How to reduce epidemic peaks keeping under control the time-span of the epidemic

Mariano Cadoni^{ab*},

^aDipartimento di Fisica, Università di Cagliari Cittadella Universitaria, 09042 Monserrato, Italy

^bI.N.F.N, Sezione di Cagliari, Cittadella Universitaria, 09042 Monserrato, Italy

7th April 2020

Abstract

One of the main challenges of the measures against the COVID-19 epidemic is to reduce the amplitude of the epidemic peak without increasing without control its timescale. We investigate this problem using the SIR model for the epidemic dynamics, for which reduction of the epidemic peak I_P can be achieved only at the price of increasing the time t_P of its occurrence and its entire time-span t_E . By means of a time reparametrization we linearize the equations for the SIR dynamics. This allows us to solve exactly the dynamics in the time domain and to derive the scaling behaviour of the size, the timescale and the speed of the epidemics, by reducing the infection rate α and by increasing the removal rate β by a factor of λ . We show that for a given value of the size (I_P , the total, I_E and average \hat{I}_P number of infected), its occurrence time t_P and entire time-span t_E can be reduced by a factor $1/\lambda$ if the reduction of I is achieved by increasing the removal rate instead of reducing the infection rate. Thus, epidemic containment measures based on tracing, early detection followed by prompt isolation of infected individuals are more efficient than those based on social distancing. We apply our results to the COVID-19 epidemic in Northern Italy. We show that the peak time t_P and the entire time span t_E could have been reduced by a factor $0.9 \leq 1/\lambda \leq 0.34$ with containment measures focused on increasing β instead of reducing α .

1 Introduction

The recent COVID-19 epidemics is posing formidable challenges both to the health and economic systems worldwide. In order to tackle the ongoing epidemic, the countries faced with the COVID-19 epidemic have used different strategies. In order to control the epidemic one has to lower the basic reproduction number (also called basic reproduction rate) ρ , eventually below the threshold, i.e $\rho < 1$. In a simple SIR Model [1, 2, 3, 4, 5, 6], which we consider in this paper, ρ depends on the infection rate α , the removal rate β and the total population N. If $\rho > 1$ the epidemic starts with the number of infected individuals I growing exponentially, it reaches a maximum I_P at a time t_P , then decreases down to zero at time t_E when the epidemic ends.

Although for severe epidemics like the COVID-19 it is very difficult to keep the reproduction number ρ from the beginning below the threshold, nevertheless containment measures soften the epidemic because they lead to a reduction of the epidemic peak I_P .

^{*}E-mail: mariano.cadoni@ca.infn.it

Basically, there are therefore three types of containment measures one can use. One can act on α by lowering it, for instance by forcing social distancing or by prophylaxis measures. One can achieve the same result increasing β , e.g. by means of a prompt strict isolation of infected individuals. Last but not least one can reduce the total population N by separating it in strictly non-communicating compartments.

The policies of different countries for fighting the COVID-19 usually contemplate a mixture of the three types of measures mentioned above. Obviously, the choice of focusing on one kind of measure instead of another depends on a number of factors, which include not only its effectiveness but also its feasibility and its social and economic impact. For instance most of the European countries have focused their COVID-19 fighting strategies on measures aimed at reducing the infection rate α . Countries like Korea and Singapore favoured instead measures aimed at rising β .

A general, unpleasant, feature of the epidemic dynamics is that the reduction of the epidemic peak I_P can be only achieved at the expenses of increasing t_P and t_E , i.e of increasing the time-span of the epidemic. On the one hand for epidemics like COVID-19 having a high rate of hospitalised infected with severe symptoms, reduction of I_P and dilution of the epidemic is necessary in order to allow the health systems to treat them properly. On the other hand, increasing t_P means increasing the time-span in which the containment measures are effective, with potentially disruptive effects both on the economies and the live of the populations in the involved countries.

