Epidemics with containment measures

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We propose a Susceptible-Infected (SI) epidemic spreading model including containment measures. In absence of containment measures the epidemics spreads exponentially fast for any value of the infectivity $\lambda > 0$. The containment measures are modeled by considering a time-dependent modulation of the bare infectivity λ leading to an effective infectivity that decays in time for each infected individual, mimicking for instance the combined effect of asymptomatic onset of the disease, testing policies and quarantine. We consider a wide range of temporal kernels for the effective infectivity and we investigate the effect of the considered containment measures. We find that not all kernels are able to push the epidemic dynamics below the epidemic threshold, with some containment measures only able to reduce the rate of the exponential growth of new infected individuals. We also propose a model with pandemic caused by a growing number of new separated foci. This model provides a stylized mathematical framework that can shed light on the role of different containment measures in mitigating and suppressing the spread of an epidemics such as COVID-19.

I. INTRODUCTION

As we write this work, half of the World is under lockdown due to the global pandemic caused by coronavirus officially called COVID-19. The scientific community is responding to this emergency by proposing modeling frameworks to study the effect of various containment measures [1–3] and testing and immunization policies [4–6], analyzing data [7, 8] and by studying different scenarios for the future evolution of the pandemic. Here we take a more abstract approach, namely we introduce and analyze a simple mathematical model of epidemic spreading mimicking containment measures that can help to understand the dynamics at the onset of a pandemic.

The study of epidemics has a fascinating history [9, 10]. Epidemic modeling goes back to Daniel Bernoulli who modeled the spread of smallpox [11]. The modeling literature grows at an accelerating pace as the new challenges ranging from HIV [12, 13] and computer viruses [14], to innovation, rumor spreading [15–18] and now COVID-19 (see e.g. [1–8, 19–22] and references therein) continue to emerge. Epidemic spreading processes are described not only in mathematical biology books [23–27], but also in statistical physics [28] and network theory textbooks [29–32] and topical reviews [33]. The Susceptible-Infected (SI), Susceptible-Infected-Susceptible (SIS) and Susceptible-Infected-Recovered (SIR) are especially popular epidemic models. These models have been studied on well-mixed populations where every individual can be in contact with any other (see [23–27, 34] for a review), on networks [33] or in a meta-population framework [35] formed by several well-mixed populations interacting through a network [36]. In all these models when the infectivity λ of an infected individual exceeds the epidemic threshold λ_c, at the onset of the epidemic outbreak the spread is exponentially fast in time [29]. When $\lambda < \lambda_c$, the epidemics quickly dies out. A very interesting regime is the critical one in which the infectivity λ is close to the epidemic threshold λ_c; the behaviors in

this regime are non-trivial and not yet fully understood even in the realm of the classical models such as SIR, see [37–44], but at least it is well established that such epidemics cannot affect a finite fraction of population.

The containment measures successfully stop the spread of the epidemic if they are able to raise the value of the epidemic threshold to $\lambda_c > \lambda$. For epidemic spreading models defined on networks, the epidemic threshold depends on the network topology [29–33]. Here we take a well-mixed population approach and neglect network effects. This is justified by the consideration that before strict containment measures the contact networks in urban centers are in first approximation well-mixed due to the presence of social activities and public transportation. In presence of strict containment measures the contact network is much sparser but the spread of the disease is likely to be due to random encounters outside of the households, justifying in first approximation the well mixed approximation also in this case.

To model the onset of the epidemics in a well-mixed meta-population we consider the SI model in a well-mixed population scenario. In this framework there are only susceptible individuals that do not carry the infection and infected individual that can carry the infection. The epidemic dynamics is very simple as the only possible transition we take into account is the transition of an individual from the susceptible to the infected state if it is in contact with an infected individual. This is an idealized scenario, but it is a good approximation for modeling the onset of an epidemics. In absence of containment measures the epidemic threshold vanishes, so for every $\lambda > 0$ the epidemic spreads exponentially fast.

We model the effect of different containment measures by introducing a temporal kernel $F(\tau)$ that modulates the infectivity of each infected individual. In particular we assume that the effective infectivity $\lambda F(\tau)$ of an infected individual decays with the time τ that has elapsed after the individual got infected. Depending on the functional form of the temporal kernel $F(\tau)$ we investigate

the critical properties of the epidemic spreading process. In particular we characterize the epidemic threshold of the model with containment measures and the asymptotic scaling of the number $n(t)$ of infected individuals with time t. We therefore characterize when the containment measures are effective in pushing the dynamics in the subcritical regime, with $\lambda < \lambda_c$. Additionally we show that in the subcritical regime the total number $N(t)$ of infected individuals is constant asymptotically in time indicating that the spread of the epidemics has been halted. In the critical regime it is possible to observe a polynomial growth of $N(t)$ in a given epidemic focus. When the containment measures are too mild to achieve the halting of the epidemics, i.e. $\lambda > \lambda_c$, we quantify the impact of the adopted measures in reducing the rate of the exponential growth.

Finally we consider a mean-field meta-population approach in which we model the geographic spread of the epidemic by assuming that the number of epidemic foci increases with time. Our results show that the total number of cases across different foci of the epidemics can growth either exponentially or as a power-law of time. This result can shed light on the observation that the data about COVID-19 cases in China seem to indicate a power-law growth instead of an exponential growth of the number of cases with time [7], without requiring considerations based on the network topology [45]. Our understanding of this observation is that the Chinese data where just at the critical state of a multi-foci dynamics. This understanding of the dynamics seem to be confirmed by the fact that the epidemic spread appears to be successfully halted in China few weeks after the reported power-law dynamics, while in all the other countries where the epidemics continues to spread the grow of the number of cases is exponential.

The paper is structured as follows. In Sec. II we present the single focus SI model and we list the four temporal kernels $F(\tau)$ that are here used to mimic the containment measures. In Sec. III we provide the exact solution of the model for an arbitrary kernel $F(\tau)$ using the generating function formalism. In Secs. IV–VII we discuss in detail the solution of the model for the four considered temporal kernels: the constant kernel, the power-law kernel, the exponential kernel and the generalized exponential kernel; in Sec. VIII we discuss the multi-foci generalization of the SI dynamics. In Sec. IX we characterize the total number of infected individual in the multi-foci model. Conclusions are presented in Sec. X. Some details of calculations are relegated to Appendices.

II.   SINGLE FOCUS SI MODEL

In a typical Susceptible-Infected (SI) epidemic model it is assumed that the infectivity λ of an infected individual does not change with time as long as the infected individual is contagious. Therefore in a SI model on well mixed population the density of infected individuals increases

exponentially at the onset of the epidemics.

Here we consider an alternative approach and study a model in which an infected individual has a reproductive number that changes with time starting from the time at which the infected individual becomes infectious. This decay of the effective infectivity can be due to different causes including asymptomatic onset combined together to early testing policies and containment measures enforced once the infection becomes symptomatic.

Starting at time $t = 0$ from a single infected individual $n(0) = 1$, the average number $n(t)$ of individuals infected at time $t \geq 1$ is given by

$$n(t) = \lambda \sum_{t'=0}^{t-1} F(t - t')\, n(t'), \tag{1}$$

where $F(\tau)$ is the temporal kernel that describes how the effective infectivity of a infected individual decays as a function of the time τ elapsed since his infection. Given this SI epidemic spreading dynamics the total number of infected $N(t)$ individual at time t is given by

$$N(t) = \sum_{t'=0}^{t} n(t'). \tag{2}$$

We consider the four simple temporal kernels $F(\tau)$.

- *Constant kernel.*
  In this case the effective infectivity of an infected individual remains constant in time:

$$F(\tau) = 1. \tag{3}$$

  In this case there are no containment measures and the epidemic model reduces to the standard SI model.

- *Power-law kernel.*
  In this case the effective infectivity of an an infected individual decays as a power-law of time:

$$F(\tau) = \frac{1}{\tau^\alpha}, \tag{4}$$

  with $\alpha \geq 0$. For $\alpha = 0$ we recover the constant kernel.

- *Exponential kernel.*
  In this case the effective infectivity of an an infected individual decays exponentially in time:

$$F(\tau) = \exp\left[-\gamma \tau\right], \tag{5}$$

  with $\gamma \geq 0$. For $\gamma = 0$ we recover the constant kernel.

- *Generalized exponential kernel.*
  In this case the effective infectivity of an an infected individual decays in time as

$$F(\tau) = \exp\left[-\gamma \tau^b\right], \tag{6}$$

  with $\gamma > 0$. For $b = 1$ we recover the exponential kernel. For $b >$ the decay of this temporal kernel is faster than exponential, for $b < 1$ it is slower than exponential.

