# Exact solutions and analysis of an SIR variant with constant-time recovery

David Alexander Madore

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**Abstract**

We investigate a variant of the SIR epidemiological model in which the recovery of infected individuals takes place in constant time rather than following an exponential distribution. This model is described by a delay-differential equation: we show that the equations in question admit an exact solution in closed form (given by rational functions of an exponential of time). Using this, we investigate the qualitative differences between this modified model and classical SIR and show that, for the same reproduction number, contagiousness and expected recovery time, the constant-time recovery variant entails a sharper, more pronounced, epidemiological peak than the classical variant (exponential-process recovery), while still having the same final attack rate.

## 1 Recollections about classical SIR

**¶1.1.** Recall that the classical SIR epidemiological model, which will henceforth be designated $\mathrm{SIR}(*)$, is based on the following hypotheses:

1. immunity acquired through infection is complete and permanent, and individuals are infectious as soon as they are infected: they are successively S (susceptible, i.e., not yet infected), I (infected and therefore infectious) and R (recovered, i.e., either cured and immune or dead);

2. population is homogeneous (all individuals are equally susceptible and equally likely to be infected), constant, large enough to be treated as continuous deterministic, and contacts obey perfect mixing (in the sense that every contact is equally likely);

3. contamination and recovery follow first-order kinetics for $I + S \to I + I$ and $I \to R$ with constants $\beta$ and $\gamma$ respectively, meaning that the rate of contamination $\beta is$ is proportional to the proportion $i$ of infected and the proportion $s$ of susceptible individuals and the rate of recovery $\gamma i$ is simply proportional to the proportion of infected individuals.

**¶1.2.** It is worth spelling out (since this is the hypothesis that will be made to vary) that recovery at rate $\gamma$ means, at the individual level, that an infected individual ($I$) has a probability of recovering ($I \to R$) equal to $\gamma\, dt$ during each infinitesimal time interval of length $dt$.

In other words, each individual's recovery follows an *exponential process* with expected recovery time $\frac{1}{\gamma}$.

**¶1.3.** Using these hypotheses we model the proportions $s, i, r$ of susceptible, infected and recovered individuals using the following system of ordinary first-order nonlinear autonomous differential equations (where $x'$ denotes differentiation with respect to the time variable $t$):

$$\begin{cases} s' = -\beta is \\ i' = \beta is - \gamma i \\ r' = \gamma i \end{cases} \qquad (*)$$
$$s + i + r = 1$$

where $\beta$ and $\gamma$ are positive real parameters (the contagiousness and recovery rate).

This represents the fact that, as explained in the hypotheses, the rate of contamination is equal to $\beta is$ (leading to a decrease in $s$ and associated increase in $i$) while simultaneously the rate of recovery is $\gamma i$ (leading to a decrease in $i$ and an increase in $r$).

Naturally, realistic solutions have $s, i, r \geq 0$ though it will be interesting to relax this hypothesis, at least concerning $r$, while we discuss the existence of solutions:

**Lemma 1.4.** If $(s, i, r)$ is a solution of $(*)$ defined on an open interval $J$, and $s$ does not vanish, then we have $s = c\,\exp(-\kappa r)$ on $J$ for some constant $c$, where $\kappa := \beta/\gamma$.

*Proof.* Comparing the first equation in $(*)$ with the third, it follows that $s'/s = -\kappa r'$, which integrates to $s = c\,\exp(-\kappa r)$ on $J$, as claimed. □

**Proposition 1.5.** Maximally defined solutions to $(*)$ for which $s > 0$ and $i > 0$ at at least one point (each) are, in fact, defined on all of $\mathbb{R}$, and satisfy $s > 0$ and $i > 0$ for all times; furthermore, $s$ and $r$ have finite limits at both $+\infty$ and $-\infty$, the limits of $s$ at $+\infty$ and $-\infty$ are both (strictly!) positive, while $i(t) \to 0$ when $t \to \pm\infty$.

Also, $s$ is monotonically decreasing, $r$ is monotonically increasing, and $i$ has a unique global maximum, which is positive, and is monotonically increasing up to this maximum and monotonically decreasing afterwards.

*Proof.* Consider a maximal solution $(s, i, r)$ defined on an open interval $J = ]t_-, t_+[$ with $-\infty \leq t_- < t_+ \leq +\infty$, and assume $s > 0$ and $i > 0$ hold at least one point (each).

If $i = 0$ at some $t \in J$ then clearly $(*)$ imposes that $i = 0$ identically and $s$ and $r$ are constant. So $i > 0$ at some point gives $i > 0$ everywhere. Similarly, if $s = 0$ at some $t$ then $(*)$ imposes that $s = 0$ identically. So $s > 0$ at some point gives $s > 0$ everywhere.

Now $s' < 0$ on $J$ by the first equation in $(*)$, so $s$ is monotonically decreasing; and similarly, $r$ is monotonically increasing by the third equation. And since $i'$ can vanish at most once (namely if and when $s(t) = \gamma/\beta$), it is monotonic in the neighborhood of $t_-$ and $t_+$. So each one of $s, i, r$ has (possibly infinite) limits at $t_-$ and $t_+$, which we call $s(t_-)$ and so on.

Letting $\kappa := \beta/\gamma > 0$, by lemma 1.4 we have $s = c\,\exp(-\kappa r)$ on $J$ for some constant $c > 0$.