In this paper we investigate, in the framework of the SIR model, the impact of containment measures, which act on α and β , on I_P , t_P , t_E and on the epidemic speed $(d\rho/dt|_P)$. The structure of the paper is as follows. In Sect 2, by means of a time reparametrization, we are able to linearize the equations for the SIR dynamics. This allows us to solve exactly the dynamics in the time domain and to derive the scaling behaviour of I_P , t_P , t_E and $d\rho/dt|_P$, by reducing the infection rate $\alpha \to \alpha/\lambda$ or by increasing the removal rate $\to \lambda \beta$. We show that keeping I_P fixed its occurrence time t_P and t_E can be be reduced by a factor $1/\lambda$ by acting on the removal rate β instead on the infection rate α . This will be discussed in Sect. 3. In Sect. 4 we discuss approximate solutions of the SIR model in which the reproduction number $\rho < e$. In Sect. 5 we apply our results to the COVID-19 epidemic in Northern Italy. We show that the peak time t_P and the entire time-span of the epidemic could have been reduced by a factor $0.9 \le 1/\lambda \le 0.34$ with containment measures focused on increasing β instead of the reducing α . Finally, in Sect. 6 we state our conclusions.

2 SIR model: time reparametrization and linearization

The SIR model describes the deterministic dynamics of an infective epidemic, characterized by the fact that individuals, which have been infected and have recovered gain permanent immunity [1, 2, 3, 4, 5, 6]. Although the model is quite simple, it can be used to give at least rough estimates of epidemic dynamics, and in particular of the COVID-19 epidemic [7, 9, 10, 11]. A generalisation of the SIR model to take into account a large number of asymptomatic infectives -hence more apt to describe the COVID-19 epidemichas been proposed in Ref. [9, 10, 11].

The homogeneous and isolated population of N individuals exposed to the epidemic, is characterised at time t by the number of susceptible S(t), infected and infectives I(t) and removed (recovered, dead or isolated) R(t) individuals, with the conservation law N = S(t) + I(t) + R(t). The timescale of the epidemics is assumed to be relatively short so that N can be assumed constant.

The dynamic describing the evolution of the epidemic is deterministic and described by the following, non linear, dynamical system:

$$\frac{dS}{dt} = -\alpha SI,\tag{1a}$$

$$\frac{dS}{dt} = -\alpha SI,$$

$$\frac{dI}{dt} = \alpha SI - \beta I,$$
(1a)

$$\frac{dR}{dt} = \beta I. \tag{1c}$$

The infective epidemic is characterised by two parameters: (1) The infection rate (also called contact rate) α , which gives the transition rate between the class of susceptible and that of infected; (2) the removal

rate β , which gives the transition rate between the class of infected and that of removed $(1/\beta)$ gives the characteristic time for the removal of infected from the dynamics).

From equation (1b) it is immediately evident that number of infected individuals grows, i.e the epidemic spreads, only if

$$S > \gamma, \qquad \gamma := \frac{\beta}{\alpha},$$
 (2)

where γ is the epidemic threshold. Equivalently, one can introduce the basic reproduction number $\rho(t)$:

$$\rho(t) = \frac{S(t)}{\gamma},\tag{3}$$

which represents the expected number of new infections generated by a single infection. The epidemic spreads if $\rho > 1$. The parameters α and β depend on several factors. Some of them are attributes of the pathogen causing the disease and cannot be changed. Other are influenced by the social behaviour of the individuals and can be therefore changed with containment and prophylaxis measures.

The system (1a),(1b),(1c) is difficult to solve, analytically in the time domain. Usually one proceeds by eliminating dt from Eqs. (1a),(1b), then after integration one easily finds the function $I(S) = I_0 + (S_0 - S) + \gamma \log(S/S_0)$, where I_0, S_0 are the initial data (see e.g. Ref. [10]). This form of I(S) allows one to derive some qualitative and quantitative features of the epidemic dynamics but not its explicit time evolution. This latter can be only obtained by numerical integration of Eqs. (1a),(1b),(1c). For instance, one can easily find that, if initially we are above the threshold $\rho_0 > 1$, I(t) grows till it reaches a maximum I_P , then it goes down to zero at a time t_E when the epidemic ends.

