Computational Science and Engineering
(International Master's Program)

Technische Universität München

Master's Thesis

Simulation of periodic contact patterns on the effect of contact tracing

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Submission Date:	May 15th, 2021

I hereby declare that this thesis is entirely the result of my own work except where otherwise indicated. I have only used the resources given in the list of references.

May 15th, 2021 Saitel Daniela Agudelo Sanabria

Acknowledgments

I would like to acknowledge my thesis advisor Prof. Dr. Johannes Müller. I am deeply grateful for this opportunity, for the knowledge and experience he shared with, and for his guidance through every stage of this research project. I also want to thank Oriolson, Andreas, Shubham, Alejandra, Laura, and Santiago for their friendship and support throughout the master's program. Finally, I want to thank my family for their love and understanding. Special thanks to my grandparents. I have no words to describe how much I appreciate all of their efforts and faith in me. My gratitude also goes out to Nicolas for his love and encouragement over the past few years.

"Nothing in life is to be feared, it is only to be understood. Now is the time to understand more, so that we may fear less."

-Marie Curie

Abstract

Contact tracing is a mechanism for the control of outbreaks. It aims to break transmission chains by tracing and isolating people exposed to an infectious individual. In this research work, we investigate how the performance of contact tracing is affected by periodic contact patterns, which are present in events that favor the propagation of diseases and events that aim to control them.

To answer the research question, we constructed a mathematical model that incorporates periodicity for each type of contact tracing: backward - which identifies the infectors, forward - which identifies the infectees, and full - which identifies both. The models incorporate the non-recursive and recursive variants of contact tracing. In the non-recursive variant, the process stops after the contacts of one case are identified, whereas the recursive variant follows a chain of infections.

We reformulate the question as a set of optimization problems and solve it for the non-recursive backward contact tracing scenario. Our results indicate that periodic contact rates positively affect the effectiveness of backward contact tracing. The existence of periodicity increases the probability of extinction of the epidemic and minimizes the effective reproduction number. The models and optimization problems we propose here can be employed in future research to explore more complex scenarios and other types of contact tracing.

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Part I.

Introduction and Background Theory

1. Introduction

In the last twenty years, we have endured outbreaks of SARS (severe acute respiratory syndrome), H1N1 (novel influenza virus), Cholera, MERS (Middle East respiratory syndrome), Ebola, Zika virus, yellow fever, plague, and most recently, the Novel Coronavirus SARS-CoV-2 [63, 64]. In 1995, Morse adverted the existence of environmental and socioeconomic driven factors that increased people's exposure to pathogens and predicted that the likelihood of emerging infections would increase [47]. This affirmation was supported later by a study that analyzed the number of emerging infectious disease events between 1994 and 2004 [29].

The lifestyle that we maintain in the twenty-first century eases the transmission of new and re-emerging epidemics. Control strategies that used to be suitable are no longer adequate. On the one hand, ecological changes, often related to the production of food and other goods, favor the increase of the pathogen population and increases the exposition of humans to zoonotic diseases [47]. On the other hand, human-to-human interactions are much more numerous due to urbanization, high mobility of people, and the possibility to travel more often and to further destinations [47].

Besides the direct health consequences of outbreaks, people face collateral health problems, economic setbacks, stress, and anxiety [41]. Governments deal with economic and political instability, social tension, and administrative reverses, exacerbated by inequality [41]. Health interventions aim to prevent epidemics, mitigate their impact and control their transmission. The selection of a specific set of measures often involves an optimization problem where the best cost-benefit ratio is required.

There are multiple methods to slow down and limit the spread of an infectious disease. When medical approaches, like vaccination and preventive treatment, are not widely or at all available, non-medical methods come into place. On a personal level, health professionals recommend good hygiene practices like handwashing and wearing protective equipment (if applicable) [55]. Local authorities might encourage self-quarantine and social distancing, and in critical scenarios, impose gathering, commerce, and travel restrictions, along with temporary lock-downs [55]. Additionally, *screening programs* test large groups of people, contributing to identifying new infection nodes [65]. Tracking people exposed to the infection through positive or probable cases reduces the transmission rate of the disease further. The present document focuses on the latter strategy, known as contact tracing.

Contact tracing is a control method implemented to slow down and, potentially, stop the spread of communicable diseases like Ebola, Lassa Fever, and Monkeypox [63]. It is the process by which people who have been in contact with infected individuals can be

identified, informed, tested, and offered health care if they need it [62]. Communities benefit from this procedure by preventing clusters from starting outbreaks.

Tracing exposed people is only possible after identifying at least one case through direct medical observation or as the result of a screening program. These programs investigate the probability of being at high risk of a health problem. Asymptomatic individuals, who have no symptoms, are invited to be tested; if a person tests positive, he/she receives advice and treatment options [65]. When contact tracing is a coupled strategy, this person triggers a new tracking process.

Digital tools allow for infection self-reporting, tracking, and notification of risky contacts, with a reduced delay between infection detection and quarantine or treatment [13]. Digital contact tracing (DCT) is a promising alternative to traditional contact tracing. Recent studies found DCT to be more effective than its traditional counterpart if sufficient users participate [13, 16, 23]. The ethical, legal, social, and technical concerns surrounding the development of such tools were summarized in [1]. The discussion ranges from obligatoriness to availability and yields different results in different contexts. The collection, storage, access, and security of the data are central topics. On the technical side, the focus is the accuracy of the technologies and algorithms.

The proper strategy or combination of strategies to face a particular disease under budget constraints has been studied in several publications using mathematical models. Armbruster *et al.* , for instance, proposed a method to determine the optimal investment in contact tracing vs. screening, using a network-structured model [4]. Pandey *et al.* analyzed isolation, sanitary burials, contact-tracing, and reduction of hospital-acquired infections as individual and combined strategies with different levels of success in the context of the Ebola epidemic in West Africa using a stochastic model [56]. They concluded that only the aggregation of measures is sufficiently strong to control the epidemic. Many more examples of such efforts are review in [49]. Pasquini *et al.* compared contact tracing cost-effectiveness with quarantine, vaccination, social distancing, hospitalization, and other five groups of strategies used in the H1N1 pandemic of 2009. They found contact tracing to be the most cost-effective intervention, followed by the use of face masks, surveillance, and vaccination [57].

Other research work has analyzed previous outbreaks and identified problems in the health policies. Kretzschmar *et al.* investigated the effect of having testing and tracing delays for COVID-19 [38]. Their findings revealed that reducing the testing delay must be a priority. This conclusion coincides with the empirical observations of an epidemiological study for Gonorrhea conducted in British Columbia [54]. Other works have studied the delay in the implementation of contact tracing, finding that the sooner is the better [44, 40].

Previous studies have investigated the impact of disease and community-specific factors on the effectiveness of contact tracing. A prevalence threshold under which contact tracing is most effective was studied in [18, 4, 5]. The performance of different contact tracing variants under specific contact network structures was considered in [30, 31, 33]. To the best of our knowledge, no publications are addressing the influence of periodic contact patterns.

Everyday activities, such as working, shopping, studying, and eating, create repetitive interaction patterns - i.e., recurrent situations in which a different number of individuals gather in the same space. In this study, we investigate how periodic contact patterns between individuals affect the performance of contact tracing. Chapter 2 briefly explains the associated mathematical tools and algorithms. Chapter 3 introduces contact tracing, discusses different mathematical representations, and describes relevant epidemiological concepts. Chapter 4 proposes a variation of the model in [50] in which the variables depend on time and are T-periodic. It also presents a demonstration of the model using constant and variable parameters. In Chapter 5, we reformulate the research question as a set of optimization problems. Finally, we summarize and discuss the results in Chapter 6.

2. Mathematical and Modelling Toolbox

2.1. Probability

Below we present some simple probability concepts and theorems used later to build up and explain our propositions.

Definition 2.1 (Conditional probability)**.** *Let E be a σ-algebra and Ω a non-empty set, with $\Omega \in E$. Let A and $B \in E$ with $P(B) > 0$, then the probability of A conditioned on B ($P(A \mid B)$) is defined as:*

$$P(A \mid B) = \frac{P(A \cap B)}{P(B)} \tag{2.1}$$

Theorem 2.1 (Bayes' Theorem)**.** *Let A and $B \in E$, $P(A), P(B) > 0$. Then:*

$$P(A \mid B) = \frac{P(B \mid A)P(A)}{P(B)} \tag{2.2}$$

Definition 2.2 (Expected value)**.** *Let A and B be continuous random variables. Then:*

$$E(A) = \int_{-\infty}^{\infty} E(A|B = b) \cdot f_B(b)\mathrm{d}b \tag{2.3}$$

This definition comes from the partition theorem [12].

2.2. Quadrature

Quadrature is the field of mathematics that studies the approximation of definite integrals. The integrand is interpolated over an interval using polynomial interpolation. This section reviews the Gauss-Laguerre quadrature and one type of Newton-Cotes Quadrature: Simpson's rule, according to the information in [26] and [19], respectively.

In Section 4, the numerical approximation of integrals is performed using Simpson's rule only. This section is intended to support the arguments for that decision.

2.2.1. Gauss-Laguerre Quadrature

Gauss-Laguerre Quadrature is a technique used to approximate numerically the value integrals of the form:

$$\int_0^{+\infty} \exp(-x) \cdot f(x) \mathrm{d}x \tag{2.4}$$

The approximation is given by:

$$\int_0^{+\infty} \exp(-x) \cdot f(x) \mathrm{d}x \approx \sum_{i=1}^{n} \omega_i \cdot f(x_i) \tag{2.5}$$

where x_i is the $i-th$ root of the Laguerre polynomial $L_n(x)$ and the weights are calculated as:

$$\omega_i = \frac{x_i}{(n+1)^2 \left[L_{n+1}(x_i)\right]^2} \tag{2.6}$$

The error estimation is:

$$E(h) = \frac{(n!)^2}{(2n)!} f^{(2n)}(\xi),\ 0 < \xi < \infty \tag{2.7}$$

This quadrature method integrates exactly all polynomials of degree $2n-1$.

2.2.2. Newton-Cotes Quadrature

Newton-Cotes quadrature, named after Isaac Newton and Roger Cotes, evaluates the integrand of an integral in $n+1$ equidistant points.

Let f be a function defined on the interval $[a, b]$. Then, the integral of f in the interval can be calculated as:

$$\int_a^b f(x) \mathrm{d}x \approx \sum_{i=0}^{n} \omega_i f(x_i) \tag{2.8}$$

where the weights (ω_i) are the interals of the Lagrange characteristic polynomial according to the specific the nodes of the quadrature (x_i) are given by:

$$x_i = i \cdot \frac{b-a}{n},\ i \in [0, n] \tag{2.9}$$

Simpson's Rule

Simpson's rule, also known as Composite Kepler's rule, is a type of Newton-Cotes Quadrature. The interval of integration is divided into $n/2$ subintervals of equal length and discretized using $h = (b-a)/n$.

$$\int_a^b f(x)\mathrm{d}x \approx \frac{h}{3}\left[f(a) + 4f(a+h) + 2f(a+2h) + ... + 4f(b-h) + f(b)\right]$$
$$= \frac{h}{3}\left[f(x_0) + 2\sum_{j=1}^{n/2-1} f(x_{2j}) + 4\sum_{j=1}^{n/2} f(x_{2j-1}) + f(x_n)\right] \quad (2.10)$$

where $x_j = a + jh$, $j = 0, .., n$

This quadrature rule integrates polynomials of degree ≤ 3 in a definite interval $[a, b]$ exactly.

The estimate of the error is:

$$E(h) = -\frac{h^4}{180}(b-a)f^{(4)}(\xi),\ \xi \in]a, b[\quad (2.11)$$

2.3. Runge-Kutta methods

In this section we describe Runge-Kutta method according to [7, 8], and using the notation in [17].

Let us consider the following initial value problem (IVP) of an ordinary differential equation (ODE):

$$\frac{\mathrm{d}x(t)}{\mathrm{d}t} = f(t, x(t)) \quad (2.12)$$
$$x(t_0) = x_0 \quad (2.13)$$

The aim is to find $x \in \mathbb{C}^1(I, \mathbb{R}^n)$ on an open and connected interval $I =]t_0, t_f[\subset \mathbb{R}$. If the exact solution does not exist or is hard to obtain, the numerical solution can be calculated iteratively using a numerical method.

The problem in Eq. 2.13 is equivalent the following formulation

$$\frac{x(t+h) - x(t)}{h} = \frac{1}{h}\int_t^{t+h} f(\varepsilon, x(\varepsilon))\mathrm{d}\varepsilon \approx \phi(t, x, h) \quad (2.14)$$

The integration interval is discretized and the solution is approximated in $N+1$ equidistant grid points. The discretization step is $h = t_i - t_{i-1}$. The increment function $\phi(t, x, h)$, is the numerical approximation of the exact solution.

Runge-Kutta Methods are a family of numerical methods, developed by Carl Runge and Wilhalm Kutta, for the numerical solution of such IVP (Eq. 2.13). The approximation of the exact solution for s stages is given by:

$$\phi(t, x, h) = \sum_{k=1}^{s} b_k \cdot f_k(t, x, h) \tag{2.15}$$

In the explicit version, f_k is calculated using the previously calculated values: $f_1, ..., f_{k-1}$.

$$f_k(t, x, h) := f(t + c_k \cdot h, x + h \sum_{j=1}^{k-1} \alpha_{kj} \cdot f_j(t, x, h)) \tag{2.16}$$

while the implicit method, $f_k, ..., f_s$ must be found by solving a root finding problem, as they are not explicitly given.

$$f_k(t, x, h) := f(t + c_k \cdot h, x + h \sum_{j=1}^{s} \alpha_{kj} \cdot f_j(t, x, h)) \tag{2.17}$$

The coefficients b_k, α_{kj}, c_k in Eq. 2.15, Eq. 2.16 and Eq. 2.17 are usually given in a Butcher tableau and chosen so that $\phi(t, x, h)$ coincides with the Taylor expansion of the exact solution up to the order of the method. The Butcher tableau is a representation where A is the Runge-Kutta matrix, b is a vector of weights, and c is a vector of nodes (See Eq. 2.18). For the explicit Runge-Kutta methods, in contrast to implicit ones, A is a lower triangular matrix.

$$\begin{array}{c|c} c & A \\ \hline & b \end{array} \tag{2.18}$$

We define $\eta_i = \eta(t_i)$ as the approximation of the solution $x_i = x(t_i)$, which is only defined at the grid points. Then in each step of the algorithm we calculate:

$$\eta(t + h) = \eta(t) + h \sum_{k=1}^{s} b_k \cdot f_k(t, \eta(t), h) \tag{2.19}$$

The error of the approximation can be controlled using two Runge-Kutta methods with different orders: p_a and p_b, with $p_a < p_b$. The algorithms that use this strategy are also known as Fehlberg methods. In theory, the error is estimated using the p_b-order accurate method and the step is calculated using the p_a-order method. In practice, however, we often find algorithm implementations where the error is controlled using the lower-order method, and the steps are taken using the higher-order one.

In Section 4.5, we use a specific implementation of the implicit Runge-Kutta method, know as Radau IIA of order 5, to solve an initial value problem (IVP). An embedded 3-order formula is used to control the error [60]. Next, we present a summary of the characteristics of the method, according to the description in [6]

Radau IIA is computed using Radau Quadrature, and the nodes c are calculated as the zeros of the following polynomial:

$$\frac{d^{s-1}}{dx^{s-1}}(x^{s-1}(x-1)^s) \tag{2.20}$$

The Butcher table for Radau IIA of order 3 is given as:

$$\begin{array}{c|cc} 1/3 & 5/12 & -1/12 \\ 1 & 3/4 & 1/4 \\ \hline & 3/4 & 1/4 \end{array} \tag{2.21}$$

Likewise, we present the Butcher table for Radau IIA of order 5:

$$\begin{array}{c|ccc} \frac{2}{5}-\frac{\sqrt{6}}{10} & \frac{11}{45}-\frac{7\sqrt{6}}{360} & \frac{37}{225}-\frac{169\sqrt{6}}{1800} & -\frac{2}{225}+\frac{\sqrt{6}}{75} \\ \frac{2}{5}+\frac{\sqrt{6}}{10} & \frac{37}{225}+\frac{169\sqrt{6}}{1800} & \frac{11}{45}+\frac{7\sqrt{6}}{360} & -\frac{2}{225}-\frac{\sqrt{6}}{75} \\ 1 & \frac{4}{9}-\frac{\sqrt{6}}{36} & \frac{4}{9}+\frac{\sqrt{6}}{36} & \frac{1}{9} \\ \hline & \frac{4}{9}-\frac{\sqrt{6}}{36} & \frac{4}{9}+\frac{\sqrt{6}}{36} & \frac{1}{9} \end{array} \tag{2.22}$$

As every implicit method, Radau IIA has excellent stability properties, which makes it suitable for the numerical treatment of stiff problems.

2.4. Numerical calculation of eigenvalues and eigenvectors: The power iteration

The eigenvalues and eigenvectors of a transformation provide information on how a system behaves. An $n \times n$ matrix $A \in \mathbb{C}$ always have n eigenvalues, which are not necessarily real nor distinct. For $n > 4$, the eigenvalues can not be computed exactly with a finite number of operations [24]. Many iterative methods have been developed to calculate them numerically. The QR, Arnoldi factorization, divide and conquer, and Jacobi algorithms, for example, allow for the approximation of all the eigenvalues of a suitable matrix A [24, 21, 22]. The power, inverse, and Rayleigh quotient iteration, on the other hand, approximate only one eigenpair [24]. In this section we review the power iteration.

The power iteration is an iterative method that converges to the largest eigenvalue and eigenvector of matrix $A \in \mathbb{C}^{n\times n}$ (See Algorithm 1).

The algorithm starts with an arbitrary non-zero initial vector, which is multiplied repeatedly by the matrix A. Eventually, the multiplication converges to the largest eigenvalue and the corresponding eigenvector. The proof, which is based on vector decomposition is given in [24].

The residual of the eigenvector is calculated as:

$$\vec{r}^(k) = A \cdot \vec{q}^{(k)} - v^{(k)} \cdot \vec{q}^{(k)} \tag{2.23}$$

and the iteration is stopped when $\left\|\vec{r}^{(k)}\right\|_2 < tol$, with tol is sufficiently small [59, 58].

Algorithm 1 Power iteration [17]

Input: $A \in \mathbb{C}^{n\times n}$, $\vec{q}^{(0)} \in \mathbb{C}^n$
for $k = 1, 2, ...$ **do**
$\vec{z}^{(k)} = A\vec{q}^{(k-1)}$
$\vec{q}^{(k)} = \vec{z}^{(k)} / \left\|\vec{z}^{(k)}\right\|_2$
$v^{(k)} = \vec{q}^{(k)H} A\vec{q}^{(k)}$
end for

The vector is normalized each iteration to avoid overflow and underflow. [24].

2.5. Optimization for a problem with side condition

In Chapter 5 we construct two optimization problems with side condition. This section provides a summary of the algorithm in [36] to explain how sequential least squares programming (SLSQP) allows us to solve them.

Sequential Least Square Programming is an iterative method to solve constrained nonlinear optimization problems. The algorithm, described by Dieter Kraft, solves a sequence sub-problems where the Lagrange function of the problem is approximated quadratically, while the constraints are linearized [36]. The nonlinear programming problem can have equality constraints (Eq. 2.25), inequality constraints (Eq. 2.26) and bounds (Eq. 2.27).

Optimization problem 2.1 (Nonlinear problem). *Minimize*

$$f(x) \mid x \in \mathbb{R}^n \tag{2.24}$$

subject to

$$g_j(x) = 0,\ j = 1, ..., m_c \tag{2.25}$$
$$g_j(x) \geq 0,\ j = m_c + 1, ..., m \tag{2.26}$$
$$x_1 \leq x \leq x_n \tag{2.27}$$

The algorithm starts with a given initial vector x_0. The next iteration vector is calculated as:

$$x^{k+1} = x^k + \alpha^k \cdot d^k \tag{2.28}$$

where d^k is the search direction and α^k is the step length into that direction. For obvious reasons, $\alpha > 0$.

The search direction is computed using a quadratic approximation of the Lagrange function

$$\mathcal{L}(x, \lambda) = f(x) - \sum_{j=1}^{m} \lambda_j \cdot g_j(x) \tag{2.29}$$

and a linear approximation of the constraints. Here, λ is the lagrange multiplier.

Optimization problem 2.2 (Search direction: Quadratic problem)**.** *Minimize*

$$\frac{1}{2}d^T \cdot B^k \cdot d + \nabla f(x^k) \cdot d \mid d \in \mathbb{R}^n \tag{2.30}$$

subject to

$$\nabla g_j(x^k) \cdot d + g_j(x^k) = 0,\ j = 1, ..., m_c \tag{2.31}$$
$$\nabla g_j(x^k) \cdot d + g_j(x^k) \geq 0,\ j = m_c + 1, ..., m \tag{2.32}$$
$$\tag{2.33}$$

with $B = \nabla^2_{xx}\mathcal{L}(x, \lambda)$

This quadratic problem is equivalent to a least-squares problem, where the matrix B is decomposed using LDLT factorization: $B = LDL^T$. Here, L is a lower triangular matrix, and D is a diagonal matrix. The letter T indicates that the second matrix L is a transposed matrix.

Optimization problem 2.3 (Search direction: Least squares problem)**.** *Minimize*

$$\left\| (D^k)^{1/2}(L^k)^T \cdot d + (D^k)^{-1/2}(L^k)^{-1}\nabla f(x^k) \right\| \mid d \in \mathbb{R}^n \tag{2.34}$$

subject to

$$\nabla g_j(x^k) \cdot d + g_j(x^k) = 0,\ j = 1, ..., m_c \tag{2.35}$$
$$\nabla g_j(x^k) \cdot d + g_j(x^k) \geq 0,\ j = m_c + 1, ..., m \tag{2.36}$$
$$\tag{2.37}$$

The step size is obtained by minimizing the penalty function (Eq. 2.38).