Clearly $s(t_+)$ is finite since $s$ is decreasing and positive. We cannot have $s(t_+) = 0$ because then $s = c\,\exp(-\kappa r)$ would impose $r(t_+) = +\infty$ so $i(t_+) = -\infty$ contradicting $i > 0$. So $s(t_+)$ is finite and positive, so $r(t_+)$ is also finite, so $i(t_+)$ is also finite. Since all three of $s, i, r$ are finite at $t_+$, we must have $t_+ = +\infty$ otherwise $J$ would not be maximal. Furthermore, $s', i', r'$ have finite limits at $+\infty$ (by $(*)$), and these limits must be $0$ since $s, i, r$ have finite limits, so $i$ must tend to $0$.

The reasoning is analogous at $t_-$ except that we must first rule out $s(t_-) = +\infty$: and indeed, since $s = c\,\exp(-\kappa r)$ this would mean $r(t_-) = -\infty$ but with $r = o(s)$ so that $s + r$ would still tend to $+\infty$ at $t_-$ and $i_-$ to $-\infty$, again a contradiction to $i > 0$. The rest is as in the previous paragraph.

Now we know that $i$ tends to $0$ at $+\infty$ and $-\infty$ and is nonzero on $\mathbb{R}$, so it cannot be monotonic, so its derivative must vanish somewhere, and we have already pointed out that it vanishes at most once, so it vanishes exactly once: $i$ is monotonically increasing until it reaches the point where $i' = 0$, and monotonically decreasing afterwards. □

**Hypothesis 1.6.** We will now limit ourselves to solutions of $(*)$ which, on top of $s > 0$ and $i > 0$ as in the previous proposition, satisfy the hypothesis that the limit $s(-\infty)$ of $s$ when $t \to -\infty$ (i.e., the supremum of $s$) equals $1$.

This is not a loss of generality: indeed, multiplying both $s$ and $i$ by a same constant $a$ and $\beta$ by $a^{-1}$ (and replacing $r$ by $(1 - a) + ar$) will make this assumption true (for an appropriate constant $a$, viz., the inverse of $s(-\infty)$). Equivalently stated, if we start with a population which is already partially immune, we can consider the epidemic restricted to the population which is not, and consider only that fraction of the population (this, of course, changes the reproduction number $\kappa$ defined below).

This assumption having been made, $s(-\infty) = 1$ gives $r(-\infty) = 0$, and then lemma 1.4 implies $s = \exp(-\kappa r)$ (the coefficient must be 1). And since $0 < s < 1$ on $\mathbb{R}$ (because $s$ is decreasing and positive), we have $r > 0$. So, to recapitulate, we have:

$$\begin{aligned} s, i, r &> 0 \\ s(-\infty) &= 1 \\ i(-\infty) &= 0 \\ r(-\infty) &= 0 \\ s &= \exp(-\kappa r) \end{aligned}$$

**¶1.7.** The only true real parameter in $(*)$ is the ration $\kappa := \beta/\gamma$ which is termed **reproduction number** for the epidemic (generally denoted $R_0$ but this seems an inauspicious choice in a system in which the letter "R" already stands for "recovered"). Indeed, a linear scaling $t \leftarrow at$ on the time parameter $t$ will divide $\beta$ and $\gamma$ by the scaling parameter $a$, allowing to specify either one of them arbitrarily (equivalently, $\beta$ and $\gamma$ have physical dimensions of inverse time, so they are multiplied by $a^{-1}$ if times are multiplied by $a$), leaving their ratio as invariant quantity.

**¶1.8.** The hypothesis made in 1.6 implies that $\kappa > 1$. Indeed, denoting $s(+\infty)$ the limit of $s$ at $+\infty$ we have $s(+\infty) < 1$ since $s$ is decreasing, and it satisfies $s(+\infty) = \exp(-\kappa(1 - s(+\infty)))$; but if $\kappa \leq 1$ then the equation $x = \exp(-\kappa(1 - x))$ cannot have a solution $x < 1$ (indeed, for $y \neq 0$, we have $\exp y > 1 + y$, which for $y := -\kappa(1 - x)$ gives $\exp(-\kappa(1 - x)) > 1 - \kappa(1 - x)$, so if $\kappa \leq 1$ and $x < 1$ this gives $\exp(-\kappa(1 - x)) > x$); we will return to this equation shortly.

This does not mean that $(*)$ does not make sense for $\kappa \leq 1$, naturally, merely that its solutions do not satisfy $s, i, r > 0$ on $\mathbb{R}$ (with $s + i + r = 1$).

**¶1.9.** The solutions of $(*)$ do not seem to be expressible in closed form as a function of time $t$. However, we can still say a number of things about their values.

Specifically we now investigate the behavior of solutions to $(*)$ at three points: when $t \to -\infty$, at peak epidemic (when $i$ is maximal), and when $t \to +\infty$.

- When $t \to -\infty$, we have $s \to 1$ (again using the hypothesis made in 1.6). Thus, $i'/i \to \beta - \gamma$ and we conclude $i \sim c \exp((\beta - \gamma)t) = c \exp(\beta \frac{\kappa-1}{\kappa} t)$. We then get $r \sim c \frac{\gamma}{\beta-\gamma} \exp((\beta - \gamma)t) = c \frac{1}{\kappa-1} \exp(\beta \frac{\kappa-1}{\kappa} t)$.
- The unique point where $i' = 0$ (peak epidemic) is characterized by $s = \frac{1}{\kappa}$. Because $s = \exp(-\kappa r)$, it satisfies $r = \frac{\log \kappa}{\kappa}$, and consequently $i = \frac{\kappa - \log \kappa - 1}{\kappa}$.
- Finally, when $t \to +\infty$, we have $s \to \Gamma$ and $r \to 1 - \Gamma$ where $\Gamma$ is the unique solution between 0 and 1 of the equation $\Gamma = \exp(-\kappa(1 - \Gamma))$, described as $\Gamma = -\frac{\mathrm{W}(-\kappa \exp(-\kappa))}{\kappa}$ by the following proposition:

**Proposition 1.10.** For $\kappa > 1$, the transcendental equation $\Gamma = \exp(-\kappa(1 - \Gamma))$ has a unique real solution other than 1, which is (strictly) between 0 and 1. This can be expressed as $\Gamma = -\frac{\mathrm{W}(-\kappa \exp(-\kappa))}{\kappa}$ where $\mathrm{W}$ denotes the principal branch of the Lambert transcendental W function (or product logarithm), namely for $z \geq -\frac{1}{e}$ real, $\mathrm{W}(z)$ is the largest solution of $w \exp(w) = z$.

This number $\Gamma = -\frac{\mathrm{W}(-\kappa \exp(-\kappa))}{\kappa}$ is the extinction probability (and $1 - \Gamma = 1 + \frac{\mathrm{W}(-\kappa \exp(-\kappa))}{\kappa}$ the non-extinction probability) of a Galton-Watson branching process with offspring distribution given by a Poisson distribution with expected value $\kappa$. We will return to this in 4.2.

*Proof.* The function $h$ defined on $\mathbb{R}$ by $h(x) := \exp(-\kappa(1 - x)) - x$ has $h'(x) = \kappa \exp(-\kappa(1 - x)) - 1$, which vanishes exactly once at $x_0 = 1 - \frac{\log \kappa}{\kappa}$, a value between 0 and 1, so $h$ is decreasing up to $x_0$ and increasing thereafter, and since $h(x_0) = \frac{1 - \kappa - \log \kappa}{\kappa} < 0$ whereas $h$ tends to $+\infty$ at $+\infty$ and $-\infty$, it follows that $h$ vanishes exactly twice on $\mathbb{R}$, namely at 1 and at exactly one other value which is $< x_0$.

The equation $\Gamma = \exp(-\kappa(1-\Gamma))$ can be rewritten as $\Gamma = \exp(-\kappa)\ \exp(-\kappa\,\Gamma)$ or again $-\kappa\ \exp(\kappa) = -\kappa\,\Gamma\ \exp(-\kappa\,\Gamma)$. This means that $-\kappa$ and $-\kappa\,\Gamma$ are the two real solutions $w$ (both negative) of $w\ \exp(w) = z$ where $z = -\kappa\ \exp(\kappa)$, namely, two branches of the Lambert W function; the larger one is $-\kappa\,\Gamma$. So $-\kappa\,\Gamma = \mathrm{W}(\kappa\ \exp(\kappa))$, and $\Gamma = -\frac{\mathrm{W}(\kappa\ \exp(\kappa))}{\kappa}$, as claimed. □

**¶1.11.** To summarize, in the classical SIR model ($\mathrm{SIR}(*)$), for a reproduction number $\kappa > 1$, assuming as in 1.6 that the population starts with an infinitesimal proportion of infected:

- During the exponential growth phase of the epidemic, $i$ and $r$ grow with rate (logarithmic derivative) $\beta - \gamma = \beta\,\frac{\kappa-1}{\kappa}$, and the ratio $i/r$ is $\kappa - 1$ (meaning, tends to this value as $t \to -\infty$).
- At peak epidemic, $s, i, r$ are equal to $\frac{1}{\kappa}$, $\frac{\kappa - \log\kappa - 1}{\kappa}$ and $\frac{\log\kappa}{\kappa}$ respectively.
- The proportion $s(+\infty)$ of uninfected at the end of the epidemic equals $\Gamma = -\frac{\mathrm{W}(-\kappa\ \exp(-\kappa))}{\kappa}$, so the attack rate $r(+\infty)$ is given by $1 - \Gamma = 1 + \frac{\mathrm{W}(-\kappa\ \exp(-\kappa))}{\kappa}$. The proportion of infected decays exponentially with growth rate (logarithmic derivative) $\beta\Gamma - \gamma = \beta\,(\Gamma - \kappa)$ as $t \to +\infty$.

**¶1.12.** It is worth investigating what happens for $\kappa$ close to 1 and for $\kappa$ large: for this, we recall that $\mathrm{W}(-\frac{1}{e} + z) = -1 + \sqrt{2e}z^{1/2} - \frac{2e}{3}z + O(z^{3/2})$ for $z \to 0^+$ while $\mathrm{W}(z) = z - z^2 + O(z^3)$ for $z \to 0$. This gives

$$\Gamma = 1 - 2(\kappa-1) + \frac{8}{3}(\kappa-1)^2 + O((\kappa-1)^3) \text{ for } \kappa \to 1^+$$
$$\Gamma = \exp(-\kappa) + \kappa\ \exp(-2\kappa) + O(\kappa^2\ \exp(-3\kappa)) \text{ for } \kappa \to +\infty$$

As for the values of $s, i, r$ at peak epidemic (viz. $\frac{1}{\kappa}$, $\frac{\kappa - \log\kappa - 1}{\kappa}$ and $\frac{\log\kappa}{\kappa}$ respectively), they are given, for $\kappa \to 1^+$, by

$$\begin{cases} s = 1 - (\kappa-1) + (\kappa-1)^2 + O((\kappa-1)^3) \\ i = \frac{1}{2}(\kappa-1)^2 + O((\kappa-1)^3) \\ r = (\kappa-1) - \frac{3}{2}(\kappa-1)^2 + O((\kappa-1)^3) \end{cases}$$

# 2 An SIR variant with constant-time recovery

**¶2.1.** We now wish to replace the hypothesis 1.2 that recovery takes place at constant rate $\gamma$ by the hypothesis that it occurs in constant *time* $T$. In other words, we wish to assume, at the individual level, that an infected individual ($I$) will recover ($I \to R$) exactly $T$ units of time after their infection.