The function I(S) allows to determine analytically the value of the peak I_P but not the time t_P of its occurrence, nor its entire time-span t_E , nor the speed of the reproduction number $V_P := d\rho/dt|_P$, nor the average value of the number of infected individuals at the peak \hat{I}_P . t_P , t_E , V_P and \hat{I}_P have to be determined after solving numerically the dynamics. This is a quite unpleasant feature because it prevents a clear understanding of the dependence of t_P, t_E, V_P and \hat{I}_P from the parameters α, β , which is a crucial information for fighting the epidemic.

In order to solve analytically the temporal dynamics let us reparametrize the time introducing a new time coordinate τ defined by $d\tau/dt = I(\tau)$, i.e.:

$$t - t_0 = \int_{\tau_0}^{\tau} \frac{d\tau'}{I(\tau')},\tag{4}$$

where $t_0 = t(\tau_0)$ is the initial time. Using time-translations we can put without loss of generality, $\tau_0 = t_0 = 0$. The new time coordinate has a simple intuitive meaning, $\tau(t)/t$ gives the average value $\hat{I}(t)$ the number of infected at time t:

$$\hat{I}(t) := \frac{1}{t} \int_0^t I(t')dt' = \frac{\tau(t)}{t}.$$
 (5)

The time reparametrization (4) allows to linearise the system (1a),(1b),(1c):

$$\frac{dS}{d\tau} = -\alpha S,\tag{6a}$$

$$\frac{dI}{d\tau} = \alpha(S - \gamma),\tag{6b}$$

$$\frac{dR}{d\tau} = \beta. \tag{6c}$$

This can be easily integrate to give:

$$S = S_0 e^{-\alpha \tau},\tag{7a}$$

$$I = I_0 + S_0 - S_0 e^{-\alpha \tau} - \beta \tau, \tag{7b}$$

$$R = R_0 + \beta \tau, \tag{7c}$$

where S_0 , I_0 are initial data and $R_0 = N - S_0 - I_0$. In the following we will take $R_0 = 0$. The function $\tau(t)$ is defined implicitly by

 $t = \int_0^{\tau} \frac{d\tau'}{I_0 + S_0 - S_0 e^{-\alpha \tau'} - \beta \tau'}.$ (8)

Exact solutions of the SIR model, which are equivalent to our Eqs. (7a),(7b),(7c), (8) have been derived in Refs. [12, 13] using a completely different approach.

Although, the integral (8) cannot be evaluated analytically, it allows to solve the temporal dynamics of the SIR model and to investigate the scaling behaviour of the relevant quantities characterizing the epidemic when the parameters α , β change. The previous expressions allow us to compute easily all the relevant quantities for the epidemic peak. From Eqs. (6b) and (7a) one gets immediately τ_P , the τ -time coordinate of the peak. The other quantities are readily computed using Eqs. (7a),7b),(7c), (8):

$$I_P = I_0 + S_0 - \gamma - \gamma \log \left(\frac{S_0}{\gamma}\right), \tag{9a}$$

$$S_P = \gamma, \quad R_P = \gamma \log \left(\frac{S_0}{\gamma}\right), \quad V_P = -\alpha I_P,$$
 (9b)

$$\tau_P = \frac{1}{\alpha} \log \left(\frac{S_0}{\gamma} \right), \tag{9c}$$

$$t_P = \int_0^{\tau_P} \frac{d\tau'}{I_0 + S_0 - S_0 e^{-\alpha \tau'} - \beta \tau'}.$$
 (9d)

The entire time-span of the epidemic t_E can be computed setting I=0 in Eq. (7b). Because I_0 is usually small compared to S_0 it can be neglected, t_E is obtained by first finding the (higher) root τ_E of the transcendental equation:

$$S_0 - S_0 e^{-\alpha \tau_E} - \beta \tau_E = 0, \tag{10}$$

and then using Eq. (8) to compute t_E .