Optimization problem 2.4 (Step size:)**.** *Minimize*

$$\Phi(\alpha) = \phi(x^k + \alpha \cdot d^k) \mid \alpha \in \mathbb{R}^1 \tag{2.38}$$

with

$$\phi(x; \rho) = f(x) + \sum_{j=1}^{m_c} \rho_j |g_j(x)| + \sum_{j=m_c+1}^{m} \rho_j |g_j(x)| \tag{2.39}$$

and $|g_j(x)| = min(0, |g_j(x)|)$ *and* ρ_j *the penalty parameters.*

This algorithm is a good choice for nonlinear optimization problems where the optima of the function has to be found within a set of feasible options, where linear and nonlinear constraints apply.

3. Contact tracing, epidemiology and mathematics

3.1. Contact tracing

Contact tracing is a measure to control epidemics. Its goal is to identify, test, and isolate infected individuals before infection clusters appear. The method was developed in the XIX century to mitigate outbreaks of diseases transmitted through air, droplets, or direct transmission [45]. Its implementation contributed to eradicate Smallpox [46]. After two centuries, it continues to be a valuable asset to counteract the spread of HIV/AIDS, Chancroid, Chlamydia, Gonorrhea, Tuberculosis, and many other pathologies [9]. This section summarizes the characteristics of contact tracing and gives a brief overview of the mathematical models used to analyze its performance. We present a more detailed review of Müeller *et al.* , which we adapt in Chapter 4.

The tracing process starts with the detection of an infected person. The discovery of a case can happen in several ways, for example: by symptom evaluation, testing, or as the result of a screening program. This person is called the *index case*, which means he/she represents the starting point of the transmission chain that we want to investigate [10]. The index case provides information about his/her contact persons in a particular period that depends on the incubation, latent, and to-recovery time of the disease [9]. The person who transmitted the infection to the index case is the *infector*, while those who receive it are the *infectees* [25]. Through contact tracing, one can find the infector, the infectees, or both. These three different objectives determine three variants: backward, forward, and full tracing (See Figure 3.1).

Backward contact tracing aims to locate the infector of the index case, while forward contact tracing intends to find the infectees. Hethcote and Yorke introduced these concepts in 1984 as *Infector tracing* and *Infectee tracing* [25]. In full contact tracing, both infector and infectees are objectives. Among the contacts referred by the index case, those at risk of exposure are reached by the health officials and receive medical advice and treatment [62]. If the process is recursive, they become index cases, and a new tracing cycle starts.

The probability of finding exposed contacts and avoid further infections depends on the ability of the index case to recall their contacts and their willingness to provide the information. In the traditional way to do contact tracing (manually), healthcare providers have the responsibility of gaining the patient's trust and persuading them to cooperate [9]. Mooney narrates the perks of that task from a historical prospect and concludes that the lack of resources to assure patients makes it increasingly difficult [46].

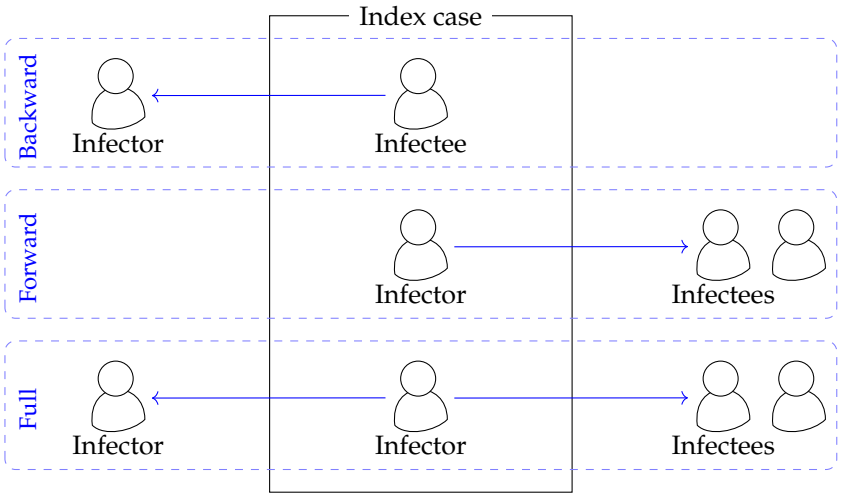


Figure 3.1. Contact tracing diagram The index case triggers a contact tracing process and help to find other infected individuals.

Another factor that affects the success of contact tracing is the agility of the process. Kretzschmar *et al.* investigated how testing and tracing delays affect the effectiveness of contact tracing, finding that the percentage of prevented infections reduces from 79.9% without delays to 21% when both the testing and tracing delay equal three days [38].

Digital contact tracing appears to be a suitable solution to the problems mentioned earlier. People no longer need to recall their interactions as they can be recorded based on proximity using technology like bluetooth [43]. Technology aids the process by reducing the tracing delay [38]. Not only is the notification process automatic, but it can also be anonymized [43]. This feature eases the process for patients and health authorities as there is no need to ask for or reveal personal information.

3.2. Mathematical models for contact tracing

The analysis of epidemiological phenomena benefits from mathematical tools. Mathematical models are helpful to assess disease-related hypotheses and to compare the performance of different health policies. Contact tracing aims to contribute to control the spread of infections, improve the surveillance process, and target specific groups of people who, otherwise, would not receive treatment. The central question here is: how effective is it?

In Table 3.1, we present a comparison between the main modeling strategies for contact tracing according to the review in [49] and the articles cited there.

Table 3.1.: Contact tracing models: comparison

Characteristic	Individual-based models (IBM)	Pair approximation models	Models based on the branching process
Idea	It consists of nodes connected through edges and a set of rules that define each node's behavior.	It consists of a set of ordinary differential equations that describe the dynamics of the system. There is one equation for each epidemiological state and one for each pair-wise correlation.	It consists of a set of equations that describe the system's dynamics at the onset of the epidemic (when the whole population is susceptible). The epidemic is approximated by a branching process, i.e., a stochastic process in which the number of infections produced by a generation is a random variable. The model can be deterministic or stochastic.
Modeling level	Individual	Population	Population
Interactions	There is an explicit contact graph. Individuals are nodes, and the connections between them are edges. A stochastic process determines when a gives two individuals come into contact.	Interactions are given as averaged rates.	Interactions are given as averaged rates.
Interaction complexity	It can describe complex inhomogeneous contact graphs.	Not suitable for complex inhomogeneous contact graphs.	Not suitable for complex inhomogeneous contact graphs.
Representation of contact tracing	The tracing process relies on the contact graph. The neighbors of an infected node have a probability p of being identified.	The model incorporates the state *Traced*, and the tracing process is modeled as an interaction that depends on the density of infectious-traced edges and the tracing rate.	The tracing process is represented through the probability of infection. This probability decreases with spontaneous recovery, observed recovery, and contact tracing.
Formulation intricacy	Low	Medium to high	Medium to high
Analytical results	Seldom available	Available	Available

Main advantage	A representation of the tracing process with high level of detail is possible.	The model reflects the slowdown of the epidemic when the likelihood of an encounter between infected individuals increases	The model allows for calculating central figures like the reproduction number and the doubling time at the start of an epidemic when they are critical to design health interventions.
Main disadvantage	The amount of data required for parameterization increases with the level of detail.	The number of equations increases with the number of states.	The results are only valid at the onset of the outbreak, when likelihood of an encounter between infected individuals is low.

The effectiveness of contact tracing has been studied in multiple publications, using different models and from multiple angles. For example, [4] analyzed the cost-effectiveness of contact tracing using an individual-based model (IBM). The impact of the network structure on the performance of contact tracing was examined in [32] and [34] using an IBM, and in [30] and [27] using a pair-approximation model. The effect of testing and tracing delays was investigated using an IBM in [38], and from the branching process perspective, in [37] and [48].

In this document, we aim to investigate the performance of contact tracing from a new perspective. We consider periodic contact patterns and assess their impact on the effectiveness of contact tracing. To do that, we modify the branching-process-based model proposed in [50]. A review is provided in Section 3.4. Section 3.3, introduces a fundamental metric in epidemiology, which is used in this study as a performance metric: the reproduction number.

3.3. Effectiveness metric: Reproduction number

The reproduction number is a central figure in epidemiology that is helpful to assess the effectiveness of a control strategy to manage an epidemic. There are two kinds of reproduction numbers or reproduction ratios: basic reproduction number (R_0) and effective reproduction number (R_∞). In this section, we provide their definitions. In Chapter 5, we use the effective reproduction number as the objective function and the basic reproduction number as a constraint in a set of optimization problems.

Definition 3.1 (Basic reproduction number (R_0))**.** *Average number of secondary infections caused by a typical individual during the early phase of an outbreak [3].*

In Definition 3.1, the term *typical individual* excludes individuals who have a different ability to carry and transmit the virus. The basic reproduction number depends on the infectious agent and the community where it is spreading [3].

Theorem 3.1 (Threshold Theorem). *If $R_0 < 1$ the disease-free equilibrium is an absorbing state. The epidemic dies out.*

If $R_0 > 1$ there are two equilibria: the disease-free equilibrium and the endemic equilibrium. Because the latter is attractive, the disease is able to spread.

The basic reproduction number R_0 can be in interpreted as the product of the probability of transmission (m), the rate of contacts per time (k), and the average duration of the infectious period (l) [42].

$$R_0 = m \cdot k \cdot l \tag{3.1}$$

Health policies aim to tackle one of these three aspects. In [14] some examples are given regarding COVID-19 pandemic: Mask-wearing reduces m, social distancing reduces k, and early diagnosis and isolation reduces l. Thus, control measures, modify the behaviour of the reproduction number in time.

Definition 3.2 (Effective reproduction number (R_∞)). *Average number of secondary infections caused by a typical individual in a non-entirely susceptible population [20].*

$$R_\infty = \lim_{t \to \infty} R_t \tag{3.2}$$

Theorem 3.1 also applies for R_∞. The objective is to make the effective reproduction number go below one so that the epidemic dies out.

The reproduction number can be calculated via linearization or using the next generation operator [52]. In both cases, we find an operator, which has the reproduction number as the dominant eigenvalue. In Section 4.4, we use a more specific procedure to deal with the periodic nature of our model when calculating the reproduction number.

3.4. Model for contact tracing without periodicity

In this section, we summarize the model proposed by Müller *et al.* in [50]. In Chapter 4 we present an extension of this model, where the probability of infection depends on the age of infection of an individual (a) and on time (t), in a periodic fashion. The effect of periodicity on the effectiveness of contact tracing as a control strategy is explored in Chapter 5.

A stochastic SIRS model is used to represent the course of an epidemic. For very large populations, the stochastic equations that represent the process can be approximated by ordinary differential equations (ODEs) [51].

Definition 3.3 (SIRS Model). *The SIRS model (See Figure 3.2) is a variation of the Kermack-McKendrick Model, also known as the SIR model. In this compartmental model, individuals have one of three states: susceptible (S), infected (I), or removed (R). The total population (N) at a time t is $N(t) = S(t) + I(t, a) + R(t)$.*

Susceptible individuals are healthy and become infected when they come into contact with an infected individual. Infectious individuals infect susceptible people at rate $\beta \cdot S/N$, and become removed if they recover from the disease spontaneously, recover after observation, or are discovered via contact tracing. In the last two cases, the assumption is that patients receive medical advice and treatment. Therefore, they are no longer contributing to the spread of the pathogen. Removed people lose immunity at rate γ and become susceptible again.

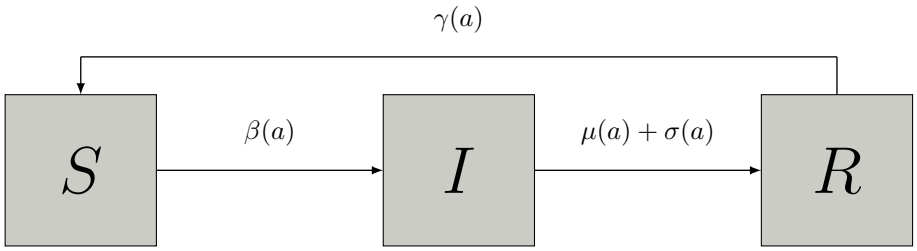


Figure 3.2. Compartmental view of the infection process Susceptible individuals become infectious at rate $\beta(a)$. Infected individuals recover spontaneously at rate $\mu(a)$ or are detected via observation or screening at rate $\sigma(a)$.

In this model the following assumptions hold:

- There are no immune individuals before the epidemic starts.
- Individuals are similar and mixed up uniformly
- There are not behaviour changes in the course of the epidemic.
- Births and deaths are neglected as they occur on a much slower time scale.
- The latent period (time from exposure to infectiousness) and the incubation period (time from exposure to symptoms onset) are equal.

The authors analyze the epidemic at its onset when almost all the individuals are susceptible ($S \approx N$). The initial phase can be approximated by a Galton-Watson process (branching process), i.e., a stochastic process where the infected individuals in a generation produce a random number of infections in the next generation. This type of analysis is helpful to predict the course of an outbreak and select appropriate control strategies. In principle, the results are only valid at the onset [50]—afterward, the number of people who have already been infected increases; therefore, contacts between infected individuals, which do not produce new infections, are more likely. However, the authors provide a deterministic approximation based on heuristic arguments.

Remark 3.1 (Onset of the epidemic and Galton-Watson Process). *Here, we review the Galton Process according to [50] and [51] using the same notation as the authors.*

Let X be a random variable that counts the number of infectees of an infector. If the number of infected individuals in the generation n is Z_n, then the next generation is conformed by their infectees:

$$Z_{n+1} = \sum_{i=1}^{Z_n} X_i \tag{3.3}$$

The probability generating function for the generation i is given by:

$$f_i(s) = \sum_{j=0}^{\infty} s^j \cdot P(X = j) \tag{3.4}$$

where $P(X = j)$ is the probability of having j infectees in the generation i.

For an asymptotically stationary Galton-Watson process, there exist a function $g_i(s)$, such that:

$$f_i(s) = g_{i-1}(f_{i-1}(s)) \tag{3.5}$$

$$g_i(s) \to g_\infty(s)\ , for\ i \to \infty \tag{3.6}$$

The expected number of infections of the generation i (R_i) is given by:

$$R_i = \frac{\mathrm{d}}{\mathrm{d}s} g_i(s)|_{s=1} \tag{3.7}$$

As $i \to \infty$, $R_i \to R_\infty$, where R_∞ is the effective reproduction number.

Following the Threshold theorem (See Theorem 3.1), if $R_0 < 1$ the epidemic is not able to invade and no outbreak is produced. If $R_0 > 1 > R_\infty$, the disease spreads initially but dies out eventually. If $R_\infty > 1$, the disease is able to spread to all the population.

The model in [50], describes the backward, forward, and full contact tracing variants (See Figure 3.1). The process always starts with one individual called the *index case*, who is asked to provide information about their contacts in the days around the probable day of infection. In backward contact tracing, the aim is to find the infector. In forward contact tracing, the aim is to find the infectees. Both infector and infectees are wanted in the latter case.

Next, we enunciate the model propositions without proof. For the details of the proof, the reader can refer to the original publication [50]. In Chapter 4, we use and build on these ideas to prove the propositions for our model.

3.4.1. Without contact tracing

The probability of infection equals one when the age of infection is equal to zero, in other words when the person has just been infected. In absence of contact tracing, it decreases with the rate of spontaneous recovery ($\mu(a)$) and the rate of observed recovery ($\sigma(a)$).

Proposition 3.1 (Probability of infection without contact tracing [50]).

$$\hat{\kappa}(a) = \exp\left(-\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a})\mathrm{d}\tilde{a}\right) \tag{3.8}$$

3.4.2. Backward Tracing

In backward tracing, the focus is on the probability of infection of the infector at age of infection a. It equals one at $a = 0$ and decreases with the spontaneous recovery rate $\mu(a)$, the observed recovery rate $\sigma(a)$, and the removal rate due to contact tracing.

The infectee might not be able to refer all his/her contacts, so p is introduced to represent the probability for the infector to be identified. The integral term that multiplies p in Eq. 3.9 and Eq. 3.10 represents the existence of a still infectious infectee who is detected. In the recursive case, the infectee is found via contact tracing, while in the non-recursive case, the infectee is observed.

Proposition 3.2 (Probability of infection under recursive backward contact tracing [50]). *The probability of infection under recursive backward contact tracing is given by:*

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^{-}(a) = -\kappa^{-}(a)\left(\mu(a) + \sigma(a) + p\int_0^a \beta(a-b)\cdot\left(-\kappa^{-\prime}(b) - \kappa^{-}(b)\cdot\mu(b)\right)\mathrm{d}b\right) \tag{3.9}$$

with $\kappa^{-}(0) = 1$.

Proposition 3.3 (Probability of infection under non-recursive backward contact tracing [50]).

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^{-}(a) = -\kappa^{-}(a)\left(\mu(a) + \sigma(a) + p\int_0^a \beta(a-b)\cdot\kappa^{-}(b)\cdot\sigma(b)\,\mathrm{d}b\right) \tag{3.10}$$

with $\kappa^{-}(0) = 1$.

3.4.3. Forward Tracing

Forward contact tracing aims to find the infectees of a subject. Here, the probability of infection for an infectee depends on the generation, in other words, of the length of the

transmission chain between infector and infectee. Eq. 3.11 and Eq. 3.12 describe the probability of infection for an infectee of the i-th generation, conditioned to the age of the infector at the infection event. The condition is removed using Definition 2.2.

The infectee is still infected provided that he/she has not yet recovered nor been observed or traced. The computation is done by multiplying the probability of infection without contact tracing and the probability of having an unsuccessful tracing process. Here, again, p represents the probability of a successful tracing event. The integral depicts the existence of a still infectious infector. In the recursive version, the infector is found via contact tracing, while in the non-recursive version, he/she is observed.

Proposition 3.4 (Probability of infection under recursive forward contact tracing [50]). *For $i > 0$:*

$$\kappa_i^+(a|b) = \hat{\kappa}(a)\left(1 - p \cdot \int_0^a \left(-\frac{{\kappa_{i-1}^+}'(\tilde{a}+b)}{\kappa_{i-1}^+(\tilde{a}+b)} - \mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}^+(\tilde{a}+b)}{\kappa_{i-1}^+(b)}d\tilde{a}\right) \tag{3.11}$$

with $\kappa_i^+(0) = 1$.

Proposition 3.5 (Probability of infection under non-recursive forward contact tracing [50]). *For $i > 0$:*

$$\kappa_i^+(a|b) = \hat{\kappa}(a)\left(1 - p \cdot \int_0^a \sigma(\tilde{a}+b) \cdot \frac{\kappa_{i-1}^+(\tilde{a}+b)}{\kappa_{i-1}^+(b)}d\tilde{a}\right) \tag{3.12}$$

with $\kappa_i^+(0) = 1$.

Forward contact tracing is not suitable for tracing the primary infected individual, as he/she did not acquire the pathogen from another person. In this case, only backward contact tracing is suitable.

3.4.4. Full Tracing

Full contact tracing combines backward and forward contact tracing. The probability of infection of a target individual depends on the generation and equals one at age of infection $a = 0$. At all ages, it can be calculated as the multiplication of the probability of infection under backward contact tracing and the probability of an unsuccessful forward contact tracing event. All in all, the target individual is still infectious if he/she has not recovered, has not been observed, and has not been traced.

Proposition 3.6 (Probability of infection under recursive full contact tracing [50]). *For $i > 0$:*

$$\kappa_i(a|b) \quad = \quad \kappa^-(a)\left(1 - p \cdot \int_0^a \left(-\frac{{\kappa_{i-1}}'(\tilde{a}+b)}{\kappa_{i-1}(\tilde{a}+b)} - \mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}(\tilde{a}+b)}{\kappa_{i-1}(b)}d\tilde{a}\right) \tag{3.13}$$

with $\kappa_i^+(0) = 1$.

Proposition 3.7 (Probability of infection under non-recursive full contact tracing [50]). *For $i > 0$:*

$$\kappa_i(a|b) = \kappa^-(a))\left(1 - p \cdot \int_0^a \sigma(\tilde{a}+b) \cdot \frac{\kappa_{i-1}(\tilde{a}+b)}{\kappa_{i-1}(b)}d\tilde{a}\right) \tag{3.14}$$

with $\kappa_i^+(0) = 1$.

Part II.

Contact tracing with periodic patterns

4. Model definition

In Chapter 3 we introduced contact tracing as a tool to detect and break chains of infection. In this chapter, we aim to build on the mathematical model presented in Section 3.4 by incorporating periodic contact patterns and investigating their effect on the impact of contact tracing.

4.1. Introducing periodicity

Our daily routines are full of interactions, but not all of these are equal. The number of contacts and the length of the interaction is different at different times of the day. One encounters few people on the elevator, dozens at work, and hundreds on public transportation, especially during rush hour. All these interactions vary in span. Further, there is an implicit periodicity that comes from the similarity of everyday routines in related groups. Likewise, health services and programs operate only on particular days and times. All in all, there is periodicity in the activities that favor the propagation of a pathogen and in the activities that aim to prevent it. How does it change the effect of contact tracing?

The model in Section 3.1 is modified to represent the periodicity of contact patterns as a time dependency. The contact rate $\beta(a,t)$, the unobserved recovery rate $\mu(a,t)$, and the observed recovery rate $\sigma(a,t)$ are redefined according to the following ideas:

- The transmission rate β depends on the age of infection (a) and, in a periodic manner, on time (t). The dependency on the age of infection reflects the change of pathogen load in the individuals as the disease progresses. The dependency on time reflects the contact rate. An individual is more likely to infect others when the pathogen load is high. He/she also has a higher probability of meeting with others at certain times of the day, e.g., while working.
- The unobserved recovery rate μ depends only on the time of infection (a). This dependency reflects the time the body needs to fight a pathogen. Note that μ does not depend on time (t), given that the immune system response is stable for long periods [15].
- The observed recovery rate σ depends on the age of infection (a) and is T-periodic in time. It depends on the age of infection because the probability of detecting an individual is higher when the symptoms appear. Furthermore, the time dependency models the detection through screening and medical examination, usually taking place on specific days and times.