In other words, each individual's recovery time is constant and equal to $T$.

**¶2.2.** Let us call $\mathrm{SIR}(\dagger)$ the model based on the same hypotheses as the classical $\mathrm{SIR}(*)$, but with 2.1 replacing 1.2. To write down the equations for $\mathrm{SIR}(\dagger)$, we note that the rate of contamination term $\beta is$ is unmodified, but the rate of recovery, instead of being $\gamma i$, is now given by the rate of contamination $T$ units of time back in the past.

In other words, if we write $f_T(t) := f(t - T)$ for the translation by $T$ of a function $f$ of time, we must replace $\gamma i$ in $(*)$ by the term $(\beta is)_T = \beta i_T s_T$ representing contaminations $T$ units of time back.

This gives the following equations for $\mathrm{SIR}(\dagger)$:

$$\begin{cases} s' = -\beta is \\ i' = \beta is - \beta i_T s_T \\ r' = \beta i_T s_T \end{cases} \qquad (\dagger)$$
$$s + i + r = 1$$

where $\beta$ and $T$ are positive real parameters (the contagiousness and recovery time).

**¶2.3.** This is now no longer a differential equation but a delay-differential equation because the derivatives of $s, i, r$ at a given time $t$ depend no longer merely on their values at time $t$ but also on their values at time $t - T$.

The question of uniqueness of solutions to such a system is much more problematic to answer (or even state) than for an ODE system such as $(*)$: indeed, there is no clear notion of "initial conditions" at a point, the functions $i$ and $s$ could be initially defined on an interval of length $T$ and then extended forward in time using (†), one interval of length $T$ at a time, at the cost of introducing irregularities at the endpoints of these intervals. Clearly this is not "right", but what is meant by "right" is unclear. Rather, we would want solutions to (†) that are of high regularity (ideally, real-analytic) on all of $\mathbb{R}$, satisfy $s, i, r > 0$, and behave when $t \to -\infty$ in a way that befits an epidemic starting from an infinitesimal seed, viz., $s \to 1$ and $i, r$ growing exponentially. This is what will be described, but it is not clear to the author in what way the solution to be presented below is unique.

**¶2.4.** To describe a closed-form solution to (†), we introduce the following notations:

- $\kappa := \beta T$ is the **reproduction number**, which we assume satisfies $\kappa > 1$,
- $\Gamma := -\frac{\mathrm{W}(-\kappa \exp(-\kappa))}{\kappa}$ is the unique solution other than 1 to the transcendental equation $\Gamma = \exp(-\kappa(1-\Gamma))$, as described in 1.10,
- $X := \exp(\beta\,(1-\Gamma)\,t)$ is an exponential function of time in which the solutions of (†) will be expressed (note that subtracting $T$ from $t$ amounts to multiplying $X$ by $\Gamma$, which motivates this particular change of variables).

The proposed solution is then given by:

$$\begin{cases} s = \dfrac{(1-\Gamma)^2 + \Gamma c X}{(1-\Gamma)^2 + cX} \\ i = \dfrac{(1-\Gamma)^4 cX}{((1-\Gamma)^2 + cX)((1-\Gamma)^2 + \Gamma cX)} \\ r = \dfrac{\Gamma(1-\Gamma)cX}{(1-\Gamma)^2 + \Gamma cX} \end{cases}$$

where $c$ is an arbitrary positive real parameter which merely serves to translate the solution.

We can check that:

- $s, i, r$ are indeed solution to (†). To this effect, note that differentiating with respect to $t$ is the same as differentiating with respect to $X$ followed by multiplying by $\beta\,(1-\Gamma)X$, and remember that subtracting $T$ from $t$ amounts to multiplying $X$ by $\Gamma$. It is then a simple computation matter to check the solutions.
- $s, i, r$ are all well-defined and positive for all $t \in \mathbb{R}$; and we have $s \to 1$ as $t \to -\infty$ (i.e. $X \to 0$). That is, we have the analogue of 1.6. Indeed, each of the subexpressions $(1-\Gamma)$, $\Gamma$ and $cX$ are positive, and the limit of $s$ as $X \to 0$ is easily checked as the ratio of the lowest-order terms in $X$ in the numerator and denominator.

Note that if we wish to find more general solutions, we can, as explained in 1.6 multiply both $s$ and $i$ by a same constant $a$ and $\beta$ by $a^{-1}$ (and replace $r$ by $(1-a) + ar$). But we will be content with studying solutions such that $s \to 1$ as $t \to -\infty$.

**¶2.5.** Just as in the case of $(*)$ we could express $s$ in function of $r$ (and independently of time) as $\exp(-\kappa r)$ (and consequently $i$ as $1 - r - \exp(-\kappa r)$), we can also find such an expression in our solution of (†) by solving $cX = \frac{(1-\Gamma)^2 r}{\Gamma(1-\Gamma-r)}$ and substituting it in the other values, giving

$$s = \frac{\Gamma}{\Gamma + r} \quad \text{and} \quad i = \frac{r(1-\Gamma-r)}{\Gamma + r}$$

**¶2.6.** We now investigate the behavior of our solutions to (†) at three points: when $t \to -\infty$, at peak epidemic (when $i$ is maximal), and when $t \to +\infty$ (compare with 1.9 and 1.11).