An other important quantity, which describes the intensity of the epidemics is the total number I_E of individuals that are infected over the whole time-span of the epidemics. Taking into account that I_0 is rather small and that initially $S_0 = N$, I_E can expressed in terms of the lower root S_E of the transcendental equation obtained by setting I = 0 in Eq. (7b) (see Ref. [9]) for details. We have

$$I_E = N - S_E, (11)$$

where S_E is the lower root of the transcendental equation

$$N - S_E + \gamma \log \left(\frac{S_E}{N}\right) = 0. {12}$$

3 Scaling behaviour of epidemic parameters

In this section we investigate the scaling behavior of the peak quantities (9a...(9d), the total number of infected (11), I_E and t_E by changing of the parameters α and β . It is already known that Eqs. (1a)...(1c) are invariant under the scaling $\alpha \to \lambda \alpha$, $\beta \to \lambda \beta$, $t \to \lambda^{-1}t$ [9]. This scaling transformation leaves invariant the epidemic threshold γ and tells us that we can increase (reduce) the timescale of the epidemic by simultaneously reducing (increasing) both α and β . However, this is not what we are interested in. Actually, we want to know what happens to the epidemic parameters listed above when we increase the threshold γ . Let us first observe that both the number of infected at the peak I_P (see Eq. (9a) and the total number of infected I_E (see Eq. (11) are decreasing functions of the parameter γ . In fact, we get from Eq. (9a) and Eq. (12),

$$\frac{dI_P}{d\gamma} = -\log\left(\frac{S_0}{\gamma}\right), \quad \frac{dS_E}{d\gamma} = -\left(\frac{\gamma}{S_E} + 1\right)^{-1}\log\left(\frac{S_E}{N}\right). \tag{13}$$

We see that above the epidemic threshold $(S_0/\gamma > 1)$, $dI_P/d\gamma$ is always negative, while being $S_E < N$, $dS_E/d\gamma$ is always positive.

It follows that if we want to reduce the peak and the total number of infected we have to increase γ by a factor $\lambda > 1$. Because one can increase γ either by reducing α or by increasing β , we have to compare the effects on the peak parameters of these two different ways of increasing γ .

We are therefore lead to consider two different scaling transformations: transformation $T^{(1)}$, which reduces the infection rate:

$$\alpha \to \lambda^{-1} \alpha, \quad \gamma \to \lambda \gamma, \quad \lambda \geqslant 1$$
 (14)

and transformation $T^{(2)}$, which increases the removal rate:

$$\beta \to \lambda \beta, \quad \gamma \to \lambda \gamma, \quad \lambda \geqslant 1.$$
 (15)

The peak quantities in Eqs. (9a...(9d) and the τ_E of Eq. (10) do not transform in a simple way under $T^{(1)}$ and $T^{(2)}$, however the ratios of $T^{(1)}$ and $T^{(2)}$ -transformed quantities follow simple scaling laws. In particular, they remain invariant whenever the quantity depends only on their ratio γ and not on α and β

Using the following notation to denote rescaled quantities: $I_P^{(1)} = I_P(\lambda^{-1}\alpha)$, $I_P^{(2)} = I_P(\lambda\beta)$ and similarly for the others quantities, we get,

$$I_P^{(1)} = I_P^{(2)}, S_P^{(1)} = S_P^{(2)}, R_P^{(1)} = R_P^{(2)}, (16a)$$

$$\begin{split} I_P^{(1)} &= I_P^{(2)}, & S_P^{(1)} &= S_P^{(2)}, & R_P^{(1)} &= R_P^{(2)}, \\ V_P^{(2)} &= \lambda V_P^{(1)}, & \tau_P^{(2)} &= \lambda^{-1} \tau_P^{(1)}, & \tau_E^{(2)} &= \lambda^{-1} \tau_E^{(1)}. \end{split} \tag{16a}$$

The transformation law for t_P and t_E can be derived by first acting with the transformation $T^{(1)}$ on the integral (9d), then acting with $T^{(2)}$ on the same integral and finally redefining the integration variable in the second integral $\tau' \to \lambda^{-1} \tau'$. One obtains in this way:

$$t_P^{(2)} = \lambda^{-1} t_P^{(1)}, \qquad t_E^{(2)} = \lambda^{-1} t_E^{(1)}.$$
 (17)

Finally, using Eqs. (5), (16b), (17) and taking into account that I_E depends only on γ (see Eqs. (11),(12)) we can easily show that the average number of infected I_P and the total number of infected I_E are invariant, i.e.