Definition 4.1. *The contact rate $\beta(a,t)$ and the detection rate $\sigma(a,t)$ depend on the age of infection and on time. The time dependency is assumed to be independent of the age of infection dependency. The spontaneous or unobserved recovery rate $\mu(a)$ only depends on the age of infection.*

$$\beta(a,t) = \beta_1(a) \cdot \beta_2(t) \tag{4.1}$$

$$\mu(a,t) = \mu(a) \tag{4.2}$$

$$\sigma(a,t) = \sigma_1(a) \cdot \sigma_2(t) \tag{4.3}$$

We assume that $\beta_2(t)$ and $\sigma_2(t)$ are T-periodic, with $T > 0$.

Remark 4.1. *The age of infection and the time are coupled. That means that if an individual has an age of infection a at time t, after h units of time, he/she will have an age of infection of $a + h$ units.*

Remark 4.2. *For the effect of contact tracing, infectees are no longer infectious if:*

- *The individuals recover spontaneously or in an unobserved way. In our model, this happens at rate $\mu(a)$.*
- *The individuals are detected by a screening program or via observation. In our model this happens at rate $\sigma(a,t)$.*
- *The individuals are identified via contact tracing. In our model, the tracing process has a probability p of being successful.*

In the case of detection and contact tracing, we assume that the individual does not generate new infections because he/she receives advice and treatment.

4.2. Chain of infection with periodic patterns

Fig. 4.1 illustrates the infection process with periodic patterns. The compartment S represent the *susceptible* state. Susceptible individuals are exposed to the pathogen and become infectious after the latent period. This happens at rate $\beta(a,t)$. The compartment I represents the *infected* state. Infected individuals develop symptoms at the end of the incubation period. In general, the latent period and the incubation period are different. However, for the sake of simplicity, we consider them equal. Individuals recover spontaneously at rate $\mu(a)$, are observed or screened at rate $\sigma(a,t)$, and are traced with probability p. In any case, they move to the compartment R, representing the *recovered* state. After the recovery, individuals obtain permanent or waning immunity.

The center of this analysis is around the onset of the outbreak. For this reason, having a SIR or a SIRS model makes no difference. We assume interhuman transmission with no vertical transmission. Likewise, the latent and incubation periods are considered equal. The population N is fixed, with no births or deaths. As in [50], we neglect delays in the contact tracing process and assume it is faster than the infection process.

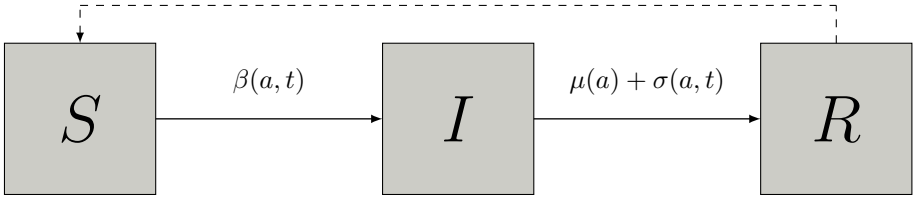


Figure 4.1. Compartmental view of the infection process Susceptible individuals become infectious at rate $\beta(a,t)$. Infected individuals recover spontaneously at rate $\mu(a)$ or are detected via observation or screening at rate $\sigma(a,t)$. The possibility of having waning immunity is depicted as a dashed line.

After being tracked, infected contacts receive medical care and start their recovery process. Therefore they are no longer considered infectious. Note that individuals recover even in the absence or failure of the contact tracing process (See Remark 4.2). In the next section, we explore the probability of recovery without contact tracing.

4.3. Probability of infection without contact tracing

Without contact tracing, individuals recover spontaneously at rate $\mu(a)$ or are detected and removed from the infected compartment at rate $\sigma(a,t)$. We are interested in the transition from the *infected* state to the *recovered* state in absence of contact tracing. Specifically, we want to calculate the probability for an individual who became infected at time t_0 to be infectious at the age of infection a and time $t = t_0 + a$.

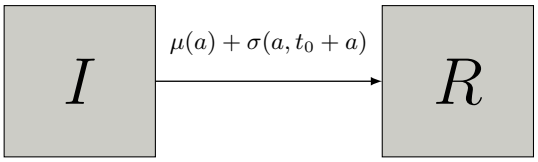


Figure 4.2. Compartmental view of the recovery process. Infected individuals recover spontaneously at rate $\mu(a)$ or are detected via observation or screening at rate $\sigma(a, t_0 + a)$. This figure zooms into the part of Figure 4.1 that is relevant for this section.

Proposition 4.1 (Probability of infection without contact tracing)**.** *The probability for an infected individual has an age of infection a, and was infected at time t_0 to recover after an interval h is:*

$$\hat{\kappa}(a;t_0) = \exp\left(-\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a}, t_0 + \tilde{a})\mathrm{d}\tilde{a}\right) \tag{4.4}$$

Proof. Because contact tracing is excluded, individuals are not traced ($p = 0$). We describe the state of health of an individual with the random variable I_{a,t_0}, defined as follows:

$$I_{a,t_0} = \begin{cases} 0 & \text{not infected (recovered)} \\ 1 & \text{infected} \end{cases} \tag{4.5}$$

We assume that the individual is infected at the age of infection a: $\hat{\kappa}(a;t_0) = 1$, and ask for the probability of recovery after h time units. From the system dynamics, we know that an infected individual can either recover or remain infected. The sum of the probability to recover and the probability to remain infected after h time units is equal to the probability of being infected at age of infection a as indicated in Eq. 4.6.

$$P(I_{a,t_0} = 1) = P(I_{a+h,t_0} = 1 | I_{a,t_0} = 1) + P(I_{a+h,t_0} = 0 | I_{a,t_0} = 1) \tag{4.6}$$

The recovery probability for an individual at the age of infection $a+h$, given that he/she was infectious at the age of infection a, is:

$$P(I_{a+h,t_0} = 0 | I_{a,t_0} = 1) = (\mu(a) + \sigma(a,t)) \cdot h \tag{4.7}$$

The condition on $P(I_{a+h,t_0} = 0 | I_{a,t_0} = 1)$ is removed using Theorem 2.1:

$$P(I_{a+h,t_0} = 0 | I_{a,t_0} = 1) = \frac{P(I_{a+h,t_0} = 0 \wedge I_{a,t_0} = 1)}{P(I_{a,t_0} = 1)} \tag{4.8}$$

Plugging Eq. 4.6 and Eq. 4.7 into Eq. 4.8, we get an expression in terms of the probability of infection.

$$\frac{P(I_{a+h,t_0} = 1) - P(I_{a,t_0} = 1)}{h} = -(\mu(a) + \sigma(a, t_0 + a)) \cdot P(I_{a,t_0} = 1) \tag{4.9}$$

We define $\hat{\kappa}(a;t_0) = P(I_{a,t_0} = 1)$ and take the limit when h tends to 0. Then the expression in Eq. 4.9 becomes an ordinary differential equation.

$$\frac{\mathrm{d}\hat{\kappa}(a;t_0)}{\mathrm{d}a} = -(\mu(a) + \sigma(a, t_0 + a)) \cdot \hat{\kappa}(a;t_0) \tag{4.10}$$

The derivative with respect to a shows how the probability of infection changes as the infected individuals move from one age of infection to the next one.

The solution to this equation can be easily calculated by integration.

$$\int_{\hat{\kappa}(0;t_0)}^{\hat{\kappa}(a;t_0)} \frac{\tilde{\mathrm{d}}\kappa(a;t_0)}{\tilde{\kappa}(a;t_0)} = -\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a}, t_0 + \tilde{a})\, \mathrm{d}\tilde{a} \tag{4.11}$$

$$\ln\left(\frac{\hat{\kappa}(a;t_0)}{\hat{\kappa}(0;t_0)}\right) = -\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a}, t_0 + \tilde{a})\, \mathrm{d}\tilde{a} \tag{4.12}$$

$$\hat{\kappa}(a;t_0) = \hat{\kappa}(0;t_0) \cdot \exp\left(-\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a}, t_0 + \tilde{a})\, \mathrm{d}\tilde{a}\right) \tag{4.13}$$

A person has age of infection zero when he/she becomes infected. At that point we know for sure that they are infected, therefore $\hat{\kappa}(0;t_0)) = 1$. Using this piece of information we complete the proof.

$$\hat{\kappa}(a;t_0) = \exp\left(-\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a}, t_0 + \tilde{a})\, \mathrm{d}\tilde{a}\right) \tag{4.14}$$

□

4.4. Reproduction number

4.4.1. Basic reproduction number

We are interested in the average number of secondary cases produced by one typical infected. Chapter 5 uses this figure in the formulation of a set of optimization problems that will be helpful to get some insight into the impact of periodic contact patterns over the effectiveness of contact tracing. We calculate the reproduction number using the next generation operator described in [52].

Eq. 4.15-4.16 describe the infectious compartment in a deterministic representation of the system, linearized in the onset of the epidemic, when most of the population is susceptible $S \approx N$.

$$(\partial t + \partial a) I(a,t) = -(\mu(a) + \sigma(a,t)) \cdot I(a,t) \tag{4.15}$$

$$I(0,t) = \int_0^\infty \beta(a,t) \cdot I(a,t) \mathrm{d}a \tag{4.16}$$

We aim to re-formulate the model as a renewal equation. As explained in [53], we follow a cohort along a characteristic line $a = t + \textit{constant}$ in the age-time space:

$$\frac{\mathrm{d}I(a+\tau, t+\tau)}{\mathrm{d}\tau} = I_a(a+\tau, t+\tau) + I_t(a+\tau, t+\tau) \tag{4.17}$$

$$\frac{\mathrm{d}I(a+\tau, t+\tau)}{\mathrm{d}\tau} = -(\mu(a+\tau) + \sigma(a+\tau, t+\tau)) \cdot I(a+\tau, t+\tau) \tag{4.18}$$

$$\int_{I(a,t)}^{I(a+\tau,t+\tau)} \frac{\mathrm{d}I(a+\tau,t+\tau)}{I(a+\tau,t+\tau)} = -\int_0^\tau \mu(a+\tau)+\sigma(a+\tau,t+\tau)\,\mathrm{d}\tau \tag{4.19}$$

$$\ln\left(\frac{I(a+\tau,t+\tau)}{I(a,t)}\right) = -\int_0^\tau \mu(a+\tau)+\sigma(a+\tau,t+\tau)\,\mathrm{d}\tau \tag{4.20}$$

$$I(a+\tau,t+\tau) = I(a,t)\cdot\exp\left(-\int_0^\tau \mu(a+\tau)+\sigma(a+\tau,t+\tau)\right)\mathrm{d}\tau \tag{4.21}$$

The latter implies that:

$$I(a,t) = I(a-\Delta,t-\Delta)\cdot\exp\left(-\int_{a-\Delta}^{a}\mu(\tilde{a})+\sigma(\tilde{a},t)\,\mathrm{d}a\right)j \tag{4.22}$$

If $t > a$ we can choose $\Delta = a$. We obtain:

$$I(a,t) = I(0,t-a)\cdot\exp\left(-\int_0^a \mu(\tilde{a})+\sigma(\tilde{a},t)\,\mathrm{d}\tilde{a}\right) \tag{4.23}$$

For $t < a$, Δ has to be t. We get:

$$I(a,t) = I(a-t,0)\cdot\exp\left(-\int_{a-t}^{a}\mu(\tilde{a})+\sigma(\tilde{a},t)\,\mathrm{d}\tilde{a}\right) \tag{4.24}$$

We plug Eq. 4.23 and 4.24 into Eq. 4.16:

$$I(0,t) = \int_0^\infty \beta(a,t)\cdot I(a,t)\mathrm{d}a \tag{4.25}$$

$$= \int_0^t \beta(a,t)\cdot I(a,t)\mathrm{d}a + \int_t^\infty \beta(a,t)\cdot I(a,t)\mathrm{d}a \tag{4.26}$$

$$\begin{aligned} I(0,t) = &\int_0^t \beta(a,t)\cdot I(0,t-a)\cdot\exp\left(-\int_0^a \mu(\tilde{a})+\sigma(\tilde{a},t)\,\mathrm{d}\tilde{a}\right)\mathrm{d}a \\ &+\int_t^\infty \beta(a,t)\cdot I(a-t,0)\cdot\exp\left(-\int_{a-t}^{a}\mu(\tilde{a})+\sigma(\tilde{a},t)\,\mathrm{d}\tilde{a}\right)\mathrm{d}a \end{aligned} \tag{4.27}$$

The exponential corresponds to our definition of $\hat{\kappa}(a;t_0)$ (See Proposition 4.1). Therefore, we can re-write the expression as:

$$\begin{aligned} I(0,t) = &\int_0^t \beta(a,t)\cdot I(0,t-a)\cdot\hat{\kappa}(a;t_0)\,\mathrm{d}a \\ &+\int_t^\infty \beta(a,t)\cdot I(a-t,0)\cdot\exp\left(-\int_{a-t}^{a}\mu(\tilde{a})+\sigma(\tilde{a},t)\,\mathrm{d}\tilde{a}\right)\mathrm{d}a \end{aligned} \tag{4.28}$$

Following the procedure in [11], we define $u(t) = I(0,t)$. Notice this is a linear periodic integral Voltera equation of the form:

$$u(t) = \int_0^t A(a,t) \cdot u(t-a)\,\mathrm{d}a + \bar{u}(t) \tag{4.29}$$

$A(a,t) \in \mathbb{R}_+ \times \mathbb{R}_+ \to \mathbb{R} = \beta(a,t) \cdot \hat{\kappa}(a;t_0)$ is the expected number of new infections that an individual, who was infected at $t-a$, will produce per unit of time. Asymptotically, $u(t) \approx \exp(\lambda^* \cdot t) \cdot \nu(t)$, with $\lambda \in \mathbb{R}$ and ν a non-negative, nonzero T-periodic function in $\mathbb{R}^2$. We define set of such functions as ε.

$$\nu(t) = \int_0^\infty \exp(-\lambda^* \cdot a) \cdot A(a,t) \cdot \nu(t-a)\,\mathrm{d}a \tag{4.30}$$

We are interested in an operator that maps one generation into the next one. We can formulate this proposition as an eigenvalue problem. Because R_0 is the spectral radius of this linear operator with $\lambda = 0$, it maps a non-negative nonzero vector $\omega \in \varepsilon$ in the following way:

$$\int_0^\infty A(a,t) \cdot \omega(t-a)\mathrm{d}a = R_0 \cdot \omega(t) \tag{4.31}$$

Replacing A by the expected number of new infections written in terms of t and a for the model without contact tracing, we find:

$$\int_0^\infty \beta(a,t) \cdot \hat{\kappa}(a;t-a) \cdot \omega(t-a)\,\mathrm{d}a = R_0 \cdot \omega(t) \tag{4.32}$$

Note the argument of ω is different on right- and left-hand side of the equation.
We reformulate the right-hand side of Eq. 4.32 considering $a = t - t_0$.

$$\begin{aligned}&\int_{-\infty}^t \beta(t-t_0,t) \cdot \hat{\kappa}(t-t_0;t_0) \cdot \omega(t_0)\,\mathrm{d}t_0 = \\ &\int_0^t \beta(t-t_0,t) \cdot \hat{\kappa}(t-t_0;t_0) \cdot \omega(t_0)\,\mathrm{d}t_0 + \int_{-\infty}^0 \beta(t-t_0,t) \cdot \hat{\kappa}(t-t_0;t_0) \cdot \omega(t_0)\,\mathrm{d}t_0\end{aligned} \tag{4.33}$$

We reformulate the integral from $-\infty$ to 0 as the infinite sum of integrals over different periods.

$$\begin{aligned}&\int_0^t \beta(t-t_0,t) \cdot \hat{\kappa}(t-t_0;t_0) \cdot \omega(t-t_0)\,\mathrm{d}t_0 \\ &\qquad + \sum_{n=0}^{\infty} \int_0^T \beta(t+(n+1)\cdot T - t_0,t) \cdot \hat{\kappa}(t+(n+1)\cdot T - t_0;t_0) \cdot \omega(t_0)\,\mathrm{d}t_0\end{aligned} \tag{4.34}$$

Splitting the second integral:

$$\begin{aligned}\int_0^t \beta(t-t_0,t)\cdot\hat{\kappa}(t-t_0;t_0)\cdot\omega(t-t_0)\,\mathrm{d}t_0 \\ + \int_0^t \sum_{n=0}^{\infty}\beta(t+(n+1)\cdot T-t_0,t)\cdot\hat{\kappa}(t+(n+1)\cdot T-t_0;t_0)\cdot\omega(t_0)\,\mathrm{d}t_0 \\ + \int_t^T \sum_{n=0}^{\infty}\beta(t+(n+1)\cdot T-t_0,t)\cdot\hat{\kappa}(t+(n+1)\cdot T-t_0;t_0)\cdot\omega(t_0)\,\mathrm{d}t_0 \end{aligned} \tag{4.35}$$

We define:

$$H(a,t,t_0)=\sum_{n=0}^{\infty}\beta(a+n\cdot T,t)\cdot\hat{\kappa}(a+n\cdot T;t_0) \tag{4.36}$$

$$\int_0^t H(t-t_0,t,t_0)\cdot\omega(t_0)\,\mathrm{d}t_0+\int_t^T H(t-t_0+T,t,t_0)\cdot\omega(t_0)\,\mathrm{d}t_0=R_0\cdot\omega(t) \tag{4.37}$$

Approximation: With time discretized as $t_i=\frac{(i)T}{N}$, for $i=0...N-1$.

$$\frac{T}{N}\left[\sum_{j=0}^{i-1}H(t_i-t_j,t_i,t_j)W_j+\sum_{j=i}^{N-1}H(t_i-t_j+T,t_i,t_j)W_j\right]=R_0\cdot W_i \tag{4.38}$$

In Chapter 5, we use the power iteration (See Section 2.4) to solve the eigenvalue problem in Eq. 4.38 and find R_0.

4.4.2. Effective reproduction number

The implementation of contact tracing allows us to identify and quarantine individuals exposed to the pathogen through infected persons. An isolated person does not transmit the pathogen further. As a consequence, the number of secondary cases produced by an average individual is reduced.

Proposition 4.2 (Effective reproduction number)**.** *The effective reproduction number for backward contact tracing (Eq. 4.39), forward contact tracing (Eq. 4.40) and full contact tracing (Eq. 4.41), are given by:*

$$\int_0^\infty \beta(a, t_0 + a) \cdot \kappa^-(a; t_0) \cdot \omega(t_0)\, \mathrm{d}a = R_\infty^- \cdot \omega(t_0 + a) \tag{4.39}$$

$$\int_0^\infty \beta(a, t_0 + a) \cdot \kappa_\infty^+(a; t_0) \cdot \omega(t_0)\, \mathrm{d}a = R_\infty^+ \cdot \omega(t_0 + a) \tag{4.40}$$

$$\int_0^\infty \beta(a, t_0 + a) \cdot \kappa_\infty(a; t_0) \cdot \omega(t_0)\, \mathrm{d}a = R_\infty \cdot \omega(t_0 + a) \tag{4.41}$$

4.5. Backward tracing with periodic patterns

The objective of backward tracing is to identify the infector of an infectee (index case). The infector becomes infected at time t_0. He/she infects the infectee at age of infection c. At the current time $t = t_0 + a$, the infectee, who has age of infection b is observed and interrogated with the hope of finding the infector. We define Δ as the interval where the tracing event might take place. Fig. 4.3 shows the timeline of infection for backward tracing.

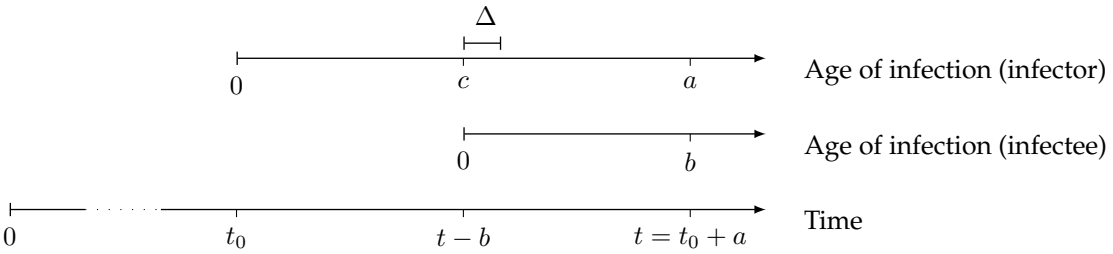


Figure 4.3. Infection timeline for backward tracing The infector is infected at time t_0. At time *t-b*, the infectee is infected by the infector, who has age of infection *c = a - b*. At the present time *t*, the infector and the infectee have age of infection a and b, respectively.

We are interested in the probability for the infector to still infectious at age of infection a, if he/she became infected at time t_0. If the infector is still infected and is found via contact tracing, he/she can be isolated to break the transmission chain.

Definition 4.2 (Probability of infection under backward tracing)**.**

$$\kappa^-(a; t_0) = P(\text{infectious at age of infection } a, \text{ if infected at time } t_0) \tag{4.42}$$

4.5.1. Recursive backward tracing

Proposition 4.3 (Probability of infection under recursive backward contact tracing)**.** *An infector that became infected at time t_0 is still infectious at the age of infection a, with a probability described by the following expression:*

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^{-}(a;t_0) = -\kappa^{-}(a;t_0)\left(\mu(a)+\sigma(a,t_0+a)+p\int_0^a \beta(a-b,t_0+a-b)\cdot \left(-\kappa^{-\prime}(b;t_0+a-b)-\kappa^{-}(b;t_0+a-b)\cdot\mu(b)\right)\mathrm{d}b\right) \tag{4.43}$$

with $\kappa^{-}(0)=1$.

