological outcomes of it in the regime where the epidemic is taking place (i.e. for r such that the population is still above the epidemic threshold);
- (B) In the case r is low enough to make the population below the epidemic threshold, what are the basic epidemiological outcomes predicted by the model, again depending on various parameters including η .

The results of these numerical investigations are summarized in Table III and Table IV respectively. We have also studied, for comparison, question (B) in the framework of the standard SIR model. The outcomes of this study are summarized in Table V.

¹²The most reliable prediction about the time-span of the epidemic and its outcome is maybe just the one based on assuming it will roughly follow the dynamics observed in other countries [13]; this of course assuming the measures will have the same impact.

	r	I_*	t_*	R_∞/S_0	U_∞/S_0	S_∞/S_0
$\xi = 1/7$ $\eta = 1/21$	1.0	$1.9 * 10^6$	60	0.14	0.85	0.01
	0.8	$1.4 * 10^6$	74	0.14	0.83	0.03
	0.6	$8.9 * 10^5$	101	0.13	0.77	0.10
	0.4	$3.3 * 10^5$	184	0.10	0.60	0.30
$\xi = 1/10$ $\eta = 1/21$	1.0	$1.3 * 10^6$	57	0.10	0.89	0.01
	0.8	$1.0 * 10^6$	70	0.10	0.87	0.03
	0.6	$6.5 * 10^5$	95	0.09	0.82	0.09
	0.4	$2.5 * 10^5$	167	0.07	0.65	0.28
$\xi = 1/7$ $\eta = 1/14$	1.0	$1.6 * 10^6$	65	0.13	0.80	0.07
	0.8	$1.1 * 10^6$	83	0.12	0.75	0.13
	0.6	$5.3 * 10^5$	124	0.10	0.62	0.28
	0.4	$5.6 * 10^4$	318	0.04	0.22	0.74
$\xi = 1/10$ $\eta = 1/14$	1.0	$1.1 * 10^6$	61	0.09	0.85	0.04
	0.8	$7.8 * 10^5$	78	0.09	0.79	0.12
	0.6	$3.9 * 10^5$	115	0.07	0.66	0.27
	0.4	$5.0 * 10^4$	273	0.03	0.28	0.69

Table III. Simulations for the A-SIR model on a population of $S_0 = 6 * 10^7$, with $\beta = 1/7$ and for the fitted initial conditions discussed in Section 4.2, for $\xi = 1/7$ and for $\xi = 1/10$, and for $\eta^{-1} = 21$ and $\eta^{-1} = 14$, for various values of the reduction factor r . We report the maximum of the (registered) infectives I_* , the time t_* at which this maximum is reached, and the fraction of the initial population which passed through the infection being registered (R_∞/S_0) or unknowingly (U_∞/S_0); the remaining fraction of population S_∞/S_0 remains not covered by immunity.

	r	t_e	R_∞/S_0	U_∞/S_0
$\xi = 1/7$ $\eta = 1/21$	0.2	443	$8.29 * 10^{-4}$	$4.96 * 10^{-3}$
	0.1	104	$2.62 * 10^{-4}$	$1.57 * 10^{-3}$
	0.05	65	$2.08 * 10^{-4}$	$1.25 * 10^{-3}$
	0.02	49	$1.87 * 10^{-4}$	$1.13 * 10^{-3}$
	0.01	46	$1.82 * 10^{-4}$	$109 * 10^{-3}$
$\xi = 1/10$ $\eta = 1/21$	0.2	539	$1.02 * 10^{-4}$	$9.17 * 10^{-3}$
	0.1	107	$2.67 * 10^{-4}$	$2.40 * 10^{-3}$
	0.05	66	$2.09 * 10^{-4}$	$1.88 * 10^{-3}$
	0.02	50	$1.88 * 10^{-4}$	$1.69 * 10^{-3}$
	0.01	46	$1.82 * 10^{-4}$	$1.64 * 10^{-3}$
$\xi = 1/7$ $\eta = 1/14$	0.2	121	$3.38 * 10^{-4}$	$2.03 * 10^{-3}$
	0.1	68	$2.24 * 10^{-4}$	$1.34 * 10^{-3}$
	0.05	53	$1.97 * 10^{-4}$	$1.18 * 10^{-3}$
	0.02	47	$1.84 * 10^{-4}$	$1.11 * 10^{-3}$
	0.01	45	$1.80 * 10^{-4}$	$1.08 * 10^{-3}$
$\xi = 1/10$ $\eta = 1/14$	0.2	126	$3.47 * 10^{-4}$	$3.12 * 10^{-3}$
	0.1	69	$2.26 * 10^{-4}$	$2.03 * 10^{-3}$
	0.05	53	$1.97 * 10^{-4}$	$1.77 * 10^{-3}$
	0.02	47	$1.84 * 10^{-4}$	$1.66 * 10^{-3}$
	0.01	45	$1.80 * 10^{-4}$	$1.63 * 10^{-3}$