$$\hat{I}_P^{(2)} = \hat{I}_P^{(1)}, \qquad I_E^{(1)} = I_E^{(2)}.$$
 (18)

An important result follows from equations (16a),(16b),(17) and (18): epidemic containment measures, which have the same effect for what concerns I_P , R_P , S_P , I_P and I_E , have different impact on the occurrence time t_P of the peak, the whole time span of the epidemic t_E and on the epidemic speed V_P . Choosing measures increasing the removal rate β by a factor λ instead of reducing the infection rate α by a factor $1/\lambda$ allows to drop t_P and t_E by a factor $1/\lambda$. For instance by implementing epidemic containment measures with $\lambda = 2$ we can reduce by a half both the time needed for the epidemic to reach the peak and the whole time-span of the epidemic. It should be noticed that this epidemic timescale reduction effect becomes more relevant for epidemics with high reproduction number $\rho_0 >> 1$. In fact the factor λ is limited by $\lambda < \rho_0$, simply because for $\lambda > \rho_0$ the epidemic does not develop at all. Thus, if we have for instance $\rho_0 = 5$ we can reduce the peak time and the entire time-span of the epidemic until a factor of 1/5.

Therefore, increasing β represents an efficient way to fight epidemics. If by increasing it we manage to bring ρ below the threshold we simply stop the epidemic, but even if we do not go so far, we can still reduce the size of an epidemic keeping under control its timescale.

The behaviour of the reproduction number speed V_P in Eq. (16b) explains clearly what is going on. If one acts on β instead on α , V_P increases, as expected, by a factor of λ . In short, increasing β instead of reducing α , allows one to speed up the epidemic dynamics keeping constant the number of infected at the peak, the average number of infected and the total number of infected. This is possible because the increasing of the removal rate allows prompt removal of infected individuals.

4 Approximate solutions for $\rho_0 < e$

In the general case the integral (8) cannot be computed analytically. Therefore the function $\tau = \tau(t)$ has to be computed numerically, by first performing numerical integration of the integral in (8) to find $t = t(\tau)$ and then inverting it. There is, however, a situation in which the integral (8) can be computed analytically and the dynamics of the epidemic until the peak, can be expressed analytically in closed form in terms of the time t, albeit in approximate form.

For $\alpha \tau \ll 1$ we can approximate the exponential in Eq. (8) by $e^{-\alpha \tau} \approx 1 - \alpha \tau$. This approximation allows to solve the integral and to invert the function $t = t(\tau)$. We find,

$$\tau \approx \frac{I_0}{\alpha(S_0 - \gamma)} \left(e^{\alpha(S_0 - \gamma)t} - 1 \right). \tag{19}$$

With this position we can easily write down the approximate form of the solutions (7a),(7b),(7c) in terms of the time t. We quote here only the form of I(t) and t_P as a function of I_P ,

$$I(t) \approx I_0 e^{\alpha(S_0 - \gamma)t}, \quad t_P \approx \frac{1}{\alpha(S_0 - \gamma)} \log \frac{I_P}{I_0}.$$
 (20)

Eqs. (19) and (20) are a good approximation only for $\alpha \tau < 1$. Because $\tau(t)$, is an increasing function of t, the approximation for the dynamics is good until the peak, if $\alpha \tau_P < 1$, which implies from equation (9c):

$$\rho_0 = \frac{S_0}{\gamma} < e. \tag{21}$$

5 Application to the COVID-19 epidemic in Northern Italy

The recent development of the COVID-19 epidemic in Northern Italy represents an interesting case for applying the results described in the previous sections. The epidemic developed in the three main regions of Northern Italy (Lombardia, Veneto and Emilia-Romagna) we consider in this paper, starting from end of February 2020 (although it may be possible that the epidemic was circulating in the regions before that date).

