

# Sideward contact tracing in an epidemic model with mixing groups

Dongni Zhang<sup>1</sup> and Martina Favero<sup>1</sup>

<sup>1</sup>Department of Mathematics, Stockholm University, 106 91 Stockholm, Sweden.

July 17, 2024

## Abstract

We consider a stochastic epidemic model with sideward contact tracing. We assume that infection is driven by interactions within mixing events (gatherings of two or more individuals). Once an infective is diagnosed, each individual who was infected at the same event as the diagnosed individual is contact traced with some given probability. Assuming few initial infectives in a large population, the early phase of the epidemic is approximated by a branching process with sibling dependencies. To address the challenges given by the dependencies, we consider sibling groups (individuals who become infected at the same event) as macro-individuals and define a macro-branching process. This allows us to derive an expression for the effective macro-reproduction number which corresponds to the effective individual reproduction number and represents a threshold for the behaviour of the epidemic. Through numerical illustrations, we show how the reproduction number varies with the mean size of mixing events, the rate of diagnosis and the tracing probability.

## 1 Introduction

Contact tracing is recognized as a crucial prevention for pandemic control. Traditional contact tracing involves *backward* tracing to identify who may have infected the index case and *forward* tracing to find out who the index case may have infected (individuals who are diagnosed are called index cases in the contact tracing). This paper concerns an innovative tracing method: “*sideward*” *contact tracing*. This method is particularly relevant in large gatherings where “superspreading events” (many people get infected at once [9]) usually occur. By detecting the gathering, sideward contact tracing can identify individuals infected at the same event as the index case.

The effect of such type of contact tracing has been analyzed, within the framework of simplicial temporal networks, in [10], where also the traditional forward and backward tracing are considered. In this paper, we focus on the concept of sideward tracing, and we consider the stochastic epidemic model with mixing groups introduced in [1]. In most epidemic models, infections are assumed to occur between pairs of individuals (one is infectious, and

the other is susceptible). Instead of having pairwise interactions, in this model, individuals make contacts via attending *mixing events* with at least two individuals involved. This allows for the possibility that an infective can infect more than one susceptible on a single occasion and thus provides a suitable framework for the study of sideward tracing.

The mathematical modelling of contact tracing presents several challenges (see an overview in [11]). This paper focuses on analyzing the initial behaviour of the epidemic with sideward tracing via a continuous-time branching process incorporating *sibling dependencies*. Generally speaking, the dependencies introduced by contact tracing complicates the analysis of related branching processes. For instance, in previous studies of models with forward and/or backward contact tracing, lifetimes of siblings are co-dependent on their parents in [3], lifetimes of siblings and parents depend on each other in [14]. In this paper, the challenge arises from the lifetimes of individuals who were infected in the same mixing event being dependent on each other due to sideward contact tracing.

To address this challenge, we consider the groups of siblings, specifically those who were born (infected) in the same birth (mixing) event, as *macro-individuals*, such macro-individual behaving independently of each other. The main idea, introduced by [4, 12], is to consider these macro-individuals as a branching process, referred to as the “macro process”, embedded into the sibling-dependent process. The key point is that macro-individuals reproduce independently, unlike single individuals, as all the dependencies are within the sibling groups. However, our definition of sibling groups differs: the sibling group in [12] consists of all the children with the same parent, whereas our sibling group comprises children born during the same birth event, so an individual can give birth to multiple sibling groups.

We study the macro process, which is a branching process with birth rates related to sizes of sibling groups, and we apply the standard branching process theory to derive a macro-reproduction number which represents the epidemic threshold and corresponds to the individual reproduction number.

The paper is structured as follows. In Section 2, the epidemic model with mixing groups is defined, as in [1], and a sideward contact tracing mechanism is introduced in the epidemic model. The approximation of the early stage of the epidemic, with and without contact tracing, is presented in Section 3. In Section 4, the limiting process for the early epidemic with contact tracing is described as a macro branching process, which allows the derivation of macro and individual reproduction numbers in Section 5. A numerical illustration of the effect of sideward tracing on the reproduction number is provided in Section 6. Finally, Section 7 presents the conclusion and discussion.

## 2 Model

### 2.1 The epidemic model with mixing groups

We describe the SIR epidemic model recently introduced and analysed in [1, 2, 5]. Initially, there are  $m_n$  number of infectives in an otherwise susceptible population of fixed size  $n$ . Differently from the standard homogeneous SIR epidemic where infections occur through pairwise interactions, infectious contacts in this model are made during temporary gatherings, referred to as “mixing events”, which occur at rate  $n\beta$ . Each mixing event involves a certain number, distributed as  $C^{(n)}$  and independent of other mixing events, of individuals chosen uniformly at random from the population. The mean size of mixing events is de-

noted by  $\mu_C^{(n)} = \mathbb{E} [C^{(n)}]$ . Naturally, it is assumed that a mixing event involves at least two individuals and at most  $n$  individuals.

Given that a mixing event is of size  $c$ , i.e. it involves  $c$  individuals, any given individual in the population attends the event with probability  $c/n$  and any given infectious individual attending the event has probability  $\pi_c$  of making an infectious contact with any given susceptible individual attending the event, where all such contacts occur independently. Any susceptible individual who is contacted by at least one infective during the event becomes infected and remains infectious for a random period of time  $T_I \sim \text{Exp}(\gamma)$  (with mean  $\mathbb{E} [T_I] = 1/\gamma$ ). Further, it is assumed that newly infected individuals cannot transmit the disease within the same event where they were infected, i.e. mixing events are treated as instantaneous, and that there is no latency period. All the processes and random variables described above are assumed to be mutually independent.

Note that it is possible to have zero or multiple infections in one single mixing event, and that, when all mixing events involve exactly two individuals, i.e.  $C^{(n)} \equiv 2$ , the above model aligns with standard homogeneously mixing SIR epidemic model with individual-to-individual rate of infection  $2\beta\pi_2/(n-1)$  and recovery rate  $\gamma$ .

## 2.2 Sideward contact tracing

This subsection aims to incorporate sideward contact tracing into the epidemic model described above. To this aim, we introduce a diagnosis rate  $\delta$ , which activates a contact tracing procedure. More precisely, infectives can be removed in three ways: due to natural recovery at rate  $\gamma$ ; due to diagnosis (excluding contact tracing) at rate  $\delta$ , or through a contact tracing mechanism described in the following. Excluding natural recovery and contact tracing, we use the term “diagnosed” in a broad sense to include various causes of removal that can trigger contact tracing. For example, an individual can be diagnosed due to the onset of symptoms or mass testing.