Table IV. Simulations for the A-SIR model on a population of $S_0 = 6 * 10^7$, with $\beta = 1/7$ and for the fitted initial conditions discussed in Section 4.2, for $\xi = 1/7$ and for $\xi = 1/10$, and for $\eta^{-1} = 21$ and $\eta^{-1} = 14$, for various values of the reduction factor r such that the population is below the epidemic threshold. We report the time t_e at which there are less than 100 known infectives, and the fraction of the initial population which passed through the infection being registered (R_∞/S_0) or unknowingly (U_∞/S_0).

r	t_e	R_∞/S_0	r	t_e	R_∞/S_0
0.20	57	$2.29 * 10^{-4}$	0.20	41	$1.77 * 10^{-4}$
0.10	49	$1.98 * 10^{-4}$	0.10	37	$1.58 * 10^{-4}$
0.05	46	$1.87 * 10^{-4}$	0.05	35	$1.51 * 10^{-4}$
0.02	44	$1.81 * 10^{-4}$	0.02	34	$1.47 * 10^{-4}$
0.01	44	$1.79 * 10^{-4}$	0.01	34	$1.46 * 10^{-4}$

Table V. Simulations for the standard SIR model on a population of $S_0 = 6 * 10^7$, with $\beta = 1/7$ (left hand side) and $\beta = 1/5$ (right hand side), and for the fitted initial conditions discussed in Section 4.2, for various values of the reduction factor r such that the population is below the epidemic threshold. We report the time t_e at which there are less than 100 known infectives, and the fraction of the initial population which passed through the infection (R_∞/S_0).

6 Conclusions

Motivated by the peculiar features of the COVID epidemics, we have considered a SIR-type model, called A-SIR model, taking into account the presence of asymptomatic infectives.

We have analyzed the available data for the COVID-19 epidemics in Northern Italy in terms of the SIR and of the A-SIR models; in particular we have fitted the model parameters based on the period 1-10 March, and considered how these models with such parameters are performing in predicting the evolution for the subsequent week, 11-17 March. As shown by Figures 3 on the one hand, and by Figures 5, 7 on the other hand, it appears that the A-SIR model is much better in predicting such (admittedly short time) evolution. In particular, this is the case with the Li *et al.* [2] estimate $\xi = 1/7$ for the ratio of clearly symptomatic versus total infections, and for the reasonable estimate $\beta^{-1} = 7$ days for the time from infection to isolation for symptomatic infectives, and $\eta^{-1} = 21$ for the time from infection to healing of asymptomatic infectives.

Looking at the full numerical integration of SIR and A-SIR equations for this set of parameters¹³ and for a population of $N = 2 * 10^7$ (which is the total population of the three most affected regions in Northern Italy [10]), see Figures 2 and 5, we have a prediction of an epidemic peak with about $1.5 * 10^6$ infected by the SIR model, reached after about 100 days; while for the A-SIR model the prediction is of a peak with about $5 * 10^5$ symptomatic infectives for $\xi = 1/7$ and with about $3.5 * 10^5$ infectives for $\xi = 1/10$, in both cases reached after about 50 days. In both cases, the mitigation measures taken at the beginning of March – which according to our analysis led to a reduction factor $r \simeq 0.6$ in the contact rate [10] – would produce a halving of the epidemic peak and a doubling of the peak time; further measures were taken one week later, and hopefully these can stop the epidemic spread.

We have studied in more detail the case which best fits the epidemiological data outside the period used to fix the model parameters; this corresponds to $\beta^{-1} = 7$ and $\xi = 1/7$ (we also considered $\xi = 1/10$). In this framework, two cases are possible: either the restrictive measures are only mitigating the epidemic, or they are capable of stopping it by raising the epidemic threshold above the population level. In the first case, a reduction of η^{-1} from 21 to 14 days produce a substantial lowering of the epidemic peak and also substantially postpones its occurrence; in the second case, the effect of such a reduction may be quite relevant if the population remains just under the threshold (see the cases with $r = 0.2$ in Table IV) or not so relevant if the reduction of the contact rate is taking the epidemic threshold well above the population level (see the cases with lower r in Table IV). In all cases, there is a marked difference with the behavior of a standard SIR model with equivalent parameters.

Finally, we would like to comment on how reliable these predictions may be

¹³We recall once again that this corresponds to the prediction of what would have happened – according to the models – if no action was taken; luckily we expect a substantially different dynamics after the general lockup, and actual predictions should rather be based on the Chinese and the Korean experiences.

considered. We can judge the adherence of a model to the real situation only *a posteriori*, which by definition is not possible in the case of an epidemic in its early phase. Moreover, in real world several types of measures are taken in order to attempt to stop, or at least slow down, the epidemic dynamics; thus a model with constant parameters, in particular constant reduction factor r , is not realistic. It appears that one could study the evolution of the COVID epidemic in China, which appears to have been stopped at the time being [11, 12], to judge if the model is able to describe this evolution. In order to do this, it would be needed to have a “mobile fit” in order to determine what has been the evolution of $r(t)$ (or equivalently of $\gamma(t)$) in that context, and numerically integrate the A-SIR equations with such a varying γ to see if these describe the evolution better (or worse) than the standard SIR model.

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