Altogether these three regions have around 20 millions of inhabitants, we will therefore take $N=2\cdot 10^7$ in our computations. We take as initial value $I_0=100$, which approximately corresponds to the known cases of COVID-19 infected Northern Italy on February 23, 2020. The determination of the initial values of the other two parameters of the SIR model α_0 and β_0 is more involved. These initial values are completely determined by the pathogen because they are not affected by the epidemic containment measures put into play. The value of α_0 can be determined from the exponential behaviour of the early dynamics, or equivalently from the initial doubling time [7, 8]. Using the raw data for the early dynamics of the epidemic in Northern Italy, Gaeta [7, 8] has given the estimate:

$$\alpha_0 = \frac{10^{-7}}{6}. (22)$$

The determination of β_0 is even more problematic. This is because we expect it to be sensitive to the presence of large cohort of asymptomatic infectives. We can estimate β_0 from the basic reproduction number ρ_0 , using Eq. (3). Rough evaluations of ρ_0 give a number between 2 and 2.5, however the indeterminacy related to the presence of large number of asymptomatic infectives may result in a much higher value for ρ_0 . To be rather conservative we assume here $\rho_0 = 3$, so that Eqs. (3) and (22) give:

$$\beta_0 = \frac{1}{9}.\tag{23}$$

The COVID-19 containment strategies put in place in Northern Italy are a mixture of social distancing, social confinement, early detection and infection tracing. Although mainly focused of social distancing, these strategies contain all the previous ingredients, which modify in different ways the parameters α and β . Social

distancing acts by reducing α , whereas systematic, prompt, and strict isolation of infected individuals as a result of early detection and tracing enhances β . Both effects rise γ and reduce in the same way the amplitude of the peak I_P , the average number of infected \hat{I}_P and the total number of infected I_E .

Because it is almost impossible to disentangle the effects of the various containment measures on α and β in the real situation, we will discuss and compare two hypothetical situations in which the rising of γ , $\gamma \to \lambda \gamma_0$, with $\gamma_0 = (2/3) \cdot 10^7$, is obtained in two fully distinct and complementary ways:

- (1) We have exclusively social confinement containment measures: β is held fixed to its initial value β_0 , whereas α_0 is reduced by a factor $1/\lambda$.
- (2) We have exclusively containment measures consisting in prompt and strict isolation of infected individuals triggered by early detection and tracing of infected: α is held fixed to its initial value α_0 , whereas β_0 is increased by a factor λ .

Being $1 \leq \lambda < \rho_0$ we consider the following values: $\lambda = 1, 1.5, 2, 2.5, 2.9$. Using Eqs. (9a)...(9d) we compute for these values of the parameters α and β the peak quantities: peak amplitude I_P , the time t_P (in days) of occurrence of the peak (computed by numerical evaluation of the integral (9d)), average number of infected \hat{I}_P and absolute value of the epidemic speed at the peak $|V_P|$ (in days⁻¹). Moreover, using Eqs. (11), (12), (10), together with Eq. (9d), we compute numerically the total number I_E of infected individuals during the epidemic and its whole time span t_E (in days). The results are shown in Table I. Our results are in accordance with the scaling behaviour given by Eqs. (16a),(16b), (17) and (18).

We see from Table I that the raising of the epidemic threshold for γ from the initial value γ_0 first to $2\gamma_0$ then to $2.9 \gamma_0$ let both the number of infected at the peak and their average number drastically sink from the order of magnitude 10^6 first to 10^5 and then to $10^3 - 10^4$. This reduction is the same independently of the fact that if it is achieved by reduction of α (way (1)) or by increase of β (way (2)). Similarly, the total number I_E of infected individuals drops from the huge value 1 $1.88 \cdot 10^7$ till $1.16 \cdot 10^7$ (for $\lambda = 2$) and then to $1.3 \cdot 10^6$ (for $\lambda = 2.9$)

On the other hand, the two ways of reducing I_P and \hat{I}_P and I_E affect differently the occurrence time of the peak t_P and the whole time span of the epidemic t_E . By acting on β (way (2)) instead of on α (way (1)) we can shorten these times by 33% (for $\lambda = 1.5$), by 50% (for $\lambda = 2$) and even reduce it by almost 1/3 (for $\lambda = 2.9$). Correspondingly, the speed of variation of the basic reproduction number $|V_P|$ will be enhanced by the same factors.