- When $t \to -\infty$, i.e., $X \to 0$, we have $s \to 1$, as has already been pointed out. Using the lowest-order terms in $X$ in the expressions of $i$ and $r$, we find $i \sim c\,\exp(\beta\,(1-\Gamma)\,t)$ and $r \sim c\,\frac{\Gamma}{1-\Gamma}\,\exp(\beta\,(1-\Gamma)\,t)$. Thus, both grow exponentially with rate (logarithmic derivative) $\beta\,(1-\Gamma)$ and the ratio $i/r$ is $\frac{1-\Gamma}{\Gamma}$ (meaning, tends to this value as $t \to -\infty$).
- The unique point where $i' = 0$ (peak epidemic) is characterized by $cX = \frac{(1-\Gamma)^2}{\sqrt{\Gamma}}$. This gives $s = \sqrt{\Gamma}$ and $i = (1-\sqrt{\Gamma})^2$ and $r = \sqrt{\Gamma}\,(1-\sqrt{\Gamma})$.
- Finally, when $t \to +\infty$, we have $s \to \Gamma$ and $r \to 1-\Gamma$ exactly as in $(*)$. The proportion of infected decays exponentially with growth date (logarithmic derivative) $\beta(\Gamma-1)$.

**¶2.7.** One notable difference between our solutions to $(\dagger)$ and those of $(*)$ is that in $(\dagger)$, the function $i$ is symmetric in time around the epidemic peak: indeed, replacing $cX$ by $\frac{(1-\Gamma)^4}{\Gamma\,cX}$ (which amounts to replacing $t$ by $2t_{\max} - t$ where $t_{\max}$ is the time of epidemic peak) leaves the expression for $i$ invariant.

**¶2.8.** As for $(*)$, we can wish to investigate what happens when $\kappa$ is close to 1 and for $\kappa$ large. The expressions given in 1.12 for $\Gamma$ are, of course, still valid. As for the epidemic peak, using the expressions $s = \sqrt{\Gamma}$ and $i = (1-\sqrt{\Gamma})^2$ and $r = \sqrt{\Gamma}\,(1-\sqrt{\Gamma})$, we get, for $\kappa \to 1^+$:

$$\begin{cases} s = 1 - (\kappa-1) + \dfrac{5}{6}(\kappa-1)^2 + O((\kappa-1)^3) \\ i = (\kappa-1)^2 + O((\kappa-1)^3) \\ r = (\kappa-1) - \dfrac{11}{6}(\kappa-1)^2 + O((\kappa-1)^3) \end{cases}$$

and for $\kappa \to +\infty$:

$$\begin{cases} s = \exp(-\frac{1}{2}\kappa) + O(\kappa\,\exp(-\frac{3}{2}\kappa)) \\ i = 1 - 2\exp(-\frac{1}{2}\kappa) + \exp(-\kappa) + O(\kappa\,\exp(-\frac{3}{2}\kappa)) \\ r = \exp(-\frac{1}{2}\kappa) - \exp(-\kappa) + O(\kappa\,\exp(-\frac{3}{2}\kappa)) \end{cases}$$

# 3 Qualitative comparison of both variants

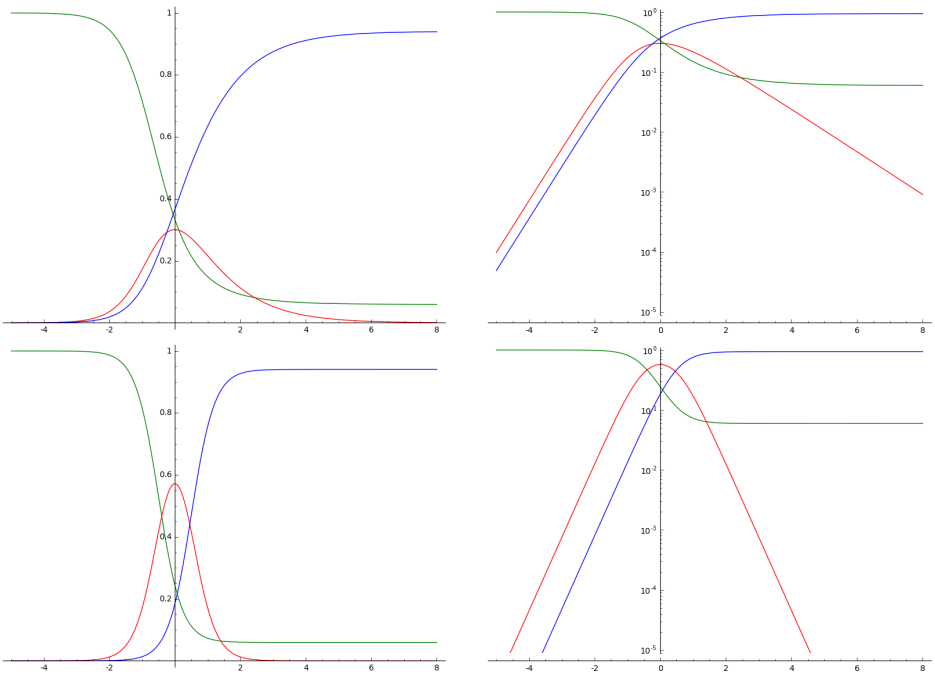
**¶3.1.** Comparing the solutions of $\mathrm{SIR}(*)$ (classical, i.e., with exponential-process recovery) and $\mathrm{SIR}(\dagger)$ (constant-time recovery), for the same values of $\kappa$ and $\beta$, we can say the following:

- initial exponential growth takes place faster in $\mathrm{SIR}(\dagger)$ (growth rate $\beta(1-\Gamma)$) than in $\mathrm{SIR}(*)$ (growth rate $\beta\,\frac{\kappa-1}{\kappa}$),
- during this initial exponential growth phase, the $i/r$ ratio is higher in $\mathrm{SIR}(\dagger)$ (namely $\frac{1-\Gamma}{\Gamma}$) than in $\mathrm{SIR}(*)$ (namely $\kappa-1$),
- the peak value of $i$ is higher in $\mathrm{SIR}(\dagger)$ (namely $(1-\sqrt{\Gamma})^2$) than in $\mathrm{SIR}(*)$ (namely $\frac{\kappa-\log\kappa-1}{\kappa}$),
- the peak is narrower in $\mathrm{SIR}(\dagger)$ than in $\mathrm{SIR}(*)$, and it is symmetric in time,
- the final attack rate, however, is the same in $\mathrm{SIR}(\dagger)$ as in $\mathrm{SIR}(*)$.

**¶3.2.** The following graphs plot values of $s, i, r$, both in linear scale and in log scale, for both models:

SIR$(*)$

SIR$(\dagger)$

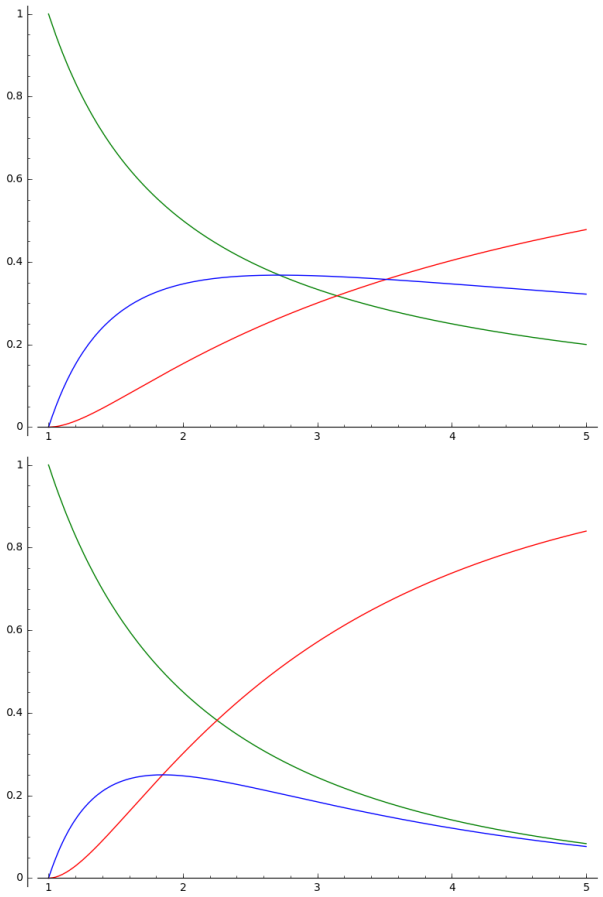


Here $s, i, r$ have been plotted in green, red and blue respectively. Abscissa is time $t$. We have used $\beta = 3$ and $\kappa = 3$, and used the time of peak epidemic as $t = 0$. For this $\kappa$ we have $\Gamma \approx 0.034$ (a final attack rate of $96.6\%$): initial exponential growth rate of $i$ and $r$ is $2.000$ and $2.821$ in SIR$(*)$ and SIR$(\dagger)$ respectively, the $i/r$ ratio during this phase is $2.0$ and $15.8$ in SIR$(*)$ and SIR$(\dagger)$ respectively; the growth rate of $i$ during its final exponential decay is $-0.821$ and $-2.821$ in SIR$(*)$ and SIR$(\dagger)$ respectively.

**¶3.3.** The following graphs plot the values at of $s, i, r$ at peak epidemic in function of the reproduction number $\kappa$ (abscissa):

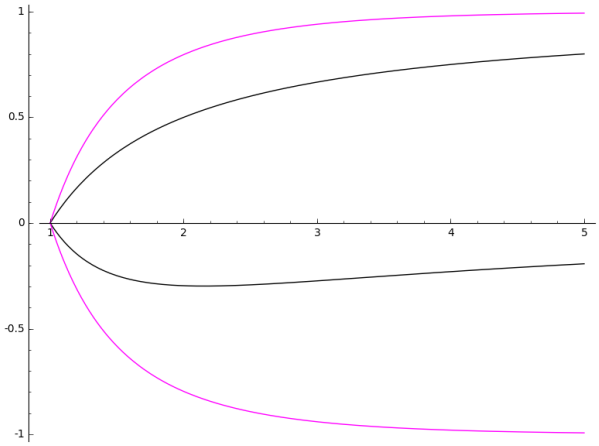
SIR$(*)$

SIR$(\dagger)$



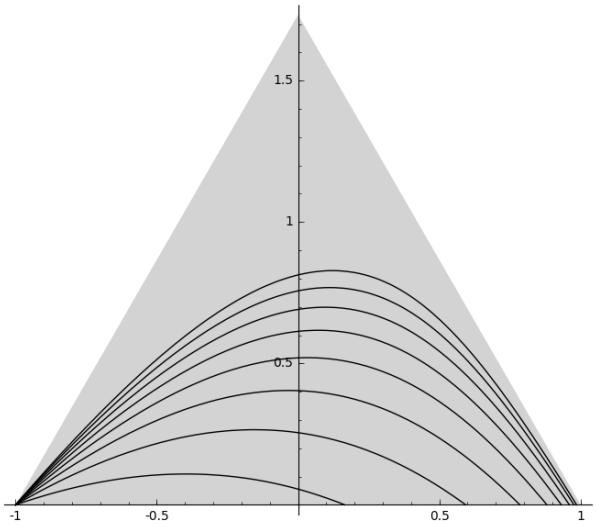
Again $s, i, r$ have been plotted in green, red and blue respectively.