III. GENERAL SOLUTION OF THE SINGLE FOCUS MODEL

A. Exact solution

The best way of analyzing recurrences such as Eq. (1) is via generating functions. Indeed, the generating function

$$\mathcal{N}(x) = \sum_{t \geq 0} n(t) x^t \qquad (7)$$

converts the recurrence Eq. (1) into a linear equation for the generating function given by

$$\mathcal{N}(x) = 1 + \lambda \mathcal{F}(x) \mathcal{N}(x), \qquad (8)$$

where

$$\mathcal{F}(x) = \sum_{\tau \geq 1} F(\tau) x^\tau, \qquad (9)$$

is the generating function of the temporal kernel. Hence Eq. (7) admits the solution

$$\mathcal{N}(x) = \frac{1}{1 - \lambda \mathcal{F}(x)}. \qquad (10)$$

The generating function $\mathcal{F}(x)$ is well-defined for $x < R$, where R is the radius of convergence. The convergence radius has an obvious lower bound, $R \geq 1$, in the relevant situations when the temporal rate $F(\tau)$ is non-decreasing function of τ.

In the case in which there exist $x = e^{-\mu} < R$ such that

$$\lambda \mathcal{F}(e^{-\mu}) = 1, \qquad (11)$$

the generating function $\mathcal{N}(x)$ has a pole at $x = e^{-\mu}$. In this case the pole is simple since the generating function $\mathcal{F}(x)$ is a strictly increasing function of x, in the non-pathological case when $F(\tau) \geq 0$. Applying the theorem of residues to Eq. (10) we deduce the exponential asymptotic,

$$n(t) \simeq A_\mu\, e^{\mu t} \qquad (12)$$

for $t \gg 1$, with growth rate μ determined by Eq. (11) and

$$A_\mu = e^\mu\, \frac{\mathcal{F}(e^{-\mu})}{\mathcal{F}'(e^{-\mu})}, \qquad (13)$$

where $\mathcal{F}' = \frac{d\mathcal{F}}{dx}$. Therefore as long as $x = e^{-\mu} < R$, for $\mu > 0$ the number of new infected individuals grows exponentially with time t; for $\mu = 0$ it remains constant in time and for $\mu < 0$ decays exponentially with time. In the interesting regimes with $\mu \geq 0$, the total number of infected individuals $N(t)$ grows as

$$N(t) \simeq \begin{cases} A_\mu (e^\mu - 1)^{-1} e^{\mu t} & \mu > 0, \\ A_0\, t & \mu = 0. \end{cases} \qquad (14)$$

When $\mu < 0$, the total number of infections saturates.

Thus if the growth rate of new infections is positive, $\mu > 0$, the total number of infections grows exponentially in time at the same rate as the number of new infections. In the critical case, $\mu = 0$, the total number of infection $N(t)$ increases linearly with time. The amplitude in this situation has a particularly neat form:

$$A_0 = \frac{\mathcal{F}(1)}{\mathcal{F}'(1)} = \frac{\sum_{\tau \geq 1} F(\tau)}{\sum_{\tau \geq 1} \tau F(\tau)}. \qquad (15)$$

Note however that if the condition $x = e^{-\mu} < R$ is not longer valid the scaling of the number $n(t)$ of new infected individuals and the scaling of the total number $N(t)$ of infected individuals can deviate significantly from the exponential behavior indicated in Eq. (12) and Eq.(14) respectively. Explicit cases where these deviations are observed will be discussed in detail in the next sections.

B. Epidemic threshold and dynamical regimes

Form the exact solution of $\mathcal{N}(x)$ given by Eq. (10) we deduce that the SI epidemic model defined by Eq. (1) has the epidemic threshold given by

$$\lambda_c = \lim_{x \to 1^-} \frac{1}{\mathcal{F}(x)}. \qquad (16)$$

Equation (10) further implies that our epidemic model exhibits different behaviors depending on whether λ is larger, equal, or smaller than λ_c.

The *supercritical regime* occurs when $\lambda > \lambda_c$. In this case, according to Eq. (11) the generating function $\mathcal{N}(x)$ has a simple pole at $x = e^{-\mu}$ with $\mu > 0$. Hence the number of new infected individuals exhibits a purely exponential asymptotic growth. In some special cases, it is possible to get exact results $n(t)$. For instance, for the constant kernel and exponential kernels, the exponential behavior is exact, i.e. valid for all $t \geq 1$.

The rate μ approaches to zero when $\lambda \to \lambda_c^+$. The behavior is particularly simple when $\mathcal{F}(x)$ is differentiable at $x = 1$, i.e. the zeroth and first moments of the temporal rate $F(\tau)$ are well-defined, i.e. $\mathcal{F}(1)$ and $\mathcal{F}'(1)$ are finite. In this situation, we expand Eq. (11) and find

$$\mu \simeq D(\lambda - \lambda_c), \qquad (17)$$

with neat general expressions for the epidemic threshold λ_c and amplitude D:

$$\lambda_c = \frac{1}{\mathcal{F}(1)}, \quad D = \frac{1}{\lambda_c^2 \mathcal{F}'(1)}. \qquad (18)$$

For temporal kernels with radius of convergence $R = 1$ and $\mathcal{F}'(1) = \infty$ the behavior of μ in the $\lambda \to \lambda_c^+$ limit can be more interesting. In the majority of cases, we have observed an algebraic behavior,

$$\mu \simeq D(\lambda - \lambda_c)^\beta, \qquad (19)$$

characterized by the *dynamical exponent* $\beta \geq 1$. Alternatively the linear scaling law (17) can acquire a logarithmic correction.

The *critical regime*, $\lambda = \lambda_c$, separates the supercritical regime from the subcritical regime. If $R > 1$, then $n(t)$ saturates according to Eq. (12). If $R = 1$, the asymptotic behavior of $n(t)$ can be extracted from an asymptotic expansion of $\mathcal{N}(x)$ for $0 < 1 - x \ll 1$; the emerging asymptotic behavior of $n(t)$ could be rich and varied depending on the kernel $F(\tau)$ as we shall demonstrate in the following sections.

In the *subcritical regime*, $\lambda < \lambda_c$, the number of new infections decreases with time. Indeed, the generating function $\mathcal{N}(x)$ remains finite at $x = 1$,

$$\mathcal{N}(1) = \frac{1}{1 - \lambda/\lambda_c} < \infty. \tag{20}$$

By definition

$$\mathcal{N}(1) = \sum_{t \geq 0} n(t), \tag{21}$$

so the number of new infections $n(t)$ converges to zero:

$$\lim_{t \to \infty} n(t) = 0. \tag{22}$$

If $R = \infty$, the number of new infections $n(t)$ exhibits an asymptotic exponential decay according to Eq. (12). When the convergence radius is finite and obeys $R > 1$, more complicated behaviors can occur as we shall see. In any case we observe that given the definition of our epidemic spreading model, the number of new infected individuals $n(t)$ cannot decay faster than $F(t)$. Starting from Eq. (1),

$$\begin{aligned} n(t) &= \sum_{t'=1}^{t-1} F(t-t')n(t') \\ &= n(1)F(t-1) + n(2)F(t-2) + \ldots, \end{aligned} \tag{23}$$

and truncating the sum at the first term, we have

$$n(t) \geq F(t-1) \simeq F(t) \tag{24}$$

for $t \gg 1$.

In the following sections, we demonstrate how the general exact approach described above applies to the four kernels we analyze in detail. We will show that if there are no containment measures, $F(\tau) = 1$, the exponential emerges for any $\lambda > 0$. Thus for the constant kernel, the model is always in the supercritical regime. We will also show that containment measures modeled by sufficiently quickly decaying kernels $F(\tau)$ can be effective in containing the epidemic spread by pushing the dynamics in the subcritical regime. Less stringent containment measures are not always able to drive the model in the subcritical regime, and they have the only effect of decreasing the rate μ of the exponential growth of new cases.

IV. CONSTANT KERNEL

For the constant kernel, $F(\tau) = 1$, Eq. (1) becomes

$$n(t) = \lambda \sum_{t'=0}^{t-1} n(t'). \tag{25}$$

The initial condition is $n(0) = 1$. Equation (25) can be also written as

$$n(t) = (1 + \lambda)n(t - 1), \tag{26}$$

which is solved to yield

$$n(t) = \lambda(1 + \lambda)^{t-1} = \frac{\lambda}{1 + \lambda} e^{\mu t} \tag{27}$$

with

$$\mu = \ln(1 + \lambda). \tag{28}$$

For any infectivity $\lambda > 0$, the rate μ is always positive. The number of new infections $n(t)$ exhibits a pure exponential growth. The total number of infected individuals $N(t)$ also grows exponentially with time:

$$N(t) = \sum_{t'=0}^{t} n(t') = (1 + \lambda)^t = e^{\mu t}. \tag{29}$$

Therefore for any $\lambda > 0$ the system is in the supercritical regime.