Proof. As stated in Remark 4.2, the probability of infection for an individual with age of infection a who became infected at time t_0 decreases with spontaneous recovery, observed recovery, and identification via contact tracing.

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^{-}(a;t_0) = -\kappa^{-}(a;t_0)\,(\text{unobserved recovery} + \text{observed recovery} + \text{successful recursive contact tracing}) \tag{4.44}$$

Spontaneous recovery and observed recovery occur at rate $\mu(a)$ and $\sigma(a,t)$, respectively, with $t = a + t_0$. Thus, Eq. 4.44 becomes:

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^{-}(a;t_0) = -\kappa^{-}(a;t_0)\,(\mu(a)+\sigma(a,t_0+a) + \text{successful recursive contact tracing}) \tag{4.45}$$

For a backward contact tracing event to be triggered in a Δ interval, the infection event should exist, the infectee should be still infected, and he/she should be detected. The infection event occurs when the infector has age of infection $c = a - b$ at rate $\beta(a-b,t_0+a-b)$. The infectee is still infected at age of infection b with probability $\kappa^{-}(b;t_0+a-b)$, and he/she triggers a contact tracing cycle in the interval Δ with probability p. Lastly, the rate of observation is the removal rate minus the rate of unobserved recovery, as depicted in Eq. 4.46. The removal rate due to a successful recursive contact tracing event is the multiplication of the figures, as shown in Eq. 4.47.

$$\frac{-\kappa^{-}(b;t_0+a-b)'}{\kappa^{-}(b;t_0+a-b)} - \mu(b) \tag{4.46}$$

$$p\cdot\beta(a-b,t_0+a-b)\cdot\kappa^{-}(b;t_0+a-b)\cdot\left(\frac{-\kappa^{-\prime}(b;t_0+a-b)}{\kappa^{-}(b;t_0+a-b)}-\mu(b)\right)\cdot\Delta \tag{4.47}$$

This removal contribution can be calculated for all intervals Δ, integrating from 0 to a.

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\left(\mu(a)+\sigma(a,t_0+a)\right. \\ \left. +p\int_0^a \beta(a-b,t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\left(\frac{-\kappa^{-\prime}(b;t_0+a-b)}{\kappa^-(b;t_0+a-b)}-\mu(b)\right)\mathrm{d}b\right) \quad (4.48)$$

Finally, the term $\kappa^-(b;t_0+a-b)$ in Eq. 4.48 is multiplied with the subsequent term in parenthesis to obtain the expression in Proposition 4.3. □

Explicit calculation of κ^-

One can calculate κ^- explicitly from Eq. 4.43. First, Eq. 4.43 is divided by $\kappa^-(a;t_0)$.

$$\frac{\mathrm{d}\kappa^-(a,t_0)}{\kappa^-(a;t_0)} = -\Big[\mu(a)+\sigma(a,t_0+a)+p\int_0^a \beta(a-b,t_0+a-b)\cdot \\ \left(-\kappa^{-\prime}(b;t_0+\tilde{a}-b)-\kappa^-(b;t_0+a-b)\cdot\mu(b)\right)\mathrm{d}b\Big] \quad (4.49)$$

Then, both sides of the equation are integrated:

$$\int_{\kappa^-(0,t_0)}^{\kappa^-(a;t_0)}\frac{\mathrm{d}\tilde{\kappa}^-(a;t_0)}{\tilde{\kappa}^-(a;t_0)} = -\int_0^a\Big[\mu(\tilde{a})+\sigma(\tilde{a},t_0+\tilde{a})+p\int_0^{\tilde{a}}\beta(\tilde{a}-b,t_0+\tilde{a}-b)\cdot \\ \left(-\kappa^{-\prime}(b;t_0+\tilde{a}-b)-\kappa^-(b;t_0+\tilde{a}-b)\cdot\mu(b)\right)\mathrm{d}b\Big]\mathrm{d}\tilde{a} \quad (4.50)$$

$$\ln\left(\frac{\kappa^-(a;t_0)}{\kappa^-(0,t_0)}\right) = -\int_0^a\Big[\mu(\tilde{a})+\sigma(\tilde{a},t_0+\tilde{a})+p\int_0^{\tilde{a}}\beta(\tilde{a}-b,t_0+\tilde{a}-b)\cdot \\ \left(-\kappa^{-\prime}(b;t_0+\tilde{a}-b)-\kappa^-(b;t_0+\tilde{a}-b)\cdot\mu(b)\right)\mathrm{d}b\Big]\mathrm{d}\tilde{a} \quad (4.51)$$

Lastly, the exponential function is used on both sides of the equation, and the initial condition is inserted to obtain:

$$\kappa^-(a;t_0) = \exp\left(-\int_0^a\mu(\tilde{a})+\sigma(\tilde{a},t_0+\tilde{a})\,\mathrm{d}\tilde{a}\right)\cdot\exp\Big(-p\cdot\int_0^a\int_0^{\tilde{a}}\beta(\tilde{a}-b,t_0+\tilde{a}-b)\cdot \\ \left(-\kappa^{-\prime}(b;t_0+\tilde{a}-b)-\kappa^-(b;t_0+\tilde{a}-b)\cdot\mu(b)\right)\mathrm{d}b\,\mathrm{d}\tilde{a}\Big) \quad (4.52)$$

$$\kappa^-(a;t_0) = \hat{\kappa}(a;t_0)\cdot\exp\Big(-p\cdot\int_0^a\int_0^{\tilde{a}}\beta(\tilde{a}-b,t_0+\tilde{a}-b)\cdot \Big(-\kappa^{-\prime}(b;t_0+\tilde{a}-b)-\kappa^-(b;t_0+\tilde{a}-b)\cdot\mu(b)\Big)\,\mathrm{d}b\,\mathrm{d}\tilde{a}\Big) \tag{4.53}$$

Numerical calculation

For the numerical calculation of this probability, we are interested in the integrodifferential expression in Proposition 4.3.

To ease the computation, we remove the derivative from κ^- using integration by parts as shown in the following steps:

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\Big(\mu(a)+\sigma(a,t_0+a)+p\int_0^a\beta(a-b,t_0+a-b)\cdot \Big(-\kappa^{-\prime}(b;t_0+a-b)-\kappa^-(b;t_0+a-b)\cdot\mu(b)\Big)\,\mathrm{d}b\Big) \tag{4.54}$$

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\Big(\mu(a)+\sigma(a,t_0+a)-p\int_0^a\beta(a-b,t_0+a-b)\cdot \kappa^{-\prime}(b;t_0+a-b)\mathrm{d}b - p\int_0^a\beta(a-b,t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\cdot\mu(b)\mathrm{d}b\Big) \tag{4.55}$$

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\Big(\mu(a)+\sigma(a,t_0+a)-p\cdot\beta(a-b,t_0+a-b)\cdot \kappa^-(b;t_0+a-b)\big|_{b=0}^{b=a}+p\int_0^a\beta'(a-b,t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\mathrm{d}b -p\int_0^a\beta(a-b,t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\cdot\mu(b)\mathrm{d}b\Big) \tag{4.56}$$

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\Big(\mu(a)+\sigma(a,t_0+a)-p\cdot\beta(0,t_0)\cdot\kappa^-(a;t_0) +p\cdot\beta(a,t_0+a)\cdot\kappa^-(0;t_0+a) +p\int_0^a\kappa^-(b;t_0+a-b)\left[\beta'(a-b,t_0+a-b)-\beta(a-b,t_0+a-b)\cdot\mu(b)\right]\mathrm{d}b\Big) \tag{4.57}$$

The approximation of the integral is done using Newton-Cotes. For that purpose, we define:

$$\Theta(a, b, t_0) = \kappa^-(b; t_0 + a - b) \left[\beta'(a - b, t_0 + a - b) - \beta(a - b, t_0 + a - b) \cdot \mu(b)\right] \tag{4.58}$$

and obtain:

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a; t_0) \approx -\kappa^-(a; t_0)\Bigg(&\mu(a) + \sigma(a, t_0 + a) - p \cdot \beta(0, t_0) \cdot \kappa^-(a; t_0) \\ &+p \cdot \beta(a, t_0 + a) \cdot \kappa^-(0; t_0 + a) + p \cdot \sum_{k=0}^{m} \omega_k \cdot \Theta(a, b_k, t_0)\Bigg)\end{aligned} \tag{4.59}$$

where ω_k are the weights of Simpson's rule (See Section 2.2.2), $b_0 = 0$ and $b_m = a$.

With this formulation we proceed to solve the problem via an Implicit Runge-Kutta method (See Section 2.3).

Figure 4.4 shows the probability of infection $\kappa^-(a; t_0)$ under recursive backward contact tracing. We explore two cases. The first one (on the left) deals with a non-periodic scenario; thus, the parameters are constant. The second one has time-dependent variable parameters, which are T-periodic with $T = 7$. The contact rate ($\beta(a, t)$) is piece-wise defined and presented as a vector where each element represents the contact rate of a day of the week.

Table 4.1 shows the basic and effective reproduction number for each case.

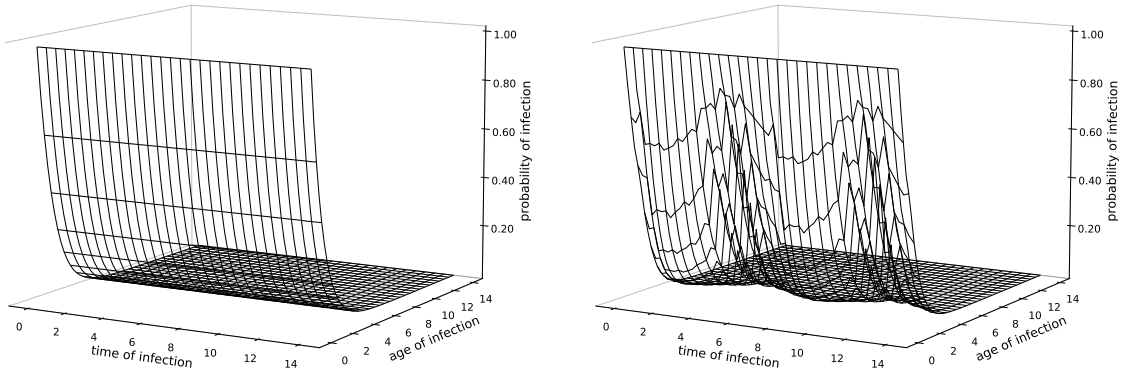
Table 4.1.: Basic and effective reproduction number for the example in Figure 4.4

Case	R_0	R_∞
non-periodic	3.5778	2.8949
periodic	14.0107	6.9054

The code for this simulation is available in [2].

4.5.2. Non-recursive backward tracing

Non-recursive contact tracing has only one tracing step. The process ends after the infector of a detected infectee is identified. In this setting, the infector can be found via backward contact tracing only if the infectee is detected via screening or observation. That occurs at rate ($\sigma(a - c, t) = \sigma(b, t_0 + a)$). Hence, the probability of infection for an individual with age of infection a who became infected at time t_0 is given by Proposition 4.4.



(a) **Probability of infection under recursive backward contact tracing with constant parameters** The parameter values are $p = 1/3$, $\beta(a,t) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, and $\sigma(a,t) = 0.5/\text{day}$

(b) **Probability of infection under recursive backward contact tracing with variable parameters** The parameter values are $p = 1/3$, $\beta(a) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, $\sigma(a) = 0.5/\text{day}$. $\beta(t) = [1, 3, 3.5, 4, 3, 2, 1]$, and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$. $\beta(t)$ and $\sigma(t)$ have period $T = 7\text{days}$.

Figure 4.4. Probability of infection $\kappa^{-}(a; t_0)$ under recursive backward contact tracing with constant (a) and variable (b) parameters.

Proposition 4.4 (Probability of infection under one-time backward contact tracing)**.** *An infector that became infected at time t_0 is still infectious at the age of infection a, with a probability described by the following expression:*

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\Bigg(\mu(a) + \sigma(a, t_0+a) + p\int_0^a \beta(a-b, t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\cdot\sigma(b,t_0+a)\,\mathrm{d}b\Bigg) \quad (4.60)$$

with $\kappa^-(0) = 1$.

Proof. The proof follows the same procedure as for Proposition 4.3. The difference is that because the process is non-recursive, the infectee can only be found via observation or screening.

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\,(\text{unobserved recovery} + \text{observed recovery} + \text{successful one-time contact tracing}) \quad (4.61)$$

Spontaneous recovery occurs at rate $\mu(a)$, while observed recovery occurs at rate $\sigma(a, t_0+a)$, so we obtain:

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\cdot(\mu(a) + \sigma(a,t_0+a) + \text{successful one-time contact tracing}) \quad (4.62)$$

The removal rate due to successful contact tracing is the multiplication of the occurrence rate of the infection event $\beta(a-b, t_0+a-b)$, the probability for the infectee to still infected $\kappa^-(b;t_0+a-b)$, the rate of detection $\sigma(b,t_0+a)$, and the probability for the infectee to trigger a contact tracing event in the interval Δ: p.

$$p\cdot\beta(a-b,t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\cdot\sigma(b,t_0+a)\cdot\Delta \quad (4.63)$$

This removal contribution can be calculated for all intervals Δ, by integrating from 0 to a.

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a;t_0) = -\kappa^-(a;t_0)\Bigg(\mu(a) + \sigma(a,t_0+a) + p\cdot\int_0^a \beta(a-b,t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\cdot\sigma(b,t_0+a)\mathrm{d}b\Bigg) \quad (4.64)$$

□

Explicit calculation of κ^-

We calculate κ^- explicitly from Eq. 4.60, following the same procedure as for the recursive version of backward contact tracing. The calculations are as follows:

$$\frac{\mathrm{d}\kappa^-(a,t_0)}{\kappa^-(a;t_0)} = -\Big[\mu(a) + \sigma(a, t_0+a) \\ +p\cdot\int_0^a \beta(a-b, t_0+a-b)\cdot\kappa^-(b;t_0+a-b)\cdot\sigma(b,t_0+a)\,\mathrm{d}b\Big] \quad (4.65)$$

Using integration on both sides of the equation:

$$\int_{\kappa^-(0,t_0)}^{\kappa^-(a;t_0)} \frac{\mathrm{d}\tilde{\kappa}^-(a;t_0)}{\tilde{\kappa}^-(a;t_0)} = -\int_0^a \Big[\mu(\tilde{a}) + \sigma(\tilde{a}, t_0+\tilde{a}) \\ +p\cdot\int_0^{\tilde{a}} \beta(\tilde{a}-b, t_0+\tilde{a}-b)\cdot\kappa^-(b;t_0+\tilde{a}-b)\cdot\sigma(b,t_0+\tilde{a})\,\mathrm{d}b\Big]\,\mathrm{d}\tilde{a} \quad (4.66)$$

$$\ln\left(\frac{\kappa^-(a;t_0)}{\kappa^-(0,t_0)}\right) = -\int_0^a \Big[\mu(\tilde{a}) + \sigma(\tilde{a}, t_0+\tilde{a}) \\ +p\cdot\int_0^{\tilde{a}} \beta(\tilde{a}-\tilde{b}, t_0+\tilde{a}-\tilde{b})\cdot\kappa^-(\tilde{b};t_0+\tilde{a}-\tilde{b})\cdot\sigma(\tilde{b},t_0+\tilde{a})\,\mathrm{d}\tilde{b}\Big]\,\mathrm{d}\tilde{a} \quad (4.67)$$

Lastly, we use the exponential function on both sides of the equation and the initial condition to obtain:

$$\kappa^-(a;t_0) = \exp\left(-\int_0^a \mu(\tilde{a}) + \sigma(\tilde{a}, t_0+\tilde{a})\,\mathrm{d}\tilde{a}\right)\cdot \\ \exp\left(-p\cdot\int_0^a\int_0^{\tilde{a}} \beta(\tilde{a}-\tilde{b}, t_0+\tilde{a}-\tilde{b})\cdot\kappa^-(\tilde{b};t_0+\tilde{a}-\tilde{b})\cdot\sigma(\tilde{b},t_0+\tilde{a})\,\mathrm{d}\tilde{b}\,\mathrm{d}\tilde{a}\right) \quad (4.68)$$

$$\kappa^-(a;t_0) = \hat{\kappa}(a;t_0)\cdot \\ \exp\left(-p\cdot\int_0^a\int_0^{\tilde{a}} \beta(\tilde{a}-\tilde{b}, t_0+\tilde{a}-\tilde{b})\cdot\kappa^-(\tilde{b};t_0+\tilde{a}-\tilde{b})\cdot\sigma(\tilde{b},t_0+\tilde{a})\,\mathrm{d}\tilde{b}\,\mathrm{d}\tilde{a}\right) \quad (4.69)$$

Numerical calculation

The computation of the expression in Eq. 4.4 uses Newton-Cotes. We define:

$$\Theta(a, b, t_0) = \beta(a - b, t_0 + a - b) \cdot \kappa^-(b; t_0 + a - b) \cdot \sigma(b, t_0 + a) \tag{4.70}$$

and obtain:

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa^-(a; t_0) \approx -\kappa^-(a; t_0) \left(\mu(a) + \sigma(a, t_0 + a) + p \cdot \sum_{k=0}^{m} \omega_k \cdot \Theta(a, b_k, t_0) \right) \tag{4.71}$$

where ω_k are the weights of Simpson's rule (See Section 2.2.2), $b_0 = 0$ and $b_m = a$.

This initial value problem is solved using an Implicit Runge-Kutta method described in Section 2.3.

Figure 4.5 shows the probability of infection $\kappa^-(a; t_0)$ under non-recursive backward contact tracing. The picture on the left depicts a scenario with constant parameters; thus, there is no periodicity. The picture on the right portrays a scenario with time-dependent variable parameters. These parameters are T-periodic with $T = 7$. The contact rate ($\beta(a, t)$) is piece-wise defined and presented as a vector where each element represents the contact rate of a day of the week.

Table 4.2 shows the basic and effective reproduction number for each case.

Table 4.2.: Basic and effective reproduction number for the example in Figure 4.5

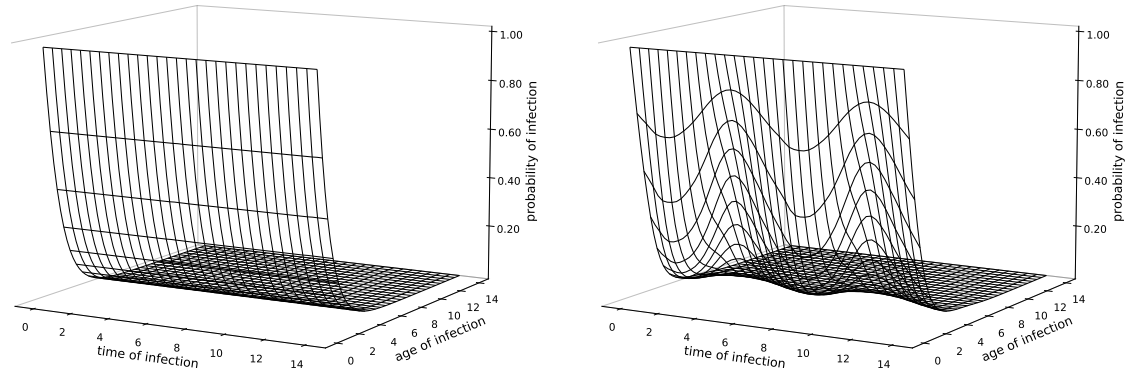
Case	R_0	R_∞
non-periodic	3.5778	3.0039
periodic	14.0107	9.5364

The code for this simulation is available in [2].

4.6. Forward tracing with periodic patterns

Forward contact tracing aims to identify newly infected individuals through their infectors. Consequently, only infectees from generation one and the next generations can be found via forward tracing. Tracing the primary infected individual is possible only through backward contact tracing.

Fig. 4.6 shows the timeline of an infection event. The infector becomes infected at time $t_0 - b$. At time t_0, when the focal individual (the infectee) becomes infected, the infector has age of infection b. At the current time $t = t_0 + a$, the infector and the infectee have age of infection $a + b$ and a, respectively. Because we do not know the exact moment the tracing event occurs, we use Δ to represent an interval where it might occur.



(a) **Probability of infection under non-recursive backward contact tracing with constant parameters** The parameter values are $p = 1/3$, $\beta(a,t) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, and $\sigma(a,t) = 0.5/\text{day}$

(b) **Probability of infection under non-recursive backward contact tracing with variable parameters** The parameter values are $p = 1/3$, $\beta(a) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, $\sigma(a) = 0.5/\text{day}$. $\beta(t) = [1, 3, 3.5, 4, 3, 2, 1]$, and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$. $\beta(t)$ and $\sigma(t)$ have period $T = 7\text{days}$.

Figure 4.5. Probability of infection $\kappa^-(a; t_0)$ under non-recursive backward contact tracing with constant (a) and variable (b) parameters.

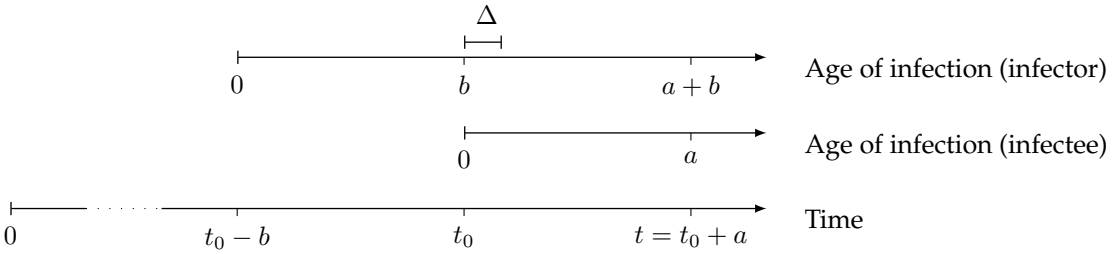


Figure 4.6. Infection timeline for forward tracing The infector is infected at time $t_0 - b$. At time t_0, the infectee is infected by the infector, who has age of infection b. At the present time $t = t_0 + a$, the infector and the infectee have age of infection $a + b$ and a, respectively.