We assume that when an infectious individual is diagnosed, they are removed (isolated) and the mixing event where they were infected is immediately detected, triggering the sideward contact tracing procedure. Each of the individuals who were infected at the detected event is immediately traced, independently with probability  $p$ , and they are tested; if infected, they are removed to stop spreading the infection. Table 2.1 lists the important model parameters.

The sideward contact tracing procedure described above only focuses on those infected at the same mixing event as the index case (the one whose diagnosis triggers the tracing), whereas it neglects those who were already infectious at the mixing event. Thus, neither the infector of the index case nor the individuals infected by the index case (infectees) can be traced by this procedure. Moreover, we assume no delays are associated with the tracing process.

Note that the epidemic model with diagnosis rate  $\delta$ , but without contact tracing, i.e.  $p = 0$ , corresponds to the epidemic model defined in Section 2.1, where the infectious period follows  $T_I \sim \text{Exp}(\gamma + \delta)$ . In addition, if all the mixing events involve exactly two individuals, sideward tracing has no effect, since no one will be contact traced.

**Table 2.1** Key quantities related to the epidemic model

Parameter	Description
$n$	population size
$n\beta$	rate of mixing events
$\gamma$	rate of natural recovery
$\delta$	rate of diagnosis
$p$	probability of being contact traced in a detected event
$\pi_c$	infection probability within a mixing event of size $c$
$C^{(n)}, \mu_C^{(n)}$	size of a mixing event and its expected value

### 3 Early epidemic approximation

In this section, first, we explain heuristically how the early stages of the epidemic involving mixing groups can be approximated by a branching process; see [2] for a more detailed explanation and rigorous proof. Furthermore, we describe how sideward contact tracing modifies the branching process by modifying the lifespan of individuals and by introducing dependencies between siblings who are born in the same birth event (corresponding to individuals who are infected during the same mixing event). The dependencies lead to challenges in the analysis of the reproduction number, which are highlighted here and addressed in the next section.

We consider the early phase of an epidemic with mixing groups in a large population of size  $n$  with a few initial infectives, i.e.  $m_n = m$  for all sufficiently large  $n$ . We make asymptotic assumptions that the events size  $C^{(n)}$  converges in distribution to  $C$ , as  $n \rightarrow \infty$ , where  $C$  has probability distribution  $p_C(c) := \mathbb{P}(C = c)$ ,  $c = 2, 3, \dots$ , with finite mean  $\mu_C$  satisfying  $\mu_C^{(n)} \rightarrow \mu_C$ , as  $n \rightarrow \infty$ .

Since mixing events are formed by choosing individuals uniformly at random from the large population and we focus on the beginning of an epidemic, with a probability close to 1, each mixing event that involves at least one infective consists of only one infective and all others being susceptible. Further, note that an event of size  $c$  includes a given typical infective with probability  $c/n$  and recall that mixing events occur at rate  $n\beta$ . Consequently, mixing events involving one typical infective occur at rate

$$\sum_{c=2}^{\infty} n\beta \frac{c}{n} \mathbb{P}(C^{(n)} = c) = \beta \mu_C^{(n)} \rightarrow \beta \mu_C, \quad \text{as } n \rightarrow \infty.$$

In addition, the size of a mixing group involving a typical infective is the size-biased version of  $C^{(n)}$ , which converges to the size-biased version of  $C$ , denoted by  $\tilde{C}$ , with probability distribution

$$p_{\tilde{C}}(c) := \mathbb{P}(\tilde{C} = c) = \frac{cp_C(c)}{\mu_C}, \quad (c = 2, 3, \dots).$$

Under the above asymptotic assumptions and additional integrability conditions, Theorem 3.1 in [2] proves that, as the population size  $n \rightarrow \infty$ , the number of infectives in the early stages of the epidemic with mixing groups, without considering sideward contact tracing, is approximated by a branching process  $\mathcal{B}$  which we describe in the following.

There are  $m$  ancestors in the branching process  $\mathcal{B}$ . Alive individuals in  $\mathcal{B}$  correspond to infectious individuals and a *birth event* corresponds to a mixing event containing one single infective in an otherwise susceptible group in the epidemic. Once born, an individual has lifetime distribution  $T_I \sim \text{Exp}(\gamma)$ , during which they give birth at rate  $\beta\mu_C$ . It follows that the number of birth events produced by one typical individual during their lifetime is geometrically distributed as  $G$ , with

$$\mathbb{P}(G = k) = \left( \frac{\beta\mu_C}{\beta\mu_C + \gamma} \right)^k \frac{\gamma}{\beta\mu_C + \gamma}, \quad (k = 0, 1, \dots) \quad (1)$$

which has mean

$$\mathbb{E}[G] = \frac{\beta\mu_C}{\gamma}. \quad (2)$$

Denote by  $\tilde{Z}_i, i = 1, \dots, G$ , the number of offspring produced at  $i$ -th birth event, which are i.i.d. random variables, independent of  $G$  and equal in distribution to  $\tilde{Z}$ , described in the following. Given that the size of a mixing group is equal to  $c$  which happens with probability  $p_{\tilde{C}}(c)$ , there are  $c - 1$  susceptibles in the group, each infected with probability  $\pi_c$  independently. Thus, the number of individuals infected at an event of size  $c$ , the number of offspring in a birth event of size  $c$ , follows a binomial distribution, that is,

$$\tilde{Z} | \tilde{C} = c \sim \text{Bin}(c - 1, \pi_c).$$

Note that

$$\mathbb{E}[\tilde{Z}] = \mathbb{E}\left[\mathbb{E}[\tilde{Z} | \tilde{C}]\right] = \mathbb{E}[(\tilde{C} - 1)\pi_{\tilde{C}}] = \sum_{c=2}^{\infty} (c - 1)\pi_c p_{\tilde{C}}(c). \quad (3)$$

Finally, the total number of offspring produced by one typical individual during their lifetime is given by

$$\sum_{i=1}^G \tilde{Z}_i,$$

and the *basic reproduction number* in the epidemic with mixing groups, without contact tracing, corresponds to the mean number of offspring in the limiting branching process, i.e.

$$R_0 = \mathbb{E}[G] \mathbb{E}[\tilde{Z}] = \frac{\beta\mu_C}{\gamma} \mathbb{E}[(\tilde{C} - 1)\pi_{\tilde{C}}] = \frac{\beta}{\gamma} \sum_{c=2}^{\infty} c(c - 1)\pi_c p_C(c), \quad (4)$$

using Equation (2) and (3). A major outbreak in the epidemic is associated to the non-extinction of the approximating branching process  $\mathcal{B}$  and occurs with non-zero probability if and only if  $R_0 > 1$  [2].