The above qualitative predictions can be also deduced from our general formalism. Indeed, for the constant kernel $F(\tau) = 1$, we have $\mathcal{F}(x) = \frac{x}{1-x}$ and Eq. (16) implies that the epidemic threshold is vanishes: $\lambda_c = 0$. We also notice that for $0 < \lambda \ll 1$, the exponential rate μ given by Eq. (28) is asymptotically

$$\mu = \lambda + O(\lambda^2). \tag{30}$$

Thus the rate μ follows the power-law scaling (19) with $\lambda_c = 0$, $D = 1$ and $\beta = 1$.

V. POWER-LAW KERNEL

The power-law kernel exemplifies kernels with slow decay in time. Below we show that for $\alpha \leq 1$, the epidemic threshold vanishes, $\lambda_c = 0$, and $n(t)$ exhibits an exponential asymptotic growth for any value of $\lambda > \lambda_c = 0$. Thus the containment measures can be effective in pushing the dynamics in the subcritical regime only if $\alpha > 1$. For any $\alpha > 1$, the epidemic threshold is indeed positive, $\lambda_c > 0$, so the containment measures bring the epidemics to the subcritical regime when $\lambda < \lambda_c$.

A. Epidemic threshold

We tacitly assume that $\alpha > 0$ since $\alpha = 0$ reduces to the constant kernel. When $F(\tau) = \tau^{-\alpha}$, the generating function $\mathcal{F}(x)$ is a polylogarithmic function of order α:

$$\mathcal{F}(x) = \mathrm{Li}_\alpha(x) = \sum_{n \geq 1} \frac{x^n}{n^\alpha}. \tag{31}$$

According to the general solution of the model given in Sec. III A the generating function $\mathcal{N}(x)$ becomes

$$\mathcal{N}(x) = \frac{1}{1 - \lambda \, \mathrm{Li}_\alpha(x)}, \tag{32}$$

and the epidemic threshold of this model is given by

$$\lambda_c = \lim_{x \to 1^-} \frac{1}{\mathrm{Li}_\alpha(x)}. \tag{33}$$

In Fig. 1 the epidemic threshold λ_c is plotted as a function of the power-law exponent α.

Since $\mathrm{Li}_\alpha(x)$ diverges at $x = 1$ when $\alpha \leq 1$, we conclude that $\lambda_c = 0$ when $0 \leq \alpha \leq 1$. The most gentle logarithmic divergence occurs in the marginal case of $\alpha = 1$ when $\mathrm{Li}_1(x) = -\ln(1-x)$. Thus for any $\lambda > 0$, the epidemic is in the supercritical regime when $0 < \alpha \leq 1$. According to Eq. (12), the number $n(t)$ of new infected individuals grows exponentially with time at rate $\mu > 0$ given by Eq. (11). The larger the decay exponent α, the more stringent are the containment measures, so the rate μ is a decreasing function of α. Hence for $0 < \alpha < 1$ the containment measures mitigate the spread of the epidemics but cannot stop its exponential growth.

For $\alpha > 1$, the finite epidemic threshold is finite:

$$\lambda_c = \frac{1}{\mathrm{Li}_\alpha(1)} = \frac{1}{\zeta(\alpha)} > 0, \tag{34}$$

where $\zeta(\alpha) = \sum_{n \geq 1} n^{-\alpha}$ is the zeta function. Thus for $\lambda < \lambda_c$, the containment measures are able to push the the dynamics in the subcritical regime stopping the exponential growth. Since $\zeta(\alpha) > 1$ for all $\alpha > 1$, the epidemic threshold λ_c is bounded from above, viz.

$$\lambda_c < 1. \tag{35}$$

The zeta function has a simple pole at $\alpha = 1$, and near the pole it admits an expansion

$$\zeta(\alpha) = \frac{1}{\alpha - 1} + \gamma_E + O(\alpha - 1) \tag{36}$$

where $\gamma_E = 0.5772156649\ldots$ is the Euler-Mascheroni constant. Using this expansion one deduces the scaling of the epidemic threshold when $0 < \alpha - 1 \ll 1$:

$$\lambda_c = \alpha - 1 - \gamma_E(\alpha - 1)^2 + O[(\alpha - 1)^3]. \tag{37}$$

We now discuss in detail the supercritical, critical and subcritical regimes for the power-law kernel with decay exponent $\alpha > 0$.
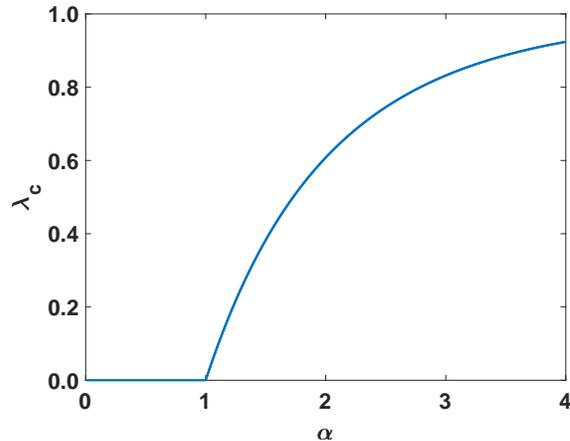

FIG. 1: The epidemic threshold λ_c versus the exponent α characterizing the power-law kernel (4). The epidemic threshold vanishes, $\lambda_c = 0$, for $0 < \alpha \leq 1$; when $\alpha > 1$, the epidemic threshold is an increasing function of α obeying $\lambda_c \leq 1$.

B. Supercritical regime

The general solution of the model, Sec. III A, implies that in the supercritical regime the number $n(t)$ individuals infected at time t obeys the asymptotic scaling

$$n(t) \simeq A_\mu \, e^{\mu t} \tag{38}$$

with $\mu > 0$ satisfying Eq. (11) which becomes

$$1 = \lambda \, \mathrm{Li}_\alpha(e^{-\mu}). \tag{39}$$

The amplitude A_μ in (38) is given by (13) which gives

$$A_\mu = e^\mu \, \frac{\mathrm{Li}_\alpha(e^{-\mu})}{\mathrm{Li}_{\alpha-1}(e^{-\mu})}. \tag{40}$$

In Fig. 2(a) we provide numerical evidence of the exponential grow of $n(t)$ in the supercritical regime $\lambda > \lambda_c$. Both $n(t)$ and $N(t)$ exhibit the exponential growth with the same growth rate:

$$N(t) \simeq C e^{\mu t}, \tag{41}$$

with $C = A_\mu/(e^\mu - 1)$. Note that despite the fact in the supercritical regime the epidemics spreads exponentially in time, the value of $\mu > 0$ determining the exponential grow varies as a function of α and λ.

For $\alpha \neq 1$, the rate μ is a function of λ, and α is implicitly determined by Eq. (39). This transcendental equation does not admit a general explicit solution. One exception is the marginal case of $\alpha = 1$ when the polylogarithmic function becomes $\mathrm{Li}_1(x) = -\ln(1-x)$. Combining this with Eq. (39) we extract an explicit expression

$$\mu = -\ln(1 - e^{-1/\lambda}) \tag{42}$$

in the marginal case of $\alpha = 1$.

We now present various asymptotic expansion of μ for different values of α. In particular, we analyze the scaling of μ for $\lambda \to \infty$ and for $\lambda \to \lambda_c^+$ at $\alpha \geq 0$.
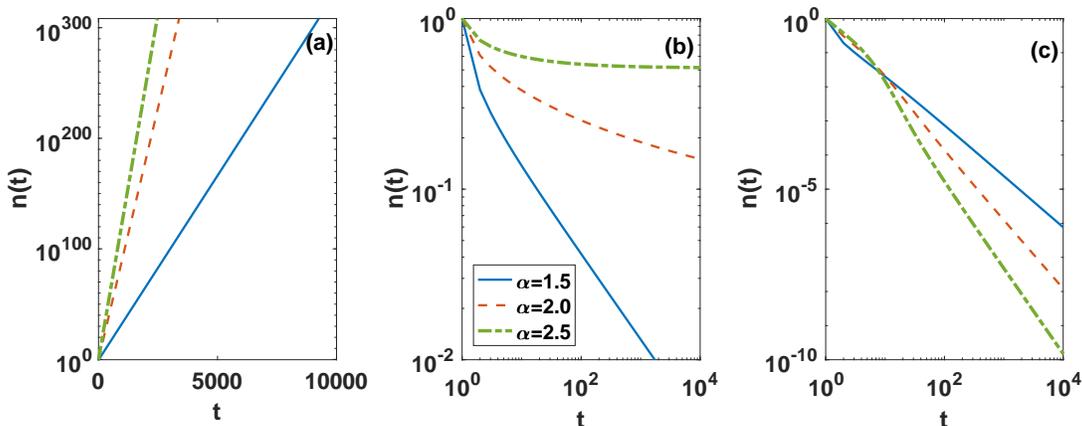
FIG. 2: The number $n(t)$ of new infected individuals for the power-law kernel given by Eq. (4) is plotted versus time t for $\alpha = 1.5, 2.0, 2.5$. Panel (a) refers to the supercritical regime with $\lambda = 1.5\lambda_c$; panel (b) refers to the critical regime with $\lambda = \lambda_c$; panel (c) refers to the subcritical regime with $\lambda = 0.5\lambda_c$.