We want to study the probability for an infectee of the generation $i > 0$, who was was infected at time t_0, of being infectious at age of infection a under forward contact tracing: $\kappa_i^+(a; t_0)$.

Definition 4.3 (Probability of infection under forward tracing). *For $i > 0$:*

$$\kappa_i^+(a; t_0) = P(\text{infectious at age of infection } a, \text{ if infected at time } t_0) \tag{4.72}$$

4.6.1. Recursive forward tracing

This section presents recursive forward contact tracing. In Definition 4.3 we introduced $\kappa_i^+(a; t_0)$ as the probability for the infectee of the generation i be infectious at age of infection a, given that he/she was infected at time t_0. If the infectee is still infectious when traced, he/she can prevent further infections through self-isolation. If the contact tracing process is recursive, the individual traced via forward contact tracing will become the index case and help identify infectees in the next generations.

The probability of infection of an infectee depends on the infector's age of infection at the infection event. The conditioned probability $\kappa_i^+(a; t_0|b)$ is given by Proposition 4.5.

Proposition 4.5 (Probability of infection under recursive forward contact tracing). *An infectee that became infected at time t_0 is still infectious at the age of infection a, with a probability described by the following expression:*

$$\kappa_i^+(a; t_0|b) = \hat{\kappa}(a; t_0)\left(1 - p \cdot \int_0^a \left(-\frac{{\kappa_{i-1}^+}'(\tilde{a} + b; t_0 - b)}{\kappa_{i-1}^+(\tilde{a} + b; t_0 - b)} - \mu(\tilde{a} + b)\right) \frac{\kappa_{i-1}^+(\tilde{a} + b; t_0 - b)}{\kappa_{i-1}^+(b; t_0 - b)} d\tilde{a}\right) \tag{4.73}$$

with $\kappa_i^+(0) = 1$.

Proof. An infectee of the generation i is still infectious if he has not recovered spontaneously, has not been observed, and has not been traced (See Remark 4.2). Thus, $\kappa_i^+(a; t_0|b)$ is the multiplication of the probability to be infectious in absence of contact tracing $\hat{\kappa}(a; t_0)$ (See Proposition 4.1), and the probability of unsuccessful contact tracing.

$$\kappa_i^+(a; t_0|b) = \hat{\kappa}(a; t_0)(1 - \text{successful recursive contact tracing}) \tag{4.74}$$

Let us calculate the probability of having a successful contact tracing event embedded in a recursive process. The tracing process starts when the infector, who should be still infectious, is found. The infector is infectious at time t_0 and age of infection b. The probability

that he/she is still contagious can be calculated as a conditional probability:

$$\kappa_{i-1}^{+}(a+b;t_0-b|b)=\frac{\kappa_{i-1}^{+}(a+b;t_0-b)}{\kappa_{i-1}^{+}(b;t_0-b)} \tag{4.75}$$

The infector is found if her/his recovery occurs in a non-spontaneous way. In other words, the infector is observed or traced. In Eq. 4.76 this probability is written as the recovery rate minus the spontaneous recovery rate.

$$\frac{-\kappa_{i-1}^{+}(a+b;t_0-b)'}{\kappa_{i-1}^{+}(a+b;t_0-b)}-\mu(a+b) \tag{4.76}$$

The probability for a successful contact tracing event in the interval Δ is the multiplication of the probability for the infector to be infectious at $t=t_0+a$ (Eq. 4.75), the probability for he/she being observed (Eq. 4.76) and the probability for the infectee to be traced via contact tracing in an interval Δ: p. As we mentioned before, except for the primary individual, every subject has a probability p of being traced via forward contact tracing.

$$p\cdot\left(-\frac{\kappa_{i-1}^{+}{}'(a+b;t_0-b)}{\kappa_{i-1}^{+}(a+b;t_0-b)}-\mu(a+b)\right)\frac{\kappa_{i-1}^{+}(a+b;t_0-b)}{\kappa_{i-1}^{+}(b;t_0-b)}\cdot\Delta \tag{4.77}$$

To account for this probability in all the Δ intervals, we integrate the previous expression from 0 to a.

$$\int_0^a p\cdot\left(-\frac{\kappa_{i-1}^{+}{}'(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^{+}(\tilde{a}+b;t_0-b)}-\mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}^{+}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^{+}(b;t_0-b)}\mathrm{d}\tilde{a} \tag{4.78}$$

Lastly, we plug this expression into Eq. 4.74 and we obtain Eq. 4.73.

□

Explicit calculation of κ_i^+

Corollary 4.1. *The probability for an infectee of the generation $i>0$ to be infectious at age of infection a, provided he/she was infected at t_0 is described by the following expression:*

$$\kappa_i^{+}(a;t_0)=\int_0^{\infty}\kappa_i^{+}(a;t_0|b)\cdot\varphi_{i-1}(b;t_0-b)\mathrm{d}b \tag{4.79}$$

with $\varphi_{i-1}(b;t_0-b)$ defined as:

$$\varphi_{i-1}(b;t_0-b)=\frac{\beta(b,t_0)\cdot\kappa_{i-1}^{+}(b;t_0-b)}{\int_0^{\infty}\beta(\tilde{b},t_0)\cdot\kappa_{i-1}^{+}(\tilde{b};t_0-\tilde{b})\,d\tilde{b}} \tag{4.80}$$

Proof. In the previous part, we calculated the probability of infection for an infectee in the i-th conditioned on the age of the infector at the infection event $\kappa_i^+(a;t_0|b)$. Here, we remove the condition to obtain $\kappa_i^+(a;t_0)$.

Let us define a random variable B_{i-1} that represents the age of the infector when he/she infects the infectee. We can compute $\kappa_i^+(a;t_0)$ as the expected value of the probability of infection given the value of B_{i-1}: $\kappa_i^+(a;t_0|B_{i-1})$

$$\kappa_i^+(a;t_0) = E(\kappa_i^+(a;t_0|B_{i-1})) \tag{4.81}$$

Using the definition of a conditional expectation (See 2.2), we calculate $\kappa_i^+(a;t_0)$ as the product of $\kappa_i^+(a;t_0|B_{i-1})$ and the probability distribution for the age of the infector $\varphi_{i-1}(b;t_0-b)$, integrated across all possible ages of infection (See Eq. 4.79).

This distribution of B_{i-1} is calculated as a normalized infection rate (See 4.80). The net infection rate at time t_0 is given by the multiplication of the rate at which the infectors (in the generation $i-1$) produced newly infected individuals (in the generation i): $\beta(b,t_0)$, and the probability of infection for an individual in the previous generation $\kappa_{i-1}^+(b;t_0-b)$. We normalized this expression dividing by:

$$\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}^+(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b} \tag{4.82}$$

□

Applying Corollary 4.1 to Eq. 4.73, we find:

$$\begin{aligned}\kappa_i^+(a;t_0) = \int_0^\infty \hat{\kappa}(a;t_0)\cdot \\ \left(1-p\cdot\int_0^a\left(-\frac{{\kappa_{i-1}^+}'(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^+(\tilde{a}+b;t_0-b)}-\mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}^+(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^+(b;t_0-b)}\,\mathrm{d}\tilde{a}\right)\cdot \\ \frac{\beta(b,t_0)\cdot\kappa_{i-1}^+(b;t_0-b)}{\int_0^\infty\beta(\tilde{b},t_0)\cdot\kappa_{i-1}^+(\tilde{b};t_0-\tilde{b})\,\mathrm{d}\tilde{b}}\,\mathrm{d}b\end{aligned} \tag{4.83}$$

$$\begin{aligned}\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0)\Bigg(\int_0^\infty\frac{\beta(b,t_0)\cdot\kappa_{i-1}^+(b;t_0-b)}{\int_0^\infty\beta(\tilde{b},t_0)\cdot\kappa_{i-1}^+(\tilde{b};t_0-\tilde{b})\,\mathrm{d}\tilde{b}}\,\mathrm{d}b \\ -p\int_0^\infty\int_0^a\left(-\frac{{\kappa_{i-1}^+}'(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^+(\tilde{a}+b;t_0-b)}-\mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}^+(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^+(b;t_0-b)}\cdot \\ \frac{\beta(b,t_0)\cdot\kappa_{i-1}^+(b;t_0-b)}{\int_0^\infty\beta(\tilde{b},t_0)\cdot\kappa_{i-1}^+(\tilde{b};t_0-\tilde{b})\,\mathrm{d}\tilde{b}}\,\mathrm{d}\tilde{a}\Bigg)\,\mathrm{d}b\end{aligned} \tag{4.84}$$

The integral in the denominator is constant in b; therefore, we can move it out of the integral in b and pre-calculate its value. In the equations that come next, the integral in the denominator is denoted as $d(t_0)$.

The numerator and the denominator in the first part of the expression are equal. Therefore we obtain:

$$\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0)\Bigg(1 - \frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\left(\int_0^a -{\kappa_{i-1}^+}'(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a} - \int_0^a \mu(\tilde{a}+b)\cdot\kappa_{i-1}^+(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\right)\mathrm{d}b\Bigg) \tag{4.85}$$

with

$$d(t_0) = \int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}^+(\tilde{b};t_0-\tilde{b})\,\mathrm{d}\tilde{b} \tag{4.86}$$

The first integral can be re-written as:

$$-\int_0^\infty \beta(b,t_0)\int_0^a {\kappa_{i-1}^+}'(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b \tag{4.87}$$

$$= -\int_0^\infty \beta(b,t_0)\int_b^{a+b} {\kappa_{i-1}^+}'(\tilde{a};t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b$$

$$= -\int_0^\infty \beta(b,t_0)\left(\kappa_{i-1}^+(a+b;t_0-b) - \kappa_{i-1}^+(b;t_0-b)\right)\mathrm{d}b \tag{4.88}$$

$$= -\int_a^\infty \beta(b-a,t_0)\cdot\kappa_{i-1}^+(b;t_0-b+a)\,\mathrm{d}b + \int_0^\infty \beta(b,t_0)\cdot\kappa_{i-1}^+(b;t_0-b)\,\mathrm{d}b \tag{4.89}$$

$$= -\int_a^\infty \beta(b-a,t_0)\cdot\kappa_{i-1}^+(b;t_0-b+a)\,\mathrm{d}b + d(t_0) \tag{4.90}$$

Thus:

$$\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0)\Bigg(1 - \frac{p}{d(t_0)}\Bigg\{d(t_0) - \int_a^\infty \beta(b-a,t_0)\cdot\kappa_{i-1}^+(b;t_0-b+a)\,\mathrm{d}b - \int_0^\infty \beta(b,t_0)\int_0^a \mu(\tilde{a}+b)\cdot\kappa_{i-1}^+(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b\Bigg\}\Bigg) \tag{4.91}$$

Finally we obtain:

$$\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0)\Bigg(1 - p + \frac{p}{d(t_0)}\int_a^\infty \beta(b-a,t_0)\cdot\kappa_{i-1}^+(b;t_0-b+a)\,\mathrm{d}b - \frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\int_0^a \mu(\tilde{a}+b)\cdot\kappa_{i-1}^+(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b\Bigg) \tag{4.92}$$

Numerical calculation

$\hat{\kappa}$ is a decreasing exponential function (See Proposition 4.1). The value of κ_i^+ is very similar to the value of $\hat{\kappa}$, only decreased due to the implementation of contact tracing. Let us rewrite the previous expression for κ_i^+ as the multiplication of $\hat{\kappa}$ and another function f_i^+.

$$\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0) \cdot f_i^+(a;t_0) \tag{4.93}$$

Thus:

$$\begin{aligned} f_i^+(a;t_0) = 1 - p + \frac{p}{d(t_0)} \int_a^\infty \beta(b-a,t_0) \cdot \hat{\kappa}(b;t_0-b+a) \cdot f_{i-1}^+(b;t_0-b+a)\,\mathrm{d}b \\ - \frac{p}{d(t_0)} \int_0^\infty \beta(b,t_0) \int_0^a \mu(\tilde{a}+b) \cdot \hat{\kappa}(\tilde{a}+b;t_0-b) \cdot f_{i-1}^+(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b \end{aligned} \tag{4.94}$$

Reformulating the problem as in Eq. 4.94, we are able to reduce the computational complexity of the calculation.

As the primary infected can only be traced via backward contact tracing, the probability of infection for generation 0 is characterized by κ^-. We write κ^- in terms of $\hat{\kappa}$ and another function f_0.

$$\kappa^- = \hat{\kappa}(a;t_0) \cdot f_0(a,t_0) \tag{4.95}$$

f_0 can be derived from the explicit calculation of κ^- in Eq. 4.53:

$$f_0(a;t_0) = \frac{\kappa^-(a;t_0)}{\hat{\kappa}(a;t_0)} \tag{4.96}$$

In the first iteration we have:

$$\kappa_1^+ \approx \hat{\kappa} \cdot f_1^+(a,t_0) \tag{4.97}$$

Subsequent iterations use f_{i-1}^+, as depicted in Eq. 4.94.

We calculate the expression in Eq. 4.94 using quadrature. Although Gauss-Laguerre quadrature (See Section 2.2.1) numerically evaluates integrals from 0 to ∞ (like the outer integral), the roots of the polynomial are not equidistant. Having equidistant evaluation points is desired because one can reuse them throughout the computations. Thus, we choose Newton-Cotes quadrature (See Section 2.2.2) to compute the integrals numerically.

In order to get unambiguous expressions, we define:

$$\Phi_1(b,a,t_0) = \beta(b-a,t_0) \cdot \hat{\kappa}(b;t_0-b+a) \cdot f_{i-1}^+(b;t_0-b+a) \tag{4.98}$$

$$\Theta(b,\tilde{a},t_0) = \mu(\tilde{a}+b) \cdot \hat{\kappa}(\tilde{a}+b;t_0-b) \cdot f_{i-1}^+(\tilde{a}+b;t_0-b) \tag{4.99}$$

$$\Phi_2(b, a, t_0) = \beta(b, t_0) \cdot \int_0^a \Theta(b, \tilde{a}, t_0)\, \mathrm{d}\tilde{a} \tag{4.100}$$

Considering that $\Phi_k(b, a, t_0), k \in \{1, 2\}$, decreases exponentially fast, we truncate the improper integrals in Eq. 4.114 to L, which is chosen sufficiently large. Hence, the approximations of the integrals in Eq. 4.94 are given by Eq 4.101, 4.102, and 4.103.

$$\int_a^\infty \Phi_1(b, a, t_0)\, \mathrm{d}b \approx \int_a^L \Phi_1(b, a, t_0)\, \mathrm{d}b \approx \sum_{i=0}^{n} \omega_i \cdot \Phi_1(b_i, a, t_0) \tag{4.101}$$

with the weights of the Simpson's Rule are denoted by ω_i, $b_0 = a$ and $b_n = L$.

$$\int_0^a \Theta(b, \tilde{a}, t_0)\mathrm{d}\tilde{a} \approx \sum_{k=0}^{m} \omega_k \cdot \Theta(b, \tilde{a}_k, t_0) \tag{4.102}$$

where ω_k are the weights of the Simpson's Rule, $b_0 = 0$ and $b_m = a$.

$$\int_0^\infty \Phi_2(b, a, t_0)\, \mathrm{d}b \approx \int_0^L \Phi_2(b, a, t_0)\, \mathrm{d}b \approx \sum_{j=0}^{l} \omega_j \cdot \Phi_2(b_j, a, t_0) \tag{4.103}$$

where ω_j are the weights of the Simpson's Rule, $b_0 = 0$ and $b_l = L$.
Similarly, to calculate d we define:

$$\Phi_d(\tilde{b}, t_0) = \beta(\tilde{b}, t_0) \cdot \hat{\kappa}(\tilde{b}; t_0 - \tilde{b}) \cdot f_{i-1}^{+}(\tilde{b}; t_0 - \tilde{b}) \tag{4.104}$$

and obtain the following approximation:

$$d(t_0) = \int_0^\infty \Phi_d(\tilde{b}, t_0)\mathrm{d}\tilde{b} \approx \int_0^L \Phi_d(\tilde{b}, t_0)\mathrm{d}\tilde{b} \approx \sum_{j=0}^{l} \Phi_d(\tilde{b}_j, t_0) \tag{4.105}$$

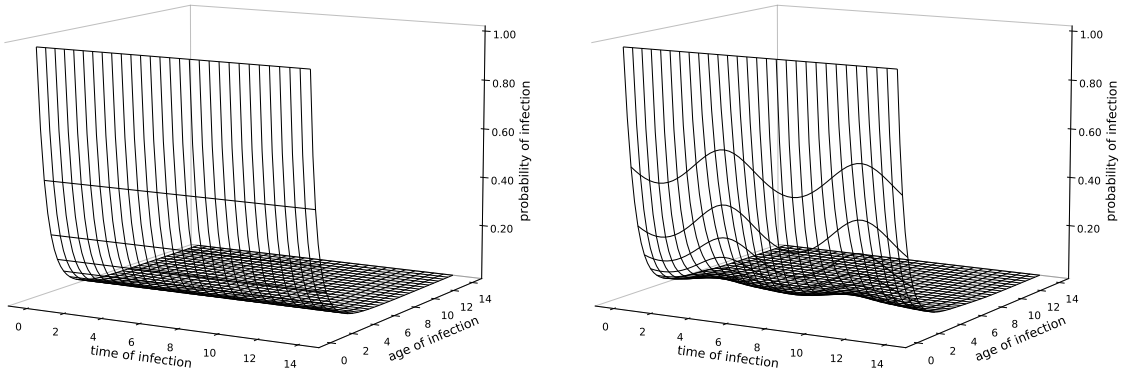
where ω_j are the weights of Simpson's Rule, $b_0 = 0$, $b_l = L$ and $l + 1$ points are used to calculate the approximations.

Putting these computations together, we obtain the following approximation for Eq. 4.94:

$$f_i^{+}(a; t_0) \approx 1 - p + \frac{p}{d(t_0)} \cdot \sum_{i=0}^{n} \omega_i \cdot \Phi_1(b_i, a, t_0) - \sum_{j=0}^{l} \omega_j \cdot \Phi_2(b_j, a, t_0) \tag{4.106}$$

with $d(t_0)$ calculated as in Eq. 4.105.

Figure 4.7 shows the probability of infection $\kappa_\infty^{+}(a; t_0)$ under recursive forward contact tracing. The picture on the left shows the case where all the parameters are constant, and there is no periodicity. The picture on the right exhibits $\kappa_\infty^{+}(a; t_0)$, with the time-dependent parameters being T-periodic with $T = 7$. The contact rate ($\beta(a, t)$) is piece-wise defined and presented as a vector where each element represents the contact rate of a day of the week.



(a) **Probability of infection under recursive forward contact tracing with constant parameters** The parameter values are $p = 1/3$, $\beta(a,t) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, and $\sigma(a,t) = 0.5/\text{day}$

(b) **Probability of infection under recursive forward contact tracing with variable parameters** The parameter values are $p = 1/3$, $\beta(a) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, $\sigma(a) = 0.5/\text{day}$. $\beta(t) = [1, 3, 3.5, 4, 3, 2, 1]$, and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$. $\beta(t)$ and $\sigma(t)$ have period $T = 7\text{days}$.

Figure 4.7. Probability of infection $\kappa_\infty^+(a; t_0)$ under recursive forward contact tracing with constant (a) and variable (b) parameters.

Table 4.3.: Basic and effective reproduction number for the example in Figure 4.7

Case	R_0	R_∞
non-periodic	3.5778	2.2201
periodic	14.0107	7.8217

Table 4.3 shows the basic and effective reproduction number for each case.
The code for this simulation is available in [2].

4.6.2. Non-recursive forward tracing

In Section 4.6.1, we explored the scenario where an infectee of the generation i, who was identified through individuals of the previous generations, becomes the index case and causes a new iteration in the tracing process. This section analyzes a forward contact tracing process that has only one step. The infector is detected either through a screening program or after the observation of symptoms. Once the infectees mentioned as contacts by the index case are identified, the process stops.

Proposition 4.6 (Probability of infection under one-time forward contact tracing)**.** *The probability of infection for an infectee who has age of infection a at time $t = t_0 + a$ and became infected at time t_0 is:*

$$\kappa_i^+(a; t_0|b) = \hat{\kappa}(a; t_0)\left(1 - p \cdot \int_0^a \sigma(\tilde{a} + b, t_0 + a) \cdot \frac{\kappa_{i-1}^+(\tilde{a} + b; t_0 - b)}{\kappa_{i-1}^+(b; t_0 - b)} d\tilde{a}\right) \tag{4.107}$$

with $\kappa_i^+(0) = 1$.

Proof. One-step forward contact tracing has only one index case: an infector, who has an observed recovery. One possibility is that he/she seeks medical attention, and the infectiousness becomes evident after the examination. Other is that a screening program spots the infector.

As for the recursive version of forward contact tracing, the conditioned probability of infection is the multiplication of $\hat{\kappa}(a; t_0)$ and the probability of having an unsuccessful contact tracing ($1 -$ successful recursive contact tracing). We obtain the following expression:

$$\kappa_i^+(a; t_0|b) = \hat{\kappa}(a; t_0)(1 - \text{successful non-recursive contact tracing}) \tag{4.108}$$

For the one-step contact tracing process to be successful, the infector should still be infectious at time $t = t_0 + a$ and be observed. The probability for the infector remain infectious was calculated before (See Eq. 4.75).