If we consider the epidemic with diagnosis rate  $\delta$  but without contact tracing, the branching process remains the same except for the lifetime of individuals (corresponding to the infectious period)  $T_I \sim \text{Exp}(\gamma + \delta)$ . Thus, in this case  $R_0$  is given by

$$R_0 = \frac{\beta\mu_C}{\gamma + \delta} \mathbb{E}[(\tilde{C} - 1)\pi_{\tilde{C}}] = \frac{\beta\mu_C}{\gamma + \delta} \mathbb{E}[C(C - 1)\pi_C]. \quad (5)$$

When sideward tracing is introduced, individuals who were infected during the same mixing event (*siblings*) depend on each other since their infectious periods can be shortened

if one of the others is diagnosed. Consequently, as  $n \rightarrow \infty$ , the initial phase of the epidemic with sideward tracing is approximated by a different process  $\mathcal{B}_{CT}$ , which corresponds to the branching process  $\mathcal{B}$  described above with modified lifespan distributions and *sibling dependencies*. The process  $\mathcal{B}_{CT}$  is described as follows. As in  $\mathcal{B}$ , each alive individual in  $\mathcal{B}_{CT}$  gives birth events at rate  $\beta\mu_C$ , a birth event in  $\mathcal{B}_{CT}$  corresponds to a mixing event consisting of one infective in an otherwise susceptible group in the epidemic, and the number of offspring produced at each birth event are i.i.d. as  $\tilde{Z} \sim \text{MixBin}(\tilde{C} - 1, \pi_{\tilde{C}})$ . However, lifespan distributions in  $\mathcal{B}_{CT}$  differ from those in  $\mathcal{B}$ .

Consider one typical individual in  $\mathcal{B}_{CT}$  and their siblings, the individuals who were born in the same birth event. Note that individuals who were born from the same parent in other birth events are *not* called siblings here. The individual dies due to either of the following events happening: natural death (natural recovery in the epidemic) at rate  $\gamma$ ; removal by diagnosis at rate  $\delta$ ; or removal by contact tracing (one of their siblings is removed due to diagnosis and tracing is successful) which happens at rate  $(i - 1)\delta p$ , given that  $(i - 1)$  of their siblings are currently alive.

The number  $G_{CT}$  of birth events produced by the typical individual in  $\mathcal{B}_{CT}$  has a distribution which is not straightforward to compute, unlike the above  $G$  without contact tracing. Consequently, when considering sideward contact tracing, the reproduction number becomes challenging to compute, despite the simple expression

$$R_e^{(ind)} = \mathbb{E} \left[ \sum_{j=1}^{G_{CT}} \tilde{Z}_j \right] = \mathbb{E} [G_{CT}] \mathbb{E} [\tilde{Z}]. \quad (6)$$

Table 3.1 lists the important quantities related to the process  $\mathcal{B}$  and  $\mathcal{B}_{CT}$ .

Furthermore, because of dependencies between individuals, it is not obvious a priori, whether the reproduction number above represents a threshold for the behaviour of  $\mathcal{B}_{CT}$  and of the epidemic (we will show in Section 5.2 that it actually does). To address these challenges and to analyse the threshold behaviour of the epidemic, in the next section, we construct a macro branching process, embedded in  $\mathcal{B}_{CT}$ , by considering sibling groups as macro-individuals, which are independent.

**Table 3.1** Key quantities related to the limiting branching processes (large population)

Notation	Description
$\mathcal{B}$	approximating branching process for epidemic without contact tracing
$C, \mu_C$	limiting size of a mixing event and its expected value
$\tilde{C}$	size of a birth event produced by a typical individual
$\beta\mu_C$	rate of birth events
$\tilde{Z}$	number of offspring in a typical birth event
$G$	number of birth events by a typical individual in $\mathcal{B}$
$\mathcal{B}_{CT}$	approximating branching process with sibling dependencies for epidemic with contact tracing
$G_{CT}$	number of birth events by a typical individual in $\mathcal{B}_{CT}$

## 4 The macro branching process

In the limiting process  $\mathcal{B}_{CT}$ , offspring born at the same birth event constitute a *sibling group*. While dependency is within each sibling group, sibling groups are independent of their parents and, since each group is produced at independent birth events, sibling groups are independent of each other. By identifying sibling groups as “macro-individual”, we define the *macro process*  $\mathcal{M}$  which is a branching process in terms of independent (macro) individuals.

A sibling group is born when a birth event occurs, and the number of offspring produced at the event is then the initial size of the sibling group, distributed as

$$Y_0 \stackrel{d}{=} \tilde{Z} \sim \text{MixBin}(\tilde{C} - 1, \pi_{\tilde{C}}). \quad (7)$$

Note that a sibling group can also have size zero, in that case, it certainly produces no offspring. In addition, each of the  $m$  ancestors in  $\mathcal{B}_{CT}$  produces independently a number of birth events distributed as  $G$ , since the ancestors will not be contact traced. It could happen that the  $m$  ancestors produce no birth events, in this case the process  $\mathcal{B}_{CT}$  dies out just after all the ancestors die and  $\mathcal{M}$  is not needed. Turning to the more interesting case where the  $m$  ancestors in  $\mathcal{B}_{CT}$  give  $g > 0$  number of birth events, the macro process  $\mathcal{M}$  is thus initiated with a number  $g$  of macro-individuals. Without loss of generality, in the following we assume that the macro process  $\mathcal{M}$  starts with one macro-individual, and we focus on the initial conditions in Section 5.3, where we study the extinction probability.