1. Scaling of μ for $\lambda \to \infty$

For the constant temporal kernel, the growth rate reads $\mu = \ln(1 + \lambda)$, see Eq. (28), so it diverges logarithmically as $\lambda \to \infty$. The presence of non-trivial power-law containment measures ($\alpha > 0$), the rate μ also diverges logarithmically as we now demonstrate. Indeed, combining the definition (31) of the polylogarithmic function,

$$\mathrm{Li}_\alpha(e^{-\mu}) = e^{-\mu} + 2^{-\alpha}e^{-2\mu} + \ldots,$$

with Eq.(39) we find

$$\mu = \ln(1 + \lambda) - \frac{1 - 2^{-\alpha}}{\lambda} + O(\lambda^{-2}). \tag{43}$$

This analytical prediction is supported by numerical results, see Fig. 3 where we plot $\ln(1 + \lambda) - \mu$ versus α. In the limit $\lambda \to \infty$ we observe the same leading term as for $\alpha = 0$ with an α-dependent sub-leading correction of order of $1/\lambda$. Thus the containment measures lead only to sub-leading corrections to a diverging value of μ.

2. Scaling of μ when $\lambda \to \lambda_c^+$

Here we examine the behavior of the growth rate μ in the $\lambda \to \lambda_c^+$ limit. The linear scaling (17) occurs when $\alpha > 2$. A more general scaling law (19) with dynamical exponent $\beta > 1$ occurs in the range $0 < \alpha < 2$. There are two anomalies: when $\alpha = 1$, the exponent β diverges, while when $\alpha = 2$, there is an additional logarithmic correction to the linear scaling (17). We now derive these results and establish the dependence of the amplitude D and the exponent β on α.

(a) Case $0 \leq \alpha < 1$.
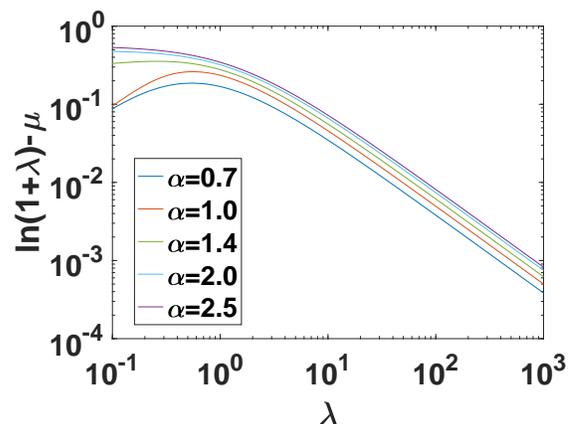From the definition (31) of the polylogarithmic

FIG. 3: The discrepancy $\ln(1 + \lambda) - \mu$ between the growth rate μ and its universal leading behavior is plotted versus λ for the model with power-law kernel (4). The results for different values of the exponent $\alpha = 0.7, 1.0, 1.4, 2.0, 2.5$ are shown.

function one extracts the expansion

$$\mathrm{Li}_\alpha(x) = (1 - x)^{\alpha-1}\Gamma(1 - \alpha) + O(1) \tag{44}$$

when $x \to 1^-$. Substituting this expansion into Eq. (39) we obtain

$$\mu \simeq D\lambda^{\frac{1}{1-\alpha}}, \quad D = [\Gamma(1 - \alpha)]^{1/(1-\alpha)}. \tag{45}$$

Thus $\lambda_c = 0$ and $\beta = (1 - \alpha)^{-1}$.

(b) Case $\alpha = 1$.
The epidemic threshold also vanishes in this case, $\lambda_c = 0$, and the explicit solution (42) leads to the exponential scaling

$$\mu = e^{-1/\lambda} + O(e^{-2/\lambda}). \tag{46}$$

Thus the exponent β is effectively infinite.

(c) *Case* $1 < \alpha < 2$.

The epidemic threshold is $\lambda_c = 1/\zeta(\alpha)$. The polylogarithmic function admits the asymptotic expansion

$$\mathrm{Li}_\alpha(x) = \zeta(\alpha) + (1-x)^{\alpha-1}\Gamma(1-\alpha) + \dots \quad (47)$$

when $x \to 1^-$. By inserting this expansion into Eq. (39) we arrive at Eq. (19) with

$$\beta = \frac{1}{\alpha-1}, \quad D = \left[-\frac{\zeta^2(\alpha)}{\Gamma(1-\alpha)}\right]^{1/(1-\alpha)} \quad (48)$$

(d) *Case* $\alpha = 2$.

The polylogarithmic function $\mathrm{Li}_2(x)$ admits the asymptotic expansion

$$\mathrm{Li}_2(x) = \zeta(2) + (1-x)[\ln(1-x) - 1] + \dots \quad (49)$$

when $x \to 1^-$. By inserting Eq. (49) into Eq. (39) and recalling that $\lambda_c = 1/\zeta(2) = 6/\pi^2$, we get

$$\mu \simeq -D\frac{(\lambda-\lambda_c)}{\ln(\lambda-\lambda_c)}, \quad D = \zeta^2(2) = \frac{\pi^4}{36}. \quad (50)$$

Thus when $\alpha = 2$ the rate μ acquires a logarithmic correction to the linear in $\lambda - \lambda_c$ scaling.

(e) *Case* $\alpha > 2$.

From the definition (31) of the polylogarithmic function one extracts the expansion

$$\mathrm{Li}_\alpha(x) = \zeta(\alpha) - (1-x)\zeta(\alpha-1) + o(1-x). \quad (51)$$

Inserting this expression into Eq. (39) we find

$$\mu \simeq D(\lambda-\lambda_c), \quad D = \frac{\zeta^2(\alpha)}{\zeta(\alpha-1)}. \quad (52)$$

Thus the dynamical exponent is universal, $\beta = 1$, for all $\alpha > 2$. The prediction (52) can be also deduced by specializing the general result (18) to the power-law kernel with $\alpha > 2$.

Figure 4 shows numerical results providing evidence for the asymptotic scalings of μ as a function of $\lambda - \lambda_c$ discussed above.

C. Critical region: $\alpha > 1$ and $\lambda = \lambda_c$

An asymptotic analysis (see Appendix A for details) shows that at the epidemic threshold, $\lambda = \lambda_c = 1/\zeta(\alpha)$, the number of new infected individuals $n(t)$ exhibits the following asymptotic behaviors:

$$n(t) \simeq \begin{cases} A t^{\alpha-2} & \text{for } 1 < \alpha < 2, \\ A/\ln t & \text{for } \alpha = 2, \\ A & \text{for } \alpha > 2. \end{cases} \quad (53)$$
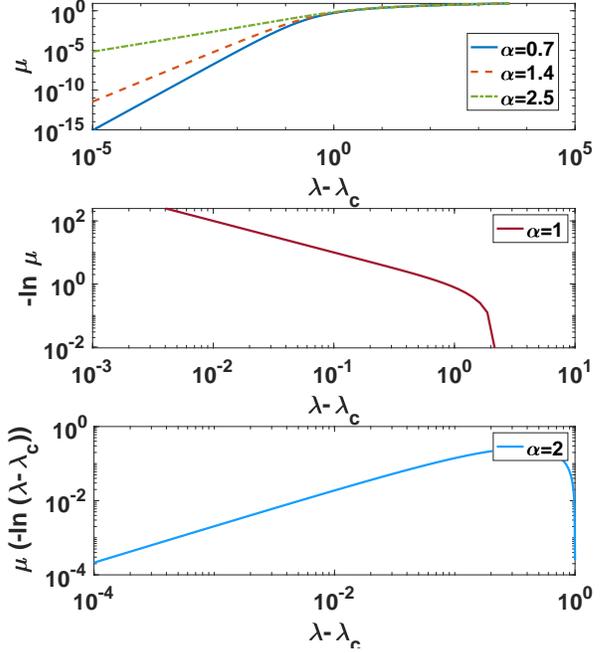

FIG. 4: The growth rate μ versus $\lambda - \lambda_c$ for the power-law kernel (4) with different values of the exponent α.