In the recursive scenario, the probability of being observed was calculated as the recovery hazard rate minus the spontaneous recovery rate (See Eq. 4.76). In the non-recursive case, this expression is equals to $\sigma(a+b, t_0+a)$ because the infector is not found via contact tracing. He/she can only be detected via screening or symptom evaluation.

$$\frac{-\kappa_{i-1}^{+}(a+b;t_0-b)'}{\kappa_{i-1}^{+}(a+b;t_0-b)} - \mu(a+b) = \sigma(a+b, t_0+a) \tag{4.109}$$

The probability for the infector to be removed from the infected compartment via contact tracing in an interval Δ is computed by multiplying the probability for the infector to be infectious at t (Eq. 4.75), the probability for he/she being observed (Eq. 4.109) and the probability for the infectee to be traced via contact tracing in an interval Δ: p.

$$p \cdot \sigma(a+b, t_0+a) \cdot \frac{\kappa_{i-1}^{+}(a+b;t_0-b)}{\kappa_{i-1}^{+}(b;t_0-b)} \cdot \Delta \tag{4.110}$$

Because the tracing event might take place in any Δ between 0 to a, we integrate over this interval. Finally we plug the integral into Eq. 4.108 and we obtain the expression in Eq. 4.107. □

Explicit calculation of κ_i^+

Corollary 4.1 also applies for the non-recursive version of forward contact tracing. The probability of infection κ_i^+ is calculated using Eq. 4.79, with $\kappa_i^+(a;t_0|b)$ as in Eq. 4.107:

$$\kappa_i^+(a;t_0) = \int_0^\infty \hat{\kappa}(a;t_0)\left(1 - p\int_0^a \sigma(\tilde{a}+b, t_0+\tilde{a}) \cdot \frac{\kappa_{i-1}^{+}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^{+}(b;t_0-b)}\,\mathrm{d}\tilde{a}\right) \cdot \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\,\mathrm{d}\tilde{b}}\,\mathrm{d}b \tag{4.111}$$

$$\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0)\left(\int_0^\infty \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}} - p\int_0^a \sigma(\tilde{a}+b, t_0+\tilde{a}) \cdot \frac{\kappa_{i-1}^{+}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}^{+}(b;t_0-b)} \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\,\mathrm{d}\tilde{a}\,\mathrm{d}b\right) \tag{4.112}$$

Finally we obtain:

$$\kappa_i^+(a;t_0) = \hat{\kappa}(a;t_0)\left(1 - \frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\int_0^a \sigma(\tilde{a}+b, t_0+\tilde{a})\cdot\kappa_{i-1}^{+}(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b\right) \tag{4.113}$$

with $d(t_0)$ defined as in Eq. 4.86

Numerical Calculation

We follow the same ideas as for the recursive version of forward contact tracing. The mathematical development is as follows:

$$f_i^+(a;t_0) = 1 - \frac{p}{d(t_0)} \cdot \int_0^\infty \beta(b,t_0) \int_0^a \sigma(\tilde{a}+b, t_0+\tilde{a}) \cdot \hat{\kappa}(\tilde{a}+b; t_0-b) \cdot f_{i-1}^+(\tilde{a}+b; t_0-b) \mathrm{d}\tilde{a}\, \mathrm{d}b \tag{4.114}$$

with $d(t_0)$ calculated in the same way as in Eq. 4.105.
For generation zero, we have:

$$f_0(a;t_0) = \frac{\kappa^-(a;t_0)}{\hat{\kappa}(a;t_0)} \tag{4.115}$$

We calculate the expression in Eq. 4.114 using Newton-Cotes (See Section 2.2.2). For the sake of clarity, we define $\Theta(b,\tilde{a},t_0)$ as:

$$\Theta(b,\tilde{a},t_0) = \sigma(\tilde{a}+b, t_0+\tilde{a}) \cdot \hat{\kappa}(\tilde{a}+b; t_0-b) \cdot f_{i-1}^+(\tilde{a}+b; t_0-b) \tag{4.116}$$

and $\Phi(b,a,t_0)$ as:

$$\Phi(b,a,t_0) = \beta(b,t_0) \cdot \int_0^a \Theta(b,\tilde{a},t_0) \mathrm{d}\tilde{a} \tag{4.117}$$

The approximation of the integral in the previous expression is:

$$\int_0^a \Theta(b,\tilde{a},t_0) \mathrm{d}\tilde{a} \approx \sum_{k=0}^{m} \omega_k \cdot \Theta(\tilde{a}_k, b, t_0) \tag{4.118}$$

where ω_k are the weights of Simpson's rule (See Section 2.2.2), $\tilde{a}_0 = 0$ and $\tilde{a}_m = a$.
Considering that $\Phi(b,a,t_0)$ decreases exponentially fast, we truncate the outer integral in Eq. 4.114 to L, which is chosen sufficiently large. The approximation is given by:

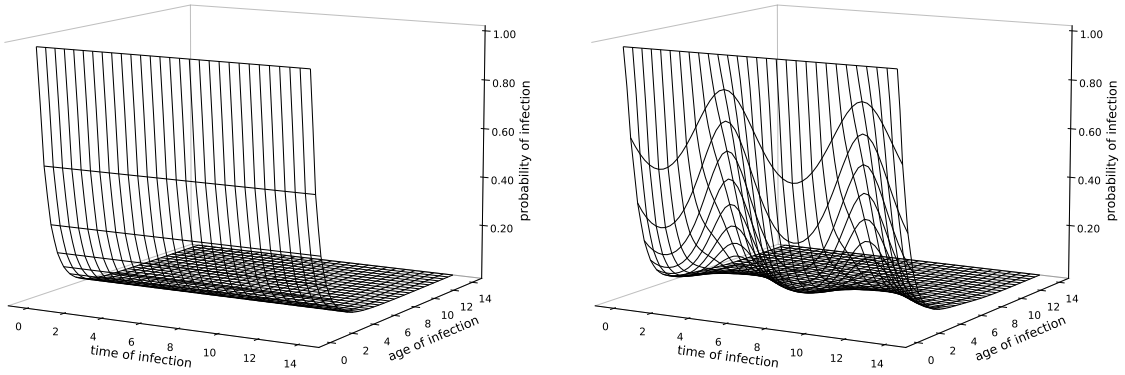
$$\int_0^\infty \Phi(b,a,t_0)\, \mathrm{d}b \approx \int_0^L \Phi(b,a,t_0)\, \mathrm{d}b \approx \sum_{j=0}^{l} \omega_j \cdot \Phi(b_j, a, t_0) \tag{4.119}$$

where ω_j are the weights of Simpson's rule, $b_0 = 0$ and $b_l = L$.
Finally, we obtain the approximation for Eq. 4.114:

$$f_i^+(a;t_0) \approx 1 - \frac{p}{d(t_0)} \cdot \sum_{j=0}^{l} \omega_j \cdot \Phi(b_j, a, t_0) \tag{4.120}$$

The evaluation of $d(t_0)$ is done as depicted in Eq. 4.105.
Figure 4.8 shows the probability of infection $\kappa_\infty^+(a;t_0)$ under non-recursive forward contact tracing. We show two cases. In the first scenario, all the parameters are constant, and

there is no periodicity (on the left). In the second one, the time-dependent parameters are T-periodic with $T = 7$ (on the right). The contact rate ($\beta(a,t)$) is piece-wise defined and presented as a vector where each element represents the contact rate of a day of the week.



(a) **Probability of infection under non-recursive forward contact tracing with constant parameters** The parameter values are $p = 1/3$, $\beta(a,t) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, and $\sigma(a,t) = 0.5/\text{day}$

(b) **Probability of infection under non-recursive forward contact tracing with variable parameters** The parameter values are $p = 1/3$, $\beta(a) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, $\sigma(a) = 0.5/\text{day}$. $\beta(t) = [1, 3, 3.5, 4, 3, 2, 1]$, and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$. $\beta(t)$ and $\sigma(t)$ have period $T = 7\text{days}$.

Figure 4.8. Probability of infection $\kappa_\infty^+(a; t_0)$ under non-recursive forward contact tracing with constant (a) and variable (b) parameters.

Table 4.4 shows the basic and effective reproduction number for each case.

Table 4.4.: Basic and effective reproduction number for the example in Figure 4.8

Case	R_0	R_∞
non-periodic	3.5778	2.5836
periodic	14.0107	11.1302

The code for this simulation is available in [2].

4.7. Full tracing with periodic patterns

Full tracing is the simultaneous implementation of backward and forward tracing. It aims to trace all contacts of the index case (including the infectees and the infector), which is only possible for generation 1 and the ones that come later. The primary infected can only be traced via backward tracing.

We are interested in the probability of infection for an individual of the generation $i > 0$ who has age of infection a and became infected at time t_0 under full contact tracing: $\kappa_i(a; t_0)$.

Definition 4.4 (Probability of infection under full tracing). *For $i > 0$:*

$$\kappa_i(a; t_0) = P(\text{infectious at age of infection } a, \text{ if infected at time } t_0) \tag{4.121}$$

4.7.1. Recursive full tracing

This section focuses on the case where the contacts of the index case who are traced via full tracing trigger another cycle of contact tracing, becoming the index case for those new iterations.

The probability of infection under full tracing $\kappa_i(a; t_0)$ depends on the generation in the same way as forward tracing. Infectees can be traced via their infector if they belong to a generation $i > 0$. Thus, we condition this probability on the age of infection of the infector: $\kappa_i(a; t_0|b)$.

Proposition 4.7 (Probability of infection under recursive full contact tracing). *An individual who became infected at time t_0 is still infected at age of infection a with probability:*

$$\kappa_i(a; t_0|b) = \kappa^-(a; t_0) \cdot \left(1 - p \cdot \int_0^a \left(-\frac{{\kappa_{i-1}}'(\tilde{a} + b; t_0 - b)}{\kappa_{i-1}(\tilde{a} + b; t_0 - b)} - \mu(\tilde{a} + b)\right) \frac{\kappa_{i-1}(\tilde{a} + b; t_0 - b)}{\kappa_{i-1}(b; t_0 - b)} d\tilde{a}\right) \tag{4.122}$$

with $\kappa_i(0) = 1$.

Proof. The probability of infection under full contact tracing decreases with spontaneous recovery, detection, tracing via backward tracing and tracing via forward tracing. In Section 4.5 we calculated $\kappa^-(a; t_0)$, which describe the probability of infection considering spontaneous recovery, detection and tracing via recursive backward tracing (See Eq. 4.53). We only need to multiply $\kappa^-(a; t_0)$ by the probability of unsuccessful recursive forward contact tracing conditioned on the age of infection of the infector:

$$\kappa_i(a; t_0|b) = \kappa^-(a; t_0)(1 - \text{successful recursive forward contact tracing } |b) \tag{4.123}$$

Replacing the probability of unsuccessful recursive forward contact tracing conditioned on the age of infection of the infector (See Eq. 4.73), we complete the proof. □

Explicit calculation of κ_i

Corollary 4.2. *The probability for an infectee of the generation $i > 0$ to be infectious at age of infection a, provided he/she was infected at t_0 is described by the following expression:*

$$\kappa_i(a;t_0) = \int_0^\infty \kappa_i(a;t_0|b) \cdot \varphi_{i-1}(b;t_0-b)\mathrm{d}b \tag{4.124}$$

with $\varphi_{i-1}(b;t_0-b)$ defined as:

$$\varphi_{i-1}(b;t_0-b) = \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})d\tilde{b}} \tag{4.125}$$

Proof. This proof follows the procedure of the proof for Corollary 4.1. We want to remove the condition of $\kappa_i(a;t_0|b)$ to obtain $\kappa_i(a;t_0)$. To do so, we define a random variable B_{i-1} that represents the age of the infector at the infection event.

$$\kappa_i(a;t_0) = E(\kappa_i(a;t_0|B_{i-1})) \tag{4.126}$$

This expression is equivalent to the product of $\kappa_i(a;t_0|B_{i-1})$ and the probability distribution for the age of the infector $\varphi_{i-1}(b;t_0-b)$, integrated across all possible ages of infection (See Eq. 4.124).

Here too, the probability distribution of B_{i-1} is calculated as a normalized infection rate, thus we divide the net infection rate $\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)$ by:

$$\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b} \tag{4.127}$$

□

The probability of infection under full contact tracing, calculated using Eq. 4.122 and Corollary 4.2 is:

$$\begin{aligned}\kappa_i(a;t_0) = \int_0^\infty \kappa^-(a;t_0)\cdot \\ \left(1-p\cdot\int_0^a\left(-\frac{{\kappa_{i-1}}'(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(\tilde{a}+b;t_0-b)}-\mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(b;t_0-b)}\mathrm{d}\tilde{a}\right)\cdot \\ \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty\beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\mathrm{d}b\end{aligned} \tag{4.128}$$

$$\kappa_i(a;t_0) = \kappa^-(a;t_0)\Bigg(\int_0^\infty \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\mathrm{d}b - p\int_0^\infty \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\cdot \int_0^a \left(-\frac{{\kappa_{i-1}}'(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(\tilde{a}+b;t_0-b)} - \mu(\tilde{a}+b)\right)\frac{\kappa_{i-1}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(b;t_0-b)}\mathrm{d}\tilde{a}\Bigg)\,\mathrm{d}b \quad (4.129)$$

$$\kappa_i(a;t_0) = \kappa^-(a;t_0)\cdot \left(1 - \frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\cdot\int_0^a -{\kappa_{i-1}}'(\tilde{a}+b;t_0-b) - \mu(\tilde{a}+b)\cdot\kappa_{i-1}(\tilde{a}+b;t_0-b)\mathrm{d}\tilde{a}\right)\mathrm{d}b \quad (4.130)$$

where $d(t_0)$ is given by Eq. 4.86.

We re-write the first integral in the same way as we did for the recursive version of forward contact tracing. Thus, we obtain:

$$\kappa_i(a;t_0) = \kappa^-(a;t_0)\Bigg(1 - p + \frac{p}{d(t_0)}\int_a^\infty \beta(b-a,t_0)\cdot\kappa_{i-1}(b;t_0-b+a)\,\mathrm{d}b - \frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\int_0^a \mu(\tilde{a}+b)\cdot\kappa_{i-1}(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b\Bigg) \quad (4.131)$$

Numerical calculation

We follow the procedure implemented in the previous sections and re-write κ_i and κ^- as the multiplication of $\hat{\kappa}$ and a function f_i.

$$\kappa_i(a;t_0) = \hat{\kappa}(a;t_0)\cdot f_i(a;t_0) \quad (4.132)$$

$$\kappa^-(a;t_0) = \hat{\kappa}(a;t_0)\cdot f_0(a;t_0) \quad (4.133)$$

Let us remember that the generation 0 can only be traced via backward contact tracing, therefore we denote the function that multiplies $\hat{\kappa}$, f_0 in Eq.4.133. Because κ^- does not depend on the generation, f_0 remains the same for all the generations traced under full contact tracing.

$$f_i(a;t_0) = f_0(a;t_0)\Bigg(1-p+\frac{p}{d(t_0)}\int_a^\infty \beta(b-a,t_0)\cdot\hat{\kappa}(b;t_0-b+a)\cdot f_{i-1}(b;t_0-b+a)\,\mathrm{d}b$$
$$-\frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\int_0^a \mu(\tilde{a}+b)\cdot\hat{\kappa}(\tilde{a}+b;t_0-b)\cdot f_{i-1}(\tilde{a}+b;t_0-b)\,\mathrm{d}\tilde{a}\,\mathrm{d}b\Bigg) \quad (4.134)$$

We compute the expression in Eq. 4.134 using Newton-Cotes (See Section 2.2.2). Because $f_i(a;t_0) = f_0(a;t_0)\cdot f_i^+(a;t_0)$, we recall the definitions (Eq. 4.86, 4.98, 4.99,4.100) and approximations (Eq. 4.101, 4.102, 4.103 4.105) in the forward contact tracing section to obtain the final expression for f_i:

$$f_i(a;t_0) \approx f_0(a;t_0)\left(1-p+\frac{p}{d(t_0)}\cdot\sum_{i=0}^{n}\omega_i\cdot\Phi_1(b_i,a,t_0)-\sum_{j=0}^{l}\omega_j\cdot\Phi_2(b_j,a,t_0)\right) \quad (4.135)$$

Figure 4.9 shows the probability of infection $\kappa_\infty^+(a;t_0)$ under recursive full contact tracing. We show two cases. In the first scenario, all the parameters are constant, and there is no periodicity (on the left). In the second one, the time-dependent parameters are T-periodic with $T = 7$ (on the right). The contact rate ($\beta(a,t)$) is piece-wise defined and presented as a vector where each element represents the contact rate of a day of the week.

Table 4.5 shows the basic and effective reproduction number for each case.

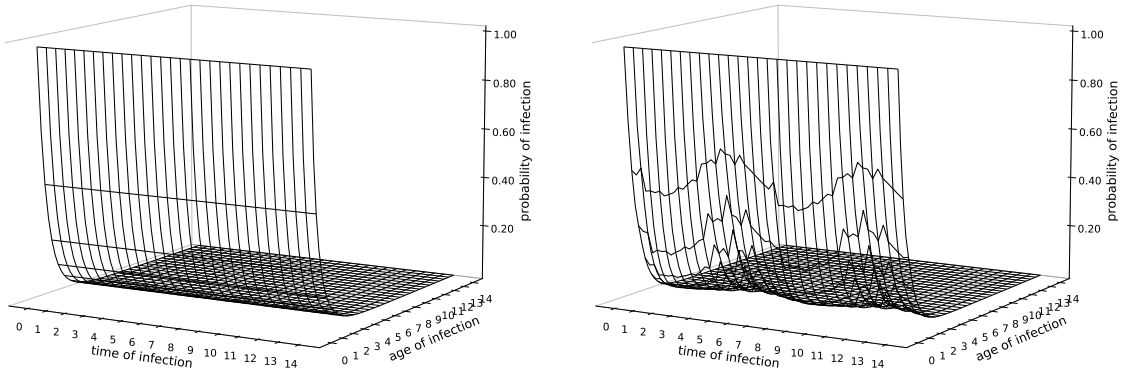
Table 4.5.: Basic and effective reproduction number for the example in Figure 4.9

Case	R_0	R_∞
non-periodic	3.5778	1.8683
periodic	14.0107	4.5302

The code for this simulation is available in [2].

4.7.2. Non-recursive full tracing

This section discusses the contact tracing process that is capable of finding the infectees and the infector of an index case in the generation $i > 0$, but only has one step. The index case is found via observation and inquired about his/her relevant contact with other individuals. The newly identified infected individuals do not trigger a new iteration of the process.



(a) **Probability of infection under recursive full contact tracing with constant parameters** The parameter values are $p = 1/3$, $\beta(a,t) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, and $\sigma(a,t) = 0.5/\text{day}$

(b) **Probability of infection under recursive full contact tracing with variable parameters** The parameter values are $p = 1/3$, $\beta(a) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, $\sigma(a) = 0.5/\text{day}$. $\beta(t) = [1, 3, 3.5, 4, 3, 2, 1]$, and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$. $\beta(t)$ and $\sigma(t)$ have period $T = 7\text{days}$.

Figure 4.9. Probability of infection $\kappa^{+}_{\infty}(a; t_0)$ under recursive full contact tracing with constant (a) and variable (b) parameters.

Proposition 4.8 (Probability of infection under one-time full contact tracing). *An individual of the generation $i > 0$, who became infected at time t_0, is infected at age of infection a with probability:*

$$\kappa_i(a;t_0|b) = \kappa^-(a;t_0)\left(1 - p\cdot\int_0^a \sigma(\tilde{a}+b, t_0+a)\cdot\frac{\kappa_{i-1}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(b;t_0-b)}d\tilde{a}\right) \tag{4.136}$$

with $\kappa_i(0) = 1$.

Proof. We develop this proof similarly as for recursive full contact tracing. The probability of infection under one-time full contact tracing can be calculated as the multiplication of probability of infection under one-time backward contact tracing (Eq. 4.69) and the probability of unsuccessful one-time forward contact tracing conditioned on the age of infection of the infector:

$$\kappa_i(a;t_0|b) = \kappa^-(a;t_0)(1 - \text{successful one-time forward contact tracing}|b) \tag{4.137}$$

Replacing the probability of unsuccessful one-time forward contact tracing conditioned on the age of infection of the infector from Eq. 4.107, we complete the proof. □

Explicit calculation of κ_i

The probability of infection κ_i for the non-recursive version of full contact tracing is calculated explicitly using Corollary 4.2. In this case $\kappa_i(a;t_0|b)$ is replaced by the Eq. 4.136. Hence, we obtain:

$$\kappa_i(a;t_0) = \int_0^\infty \kappa^-(a;t_0)\left(1 - p\cdot\int_0^a \sigma(\tilde{a}+b, t_0+a)\cdot\frac{\kappa_{i-1}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(b;t_0-b)}d\tilde{a}\right) \cdot\frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\mathrm{d}b \tag{4.138}$$

$$\begin{aligned}\kappa_i(a;t_0) = \kappa^-(a;t_0)\Bigg(&\int_0^\infty \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\mathrm{d}b \\ &-p\int_0^\infty \frac{\beta(b,t_0)\cdot\kappa_{i-1}(b;t_0-b)}{\int_0^\infty \beta(\tilde{b},t_0)\cdot\kappa_{i-1}(\tilde{b};t_0-\tilde{b})\mathrm{d}\tilde{b}}\cdot\int_0^a \sigma(\tilde{a}+b,t_0+a)\cdot \\ &\frac{\kappa_{i-1}(\tilde{a}+b;t_0-b)}{\kappa_{i-1}(b;t_0-b)}d\tilde{a}\Bigg)\,\mathrm{d}b\end{aligned} \tag{4.139}$$

$$\kappa_i(a;t_0) = \kappa^-(a;t_0)\left(1 - p\int_0^\infty \frac{\beta(b,t_0)}{d(t_0)} \cdot \int_0^a \sigma(\tilde{a}+b, t_0+a)\cdot \kappa_{i-1}(\tilde{a}+b; t_0-b)d\tilde{a}\right)\mathrm{d}b \tag{4.140}$$

with $d(t_0)$ given by Eq. 4.105.