Let  $Y(t)$  be the size of a sibling group (number of alive siblings in the group) at time  $t$  after the birth event, then the process  $\{Y(t)\}_{t \geq 0}$  is a *continuous-time Markov jump process* on the state space  $\mathbb{N}$ , with initial distribution  $Y(0) \stackrel{d}{=} Y_0$  and absorbing state 0. From a non-zero state  $i$ , two *types* of jumps can occur. One type is that the group size is decreased by 1 ( $i \rightarrow i - 1$ ), corresponding to one of the  $i$  siblings recovering naturally, or one of the  $i$  siblings being diagnosed and all the other  $i - 1$  siblings “escaping” from being traced. Another type of jump is that the group size is decreased by more than 1 ( $i \rightarrow i - j, j = 2, \dots, i$ ) corresponding to one of the  $i$  siblings being diagnosed and  $j - 1$  siblings being traced, as a consequence. The transition rates of the process from state  $i$  to  $j$ , denoted by  $q_{i,j}$ , are as follows

$$q_{i,j} = \begin{cases} i\gamma + i\delta(1-p)^j & \text{if } j = i - 1; \\ i\delta \binom{i-1}{i-j-1} (1-p)^j p^{i-j-1} & \text{if } j = 0, \dots, i - 2. \end{cases} \quad (8)$$

Each individual in a sibling group gives birth at  $\beta\mu_C$ , thus, at age  $t$  the whole group gives birth to a new sibling group at a total rate  $Y(t)\beta\mu_C$ . The whole sibling group dies when the Markov process  $\{Y(t)\}_{t \geq 0}$  reaches state 0. We can thus simply describe the macro branching process  $\mathcal{M}$  as a Crump-Mode-Jagers branching process where the birth rate of each individual at age  $t$  is independent on other individuals and distributed as  $\lambda(t) := Y(t)\beta\mu_C$ . See [7] for an overview and details on Crump-Mode-Jagers branching processes.

The main advantage of defining this macro branching process is that the macro-individuals are independent and we can apply standard theory of branching processes. In particular, its reproduction number, which we will derive in the following section, corresponds to the epidemic threshold, determining whether there could be a major outbreak in the large population limit. The probability of a major outbreak, corresponding to the probability of non-extinction of  $\mathcal{B}_{CT}$ , is discussed in Section 5.3.

**Remark 4.1.** For the macro process  $\mathcal{M}$ , it is also possible to easily express the generation time distribution by normalising  $\mathbb{E}[\lambda(t)]$  so that it integrates to 1 (dividing by the reproduction number) and to express the Malthusian parameter, or growth rate,  $r_{\mathcal{M}}$  as the unique solution of

$$\int_0^{\infty} e^{-r_{\mathcal{M}}t} \mathbb{E}[\lambda(t)] dt = 1.$$

This provides additional tools that can be used for further analyses, left to future work. For example, it is known [6] that some preventive measures (as isolation, mass testing, forward and backward contact tracing) affect the generation time distribution leading to biased estimates of the reproduction number. This suggests that a similar study on the effect of sideward contact tracing on the generation time distribution in the epidemic model and resulting biases might yield interesting results.

**Remark 4.2.** The Malthusian parameter  $r_{\mathcal{M}}$  of the macro-process  $\mathcal{M}$  not only provides information about the growth of the macro-process  $\mathcal{M}$ , but also of the single-individuals process  $\mathcal{B}_{CT}$ . In fact, letting  $I_{\mathcal{M}}(s)$  and  $I_{\mathcal{B}_{CT}}(s)$  be the number of individuals alive at time  $s$  in  $\mathcal{M}$  and  $\mathcal{B}_{CT}$  respectively, we can write

$$I_{\mathcal{B}_{CT}}(s) = \sum_{i=1}^{I_{\mathcal{M}}(s)} Y_i(s - \sigma_i(s)),$$

where  $\sigma_i(s)$  is the time of birth of the  $i^{\text{th}}$  macro-individual alive at time  $s$ , with  $s - \sigma_i(s)$  thus being its age. By interpreting the size process  $Y$  as a “characteristic” of a macro-individual, the expression above allows us to interpret  $I_{\mathcal{B}_{CT}}(s)$  as the “total characteristic” of  $\mathcal{M}$  at time  $s$ , as in [8]. Then, the classical theory of Crump-Mode-Jagers branching processes [8] implies in the supercritical case that, for large times,  $I_{\mathcal{M}}(s) \approx e^{r_{\mathcal{M}}s} W$ , with  $W$  being a positive random variable; and that  $I_{\mathcal{B}_{CT}}(s) \approx e^{r_{\mathcal{M}}s} m_Y W$ , with  $m_Y$  being a positive constant.

## 5 Main results

### 5.1 Effective macro reproduction number

In this section, our interest is to derive the important quantity,  $R_e$ , which is defined as the mean number of offspring of one typical “individual” in the macro branching process  $\mathcal{M}$ , i.e., the expected number of sibling groups produced by one typical sibling group during its lifetime (before its size decreases to 0).

By standard results from the theory of branching process, the macro branching process  $\mathcal{M}$  dies out with probability 1 if  $R_e \leq 1$ ; instead, if  $R_e > 1$ , the process  $\mathcal{M}$  explodes with a strictly positive probability. In Section 5.3, we will show that the individual process  $\mathcal{B}_{CT}$  dies out with probability 1 if and only if the macro process  $\mathcal{M}$  dies out with probability 1. Then, due to the approximation outlined in Section 3, the epidemic may result in a major outbreak with a non-zero probability if and only if  $R_e > 1$ . We therefore refer  $R_e$  to as the *effective macro reproduction number*. Denoting by  $H$  the number of sibling groups produced by a sibling group, we have  $R_e = \mathbb{E}[H]$ , which we compute more explicitly in the following.

Consider a typical sibling group and let  $\{Y_k\}_{k \in \mathbb{N}}$  be the discrete-time jump Markov chain associated to the size process  $\{Y(t)\}_{t \geq 0}$ . The Markov chain  $\{Y_k\}_{k \in \mathbb{N}}$  has initial distribution



given by Equation (7) and absorbing state 0. It follows from the transition rates of  $\{Y(t)\}$  in Equation (8) that the transition probabilities  $p_{i,j}$  (i.e., the probability of  $Y_k$  moving from state  $i$  to state  $j$ ),  $i = 1, \dots$ , are given by

$$p_{i,j} = \begin{cases} \frac{\gamma + \delta(1-p)^{i-1}}{\gamma + \delta} & \text{for } j = i - 1; \\ \frac{\delta \binom{i-1}{i-j-1} (1-p)^j p^{i-j-1}}{\gamma + \delta} & \text{for } j = 0, \dots, i - 2; \\ 0 & \text{otherwise.} \end{cases} \quad (9)$$

Let  $N$  be the number of jumps until  $\{Y_k\}_{k \in \mathbb{N}}$  reaches zero, that is,

$$N = \inf\{k \in \mathbb{N} : Y_k = 0\},$$

and let  $X_k, k = 1, \dots$ , be the number of new sibling groups produced between the  $(k - 1)$ -th jump and  $k$ -th jump. With this notation, we have

$$H = \sum_{k=1}^N X_k.$$

Table 5.1 lists the important quantities related to the macro process  $\mathcal{M}$ .