The amplitude A in Eq. (53) actually depends on α:

$$A = \begin{cases} -\zeta(\alpha)/\left[\Gamma(\alpha-1)\Gamma(1-\alpha)\right] & \text{for } 1 < \alpha < 2, \\ \zeta(2) & \text{for } \alpha = 2, \\ \zeta(\alpha)/\zeta(\alpha-1) & \text{for } \alpha > 2. \end{cases} \quad (54)$$

Thus the average number of new cases remains constant when $\alpha > 2$; otherwise, the number of new infected individuals decays with time. The predictions of Eq. (53) are confirmed by the direct numerical integration of the dynamics dictated by Eq. (1) in the critical regime $\lambda = \lambda_c$, see Fig. 2(b). By using the asymptotic expression for $n(t)$ in Eq. (53) we deduce the scaling of the total number $N(t)$ of infected individuals at time t, given by

$$N(t) \simeq \begin{cases} C t^{\alpha-1} & \text{for } 1 < \alpha < 2, \\ C t/\ln t & \text{for } \alpha = 2, \\ C t & \text{for } \alpha > 2, \end{cases} \quad (55)$$

with

$$C = \begin{cases} \zeta(\alpha)/\left[\Gamma(\alpha-1)\Gamma(2-\alpha)\right] & \text{for } 1 < \alpha < 2, \\ \zeta(2) & \text{for } \alpha = 2, \\ \zeta(\alpha)/\zeta(\alpha-1) & \text{for } \alpha > 2. \end{cases} \quad (56)$$

Thus in the critical regime, $\lambda = \lambda_c$ with $\alpha > 1$, the total number of infected individuals grows linearly when $\alpha > 2$ and sub-linearly when $1 < \alpha \leq 2$.

D. Subcritical region: $\alpha > 1$ and $\lambda < \lambda_c$

In this subcritical regime, the asymptotic behavior of new infected individuals is algebraic

$$n(t) \simeq A\,t^{-\alpha}, \quad A = \frac{\lambda}{[1 - \lambda\,\zeta(\alpha)]^2}. \qquad (57)$$

Thus the asymptotic behavior is dominated by the time dependence of the power-law kernel $F(\tau)$. One can establish (57) by performing an asymptotic analysis of the behavior of $\mathcal{N}(x)$ as $x \to 1^-$, which in turn requires the knowledge of the behavior of $\mathrm{Li}_\alpha(x)$ as $x \to 1^-$. The details are presented in Appendix B. The analysis is rather straightforward in the range $1 < \alpha \leq 2$, but become more and more tedious as α increases. We have verified (57) in details when $\alpha < 3$, and we have argued for the validity of simple general prediction (57) despite of the fact that our proof quickly becomes unwieldy, e.g. it requires the asymptotic expansion till order k and $k-$fold differentiations when $k < \alpha \leq k+1$. Our numerical results, see Fig. 2(c), are in excellent agreement with the theoretical prediction (57) for all values $\alpha > 2$ where we have performed simulations.

Using Eq. (57) we find that the total number of infected individuals $N(t)$ saturates to a constant value as

$$N(t) = A\left[\zeta(\alpha) - \frac{1}{\alpha-1}t^{1-\alpha}\right] + O(t^{-\alpha}). \qquad (58)$$

VI. EXPONENTIAL KERNEL

Let us assume that the effective infectivity of an individual decays exponentially with time, $F(\tau) = e^{-\gamma\tau}$. The constant kernel corresponds to $\gamma = 0$, so we tacitly assume that $\gamma > 0$. Equation (1) can be written as the recursive equation

$$n(t) = e^{-\gamma}(1 + \lambda)n(t-1), \qquad (59)$$

valid for any $t \geq 2$ with initial condition $n(1) = \lambda e^{-\gamma}$. Solving this equation yields

$$n(t) = \frac{\lambda}{1+\lambda}e^{\mu t}, \qquad (60)$$

with

$$\mu = \ln(1+\lambda) - \gamma. \qquad (61)$$

For the exponential kernel, the generating function

$$\mathcal{F}(x) = G_\gamma(x) = \sum_{m=1}^{\infty}\left(xe^{-\gamma}\right)^m = \frac{e^{-\gamma}x}{1 - xe^{-\gamma}}. \qquad (62)$$

has the radius of convergence $R = e^\gamma > 1$. The epidemic threshold is

$$\lambda_c = \frac{1}{G_\gamma(1)} = e^\gamma - 1. \qquad (63)$$

Thus the containment measures suppress the spreading of the epidemic when $\lambda < \lambda_c = e^\gamma - 1$.

We now use Eq. (61) and Eq. (63) to derive the properties of the three different regimes. In the supercritical phase, $\lambda > \lambda_c$, the growth rate (61) is smaller than for the constant kernel (corresponding to $\gamma = 0$). Close to the critical point the scaling of μ is similar to the scaling in for the constant kernel, namely it is linear in $\lambda - \lambda_c$:

$$\mu = D(\lambda - \lambda_c), \quad D = e^{-\gamma}. \qquad (64)$$

In the critical phase, the number of new cases is constant in time. In the subcritical phase, the number of new cases decays exponentially. The total number $N(t)$ of infected individuals is determined by Eq. (14) for any value of λ, with μ given by Eq. (61).
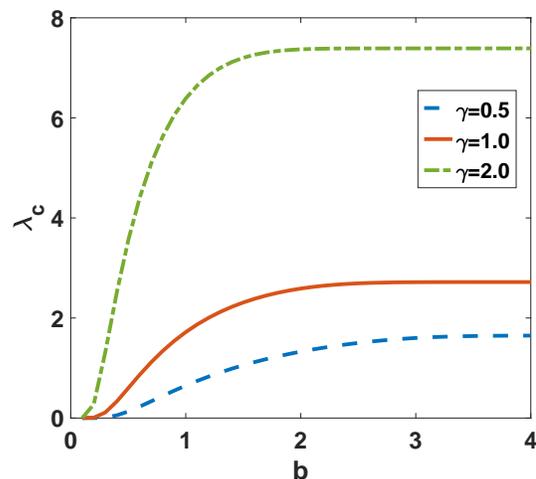

FIG. 5: The epidemic threshold λ_c for generalized exponential kernel (65) is plotted versus b for $\gamma = 0.5, 1.0, 2.0$.

VII. GENERALIZED EXPONENTIAL DECAY

In this section, we consider a two-parameter class of generalized exponential decay kernels

$$F(\tau) = \exp\left[-\gamma\tau^b\right], \quad \gamma > 0 \quad \text{and} \quad b > 0. \qquad (65)$$

In this case, the generating function $\mathcal{F}(x)$ becomes

$$\mathcal{F}(x) = G_{\gamma,b}(x) = \sum_{m\geq 1} x^m\, e^{-\gamma m^b}. \qquad (66)$$

From the general solution presented in Sec. III A we find that the generating function $\mathcal{N}(x)$ of the number of new infected individuals reads

$$\mathcal{N}(x) = \frac{1}{1 - \lambda\,G_{\gamma,b}(x)} \qquad (67)$$

and the epidemic threshold is given by

$$\lambda_c = \frac{1}{G_{\gamma,b}(1)} = \left[\sum_{m=1}^{\infty} e^{-\gamma m^b}\right]^{-1}. \tag{68}$$

In Fig. 5 we plot the epidemic threshold λ_c as a function of b for generalized exponential kernels with $\gamma = 1$.

For all values of $b > 0$ $G_{\gamma,b}(1)$ and $G'_{\gamma,b}(1)$ are finite, therefore the growth rate μ exhibits the linear scaling (17)–(18) in the $\lambda \to \lambda_c^+$ limit. Specializing Eq. (18) to the kernel (65) we get Eq. (68) with

$$D = \frac{[G_{\gamma,b}(1)]^2}{G'_{\gamma,b}(1)} = \frac{\left[\sum_{m\geq 1} e^{-\gamma m^b}\right]^2}{\sum_{m\geq 1} m\,e^{-\gamma m^b}} \tag{69}$$

The sum in Eq. (68) and Eq. (69) cannot be generally expressed through known special functions. One exception is the $b = 2$ case when recalling the definition of the Jacobi theta function

$$\theta_3(q) = \sum_{n=-\infty}^{\infty} q^{n^2} \tag{70}$$

we re-write the epidemic threshold as

$$\lambda_c = \frac{2}{\theta_3(e^{-\gamma}) - 1}. \tag{71}$$

In the general case of arbitrary $b > 0$, the asymptotic behaviors of the sums in Eq. (68) and Eq. (69) can be established when $\gamma \to 0^+$. Indeed, in this situation we replace the summation by integration and arrive to the following leading behaviors

$$\lambda_c \simeq \frac{\gamma^{1/b}}{\Gamma\left(1 + \frac{1}{b}\right)}, \quad D \simeq 2\,\frac{\Gamma^2\left(1 + \frac{1}{b}\right)}{\Gamma\left(1 + \frac{2}{b}\right)}. \tag{72}$$

The number $n(t)$ of new infected individuals follows different scaling behaviors depending on whether $b > 1$ or $b < 1$. The kernel $F(\tau)$ decays faster than exponential if $b > 1$, so the generating function $G_{\gamma,b}(x)$ has an infinite radius of convergence in this situation and the number $n(t)$ of new infected individuals follows Eq. (12). The rate μ is determined by Eq. (11) that for the kernel (65) becomes

$$\lambda\, G_{\gamma,b}(e^{-\mu}) = 1. \tag{73}$$

In Fig. 6 we show numerical results for the number $n(t)$ of new infected individuals for $b = 1.25 > 1$ in the supercritical, critical and subcritical regime. The total number $N(t)$ of infected individuals for $b > 1$ follows Eq. (14) for any value of λ, with the rate μ satisfying Eq. (73).