Numerical calculation

We re-write Eq. 4.140 using the equivalences in Eq. 4.132 and 4.133:

$$f_i(a;t_0) = f_0(a;t_0)\Bigg(1 - \frac{p}{d(t_0)}\int_0^\infty \beta(b,t_0)\cdot\int_0^a \sigma(\tilde{a}+b,t_0+a)\cdot \hat{\kappa}(\tilde{a}+b;t_0-b)\cdot f_{i-1}(\tilde{a}+b;t_0-b)d\tilde{a}\Bigg)\mathrm{d}b \tag{4.141}$$

Because $f_i(a;t_0) = f_0(a;t_0)\cdot f_i^+(a;t_0)$, we approximate Eq. 4.141 using Eq. 4.120.

$$f_i(a;t_0) \approx f_0(a;t_0)\left(1 - \frac{p}{d(t_0)}\cdot\sum_{j=0}^{l}\omega_j\cdot\Phi(b_j,a,t_0)\right) \tag{4.142}$$

Figure 4.10 shows the probability of infection $\kappa_\infty^+(a;t_0)$ under non-recursive full contact tracing. We show two cases. In the first scenario, all the parameters are constant, and there is no periodicity (on the left). In the second one, the time-dependent parameters are T-periodic with $T = 7$ (on the right). The contact rate ($\beta(a,t)$) is piece-wise defined and presented as a vector where each element represents the contact rate of a day of the week.

Table 4.6 shows the basic and effective reproduction number for each case.

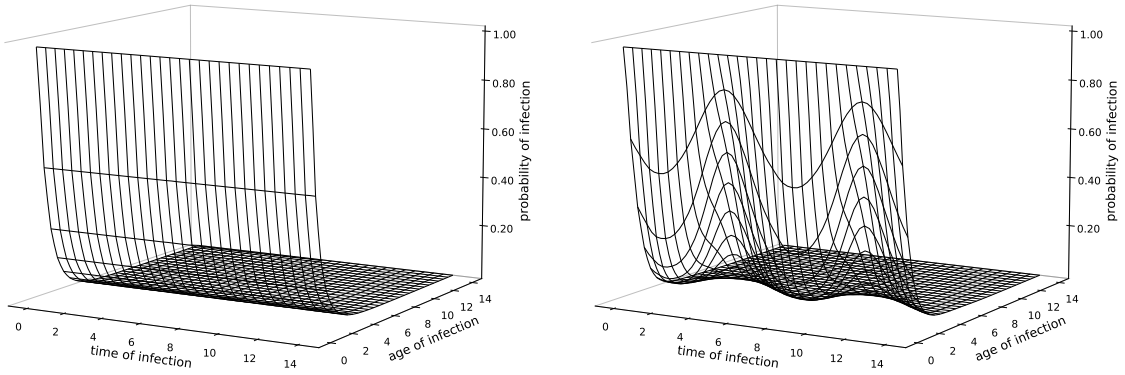
Table 4.6.: Basic and effective reproduction number for the example in Figure 4.10

Case	R_0	R_∞
non-periodic	3.5778	2.2418
periodic	14.0107	7.8456

The code for this simulation is available in [2].

4.8. Periodicity of the probability of infection

In Chapter 5 we propose a set of optimization problems to get some insight on how periodic contact patterns affect the effectiveness of contact tracing and solve them using the model for backward contact tracing described in Eq. 4.60. In preparation for that, this section explores the periodicity of the probability of infection under backward contact tracing.



(a) **Probability of infection under non-recursive full contact tracing with constant parameters** The parameter values are $p = 1/3$, $\beta(a,t) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, and $\sigma(a,t) = 0.5/\text{day}$

(b) **Probability of infection under non-recursive full contact tracing with variable parameters** The parameter values are $p = 1/3$, $\beta(a) = 1.0/\text{day}$, $\mu(a) = 0.3/\text{day}$, $\sigma(a) = 0.5/\text{day}$. $\beta(t) = [1, 3, 3.5, 4, 3, 2, 1]$, and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$. $\beta(t)$ and $\sigma(t)$ have period $T = 7\text{days}$.

Figure 4.10. Probability of infection $\kappa^{+}_{\infty}(a; t_0)$ under non-recursive full contact tracing with constant (a) and variable (b) parameters.

4.8.1. Periodicity in backward tracing

Lemma 4.1. *Two different persons who became infected at time t_0 and $t_0 + T$, respectively, have the same probability to be infected at age of infection a:*

$$\kappa^-(a; t_0 + T) = \kappa^-(a; t_0) \tag{4.143}$$

where $T > 0$

Proof. We prove the lemma using the equations for recursive backward tracing, as they are more complex than the equations for non-recursive backward contact tracing. However, the proof for non-recursive backward tracing follows the same procedure and leads to the same conclusion.

We want to know the difference between the probability of infection for two different individuals who got infected T units of time apart, with $T > 0$. The first individual is infected at t_0. At time $t = t_0 + a$ his/her probability of infection ($\kappa^-(a; t_0)$) is described by Eq. 4.43. The second individual is infected at time $t_0 + T$. At time $t = t_0 + T + a$ his probability of infection ($\kappa^-(a; t_0 + T)$) is described by:

$$\frac{\mathrm{d}\kappa^-(a; t_0 + T)}{\mathrm{d}a} = -\kappa^-(a; t_0 + T)\Bigg(\mu(a) + \sigma(a, t_0 + a + T) + p\int_0^a \beta(a - \tilde{b}, t_0 + a - \tilde{b} + T) \cdot \Big(-\kappa^{-\prime}(\tilde{b}; t_0 + a - \tilde{b} + T) - \kappa^-(\tilde{b}; t_0 + a - \tilde{b} + T) \cdot \mu(b)\Big)\,\mathrm{d}\tilde{b}\Bigg) \tag{4.144}$$

Definition 4.1 establishes that $\beta(a, t)$ and $\sigma(a, t)$ are T-periodic time-dependent parameters. Conforming to the definition of a periodic function, it holds that $\beta(a, t) = \beta(a, t + T)$ and $\sigma(a, t) = \sigma(a, t + T)$. Using this fact, we obtain the following expression:

$$\frac{\mathrm{d}\kappa^-(a; t_0 + T)}{\mathrm{d}a} = -\kappa^-(a; t_0 + T)\Bigg(\mu(a) + \sigma(a, t_0 + a) + p\int_0^a \beta(a - \tilde{b}, t_0 + a - \tilde{b}) \cdot \Big(-\kappa^{-\prime}(\tilde{b}; t_0 + a - \tilde{b} + T) - \kappa^-(\tilde{b}; t_0 + a - \tilde{b} + T) \cdot \mu(b)\Big)\,\mathrm{d}\tilde{b}\Bigg) \tag{4.145}$$

We substract Eq.4.145 from Eq. 4.43, and define $\Delta\kappa^- = \kappa^-(a; t_0 + T) - \kappa^-(a; t_0)$.

$$\begin{aligned}\frac{\mathrm{d}\Delta\kappa^-}{\mathrm{d}a} &= -\Delta\kappa^- \cdot (\mu(a) + \sigma(a, t_0 + a)) - \kappa^-(a; t_0 + T) \cdot p \cdot \\ &\int_0^a \beta(a - \tilde{b}, t_0 + a - \tilde{b}) \cdot \Big[-\kappa^{-\prime}(\tilde{b}; t_0 + a - \tilde{b} + T) - \kappa^-(\tilde{b}; t_0 + a - \tilde{b} + T) \cdot \mu(b)\Big]\,\mathrm{d}\tilde{b} \\ &+ \kappa^-(a; t_0) \cdot p \cdot \int_0^a \beta(a - \tilde{b}, t_0 + a - \tilde{b}) \cdot \Big[-\kappa^{-\prime}(\tilde{b}; t_0 + a - \tilde{b}) - \kappa^-(\tilde{b}; t_0 + a - \tilde{b}) \cdot \mu(b)\Big]\,\mathrm{d}\tilde{b}\end{aligned} \tag{4.146}$$

After adding and subtracting the following expression:

$$\kappa^-(a;t_0)\cdot p\cdot\int_0^a \beta(a-\tilde{b},t_0+a-\tilde{b})\cdot \Big[-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)\cdot\mu(b)\Big]\,\mathrm{d}\tilde{b} \quad (4.147)$$

we obtain the following expression:

$$\begin{aligned}\frac{\mathrm{d}\Delta\kappa^-}{\mathrm{d}a} &= -\Delta\kappa^-\cdot(\mu(a)+\sigma(a,t_0+a))-\kappa^-(a;t_0+T)\cdot p\cdot\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\\ &\Big(-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}+\kappa^-(a;t_0)\cdot p\cdot\\ &\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\Big(-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}\\ &-\kappa^-(a;t_0)\cdot p\cdot\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\Big(-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}\\ &+\kappa^-(a;t_0)\cdot p\cdot\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\Big(-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b})-\kappa^-(\tilde{b};t_0+a-\tilde{b})\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}\end{aligned} \quad (4.148)$$

$$\begin{aligned}\frac{\mathrm{d}\Delta\kappa^-}{\mathrm{d}a} &= -\Delta\kappa^-\cdot(\mu(a)+\sigma(a,t_0+a))-\Delta\kappa^-\cdot p\cdot\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\\ &\Big(-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}-\kappa^-(a;t_0)\cdot p\cdot\\ &\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\Big(-\Big[\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b})\Big]\\ &-\Big[\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b})\Big]\cdot\mu(b)\Big)\mathrm{d}\tilde{b}\end{aligned} \quad (4.149)$$

$$\begin{aligned}\frac{\mathrm{d}\Delta\kappa^-}{\mathrm{d}a} &= -\Delta\kappa^-\cdot(\mu(a)+\sigma(a,t_0+a))-\Delta\kappa^-\cdot p\cdot\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\\ &\Big(-\kappa^{-\prime}(\tilde{b};t_0+a-\tilde{b}+T)-\kappa^-(\tilde{b};t_0+a-\tilde{b}+T)\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}\\ &+\kappa^-(a;t_0)\cdot p\cdot\int_0^a\beta(a-\tilde{b},t_0+a-\tilde{b})\cdot\Big(\Delta\kappa^{-\prime}+\Delta\kappa^-\cdot\mu(b)\Big)\,\mathrm{d}\tilde{b}\end{aligned} \quad (4.150)$$

This integrodifferential equation is linear in $\Delta\kappa$, and therefore, if there is a solution, the solution is unique [61], provided that Eq. 4.43 has a unique solution. We assume the latter

is given. Replacing $\Delta\kappa^-$ by zero, we verify that zero is a solution of the equation. We conclude that an individual who becomes infected at time t_0 and another subject who becomes infected at time $t_0 + T$ experience the same conditions and have the same probability of infection at every age of infection. □

Part III.

Results and Conclusion

5. Optimization problem

In this section, we investigate how the effectiveness of contact tracing is affected by the periodicity of the contact patterns. Chapter 4 presents two simple scenarios to illustrate how the probability of infection behaves in a periodic setting under contact tracing. Let us now concentrate our efforts on the simplest case: non-recursive backward contact tracing with periodic contact patterns.

Later, we will see that some periodicity patterns amplify its impact while others decrease it. Our measure of *efficiency* is the reproduction number R_∞. We propose two optimization problems: find the contact rate ($\beta(t)$) that minimizes the reproduction number (R_∞) and find the contact rate that maximizes it.

In these constrained optimization problems, the value of the basic reproduction number (R_0) is fixed to a constant number r_0. Within the functions $\beta(t)$ that produce in average r_0 secondary infections per individual in the early phase of the outbreak, we look for the function $\beta(t)$ that minimizes or maximizes R_∞, respectively. We model the contact rate as a piecewise constant function, with 0 as the lower bound and no upper bound. The discretization has timestep $h = 0.25$day. We use the SLSQP optimizer (See Section 2.5) to find the optima.

This research question is explored from a qualitative rather than a quantitative perspective. The minimization and maximization problems are studied from two different perspectives. In the first one, only the contact rate ($\beta(t)$) is T-periodic with $T = 7$ days. The rest of the parameters are constant values with respect to time and age of infection. In the second approach, the observation rate ($\sigma(t)$) is also allowed to vary with respect to time in a periodic manner. The period is $T = 7$ as well.

5.1. Problem 1: Reproduction number minimization

Is there a periodic contact rate that leads to the minimum effective reproduction number? How does it look like? And, how different is the effect of non-recursive backward contact tracing when the contact rate has that structure? We aim to gain some insight into these questions by solving Problem 5.1.

Optimization problem 5.1 (R_∞ Minimization). *Prescribe $R_0 = r_0$. Minimize R_∞ over vectors $\vec{\beta} \in \Omega$ where $\Omega = \{\vec{\beta} \mid R_0 = r_0\}$, and r_0 is a constant.*

We choose $r_0 = 3$, $p = 1/3$, $\beta(a) = 1.0/day$, $\mu(a) = 0.3/day$, and $\sigma(a,t) = 0.5/day$.

The solution to this optimization problem is a delta peak (δ) with finite weight. In other words, the contact rate that minimizes the effective reproduction number allows for maximum contact only once in a period (See Figure 5.1). This behaviour is typical of bang-bang controllers [28].

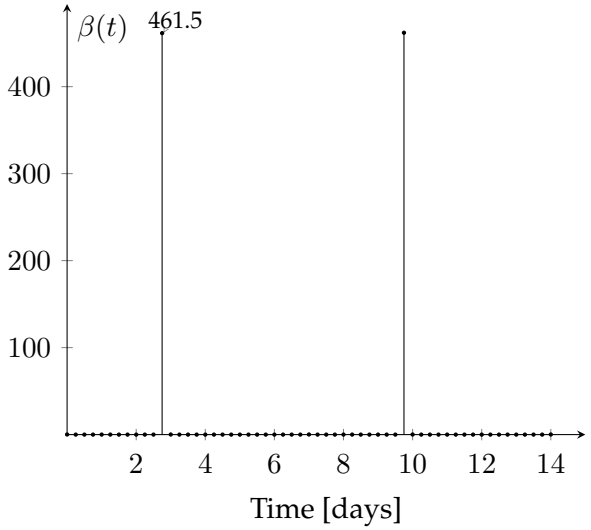


Figure 5.1. Optimal $\beta(t)$ for R_∞ minimization: Case 1 Basic reproduction number is prescribed ($R_0 = 3$). The effective reproduction number is $R_\infty = 0.0038$

The result above was determined in the setting with time-independent parameters; particularly $\sigma(a,t) = \sigma(a)$. In the context of non-recursive backward contact tracing, the infectee must be observed in order to trace the infector. In Problem 5.2 we aim to answer the following question: does the optimal contact pattern ($\beta(t)$) changes when the observation rate ($\sigma(t)$) is T-periodic?

Optimization problem 5.2 (R_∞ Minimization)**.** *Prescribe $R_0 = r_0$. Minimize R_∞ over vectors $\vec{\beta} \in \Omega$ where $\Omega = \{\vec{\beta} \mid R_0 = r_0\}$, and r_0 is a constant.*

We choose $r_0 = 3$, $p = 1/3$, $\beta(a) = 1.0/day$, $\mu(a) = 0.3/day$, $\sigma(a) = 0.5/day$ and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$.

Figure 5.2 shows that the optimal contact rate is, again, a δ peak with finite weight.

Figure 5.1 and Figure 5.2 exhibit only one point per period where the contact rate is very high, which suggests the occurrence of a super-spreading event. These events are known to cause rare and bursting outbreaks [39]. Intuitively, we understand that the higher the number of infectees, the higher the probability of tracing their (common) infector. Also, from Proposition 4.4 we verify that the likelihood of one of the infectees being identified and triggering a tracing process that removes the super-spreader from the infection compartment increases with the contact rate.

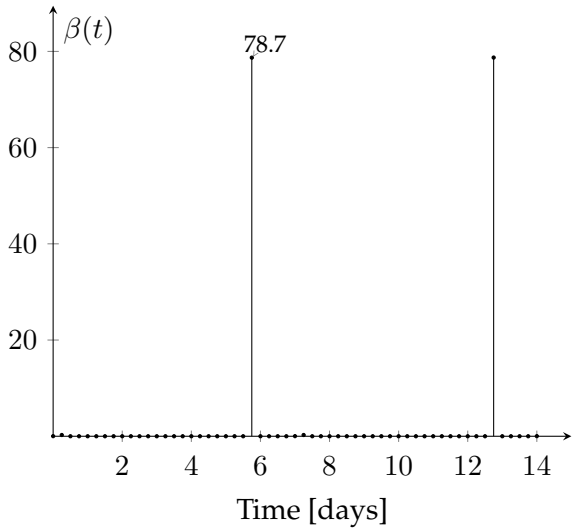


Figure 5.2. Optimal $\beta(t)$ for R_∞ minimization: Case 2 Basic reproduction number is prescribed ($R_0 = 3$). The effective reproduction number is $R_\infty = 0.0474$

Müller *et al.* made the following observation for their stochastic SIRS model (See Section 3.4) in [50]: "It is in general not possible to reduce the effective reproduction number below one with backward tracing only. [...] [The number of traced infections] grows only linearly and is, compared with the exponential growth of the branching process, in general not sufficient to stop the branching process [...]" In contrast, we observe that the minimum effective reproduction number in Problem 5.1 and Problem 5.2 is smaller than one.

The latter is a fascinating result for backward contact tracing in a periodic setting. Because the contacts reside in one point mass, the number of infections does not grow exponentially. Instead, there is a spike in the number of new cases, which drops when the health officials find and remove the super-spreader. In this scenario, the effort to trace the infectors is sufficient to force the epidemic to die out.

5.2. Problem 2: Reproduction number maximization

Let us study the contact rate shape that leads to the worst-case scenario under non-recursive backward contact tracing. Problem 5.3 and Problem 5.4 detail the case we consider.

Optimization problem 5.3 (R_∞ Maximization)**.** *Prescribe $R_0 = r_0$. Maximize R_∞ over vectors $\vec{\beta} \in \Omega$ where $\Omega = \{\vec{\beta} \mid R_0 = r_0\}$, and r_0 is a constant.*

We choose $r_0 = 3$, $p = 1/3$, $\beta(a) = 1.0/day$, $\mu(a) = 0.3/day$, and $\sigma(a,t) = 0.5/day$.

The solution to Problem 5.3 resembles a constant function. According to Proposition 4.4, having on average the same number of contacts occur every time step means that all indi-

viduals have an equal probability of being traced. Some of them are traced and removed; others remain infected for a longer period and can generate additional infections.

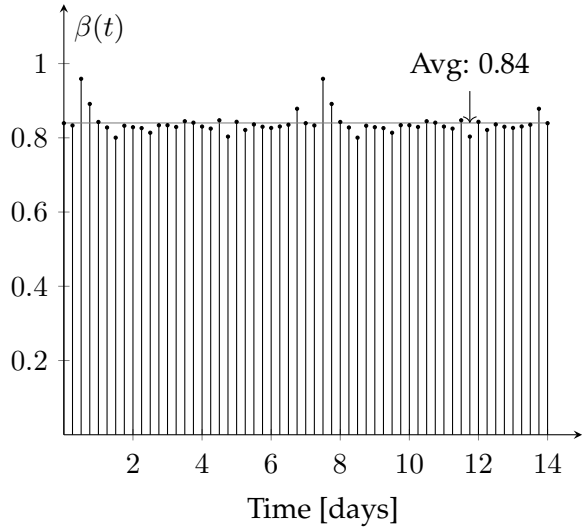


Figure 5.3. Optimal $\beta(t)$ for R_∞ maximization: Case 1 Basic reproduction number is prescribed ($R_0 = 3$). The effective reproduction number is $R_\infty = 2.5815$

We ask the same question in a different scenario, where the observation rate ($\sigma(t)$) is T-periodic in time.

Optimization problem 5.4 (R_∞ Maximization). *Prescribe $R_0 = r_0$. Maximize R_∞ over vectors $\vec{\beta} \in \Omega$ where $\Omega = \{\vec{\beta} \mid R_0 = r_0\}$, and r_0 is a constant.*

We choose $r_0 = 3$, $p = 1/3$, $\beta(a) = 1.0/day$, $\mu(a) = 0.3/day$, $\sigma(a) = 0.5/day$ and $\sigma(t) = \frac{1}{2}\sin(\frac{2\pi}{7}t) + \frac{1}{2}$.

In Figure 5.4 we observe a similar behaviour as for Figure 5.3. Here again, the number of infections increases exponentially and contact tracing effort is insufficient to make the system change its state, and the epidemic continues.

The code used to solve these optimization problems is available in [2].

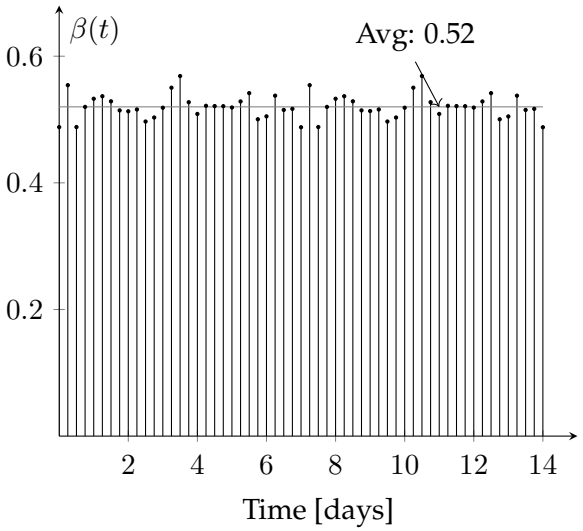


Figure 5.4. Optimal $\beta(t)$ for R_∞ maximization: Case 2 Basic reproduction number is prescribed ($R_0 = 3$). The effective reproduction number is $R_\infty = 2.7282$

6. Discussion

This study demonstrates that periodic contact patterns have an impact on the efficacy of contact tracing. In particular, periodic contact rates have a positive effect on the performance of non-recursive backward contact tracing. The existence of periodicity favors the extinction of the epidemic and minimizes the effective reproduction number. In contrast, the lack of periodicity maximizes the effective reproduction number and leads to a sustained outbreak. This result supports the statement that backward contact tracing can detect and remove super-spreaders [35].