**Table 5.1** Key quantities related to the macro branching process

Notation	Description
$\mathcal{M}$	macro branching process
$\{Y(t)\}_{t \geq 0}, \{Y_k\}_{k \in \mathbb{N}}$	process of sibling group size and its jump chain
$Y_0, \tilde{Y}_0$	initial size of a sibling group and its size-biased version
$\lambda(t) = \beta\mu_C Y(t)$	birth rate by one macro-individual at age $t$
$H$	number of macro-individuals generated by a typical macro-individual
$N$	number of jumps it takes for $\{Y_k\}_{k \in \mathbb{N}}$ to reach 0

Suppose that at some point we have  $Y_k = i \geq 1$ , then the time until next jump follows an exponential time  $Exp(i\gamma + i\delta)$ . Until the next jump, each of the  $i$  individuals currently alive in the group gives birth at rate  $\beta\mu_C$ , so the whole group gives birth at total rate  $i\beta\mu_C$ . Hence,  $X_k$  is distributed as a geometric random variable  $X$  with success probability  $\frac{i\gamma + i\delta}{i\beta\mu_C + i\gamma + i\delta} = \frac{\gamma + \delta}{\beta\mu_C + \gamma + \delta}$ , and mean

$$\mathbb{E}[X] = \frac{i\beta\mu_C}{i\gamma + i\delta} = \frac{\beta\mu_C}{\gamma + \delta}. \quad (10)$$

Crucially,  $X_k$  is independent of the current size  $i$ , thus we have a sequence of i.i.d random variables; and, naturally, it is also independent of  $N$ . It follows that

$$R_e = \mathbb{E}[H] = \mathbb{E}\left[\sum_{k=1}^N X_k\right] = \mathbb{E}[X] \mathbb{E}[N] = \frac{\beta\mu_C}{\gamma + \delta} \mathbb{E}[N]. \quad (11)$$

To derive  $\mathbb{E}[N]$ , we first condition on the initial size of the sibling group, that is,

$$\mathbb{E}[N] = \sum_{i=0}^{\infty} \mathbb{E}[N|Y_0 = i] \mathbb{P}[Y_0 = i] = \sum_{i=0}^{\infty} m_{i0} \mathbb{P}[Y_0 = i], \quad (12)$$

where  $m_{i0} := \mathbb{E}[N|Y_0 = i]$  is the expected number of jumps for  $\{Y_k\}_{k \in \mathbb{N}}$  to reach state 0, starting from state  $i$ ,  $i = 0, 1, \dots$  (naturally  $m_{00} = 0$ ). Conditioning on the first jump from a positive  $i$  to  $j = 0, 1, \dots, i-1$  (which happens with probability  $p_{i,j}$ ), the expected number of jumps from  $i$  to 0 is  $1 + m_{j0}$  (one single jump from  $i$  to  $j$  plus the expected number of jumps from  $j$  to 0). Therefore,  $m_{i0}$ ,  $i = 1, \dots$ , is determined by the following recursive relation

$$m_{i0} = \sum_{j=0}^{i-1} p_{i,j} (1 + m_{j0}) = 1 + \sum_{j=1}^{i-1} p_{i,j} m_{j0}, \quad (13)$$

where  $p_{i,j}$  are given by Equation (9).

Using Equation (11) and (12), the effective macro reproduction number is given by

$$R_e = \frac{\beta \mu_C}{\gamma + \delta} \sum_{i=0}^{\infty} m_{i0} \mathbb{P}(Y_0 = i)$$

with  $m_{i0}$  in Equation (13) and  $Y_0 \sim \text{Bin}(\tilde{C} - 1, \pi_{\tilde{C}})$ . We have thus proved the following Theorem.

**Theorem 5.1.** *The macro reproduction number  $R_e$  for the macro branching process  $\mathcal{M}$  is given by*

$$R_e = \frac{\beta}{\gamma + \delta} \sum_{c=2}^{\infty} c \mathbb{P}(C = c) \sum_{k=1}^{c-1} m_{i0} \binom{c-1}{i} \pi_c^i (1 - \pi_c)^{c-i-1}, \quad (14)$$

where  $m_{i0}$  is given by Equation (13).

Another possible approach to compute  $R_e$  from Equation (11) consists of numerically approximating the expected number of steps,  $\mathbb{E}[N]$ , through a Monte Carlo integration based on simulating the Markov chain  $\{Y_k\}_{k \in \mathbb{N}}$ .

## 5.2 Effective individual reproduction number

While it is sufficient to use the effective macro reproduction number  $R_e$  to analyse the threshold behaviour of the early epidemic, as shown in the next section, for completeness, in this section we show that  $R_e$  corresponds to the *effective individual reproduction number*  $R_e^{(ind)}$  of Equation (6).

**Proposition 5.2.** *Let  $R_e$  be the macro reproduction number of  $\mathcal{M}$  given by Equation (14), and let  $R_e^{(ind)}$  be the individual reproduction number of  $\mathcal{B}_{CT}$  given by Equation (6). Then, we have*

$$R_e^{(ind)} = R_e. \quad (15)$$

Such correspondence is trivial in the absence of contact tracing, as shown in the following remark.

**Remark 5.3.** In the situation when  $p = 0$ , the size of sibling group decreases by one at each jump (due to one individual naturally recovering or being diagnosed) and we have the transition probability  $p_{i,i-1} = 1$ . Therefore,  $m_{i0}(p = 0) = i$ , and  $\mathbb{E}[N] = \mathbb{E}[Y_0]$ . It follows with Equation (11) that

$$R_e(p = 0) = \mathbb{E}[X] \mathbb{E}[N] = \frac{\beta \mu_C}{\gamma + \delta} \mathbb{E}[Y_0].$$

Since  $Y_0 \stackrel{d}{=} \tilde{Z}$ , the expression above corresponds to Equation (5), thus

$$R_e(p = 0) = R_e^{(ind)}(p = 0).$$

*Proof of Proposition 5.2.* In general, when  $0 \leq p \leq 1$ ,  $R_e^{(ind)}$  is given by Equation (6). In order to show  $R_e^{(ind)} = R_e$ , it is thus enough to show that

$$\mathbb{E}[G_{CT}] \mathbb{E}[\tilde{Z}] = \mathbb{E}[H], \quad (16)$$

recalling that  $G_{CT}$  and  $\tilde{Z}$  are respectively the number of birth events and the number of offspring in one birth event generated by a typical individual in  $\mathcal{B}_{CT}$ ; and  $H$  is the number of macro-individuals (sibling groups) generated by a typical macro-individual in  $\mathcal{M}$ .