**¶3.4.** The following graph illustrates the growth rate of $i$ during its exponential growth phase (positive part) and exponential decay (negative part) for $\mathrm{SIR}(*)$ in black and $\mathrm{SIR}(\dagger)$ in magenta: this is for $\beta = 1$ and $\kappa$ (which is then also the expected recovery time) in abscissa:

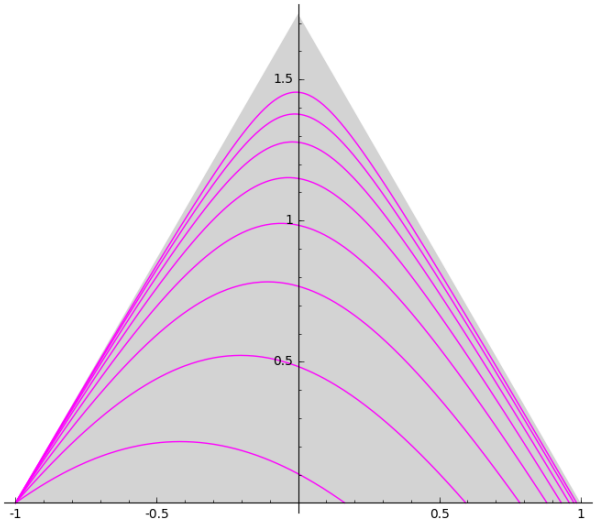



**¶3.5.** Finally, the following plots illustrate the trajectories of the solutions to $\mathrm{SIR}(*)$ (black, top) and $\mathrm{SIR}(\dagger)$ (magenta, bottom) in a triangle plot where the lower-left vertex corresponds to $(s, i, r) = (1, 0, 0)$, the top vertex to $(0, 1, 0)$ and the lower-right vertex to $(0, 0, 1)$ (thus, the state point starts from the lower-left vertex, evolves following a curve from left to right until it reaches the bottom edge at the limit when $t \to +\infty$; abscissa is $r - s$ and ordinate is $\sqrt{3}(1 - r - s)$). Eight different such trajectories have been plotted in each case, corresponding to equally spaced values of $\kappa$ from $1.5$ to $5.0$ inclusive by increments of $0.5$.

$\mathrm{SIR}(*)$




$\mathrm{SIR}(\dagger)$



# 4 Further remarks

**¶4.1.** Let us comment on the value of the reproduction number $\kappa$, defined in both $\mathrm{SIR}(*)$ and $\mathrm{SIR}(\dagger)$ as the product of the contagiousness parameter $\beta$ by the *expected* recovery (i.e., contagiousness) time $T_{\exp}$. Indeed, in an entirely susceptible population, an infected individual will infect $\beta\,dt$ more individuals during each infinitesimal time interval of length $dt$, meaning that they infect $\beta T_{\exp}$ others from their time of first infection (so long as $s$ remains close to 1): this does not depend on assumptions on the distribution of the recovery time.

However, it is important to note that whereas the number $N$ of people each infected individual infects in turn (in an entirely susceptible population) has the same expected value $\kappa$ in both $\mathrm{SIR}(*)$ and $\mathrm{SIR}(\dagger)$, the distribution is different: in $\mathrm{SIR}(*)$, like the contagiousness time itself, this follows an exponential distribution

$$\mathbb{P}(N=j) = \frac{\kappa^j}{(\kappa+1)^{j+1}}$$
$$\mathbb{E}(x^N) = \frac{1}{1-\kappa(x-1)}$$

whereas in $\mathrm{SIR}(\dagger)$ it follows a Poisson distribution

$$\mathbb{P}(N=j) = \frac{\kappa^j}{j!}\,\exp(-\kappa)$$
$$\mathbb{E}(x^N) = \exp(\kappa(x-1))$$

(The latter follows from the definition of a Poisson distribution as the count of events of a point Poisson process, while the former can be deduced from the general fact that if recovery time follows distribution with density $f(T)\,dT$ then the distribution of $N$ will be the average of the corresponding Poisson distributions, i.e., $\mathbb{P}(N=j) = \frac{1}{j!}\int_0^{+\infty}(\beta T)^j\,\exp(-\beta T)\,f(T)\,dT$, and apply this with $f(T) = \gamma\,\exp(\gamma T)$.)

**¶4.2.** Let us now comment on the reason why the final attack rate is the same, $1-\Gamma$, in the $\mathrm{SIR}(*)$ and $\mathrm{SIR}(\dagger)$ models, even though, as we have just explained, the number of individuals infected by each infected individual (in an otherwise entirely susceptible population) follow different distributions in both cases.