When $b < 1$, the kernel $F(\tau)$ decays slower than exponential and the radius of convergence of $G_{\gamma,b}(x)$ is $R = 1$. Therefore we might expect deviations from the exponential scaling described by Eq. (12) in the critical and subcritical regimes. Here we summarize the asymptotic behaviors in these regimes (see Appendix C for the derivations). In the critical regime, the asymptotic analysis

shows that the number of new infected individuals $n(t)$ saturates asymptotically for large times t (see Figure 6), with limit given by

$$\lim_{t\to\infty} n(t) = \frac{G_{\gamma,b}(1)}{G'_{\gamma,b}(1)}. \tag{74}$$

Therefore in the critical regime, the total number $N(t)$ of infected individuals grows linearly with time for $t \gg 1$.

In the subcritical regime, the asymptotic scaling analysis (see Appendix C) implies that $n(t)$ decays faster than t^{-2}. Our numerical analysis indicates that $n(t)$ decays like $F(t)$, see Fig. 6(c). Therefore in the subcritical regime, the total number $N(t)$ of infected individuals for sufficiently long times saturates to a constant value.

VIII.   MULTI-FOCI SI MODEL

An epidemic outbreak in one region of the world can spread to other regions also in presence of containment measures forming several foci of the epidemics. We thus consider a model in which the pandemic is formed by a set of separated foci i where the outbreak starts at different times t_i. A realistic meta-population model of this sort may account for the mobility of the individuals across the different locations, here we take a simplified mean-field approach and assume that the number of new foci at time $t = t_i$ is a deterministic function of t_i indicated by $\rho(t_i)$. We consider two functional forms for $\rho(t_i)$:

(A) A power-law functional form for $\rho(t_i)$

$$\rho(t_i) = B t_i^{\gamma}, \tag{75}$$

where $\gamma \geq 0$ and $B > 0$. A constant number of new foci as a function of time corresponds to $\gamma = 0$; if $\gamma > 0$, the number of new foci increases with time.

(B) An exponential functional form for $\rho(t_i)$

$$\rho(t_i) = B e^{\theta t_i} \tag{76}$$

with $\theta \geq 0$ and $B > 0$. If $\theta > 0$, the number of new foci increases exponentially as a function of time.

In both scenarios the total number of cases $I(t)$ at time t calculated across all the foci is given by

$$I(t) = \sum_{1}^{t-1} N_i(t - t_i)\rho(t_i), \tag{77}$$

where $N_i(t - t_i)$ is the total number of cases of the foci i at time t. In principle, at every foci different containment measures could be applied, in particular if they are in different countries. However, we focus on the simplest situation whhen each focus follows the same dynamics and has the same parameters. This could be a reasonable assumption for describing different foci in the same country (as for instance China in the outbreak of COVID-19).
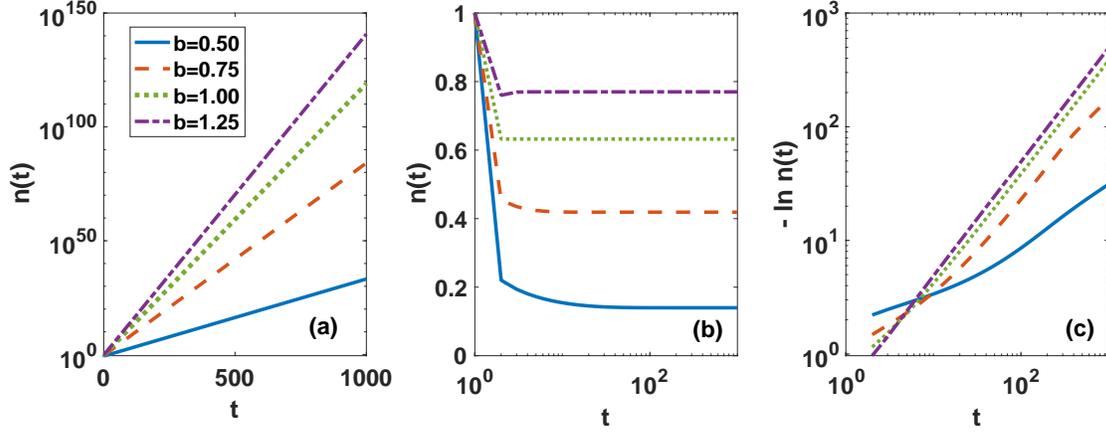
FIG. 6: The number $n(t)$ of new infected individuals is plotted versus time t for the generalized exponential kernel $F(\tau)$ given by Eq. (65) with $\gamma = 1$ and $b = 0.50, 0.75, 1.00, 1.25$. Panel (a) refers to the supercritical regime with $\lambda = 1.5\lambda_c$, panel (b) refers to the critical regime $\lambda = \lambda_c$, and panel (c) refers to the subcritical regime $\lambda = 0.5\lambda_c$.

IX. TOTAL NUMBER OF INFECTED IN THE MULTI-FOCI MODEL

In this section we calculate the total number of infected individuals $I(t)$ in the multi-foci meta-population approach. Since we assume that every foci follows the same dynamics, $I(t)$ is given by Eq. (77) with N_i being the same and just shifted to the activation time t_i, that is $N_i(t - t_i) = N(t - t_i)$ at time t.

For the kernels which we employ, the asymptotic behavior of $N(t)$ at large time can be cast in two major classes: the exponential behavior and the power-law behavior. We now separately consider these two cases.

A. Exponential case

Consider an exponential dependence of $N(t)$, i.e.

$$N(t) \simeq Ce^{\mu t}, \qquad (78)$$

where without loss of generality we consider $\mu > 0$.

(A) If the number of new foci increases as a power-law, Eq. (75), by putting Eq. (78) into Eq. (77) and limiting ourselves to the situation when the growth of $N(t)$ is exponential, $\mu > 0$, we obtain

$$I(t) \simeq \mathcal{C} \, \mathrm{Li}_{-\gamma}(e^{-\mu}) \, e^{\mu t}, \qquad (79)$$

where $\mathcal{C} = BC$ and where $\mathrm{Li}_a(x)$ is a polylogarithm with index a. Therefore for $\mu > 0$ the presence of different foci does not change the exponential trend and $I(t)$ and $N(t)$ differ only by a constant.

(B) If the number of new foci increases exponentially, Eq. (76), we put Eq. (78) into Eq. (77) to yield

$$I(t) \simeq \begin{cases} \mathcal{C}e^{\mu t} & \text{if } \mu > \theta, \\ \mathcal{C}te^{\mu t} & \text{if } \mu = \theta, \\ \mathcal{C}e^{\theta t}[e^{\theta-\mu} - 1]^{-1} & \text{if } \mu < \theta, \end{cases} \qquad (80)$$

where $\mathcal{C} = BC$. Thus the presence of different foci changes the exponential trends if and only if $\theta \geq \mu$.

B. Power-law case

We now consider the case in which the total number of infected individuals $N(t)$ in each focus of the epidemics scales as a power-law,

$$N(t) = Ct^{\nu}, \qquad (81)$$

for $t \gg 1$. We can assume that $\nu \geq 0$. Indeed, the definition of the total number $N(t)$ of infected individuals in a given focus, given by Eq. (2) implies that $N(t)$ is non-decreasing function of time, with $N(t) \geq n(0) = 1$.

(A) We now insert Eq. (81) into Eq. (77) and approximate the sum by an integral in the long time limit. Computing the integral we obtain

$$I(t) \simeq C \, B(1 + \gamma, 1 + \nu) \, t^{1+\gamma+\nu}, \qquad (82)$$

where $B(a, b)$ is the Euler beta function

$$B(a, b) = \int_0^1 dx \, x^{a-1}(1 - x)^{b-1}. \qquad (83)$$

The replacement of the sum by an integral leading to Eq. (82) is asymptotically justifiable when $\gamma > -1$. Note that both $I(t)$ and $N(t)$ grow algebraically. The presence of different foci *accelerates* the growth, $1+\gamma+\nu > \nu$ when $\gamma > -1$. This could be a scenario compatible with the finding reported in Ref. [7] regarding the COVID-19 data in China.

When $\gamma \leq -1$, we need to estimate the sum in Eq. (77) more carefully. One finds

$$I(t) \simeq Ct^{\nu} \times \begin{cases} \ln t & \gamma = -1, \\ \zeta(-\gamma) & \gamma < -1. \end{cases} \qquad (84)$$

(B) If the number of new foci increases exponentially with time (i.e. it follows Eq. (76)), by putting Eq. (81) into Eq. (77) we obtain

$$I(t) \simeq \mathcal{C}\mathrm{Li}_{-\nu}(e^{-\theta})e^{\theta t} \qquad (85)$$

where $\mathcal{C} = BC$ and the polylogarithm function $\mathrm{Li}_a(x)$ is defined in Eq. (31). Therefore in this case the total number of infected across all the foci is growing exponentially with rate θ. In other words, $I(t)$ growth in time is dominated by the rate at which new foci are established.