The initial size of a sibling group containing the typical individual we are considering is distributed as  $\tilde{Y}_0$ , the size-biased version of  $Y_0$ , i.e.

$$\mathbb{P}(\tilde{Y}_0 = i) = \frac{i \mathbb{P}(Y_0 = i)}{\mathbb{E}[Y_0]}, \quad (i = 0, 1, 2, \dots). \quad (17)$$

Furthermore, each of the individual who belongs to a sibling group with initial size  $i$ , produces, on average,  $\mathbb{E}[G_{CT} | \tilde{Y}_0 = i]$  number of sibling groups (despite being dependent, they are identically distributed). Thus the whole sibling group of initial size  $i$  generates  $i \mathbb{E}[G_{CT} | \tilde{Y}_0 = i]$  number of sibling groups on average. This implies the following relation

$$i \mathbb{E}[G_{CT} | \tilde{Y}_0 = i] = \mathbb{E}[H | Y_0 = i]. \quad (18)$$

Consequently, the expectation of  $G_{CT}$  is given by

$$\mathbb{E}[G_{CT}] = \sum_{i=0}^{\infty} \mathbb{E}[G_{CT} | \tilde{Y}_0 = i] \mathbb{P}(\tilde{Y}_0 = i) = \sum_{i=1}^{\infty} \frac{\mathbb{E}[H | Y_0 = i]}{i} \frac{i \mathbb{P}(Y_0 = i)}{\mathbb{E}[Y_0]} = \frac{\mathbb{E}[H]}{\mathbb{E}[Y_0]}.$$

Therefore, since  $\mathbb{E}[Y_0] = \mathbb{E}[\tilde{Z}]$ , the expression above proves Equation (16), and finally that

$$R_e^{(ind)} = \mathbb{E}[G_{CT}] \mathbb{E}[\tilde{Z}] = \frac{\mathbb{E}[H]}{\mathbb{E}[Y_0]} \mathbb{E}[\tilde{Z}] = \mathbb{E}[H] = R_e. \quad (19)$$

□

In conclusion, the individual reproduction number  $R_e^{(ind)}$  has the same expression as the macro reproduction number  $R_e$  and hence, despite the dependencies between individuals, it inherits from  $R_e$  the epidemic threshold property which is proven in the next section.

### 5.3 Extinction probability

The goal of this section is to ensure that the macro reproduction number  $R_e$  possesses the important threshold property and to provide an expression for the probability of non-extinction of the branching process  $\mathcal{B}_{CT}$ . As explained in Section 3, the non-extinction of  $\mathcal{B}_{CT}$  corresponds to a major outbreak in the epidemic, in the large population limit. It remains to show that the process  $\mathcal{B}_{CT}$  goes extinct with probability 1 if and only if the macro process  $\mathcal{M}$  goes extinct with probability 1; otherwise, the two processes  $\mathcal{B}_{CT}$  and  $\mathcal{M}$  explode with strictly positive probabilities.

In the following, we obtain an expression for the extinction probability of the branching process  $\mathcal{B}_{CT}$ , denoted by  $z_{\mathcal{B}_{CT}}$  when there is one ancestor, and equal to  $z_{\mathcal{B}_{CT}}^m$  when there are  $m$  ancestors. Consider now the process  $\mathcal{M}$  initiated with one macro-individual and denote by  $z_{\mathcal{M}}$  its extinction probability. By the standard theory of branching processes,  $z_{\mathcal{M}}$  is the smallest solution in  $[0, 1]$  of  $f_H(s) = s$ , with  $f_H(s)$  being the offspring probability generating function for  $\mathcal{M}$ . More explicitly,

$$f_H(s) = \mathbb{E} [s^H] = \mathbb{E} \left[ \mathbb{E} \left[ s^{\sum_{k=1}^N X_k} \mid N \right] \right] = \mathbb{E} [f_X(s)^N],$$

where

$$f_X(s) = \mathbb{E} [s^X] = \frac{\gamma + \delta}{\gamma + \delta + \beta\mu_C(1 - s)}$$

is the probability generating function of  $X$ .

To obtain the extinction probability  $z_{\mathcal{B}_{CT}}$ , we condition on the number of birth events (sibling groups/macro-individuals) produced by the single ancestor in  $\mathcal{B}_{CT}$  being equal to  $g$ . Since the ancestor cannot be contact traced, but can recover or be diagnosed, this number is geometrically distributed as  $G$  in (1) with success probability  $\frac{\gamma + \delta}{\gamma + \delta + \beta\mu_C}$ . We then consider the macro process  $\mathcal{M}$  initiated with  $g$  macro-individuals, which has extinction probability  $z_{\mathcal{M}}^g$ . Therefore, the extinction probability of the process  $\mathcal{B}_{CT}$  starting with one ancestor is given by

$$z_{\mathcal{B}_{CT}} = \mathbb{E} [z_{\mathcal{M}}^G] = \frac{\gamma + \delta}{\gamma + \delta + \beta\mu_C(1 - z_{\mathcal{M}})}, \quad (20)$$

and hence in the epidemic setting with  $m$  initial infectives, a major outbreak occurs with probability  $1 - z_{\mathcal{B}_{CT}}^m$ .

Moreover, Equation (20) indicates that  $z_{\mathcal{B}_{CT}} = 1$  is equivalent to  $z_{\mathcal{M}} = 1$ ; and the inequality  $z_{\mathcal{B}_{CT}} < 1$  is equivalent to the inequality  $z_{\mathcal{M}} < 1$ . As a consequence, the process  $\mathcal{B}_{CT}$  dies out with probability 1 if  $R_e \leq 1$ ; if  $R_e > 1$ , the process  $\mathcal{B}_{CT}$  starting with one ancestor explodes with a strictly positive probability  $(1 - z_{\mathcal{B}_{CT}})$ . This confirms that the macro reproduction number  $R_e$  has the epidemic threshold property.

We summarize the arguments above in the following theorem.

**Theorem 5.4.** *Let  $\mathcal{B}_{CT}$  be the process approximating the early stage of the epidemic defined in Section 3, and  $R_e$  be the macro reproduction number given by Equation (14), then  $R_e$  has the threshold property for  $\mathcal{B}_{CT}$ . That is, if  $R_e \leq 1$ , the process  $\mathcal{B}_{CT}$  goes extinct with probability 1; if  $R_e > 1$ , the process  $\mathcal{B}_{CT}$  starting with  $m \geq 1$  ancestor(s) explodes with a strictly positive probability  $(1 - z_{\mathcal{B}_{CT}}^m)$  and goes extinct with the complementary probability  $z_{\mathcal{B}_{CT}}^m$ , with  $z_{\mathcal{B}_{CT}}$  given by Equation (20).*

## 6 Numerical illustration

The goal of this section is to illustrate the effect of sideward contact tracing by means of some numerical examples relying on the theoretical results of the previous section.