If the containment measures can manage to increase λ above 3, we go below the threshold for ρ and the epidemic does not start at all. Obviously, in a real situation reducing α or increasing β is not performed once for all at the beginning, but occurs in steps. Our main result is that in order to try to stop the epidemic it is much more convenient to rise β instead of lowering α because even if we do not manage to stop it, we are able to reduce its size and at the same time to shorten its timescale.

λ	α/α_0	β/β_0	I_P	\hat{I}_P	$t_P(\mathrm{days})$	$ V_P (\mathrm{days})^{-1}$	I_E	$t_E ext{ (days)}$
1	1	1	$6 \cdot 10^{6}$	$1.13 \cdot 10^6$	58	10^{-1}	$1.88 \cdot 10^7$	187
1.5	0.66	1	$3.07 \cdot 10^6$	$5.74 \cdot 10^5$	109	$3 \cdot 10^{-2}$	$1.59 \cdot 10^{7}$	279
1.5	1	1.5	$3.07 \cdot 10^6$	$5.74 \cdot 10^5$	72	$5 \cdot 10^{-2}$	$1.59 \cdot 10^7$	187
2	0.5	1	$1.26 \cdot 10^{6}$	$2.45 \cdot 10^{5}$	198	10^{-2}	$1.16 \cdot 10^{7}$	453
2	1	2	$1.26 \cdot 10^6$	$2.45 \cdot 10^5$	99	$2 \cdot 10^{-2}$	$1.16 \cdot 10^7$	227
2.5	0.4	1	$2.95 \cdot 10^{5}$	$6.42 \cdot 10^4$	426	$2 \cdot 10^{-3}$	$6.2 \cdot 10^{6}$	899
2.5	1	2.5	$2.95 \cdot 10^5$	$6.42 \cdot 10^4$	170	$5 \cdot 10^{-3}$	$6.2 \cdot 10^6$	319
2.9	0.34	1	$1.12 \cdot 10^4$	$3.7 \cdot 10^3$	1592	$6 \cdot 10^{-5}$	$1.3 \cdot 10^{6}$	3213
2.9	1	2.9	$1.12 \cdot 10^4$	$3.7 \cdot 10^{3}$	549	$2 \cdot 10^{-4}$	$1.3 \cdot 10^{6}$	1107

Table I. Comparison of the effect of reduction of the infection rate $\alpha \to (1/\lambda)\alpha$ versus increase of the removal rate $\beta \to \lambda\beta$ on epidemic parameter: peak amplitude I_P , average value of infected \hat{I}_P , peak time t_P , speed of basic reproduction number $|V_P|$ at the peak, total number of infected individuals I_E and whole

¹Notice that without containment measures at the end of the epidemic almost all individuals have been infected.

time-span of the epidemics t_E . The total population is $N = 2 \cdot 10^7$, $\beta_0 = 1/9$ and $\alpha_0 = (1/6)10^{-7}$. The values of I_P , \hat{I}_P , t_P , $|V_P|$, I_E , t_E are tabulated for values of $\lambda = 1, 1.5, 2, 2.5, 2.9$. For sake of clarity we also show in the table the values of α and β corresponding to a given value of λ .

6 Concluding remarks

In this paper we have analysed, in the context of the standard SIR model for epidemic dynamics, the impact of different containment measures on size (the epidemic peak I_P , the average number of infected \hat{I}_P and the total number of infected I_E), the timescale (the occurrence time of the peak t_P and the whole time-span t_E) and the speed (time variation of the reproduction number $|V_P|$) of epidemics. Using an exact solution for the epidemic dynamics we have been able to derive the scaling behaviour of these quantities under change of the two parameters (the infection rate α and the removal rate β) of the SIR model, which can be controlled by the containment measures. This allowed us to compare the impact on size, timescale and speed of the epidemic of containment measures acting either on α or on β