X. CONCLUSIONS

We proposed an SI epidemic spreading model with effective time-dependent infectivity that models different types of containment measures. This theoretical framework can be used to investigate the onset of an epidemics and the role that a time-dependent infectivity can have on the spread of the disease. We demonstrated that different containment measures can either lead to a slowing down of the exponential spread by modulating the rate μ of the exponential growth of new case, or can bring the epidemic to an halt when they push the dynamics in the subcritical regime. In particular, exponential and generalized exponential temporal kernels always induce a finite epidemic threshold λ_c, so they are able to stop epidemics provided that $\lambda < \lambda_c$. For power-law temporal kernels, the effective infectivity is able to induce a non-vanishing epidemic threshold only if they are steep enough, viz. if the power-law exponent α determining their decay exceeds unity: $\alpha > 1$. For different temporal kernels of the infectivity, in the supercritical regime, $\lambda > \lambda_c$, the total number of infected individuals grows exponentially fast; in the subcritical regime, i.e. below the epidemic threshold, the total number of infected individuals saturates to a constant; in the critical regime, the number of infected individuals grows in time linearly or sub-linearly. These results have been obtained assuming a well-mixed approximation and by considering a single focus of the epidemic.

We also investigated the growth of the total number of cases in a pandemic formed by a growing set of epidemic foci, each having at least one infected individual. We studied the simplest situation with each focus follows the same dynamics. We showed that if the number of new foci increases a a power-law of time, in the supercritical regime the total number of cases across different foci scales like the total number of case in each focus. In the critical (and subcritical) regime, the total number of cases across different foci can grow faster than linearly. When the number of new foci increases exponentially the growth of the number of cases across different foci is always growing exponentially at the rate at which the new foci are established with the only exception when the growth rate μ of the number of new cases in each single focus is faster, in which case the total number of infected across different foci grows at rate μ.

There are many avenues for future work. An obvious generalization is to consider SIR and SIS model including the same time-dependent effective infectivity. Perhaps the most important challenge is to model stochastic characteristics and try to account for large fluctuations observed in pandemics. Stochastic characteristics are difficult to describe even in the classical SIR model in the critical regime [37–44], and they may play an important role in our model. Finally the multi-foci meta-population approach could be expanded by considering the effect of social and transportation networks.

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Appendix A: Derivation of Eq. (53)

In this Appendix we derive the announced asymptotic behaviors (53) of the number of new infected individual $n(t)$ in the critical regime for the power-law kernel . We also derive the predictions (54) for the amplitude, and additionally compute the sub-leading term in the special case of $\alpha = 2$ when the convergence to the leading asymptotic is anomalously slow.

Our starting point is Eq. (32) that we rewrite as

$$\mathcal{N}(x) = \frac{1}{1 - \lambda_c \operatorname{Li}_\alpha(x)}. \tag{A1}$$

We keep in mind known relations $\lambda = \lambda_c = 1/\zeta(\alpha)$ characterizing the critical regime of the power-law kernel in the $\alpha > 1$ range.

To establish Eq. (53) we expand the right-hand side of Eq. (A1) in the region $x \to 1^-$; the asymptotic behavior of $n(t)$ follows from this expansion. The polylogarithmic function $\operatorname{Li}_\alpha(x)$ exhibits different asymptotic behaviors in the $x \to 1^-$ limit depending on whether different values of α is smaller or larger than 2. Therefore we separately treat the cases of $1 < \alpha < 2$, $\alpha = 2$ and $\alpha > 2$.

1. Case $1 < \alpha < 2$

In this range, the polylogarithmic function $\operatorname{Li}_\alpha(x)$ admits the asymptotic expansion (47) which we insert into Eq. (A1) and arrive at

$$\mathcal{N}(x) \simeq -\frac{\zeta(\alpha)}{\Gamma(1-\alpha)} (1-x)^{1-\alpha} \tag{A2}$$

as $x \to 1^-$. Thus

$$\sum_{t \geq 0} n(t) \, x^t \simeq -\frac{\zeta(\alpha)}{\Gamma(1-\alpha)} (1-x)^{1-\alpha} \tag{A3}$$

which implies the large time behavior

$$n(t) \simeq -\frac{\zeta(\alpha)}{\Gamma(\alpha-1)\,\Gamma(1-\alpha)} \, t^{\alpha-2} \tag{A4}$$

stated in Eqs. (53)–(54) when $1 < \alpha < 2$. Using the Euler identity $\Gamma(y)\Gamma(1-y) = \pi/\sin(\pi y)$, one can also re-write (A4) as

$$n(t) \simeq \frac{(\alpha-1)\zeta(\alpha)\sin[\pi(\alpha-1)]}{\pi} \, t^{\alpha-2} \tag{A5}$$

A simple 'physical' confirmation of Eq. (A4) is obtained by substituting Eq.(A4) into the sum in the left-hand side of Eq. (A2), noting that in the $x \to 1^-$ limit the summation can be replaced by integration, computing the integral and recovering the right-hand side of (A2). A rigorous derivation of the asymptotic of the coefficients from the singular behavior of the generating function can be done by a variety of techniques, e.g. by using Tauberian theorems [46] or complex analysis [47]; see the textbook [48] for numerous examples.

2. Case $\alpha = 2$

The polylogarithmic function $\mathrm{Li}_2(x)$ has the asymptotic expansion (49) which we insert into Eq. (A1) and obtain

$$\mathcal{N}(x) \simeq \frac{\zeta(2)}{1 - \ln(1-x)} \frac{1}{1-x} , \quad \text{(A6)}$$

from which we deduce the leading asymptotic behavior reported in Eqs. (53)–(54) at $\alpha = 2$. The presence of logarithms often implies that the sub-leading term is just logarithmically smaller than the leading term, and then the sub-sub-leading term is another logarithmic factor smaller. The derivation of these sub-leading terms is a bit long, but it uses standard techniques [47, 48]; alternatively, it can be also extracted from the general results presented in [48]. Keeping just the leading and sub-leading terms yields the following asymptotic

$$n(t) \simeq \frac{\zeta(2)}{\ln t + \gamma_E + 1} \quad \text{(A7)}$$

where γ_E is the Euler-Mascheroni constant and we have dropped the terms of the order $(\ln t)^{-3}$. Using Eq. (A7) we obtain a slightly more precise version of Eq. (55) at $\alpha = 2$:

$$N(t) \simeq \frac{\zeta(2)\,t}{\ln t + \gamma_E} \quad \text{(A8)}$$

3. Case $\alpha > 2$

When $\alpha > 2$, the polylogairthmic function $\mathrm{Li}_\alpha(x)$ admits the asymptotic expansion (51) which we insert into (A1) and get

$$\mathcal{N}(x) \simeq \frac{\zeta(\alpha)}{\zeta(\alpha-1)} (1-x)^{-1}, \quad \text{(A9)}$$

implying that the number $n(t)$ of new infected individuals saturates,

$$\lim_{t\to\infty} n(t) = \frac{\zeta(\alpha)}{\zeta(\alpha-1)} , \quad \text{(A10)}$$

as stated in Eqs.(53)–(54) at $\alpha > 2$.

Appendix B: Derivation of Eq. (57)

In this Appendix we derive the announced asymptotic behavior (57) of the number of new infected individuals $n(t)$ for the power-law kernel in the subcritical regime. We start with Eq. (32) that we rewrite here for convenience

$$\mathcal{N}(x) = \frac{1}{1 - \lambda\mathrm{Li}_\alpha(x)} . \quad \text{(B1)}$$

The sub-critical regime $\lambda < \lambda_c = 1/\zeta(\alpha)$ is possible for all $\alpha > 1$. Since $\mathcal{N}(1)$ is finite, we consider the expansion of $\mathcal{N}(1) - \mathcal{N}(x)$ in the $x \to 1^-$ limit. By using Eq. (B1) it is possible to get the asymptotic expression of $n(t)$ as long as $1 < \alpha \le 2$. In the following paragraph we will show in detail how this expansion can be carried out for $1 < \alpha < 2$, and $\alpha = 2$ and how in these cases we recover the asymptotic scaling in Eq. (57). Moreover we will show how the same method in the case $\alpha > 2$ provides only a bound to the scaling of $n(t)$.