Throughout this section, we assume that the size of mixing event  $C$  follows a geometric distribution, conditioned on being larger than 2 (following the choice in [1]), that is,

$$\mathbb{P}(C = c) = (1 - \alpha)^{c-2}\alpha, \quad (c = 2, 3, \dots),$$

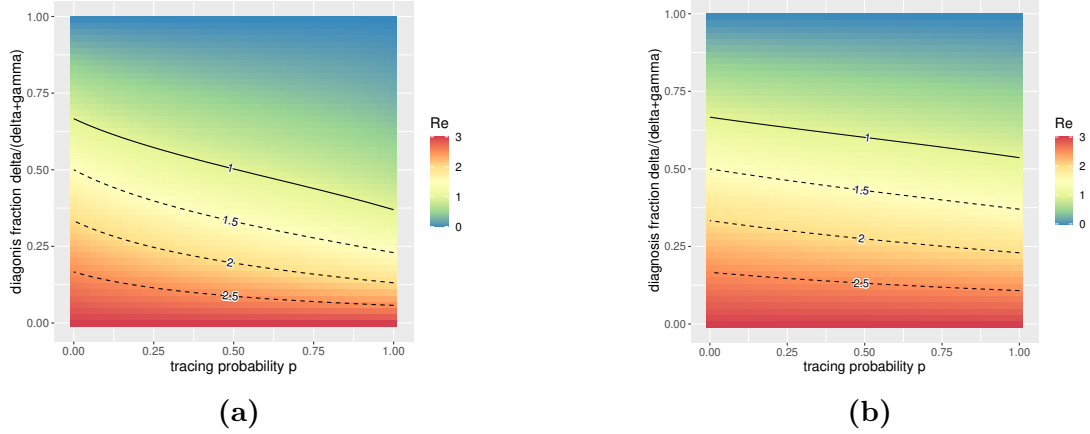
with  $\alpha = 1/(\mu_C - 1)$ . In addition, we assume that the rate of natural recovery is  $\gamma = 1/7$ .

First, we fix the average size of mixing events to be  $\mu_C = 5$ . In Figure 6.1 we examine how the fraction of diagnosis  $\delta/(\delta + \gamma)$  and tracing probability  $p$  influence the effective reproduction number  $R_e$ . The infection probability  $\pi_c$  is assumed first in Figure 6.1a independent of  $c$ , i.e.  $\pi_c = 0.5$  for all  $c$ , and then in Figure 6.1b,  $\pi_c = 2/c$ . In each of the two cases, we fix  $\beta$  such that the basic reproduction number  $R_0$  in Equation (4) equals 3. It is observed that  $R_e$  is monotonically decreasing in both  $\delta/(\delta + \gamma)$  and  $p$  as expected. Additionally,  $\delta/(\delta + \gamma)$  appears to have a stronger effect on reducing  $R_e$  compared to  $p$ . This difference becomes more evident when  $\pi_c = 2/c$ . This is because, in comparison with the deterministic choice of  $\pi_c = 0.5$ , fewer people, on average, become infected in a single mixing event of the same size and hence, more effort is required in sideward tracing to control the epidemic for a fixed reproduction number.

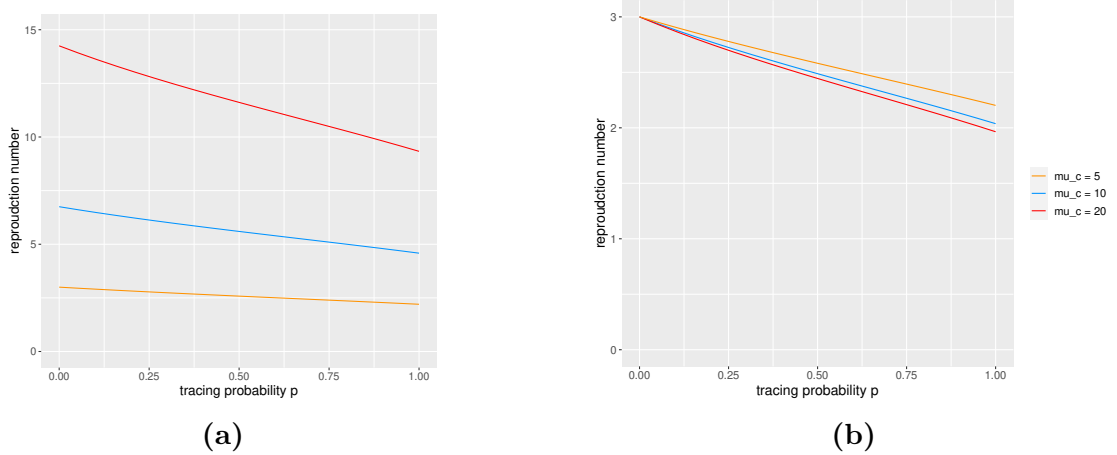
Further, we explore how the mean size of mixing events influences the effect of sideward tracing. We consider three situations of  $\mu_C = 5, 10$  and  $20$  while keeping  $\delta = \gamma = 1/7$  fixed. As shown in Figure 6.2b, if we fix  $R_0 = 3$  (different  $\beta$  for different  $\mu_C$ ), the reduction in the reproduction number due to contact tracing (i.e.  $R_0 - R_e$ ) is bigger for larger  $\mu_c$  with the same tracing probability  $p$ . This is due to sideward tracing being more effective when more individuals get infected during the same event. This observation should not suggest that larger events are preferable, in fact, imposing limitations on the gathering size plays a bigger role in the reduction of the reproduction number. For instance, as shown in Figure 6.2a, when we fix  $\beta$  to be the same for all  $\mu_C = 5, 10, 20$ , the case of  $\mu_C = 20$ , gives a much larger  $R_0$  and even with perfect tracing ( $p = 1$ ), the reproduction number remains considerably high, well above 1.