We have shown that for a given reduction of I_P , \hat{I}_P , I_E , the timescale and the speed of the epidemic are to a great extend sensitive to the kind of measures we put into play. By increasing the removal rate β instead of reducing the infection rate by a factor λ one can reduce the timescale of the epidemic by a factor $1/\lambda$ and increase the speed of the epidemics by a factor λ . In the case we have analysed in detail, namely the COVID-19 epidemic in Northern Italy, the reduction factor λ , in principle could also take values around 3

An important point we have not addressed in this paper is the determination of the exact way in which the usual containment measures used to fight epidemic, impact on the values of the parameters α and β . Whereas it is quite clear that social distancing reduces the parameter α and does not change β , the effect of other measures like, early detection and contacts tracing is not a priori evident. Early detection and contact tracing increase β only if implemented on a large scale and followed by prompt and strict isolation of the detected infectives. If this is not the case, it is likely that these measures just bring a small reduction of α

The recent analysis of Gaeta [10] of the different strategies used in Northern Italy to tackle the COVID-19 epidemic seems to confirm this result. He found that simple early detection and contact tracing, while having an impact on the epidemic peak, do not substantially affect the timescale of the epidemic. On the other hand he also showed that contact tracing if followed by prompt isolation is the only efficient way to reduce the size of the epidemic, without having to live with it a long time. The Veneto experience shows that this was one of the factors underlying the success of the containment strategy in that region. Thus the main lesson one can draw from our results is that, epidemic containment measures focused on tracing, early detection followed by prompt removal of infected individuals are more efficient to fight epidemics than those based on social distancing.

Let us conclude this paper with some comments about the range of validity of our results. The SIR model is an oversimplified model for epidemic dynamics. Generalisations of it are necessary in order to give a good descriptions of real epidemics. For instance, in the case of the COVID-19 epidemic a generalization of the SIR model seems to be necessary in order to take into account the presence of a large set of a asymptomatic infective [9, 10, 11]. On the other hand, the SIR model gives the bare bones of deterministic epidemic dynamics. For this reason we believe that, at least at qualitative level, the main result of this paper - the possibility to reduce the epidemic peak keeping under control its timescale by acting on removal rates- could remain true for generalized and improved SIR-like models.

Acknowledgements

I thank Giuseppe Gaeta for several useful discussions and comments.

References

- W.O. Kermack and A.G. McKendrick, Contributions to the Mathematical Theory of Epidemics, Proc. R. Soc. Lond. A 138 (1932), 55-83; Proc. R. Soc. Lond. A 141 (1933), 94-122
- [2] J.D. Murray, Mathematical Biology. I: An Introduction, Springer (Berlin) 2002
- [3] H.W. Hethcote, The Mathematics of Infectious Diseases, SIAM Review 42 (2000), 599-653
- [4] F. Brauer and C. Castillo-Chavez, Mathematical Models in Population Biology and Epidemiology, Springer, New York (2001)
- [5] D. J. Daley and J. Gani, *Epidemic Modeling: An Introduction*, Cambridge University Press, Cambridge (2005).
- [6] F. Brauer, P. van den Driessche, and J. Wu (Editors), Lecture Notes in Mathematical Epidemiology, Springer-Verlag, Berlin, Heidelberg (2008)
- [7] G. Gaeta, Data analysis for the COVID-19 early dynamics in Northern Italy, arXiv:2003.02062;
- [8] G. Gaeta, Data Analysis for the COVID-19 early dynamics in Northern Italy. The effect of first restrictive measures, arXiv:2003.03775
- [9] G. Gaeta, A simple SIR model with a large set of asymptomatic infectives, arXiv:2003.08720
- [10] G. Gaeta, Social distancing versus early detection and contacts tracing in epidemic management, arXiv:2003.14102
- [11] G. Gaeta, Asymptomatic infectives and R0 for COVID, arXiv:2003.14098
- [12] T. Harko, F. S. N. Lobo, M. K. Mak, Exact analytical solutions of the Susceptible-Infected-Recovered (SIR) epidemic model and of the SIR model with equal death and birth rates, Applied Mathematics and Computation, 236, (2014) 184-194, arXiv:1403.2160
- [13] J.C. Miller, A note on the derivation of epidemic final sizes, Bulletin of Mathematical Biology. 74, (2012) 9