This can seem surprising since the *(rapid) extinction* probability of the epidemic, starting from a single infected individual in an otherwise entirely susceptible population, is not the same in the stochastic versions of $\mathrm{SIR}(*)$ and $\mathrm{SIR}(\dagger)$: the latter is still $\Gamma = -\frac{\mathrm{W}(-\kappa\,\exp(-\kappa))}{\kappa}$, but the former is simply $\frac{1}{\kappa}$. Indeed, let us briefly recall what a branching Galton-Watson process is: given a probability distribution on $\mathbb{N}$ (specified by $p_j := \mathbb{P}(N=j)$ for $j \in \mathbb{N}$ nonnegative and summing to 1), build a random tree by starting with the root node and giving each node $N$ descendants where $N$ is drawn, independently for each node, according to the specified distribution; we say that the process becomes extinct when the tree in question is finite: the probability $q$ that this occurs is the smallest nonnegative solution of the equation $q = \varphi(q)$ where $\varphi(x) := \sum_{j=0}^{+\infty} p_j x^j = \mathbb{E}(x^N)$ is the generating function of the specified distribution. (Informal explanation: the probability $q$ that the process becomes extinct starting from a single node is equal to the probability that it becomes extinct starting from $N$ nodes with $N$ being equal to $j$ with probability $p_j$, so $q^j$ in this case: this gives $q = \sum_{j=0}^{+\infty} p_j q^j$ as announced.) Now the stochastic versions of $\mathrm{SIR}(*)$ and $\mathrm{SIR}(\dagger)$ behave like a Galton-Watson process as long as the population is almost entirely susceptible; using $\varphi(x) = \frac{1}{1-\kappa(x-1)}$ for $\mathrm{SIR}(*)$ and $\varphi(x) = \exp(\kappa(x-1))$ for $\mathrm{SIR}(\dagger)$ as we just described, we find extinction probabilities of $\frac{1}{\kappa}$ and $\Gamma$ respectively.

So why is the attack rate still the same in both cases and why does it coincide with the non-extinction probability of a Galton-Watson process with Poisson distribution in both cases?

To understand this better, let us offer the following informal explanation: we consider a random directed graph constructed on a (huge) number of vertices and where, for each vertex $x$, we choose a random number $N$ of edges that will lead away from it (following a specified distribution $p_j := \mathbb{P}(N=j)$ as above, with expected value $\kappa := \sum_{j=0}^{+\infty} p_j j = \varphi'(1)$ finite and $> 1$), and then choose $N$ vertices $y$ uniformly at random and a directed edge from $x$ to $y$ (all such random choices are independent). Thus, the out-degree of vertices follows the prescribed distribution $(p_j)$. This represents the graph of possible contaminations during the

epidemic. The probability that a given starting vertex $x$ has a large set of reachable vertices (*from* $x$) is the non-extinction probability of the Galton-Watson process for this distribution. However, the attack rate is modeled by the *backward* process, namely, what is fraction of vertices $x$ which are reachable *from* a large number of vertices: this is the non-extinction probability of the Galton-Watson process for the distribution of *in*-degrees of our graph; but, no matter the distribution of out-degrees, the distribution of in-degrees will be Poisson (with the same expected value $\kappa$) because each node $x$ has the same small probability of having a vertex to $y$ and we recover a Poisson process as the sum of a large number of Bernoulli processes.

The informal reasoning which we just sketched is made rigorous in [Penrose 2016]: specifically, see theorem 4 (where $\mu_\infty$ is what we called $\kappa$ and $\sigma'(\mu_\infty)$ is what we called $1 - \Gamma$, while $\sigma(F)$ is the non-extinction probability for the forward process) and the following discussion.

This discussion also helps explain why $\mathrm{SIR}(\dagger)$ is more symmetric than $\mathrm{SIR}(*)$: it has the property that the distribution of the number of people who (would have) contaminated a given individual, i.e., the distribution of in-degrees, is the same Poisson distribution as that of the number of people who (would) have been contaminated by a given individual, i.e., the distribution of out-degrees, whereas in the case of $\mathrm{SIR}(*)$ we have a Poisson distribution versus an exponential distribution.

**¶4.3.** Let us sketch a different informal explanation (or a different presentation of the same reason) why the attack rate is the same in the $\mathrm{SIR}(*)$ and $\mathrm{SIR}(\dagger)$ models. Again it hinges on the fact that if we take a huge number of nodes and construct a contamination digraph by adding a directed edge from each given node to a random number $N$ of randomly chosen other nodes, then the number of nodes eventually reached from a starting node, if it does not become rapidly extinct, does not depend on the distribution of $N$ but merely on its expected value $\kappa := \mathbb{E}(N)$. To realize this, consider a breadth-first search algorithm to go through all the reachable nodes: this algorithm uses a FIFO queue to store the nodes to be processed and, whenever it removes a node from the FIFO, adds all its out-neighbors to it, until the FIFO is empty; now if, at a given point during the execution of the algorithm, we call $r$ the fraction of all nodes hat have been marked as visited, $i$ the fraction held in the FIFO, and $s$ the remaining fraction, every time we move $dr$ (a very small fraction of the total, but still very large in absolute number) from the FIFO ($i$) to the processed nodes ($r$), by the law of large numbers, we consider a fraction $\kappa\, dr$ as out-neighbors, of which $\kappa\, s\, dr$ are new and added to the FIFO, so $\frac{di}{dr} = -1 + \kappa\, s$ while $\frac{ds}{dr} = -\kappa\, s$. These are precisely the equations defining $\mathrm{SIR}(*)$ implicitly in terms of $r$, so we get the same attack rate as for it. In other words, while the models schedule the infections in a different way in time, a breadth-first search of the contamination graph will follow the equations of the $\mathrm{SIR}(*)$ model and only depends on the expected value $\kappa$ of the number $N$ of individuals contaminated by a given individual (in an otherwise susceptible population).

# References

[Penrose 2016] Mathew D. Penrose, "The strong giant in a random digraph", *J. Appl. Probab.*, **53** (2016), 57–70, `https://doi.org/10.1017/jpr.2015.8` or `https://arxiv.org/abs/1409.4371` (preprint).