1. Case $1 < \alpha < 2$

In the $1 < \alpha < 2$ range, the deviation of $\mathrm{Li}_\alpha(x)$ from $\mathrm{Li}_\alpha(1) = \zeta(\alpha)$ scales as

$$\mathrm{Li}_\alpha(x) - \mathrm{Li}_\alpha(1) \simeq \Gamma(1-\alpha)\,(1-x)^{\alpha-1} \quad \text{(B2)}$$

when $x \to 1^-$. This is just the re-writing of Eq. (47). Using Eqs. (B1) and (B2) we find

$$\mathcal{N}(1) - \mathcal{N}(x) \simeq -\frac{\lambda\,\Gamma(1-\alpha)}{[1 - \lambda\,\zeta(\alpha)]^2} (1-x)^{\alpha-1} . \quad \text{(B3)}$$

Recalling the definition of the generating function $\mathcal{N}(x)$, we get

$$\sum_{t\ge 0} n(t)\big[1 - x^t\big] \simeq -\frac{\lambda\,\Gamma(1-\alpha)}{[1 - \lambda\,\zeta(\alpha)]^2} (1-x)^{\alpha-1} . \quad \text{(B4)}$$

Differentiating with respect to x to obtain

$$\sum_{t\ge 0} t\,n(t)\,x^{t-1} \simeq (1-x)^{\alpha-2} \frac{\lambda\,\Gamma(2-\alpha)}{[1 - \lambda\,\zeta(\alpha)]^2} \quad \text{(B5)}$$

leading to the announced asymptotic behavior (57) in the $1 < \alpha < 2$ range.

2. Case $\alpha = 2$

When $\alpha = 2$, we re-write (49) as

$$\mathrm{Li}_2(1) - \mathrm{Li}_2(x) \simeq (1-x)[\ln(1-x) - 1]. \quad \text{(B6)}$$

Using Eq. (B1) and Eq.(B6) we find

$$\mathcal{N}(1) - \mathcal{N}(x) \simeq (1-x)[\ln(1-x) - 1] \frac{\lambda}{[1 - \lambda\,\zeta(2)]^2} \quad \text{(B7)}$$

from which we deduce

$$\sum_{t\ge 0} t\,n(t)\,x^{t-1} \simeq -\ln(1-x) \frac{\lambda}{[1 - \lambda\,\zeta(2)]^2} \quad \text{(B8)}$$

leading to the announced asymptotic (57) at $\alpha = 2$.

3. Case $\alpha > 2$

For $\alpha > 2$, we re-write Eq.(51) as

$$\text{Li}_\alpha(1) - \text{Li}_\alpha(x) \simeq \zeta(\alpha - 1)(1 - x). \qquad (B9)$$

Using Eq. (B1) and Eq. (B9) we find

$$\mathcal{N}(1) - \mathcal{N}(x) \simeq \frac{\lambda\,\zeta(\alpha - 1)}{[1 - \lambda\,\zeta(\alpha)]^2}\,(1 - x). \qquad (B10)$$

The same treatment as before gives

$$\sum_{t \geq 0} t\, n(t)\, x^{t-1} \simeq \frac{\lambda\,\zeta(\alpha - 1)}{[1 - \lambda\,\zeta(\alpha)]^2} \qquad (B11)$$

which only implies that $n(t)$ should decay faster than t^{-2}.

To derive the announced asymptotic (57) for $\alpha > 2$ one should employ the expansion of $\text{Li}_\alpha(1) - \text{Li}_\alpha(x)$ which is more accurate than the leading term given by Eq. (B9). Let us first consider the region $2 < \alpha < 3$. In this range, the required more accurate form reads

$$\begin{aligned}\text{Li}_\alpha(1) - \text{Li}_\alpha(x) &= \zeta(\alpha - 1)(1 - x)\\ &- B(1 - x)^{\alpha-1} + \dots\end{aligned} \qquad (B12)$$

Differentiating Eq.(B12) twice with respect of x and using the identity

$$\frac{d^2 \text{Li}_\alpha(x)}{dx^2} = \frac{\text{Li}_{\alpha-2}(x) - \text{Li}_{\alpha-1}(x)}{x^2} \qquad (B13)$$

we obtain

$$\text{Li}_{\alpha-2}(x) - \text{Li}_{\alpha-1}(x) \simeq B(\alpha-1)(\alpha-2)(1-x)^{\alpha-3} \quad (B14)$$

in the $x \to 1^-$ limit. The leading behavior of the left-hand side of Eq. (B14) is provided by the leading asymptotic of $\text{Li}_{\alpha-2}(x)$ and it reads

$$\text{Li}_{\alpha-2}(x) \simeq \Gamma(3 - \alpha)(1 - x)^{\alpha-3} \qquad (B15)$$

Thus we fix the amplitude in (B14):

$$B = \frac{\Gamma(3 - \alpha)}{(\alpha - 1)(\alpha - 2)}. \qquad (B16)$$

Using Eq. (B1) and Eq. (B9) we obtain

$$\begin{aligned}\sum_{t \geq 0} n(t)\big[1 - x^t\big] &\simeq \frac{\lambda\,\zeta(\alpha - 1)}{[1 - \lambda\,\zeta(\alpha)]^2}\,(1 - x)\\ &- \frac{\lambda}{[1 - \lambda\,\zeta(\alpha)]^2}\,B(1 - x)^{\alpha-1}\end{aligned}$$

which we differentiate twice with respect to x to yield

$$\sum_{t \geq 0} t(t - 1)\, n(t)\, x^{t-2} \simeq (1 - x)^{\alpha-3}\,\frac{\lambda\,\Gamma(3 - \alpha)}{[1 - \lambda\,\zeta(\alpha)]^2} \quad (B17)$$

where we have also used Eq.(B16). From the above expression we confirm the announced asymptotic (57) in the range $2 < \alpha < 3$. The same tedious analysis using allows one to confirm Eq.(57) at $\alpha = 3$. In the range $3 < \alpha < 4$ one needs to use an extra term

$$\begin{aligned}\text{Li}_\alpha(1) - \text{Li}_\alpha(x) &= \zeta(\alpha - 1)(1 - x) + B_2(1 - x)^2\\ &- B_3(1 - x)^{\alpha-1} + \dots\end{aligned} \qquad (B18)$$

The most important is the singular term $B_3(1 - x)^{\alpha-1}$, with amplitude B_3 found after differentiating Eq. (B18) three times with respect of x. One then obtains

$$\sum_{t \geq 0} t(t - 1)(t - 3)\, n(t)\, x^{t-2} \sim (1 - x)^{\alpha-4} \qquad (B19)$$

from which one confirms Eq. (57) in the range $3 < \alpha < 4$.

The above tedious proof extends to all $\alpha > 2$. The simplicity of the final result, Eq. (57), hints on a possible general derivation circumventing the consideration of the infinitely many intervals $k < \alpha < k+1$ for integer $k \geq 1$, and also the separate analysis of $\alpha = k$ with integer $k \geq 2$ where the logarithms arise in the intermediate steps, but disappear from the final formula given by Eq. (57).

Appendix C: Asymptotic analysis of the generalized exponential kernel with $b < 1$

In this Appendix we discuss the derivation of the asymptotic expansion for $n(t)$ for the generalized exponential kernel with $b < 1$. In the critical regime the generating function $\mathcal{N}(x)$ satisfies

$$\mathcal{N}(x) = \frac{1}{1 - \lambda_c\, G_{\gamma,b}(x)}. \qquad (C1)$$

In the $x \to 1^-$ limit we therefore obtain

$$\mathcal{N}(x) \simeq \frac{G_{\gamma,b}(1)}{G'_{\gamma,b}(1)}\,(1 - x)^{-1}, \qquad (C2)$$

leading to the asymptotic behavior (74), namely

$$\lim_{t \to \infty} n(t) = \frac{G_{\gamma,b}(1)}{G'_{\gamma,b}(1)}. \qquad (C3)$$

In the subcritical regime, we get

$$\mathcal{N}(1) - \mathcal{N}(x) \simeq \frac{\lambda\, G'_{\gamma,b}(1)}{[1 - \lambda\, G_{\gamma,b}(1)]^2}\,(1 - x) \qquad (C4)$$

which we treat as in Appendix B to find

$$\sum_{t \geq 1} t\, n(t)\, x^{t-1} \simeq \frac{\lambda\, G'_{\gamma,b}(1)}{[1 - \lambda\, G_{\gamma,b}(1)]^2}. \qquad (C5)$$

This relation tells us that $n(t)$ decays faster than t^{-2}. To derive a more precise prediction one can use the same trick as in Appendix B, namely to establish a more precise expansion than the one provided by Eq. (C4). One

gets, however, the regular expansion, e.g. in the next order

$$\mathcal{N}(1) - \mathcal{N}(x) \simeq C_1(1-x) + C_2(1-x)^2 \qquad \text{(C6)}$$

from which we would get

$$\sum_{t \geq 1} t(t-1)\, n(t)\, x^{t-2} \simeq 2C_2$$

and conclude that $n(t)$ decays faster than t^{-3}. Proceed-ing, one finds that $n(t)$ seemingly decays faster than any power of time. Recall, that for the power-law kernel the decay of $n(t)$ in the subcritical regime is qualitatively the same as the decay of the kernel $F(\tau)$. This may occur also for the generalized exponential kernel, and our simulation results agree with this conjecture. Theoretically, however, we only established that the decay of $n(t)$ is faster than any power law.