## 7 Conclusion and discussion

This paper investigates a novel concept: sideward contact tracing. This tracing approach is incorporated into an epidemic model that includes short-term mixing events, where multiple infections can occur at a single mixing event. In contrast to traditional tracing methods, sideward tracing aims to identify those who were infected at the same event, rather than the infector or/and the infectees of the index case. The early stage of the epidemic with sideward tracing was analysed through a branching process with sibling dependence. In particular, we treated the groups of individuals infected at the same event (group of siblings) as “macro-individuals”; they behave independently, according to the principles of a branching process. The effective macro reproduction number  $R_e$  was derived as the mean number of offspring of the macro branching process. The individual reproduction number  $R_e^{(ind)}$ , related to the original individual branching process, was also obtained. The two reproduction numbers have the same expression. We also expressed the probability of a major outbreak in the



**Figure 6.1** Heatmap of  $R_e$  as function of  $\delta/(\delta + \gamma)$  in  $[0, 0.99]$  and  $p$  in  $[0, 1]$  with mean size of mixing event  $\mu_C = 5$ ,  $\gamma = 1/7$ , in (a):  $\pi_c = 0.5$  for all  $c$ , in (b):  $\pi_c = 2/c$ ; and  $\beta$  is chosen so that  $R_0 = 3$ .



**Figure 6.2** Plot of  $R_e$  against the tracing probability  $p$  for different mean size of events  $\mu_C = 5, 10$  and  $20$ . The infection probability  $\pi_c = 2/c$  for all  $c$ ,  $\delta = \gamma = 1/7$ , and in (a),  $\beta$  is fixed such that  $R_0(\mu_c = 5) = 3$ ; in (b),  $\beta$  is chosen specifically for each  $\mu_C$  such that  $R_0 = 3$ .

epidemic in terms of the non-extinction probability of the macro branching process, which is defined through standard branching process theory.

A numerical illustration reveals that the fraction of diagnosis has a more significant impact on reducing  $R_e$  than the tracing probability. This observation does not diminish the usefulness of sideward contact tracing. Note that increasing the fraction of diagnosis might be more challenging (especially in the beginning of the epidemic, requiring extensive testing efforts and more cost-effective tests) than simply increasing  $p$  (e.g. during Covid-19 time in the UK, visitors/customers were encouraged to “check-in” by scanning QR code via NHS app [13]). Furthermore, the impact of sideward tracing is more pronounced, resulting in a greater reduction in the reproductive number when the size of mixing events is larger. However, it remains crucial to impose limitations on gathering size. Without such restrictions, the reproductive number may remain excessively high, making it unfeasible to bring it below 1 even with perfect tracing.

In this paper, we restrict our attention to sideward contact tracing only to analyse its effect separately from other types of contact tracing. One promising area for further investigation is combining sideward with conventional forward and backward tracing procedures which can be employed instead to trace infectors and infectees. It would be interesting to explore how these different tracing strategies can complement each other. For instance, in a scenario where an asymptomatic infector transmits the infection to two susceptibles during a mixing event, forward tracing may fail to identify the infectees as the infector remains undiagnosed. In such cases, sideward tracing becomes crucial if one of the infectees is diagnosed, enabling the identification of the remaining infectee. On the other hand, starting from one of the infectees, backward-forward tracing could potentially identify the asymptomatic infector but then fail to contact trace the other infectee before recovery. Integrating these tracing strategies is, however, analytically challenging, particularly due to the additional dependencies it introduces between the lifespans of siblings and their parents. Another possible extension of the present model making it more realistic is to incorporate delays between the diagnosis of the index case and the notification of other siblings. Additionally, incorporating heterogeneity into the model could provide deeper insights. For example, categorizing individuals based on their levels of social activity (high, normal, or low) could reveal more about the effectiveness of sideward tracing. More socially active individuals are more likely to attend events and consequently more likely to become infected and spread the infection to others. In this case, it would be important to trace the infector as well as the siblings.

In conclusion, this paper highlights the potential effectiveness of sideward contact tracing and underscores that it can play a significant role in controlling epidemics, especially when relaxing the gathering size limitations.

## Acknowledgments

We would like to thank Tom Britton for valuable discussions and helpful comments throughout this work. Additionally, we extend our gratitude to Serik Sagitov for his constructive feedback. D.Z. acknowledges the Swedish Research Council (grant 2020-04744) for financial support. M.F. acknowledges the Knut and Alice Wallenberg Foundation (Program for Mathematics, grant 2020.072) for financial support.

## References

- [1] Frank Ball and Peter Neal, *An epidemic model with short-lived mixing groups*, *Journal of Mathematical Biology* **85** (2022), no. 6-7, 63.
- [2] ———, *Strong convergence of an epidemic model with mixing groups*, *Advances in Applied Probability* (2024), 1–34.
- [3] Frank G Ball, Edward S Knock, and Philip D O’Neill, *Threshold behaviour of emerging epidemics featuring contact tracing*, *Advances in Applied Probability* **43** (2011), no. 4, 1048–1065.
- [4] Per Broberg, *Sibling dependences in branching populations* (1987).
- [5] Roberto Cortez, *SIR model with social gatherings*, *Journal of Applied Probability* (2024), 1–18.
- [6] Martina Favero, Gianpaolo Scalia Tomba, and Tom Britton, *Modelling preventive measures and their effect on generation times in emerging epidemics*, *Journal of the Royal Society Interface* **19** (2022), 20220128.
- [7] Peter Jagers, *Branching processes with biological applications*, John Wiley & Sons, 1975.

- [8] Peter Jagers and Olle Nerman, *The growth and composition of branching populations*, *Advances in Applied Probability* **16** (1984), no. 2, 221–259.
- [9] Dyani Lewis, *Superspreading drives the covid pandemic—and could help to tame it.*, *Nature* **590** (2021), no. 7847, 544–547.
- [10] Marco Mancastroppa, Andrea Guizzo, Claudio Castellano, Alessandro Vezzani, and Raffaella Burioni, *Sideward contact tracing and the control of epidemics in large gatherings*, *Journal of the Royal Society Interface* **19** (2022), no. 190, 20220048.
- [11] Johannes Müller and Mirjam Kretzschmar, *Contact tracing—old models and new challenges*, *Infectious Disease Modelling* **6** (2021), 222–231.
- [12] Peter Olofsson, *Branching processes with local dependencies*, *Classical and modern branching processes* (1997), 239–255.
- [13] Chris Wymant, Luca Ferretti, Daphne Tsallis, Marcos Charalambides, Lucie Abeler-Dörner, David Bonsall, Robert Hinch, Michelle Kendall, Luke Milsom, and Matthew Ayres *et al.*, *The epidemiological impact of the nhs covid-19 app*, *Nature* **594** (2021), no. 7863, 408–412.
- [14] Dongni Zhang and Tom Britton, *Analysing the effect of test-and-trace strategy in an sir epidemic model*, *Bulletin of Mathematical Biology* **84** (2022), no. 10, 105.