A study carried out by Lloyd-Smith *et al.* found that diseases with high infectiousness heterogeneity have outbreaks with higher probabilities of extinction [39]. In our research, infectious heterogeneity is described by the periodic variation of the contact rate. In the solution to Problem 5.1 and Problem 5.2, we found that a δ-like contact rate minimizes the effective reproduction number. This result reflects a high level of heterogeneity, hints at the occurrence of a super-spreading event, and corroborates the findings mentioned earlier.

The fact that the contact rate is high only at one coordinate per period also supports the observation that individual-specific control measures add heterogeneity to the distribution of infectiousness [39]. Contact tracing modifies the effective infectivity by removing traced individuals and preventing new infection clusters. According to Lloyd *et al.* , when the infectiousness heterogeneity is significant, individual-specific control measures outperform population-level interventions [39].

From the optimization perspective, a δ-like contact rate resembles the control signal of a bang-bang controller [28]. This type of controller, also known as an on-off controller, makes the system switch from one state to another abruptly. In this case, the model for backward contact tracing (See Proposition 4.4) is the system, the contact rate is the control signal, and the state changes between *epidemic* and *endemic*.

The solution to Problem 5.3 and Problem 5.4 shows that a constant contact rates maximizes the effective reproduction number. Indeed, infectiousness homogeneity reduces the probability of extinction of an epidemic [39]. Under these conditions, the effect of tracing and isolation activities to containing the epidemic is reduced [39].

Our results regarding the periodicity of the contact rate extend to recursive backward contact tracing. In the model equations, the occurrence of a super-spreading event increases the probability of finding a participant who can initiate or continue a contact tracing process. Once the infector, who has high infectiousness, is traced and removed, he/she become the index case, and the aim is to find their infector.

We derived the formulation for the recursive and non-recursive variants of forward contact tracing and full contact tracing. Although we showed that periodicity affects the prob-

ability of infection under forward and full contact tracing, it is still unclear how. For forward contact tracing, the infector of a super-spreading event is the key to trace a large number of cases. Intuitively, the probability of observing the infector is much smaller than the probability of tracing the infector through one of their infectees. Thus, the observation rate ($\sigma(t)$) is likely to play a more significant role. Future studies can look for the periodic parameter that provides the best and worst performance.

The simulations in this document focus on the dependency with respect to time. Thus, we kept the parameters constant with respect to the age of infection. Nonetheless, the variation with respect to the age of infection represents changes in infectiousness, immune response, and symptoms appearance. Future research could simulate scenarios where all the parameters show variation using the model we proposed.

The finding that periodicity affects the effectiveness of contact tracing calls for the design of strategies to identify events that force a more or less constant contact rate. Since a steady contact rate hinders contact tracing performance, local governments can organize activities related to commerce, transportation, and work in a way that creates variation. In particular, the combination of digital contact tracing and movement regulations constitutes a promising alternative to control the spread of epidemics.

Bibliography

[1] Ada Lovelace Institute. Exit through the App Store? A rapid evidence review on the technical considerations and societal implications of using technology to transition from the COVID-19 crisis. Technical Report 3, Ada Lovelace Institute, 6 2020. URL: https://www.adalovelaceinstitute.org/our-work/covid-19/covid-19-exit-through-the-app-store/.

[2] Saitel Agudelo. Contact tracing and periodic contact patterns, 2021. URL: https://github.com/daniagudelos/contact_tracing.

[3] Håkan Andersson and Tom Britton. *Stochastic Epidemic Models and Their Statistical Analysis*, volume 151 of *Lecture Notes in Statistics*. Springer New York, New York, NY, 2000. doi:10.1007/978-1-4612-1158-7.

[4] Benjamin Armbruster and Margaret L. Brandeau. Contact tracing to control infectious disease: When enough is enough. *Health Care Management Science*, 10(4):341–355, 12 2007. URL: https://link.springer.com/article/10.1007/s10729-007-9027-6, doi:10.1007/s10729-007-9027-6.

[5] Benjamin Armbruster and Margaret L. Brandeau. Optimal mix of screening and contact tracing for endemic diseases. *Mathematical Biosciences*, 209(2):386–402, 10 2007. URL: https://linkinghub.elsevier.com/retrieve/pii/S0025556407000375, doi:10.1016/j.mbs.2007.02.007.

[6] Kendall E. Atkinson, Weimin Han, and David Stewart. Implicit RK methods for stiff differential equations. In *Numerical Solution of Ordinary Differential Equations*, chapter 9, pages 149–160. John Wiley & Sons, Inc., Hoboken, NJ, USA, 10 2011. doi:10.1002/9781118164495.ch9.

[7] Kendall E. Atkinson, Weimin Han, and David Stewart. Taylor and Runge-Kutta methods. In *Numerical Solution of Ordinary Differential Equations*, chapter 5, pages 67–88. John Wiley & Sons, Inc., Hoboken, NJ, USA, 10 2011. doi:10.1002/9781118164495.ch5.

[8] Kendall E. Atkinson, Weimin Han, and David Stewart. Theory of differential equations: An introduction. In *Numerical Solution of Ordinary Differential Equations*, chapter 1, pages 3–12. John Wiley & Sons, Inc., Hoboken, NJ, USA, 10 2011. doi:10.1002/9781118164495.ch1.

[9] Australasian Society for HIV Medicine. A guide to contact tracing. In *Australasian Contact Tracing Manual*, chapter 2, pages 10–20. Australasian Society for HIV Medicine (ASHM), Darlinghurst, 4 edition, 10 2010.

[10] Australasian Society for HIV Medicine. Contact tracing in context. In *Australasian Contact Tracing Manual*, chapter 1, page 9. Australasian Society for HIV Medicine (ASHM), Darlinghurst, 4 edition, 2010.

[11] Nicolas Bacaër and Souad Guernaoui. The epidemic threshold of vector-borne diseases with seasonality: The case of cutaneous leishmaniasis in Chichaoua, Morocco. *Journal of Mathematical Biology*, 53(3):421–436, 9 2006. URL: http://link.springer.com/10.1007/s00285-006-0015-0, doi:10.1007/s00285-006-0015-0.

[12] D. E. Barton, M. Abramovitz, and I. A. Stegun. *Handbook of Mathematical Functions with Formulas, Graphs and Mathematical Tables.* Dover Publications, 1964. doi:10.2307/2343473.

[13] William J. Bradshaw, Ethan C. Alley, Jonathan H. Huggins, Alun L. Lloyd, and Kevin M. Esvelt. Bidirectional contact tracing could dramatically improve COVID-19 control. *Nature Communications*, 12(1):232, 12 2021. URL: http://www.nature.com/articles/s41467-020-20325-7, doi:10.1038/s41467-020-20325-7.

[14] Tom Britton. 2020.06.18 Tom Britton - Basic reproduction numbers, effective reproduction number and herd immunity - YouTube, 6 2020. URL: https://www.youtube.com/watch?v=pC0pE5MoOjI.

[15] Petter Brodin and Mark M. Davis. Human immune system variation. *Nature Reviews Immunology*, 17(1):21–29, 1 2017. URL: http://www.nature.com/articles/nri.2016.125, doi:10.1038/nri.2016.125.

[16] Vir Bannerjee Bulchandani, Saumya Shivam, Sanjay Moudgalya, and Shivaji L Sondhi. Digital herd immunity and COVID-19. *Physical Biology*, 4 2021. URL: http://iopscience.iop.org/article/10.1088/1478-3975/abf5b4https://iopscience.iop.org/article/10.1088/1478-3975/abf5b4, doi:10.1088/1478-3975/abf5b4.

[17] Rainer Callies. Numerical Programming 2 (MA 3306) Summer Term 2019, 2019.

[18] James Clarke, K. A. Jane White, and Katy Turner. Exploring Short-Term Responses to Changes in the Control Strategy for Chlamydia trachomatis. *Computational and Mathematical Methods in Medicine*, 2012:1–10, 2012. URL: http://www.hindawi.com/journals/cmmm/2012/803097/, doi:10.1155/2012/803097.

[19] Germund Dahlquist and Åke Björck. Numerical Integration. In *Numerical Methods in Scientific Computing, Volume I*, pages 521,531. Society for Industrial and Applied Mathematics, 1 2008. doi:10.1137/1.9780898717785.

[20] Paul L. Delamater, Erica J. Street, Timothy F. Leslie, Y. Tony Yang, and Kathryn H. Jacobsen. Complexity of the basic reproduction number (R0). *Emerging Infectious Diseases*, 25(1):1–4, 1 2019. URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6302597/http://wwwnc.cdc.gov/eid/article/25/1/17-1901_article.htm, doi:10.3201/eid2501.171901.

[21] Jack J. Dongarra, Iain S. Duff, Danny C. Sorensen, and Henk A. van der Vorst. 10. Linear Eigenvalue Problems Ax=λx. In *Numerical Linear Algebra for High-Performance Computers*, pages 231–261. Society for Industrial and Applied Mathematics, 1 1998. URL: http://epubs.siam.org/doi/10.1137/1.9780898719611.ch10, doi:10.1137/1.9780898719611.ch10.

[22] Jack J. Dongarra, Iain S. Duff, Danny C. Sorensen, and Henk A. van der Vorst. 11. The Generalized Eigenproblem. In *Numerical Linear Algebra for High-Performance Computers*, pages 263–272. Society for Industrial and Applied Mathematics, 1 1998. URL: http://epubs.siam.org/doi/10.1137/1.9780898719611.ch11, doi:10.1137/1.9780898719611.ch11.

[23] Katayoun Farrahi, Rémi Emonet, and Manuel Cebrian. Epidemic contact tracing via communication traces. *PLoS ONE*, 9(5):e95133, 5 2014. URL: https://dx.plos.org/10.1371/journal.pone.0095133, doi:10.1371/journal.pone.0095133.

[24] Michael T Heath. Eigenvalue Problems. In *Scientific Computing*, pages 156–202. Society for Industrial and Applied Mathematics, Philadelphia, PA, 11 2001. doi:10.1137/1.9781611975581.

[25] Herbert W Hethcote and James A Yorke. *Gonorrhea Transmission Dynamics and Control*, volume 56 of *Lecture Notes in Biomathematics*. Springer Berlin Heidelberg, Berlin, Heidelberg, 1 1984. doi:10.1007/978-3-662-07544-9.

[26] Francis-Begnaud Hildebrand. Laguerre-Gaus Quadrature. In *Introduction to Numerical Analysis*, chapter 8, pages 392–395. Dover Publications Inc., New York, 2 edition, 6 1987.

[27] Thomas House and Matt J. Keeling. The Impact of Contact Tracing in Clustered Populations. *PLoS Computational Biology*, 6(3):e1000721, 3 2010. URL: https://dx.plos.org/10.1371/journal.pcbi.1000721, doi:10.1371/journal.pcbi.1000721.

[28] Richard J. Jagacinski and John M. Flach. Nonproportional Control. In *Control Theory for Humans*, pages 58–73. CRC Press, 10 2018. doi:10.1201/9781315144948-7.

[29] Kate E. Jones, Nikkita G. Patel, Marc A. Levy, Adam Storeygard, Deborah Balk, John L. Gittleman, and Peter Daszak. Global trends in emerging infectious diseases. *Nature*, 451(7181):990–993, 2 2008. URL: https://www.nature.com/articles/nature06536http://www.nature.com/articles/nature06536, doi:10.1038/nature06536.

[30] Matt J. Keeling and Ken T.D. Eames. Networks and epidemic models. *Journal of The Royal Society Interface*, 2(4):295–307, 9 2005. URL: https://royalsocietypublishing.org/doi/10.1098/rsif.2005.0051, doi:10.1098/rsif.2005.0051.

[31] Istvan Z. Kiss, Darren M. Green, and Rowland R. Kao. Infectious disease control using contact tracing in random and scale-free networks. *Journal of the Royal Society Interface*, 3(6):55–62, 2 2006. URL: https://royalsocietypublishing.org/doi/10.1098/rsif.2005.0079, doi:10.1098/rsif.2005.0079.

[32] Istvan Z. Kiss, Darren M. Green, and Rowland R. Kao. Infectious disease control using contact tracing in random and scale-free networks. *Journal of the Royal Society Interface*, 3(6):55–62, 2 2006. URL: https://royalsocietypublishing.org/doi/10.1098/rsif.2005.0079, doi:10.1098/rsif.2005.0079.

[33] Istvan Z. Kiss, Darren M. Green, and Rowland R. Kao. The effect of network mixing patterns on epidemic dynamics and the efficacy of disease contact tracing. *Journal of the Royal Society Interface*, 5(24):791–799, 7 2008. URL: https://royalsocietypublishing.org/doi/10.1098/rsif.2007.1272, doi:10.1098/rsif.2007.1272.

[34] Istvan Z. Kiss, Darren M. Green, and Rowland R. Kao. The effect of network mixing patterns on epidemic dynamics and the efficacy of disease contact tracing. *Journal of the Royal Society Interface*, 5(24):791–799, 7 2008. URL: https://royalsocietypublishing.org/doi/10.1098/rsif.2007.1272, doi:10.1098/rsif.2007.1272.

[35] Don Klinkenberg, Christophe Fraser, and Hans Heesterbeek. The Effectiveness of Contact Tracing in Emerging Epidemics. *PLoS ONE*, 1(1):e12, 12 2006. URL: https://dx.plos.org/10.1371/journal.pone.0000012, doi:10.1371/journal.pone.0000012.

[36] D. Kraft. *A Software Package for Sequential Quadratic Programming*. Number 28. Wiss. Berichtswesen d. DFVLR, 1988.

[37] Mirjam Kretzschmar, Susan Van Den Hof, Jacco Wallinga, and Jan Van Wijngaarden. Ring Vaccination and Smallpox Control. *Emerging Infectious Diseases*, 10(5):832–841, 5 2004. URL: http://wwwnc.cdc.gov/eid/article/10/5/03-0419_article.htm, doi:10.3201/eid1005.030419.

[38] Mirjam E. Kretzschmar, Ganna Rozhnova, Martin C J Bootsma, Michiel van Boven, Janneke H H M van de Wijgert, and Marc J M Bonten. Impact of delays on effectiveness of contact tracing strategies for COVID-19: a modelling study. *The Lancet Public Health*, 5(8):e452–e459, 8 2020. URL: https://linkinghub.elsevier.com/retrieve/pii/S2468266720301572, doi:10.1016/S2468-2667(20)30157-2.

[39] J. O. Lloyd-Smith, S. J. Schreiber, P. E. Kopp, and W. M. Getz. Superspreading and the effect of individual variation on disease emergence. *Nature*, 438(7066):355–359, 11 2005. URL: https://pubmed.ncbi.nlm.nih.gov/16292310/, doi:10.1038/nature04153.

[40] James O. Lloyd-Smith, Alison P. Galvani, and Wayne M. Getz. Curtailing transmission of severe acute respiratory syndrome within a community and its hospital. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 270(1528):1979–1989, 10 2003. URL: https://royalsocietypublishing.org/doi/10.1098/rspb.2003.2481, doi:10.1098/rspb.2003.2481.

[41] Nita Madhav, Ben Oppenheim, Mark Gallivan, Prime Mulembakani, Edward Rubin, and Nathan Wolfe. Pandemics: Risks, Impacts, and Mitigation. In *Disease Control Priorities, Third Edition (Volume 9): Improving Health and Reducing Poverty*, pages 315–345. The World Bank, 12 2017. doi:10.1596/978-1-4648-0527-1{_}ch17.

[42] Robert M May and Roy M Anderson. *Infectious diseases of humans: Dynamics and control*. Oxford University Press, 8 1991.

[43] Michelle M. Mello. Digital Technology and Contact Tracing. In Jeffrey P. Kahn Technologies, Johns Hopkins Project on Ethics, and Governance of Digital Contact Tracing, editors, *Digital Contact Tracing for Pandemic Response: Ethics and Governance Guidance*, chapter 2, pages 35–41. Johns Hopkins University Press, Baltimore, 2020.

[44] Narges Montazeri Shahtori, Tanvir Ferdousi, Caterina Scoglio, and Faryad Darabi Sahneh. Quantifying the impact of early-stage contact tracing on controlling Ebola diffusion. *Mathematical Biosciences & Engineering*, 15(5):1165–1180, 2018. URL: http://aimsciences.org//article/doi/10.3934/mbe.2018053, doi:10.3934/mbe.2018053.

[45] Graham Mooney. History of Contact Tracing - YouTube, 6 2020. URL: https://www.youtube.com/watch?v=PVsbfbdn5YI.

[46] Graham Mooney. "A Menace to the Public Health" — Contact Tracing and the Limits of Persuasion. *New England Journal of Medicine*, 383(19):1806–1808, 11 2020. URL: http://www.nejm.org/doi/10.1056/NEJMp2021887, doi:10.1056/nejmp2021887.

[47] Stephen S. Morse. Factors in the emergence of infectious diseases. *Emerging Infectious Diseases*, 1(1):7–15, 3 1995. URL: http://wwwnc.cdc.gov/eid/article/1/1/95-0102_article.htm, doi:10.3201/eid0101.950102.

[48] Johannes Müller and Bendix Koopmann. The effect of delay on contact tracing. *Mathematical Biosciences*, 282:204–214, 12 2016. URL: https://linkinghub.elsevier.com/retrieve/pii/S0025556416302693, doi:10.1016/j.mbs.2016.10.010.

[49] Johannes Müller and Mirjam Kretzschmar. Contact tracing – Old models and new challenges. *Infectious Disease Modelling*, 6:222–231, 2021. URL: https://linkinghub.elsevier.com/retrieve/pii/S2468042720301093, doi:10.1016/j.idm.2020.12.005.

[50] Johannes Müller, Mirjam Kretzschmar, and Klaus Dietz. Contact tracing in stochastic and deterministic epidemic models. *Mathematical Biosciences*, 164(1):39–64, 3 2000. URL: https://linkinghub.elsevier.com/retrieve/pii/S0025556499000619, doi:10.1016/S0025-5564(99)00061-9.

[51] Johannes Müller and Christina Kuttler. Compartmental Modelling. In *Methods and Models in Mathematical Biology*, chapter 1, pages 1–156. Springer US, 2015. URL: http://link.springer.com/10.1007/978-3-642-27251-6_1, doi:10.1007/978-3-642-27251-6{_}1.

[52] Johannes Müller and Christina Kuttler. Epidemiology. In *Methods and Models in Mathematical Biology*, chapter 4, pages 452–456. Springer Berlin Heidelberg, 2015. URL: http://link.springer.com/10.1007/978-3-642-27251-6_4, doi:10.1007/978-3-642-27251-6{_}4.

[53] Johannes Müller and Christina Kuttler. Structured Models in Ecology. In *Methods and Models in Mathematical Biology*, chapter 3, pages 380–387. Springer Berlin Heidelberg, 2015. URL: http://link.springer.com/10.1007/978-3-642-27251-6_3, doi:10.1007/978-3-642-27251-6{_}3.

[54] A. J. Nelson. Applied epidemiology of gonorrhea in British Columbia. *Public health reports*, 72(3):223–228, 3 1957. URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2031160/, doi:10.2307/4589741.

[55] Organisation for Economic Co-operation and Development. Flattening the COVID-19 peak: Containment and mitigation policies, 3 2020. URL: https://www.oecd.org/coronavirus/policy-responses/flattening-the-covid-19-peak-containment-and-mitigation-policies-e96a4226/.

[56] Abhishek Pandey, Katherine E. Atkins, Jan Medlock, Natasha Wenzel, Jeffrey P. Townsend, James E. Childs, Tolbert G. Nyenswah, Martial L. Ndeffo-Mbah, and Ali-

son P. Galvani. Strategies for containing Ebola in West Africa. *Science*, 346(6212):991–995, 11 2014. URL: https://www.sciencemag.org/lookup/doi/10.1126/science.1260612, doi:10.1126/science.1260612.

[57] Hélène Pasquini-Descomps, Nathalie Brender, and David Maradan. Value for Money in H1N1 Influenza: A Systematic Review of the Cost-Effectiveness of Pandemic Interventions. *Value in Health*, 20(6):819–827, 6 2017. URL: https://linkinghub.elsevier.com/retrieve/pii/S1098301516304922, doi:10.1016/j.jval.2016.05.005.

[58] Yousef Saad. 4. The Tools of Spectral Approximation. In *Numerical Methods for Large Eigenvalue Problems*, pages 85–114. Society for Industrial and Applied Mathematics, 1 2011. URL: http://epubs.siam.org/doi/10.1137/1.9781611970739.ch4, doi:10.1137/1.9781611970739.ch4.

[59] Yousef Saad. Perturbation Theory and Error Analysis. In *Numerical Methods for Large Eigenvalue Problems*, pages 47–84. Society for Industrial and Applied Mathematics, 1 2011. doi:10.1137/1.9781611970739.ch3.

[60] The SciPy community. scipy.integrate.Radau — SciPy v1.6.2 Reference Guide, 2021. URL: https://docs.scipy.org/doc/scipy/reference/generated/scipy.integrate.Radau.html.

[61] Abdul-Majid Wazwaz. Introductory Concepts of Integral Equations. In *Linear and Nonlinear Integral Equations*, volume 66, pages 33–63. Springer Berlin Heidelberg, Berlin, Heidelberg, 8 2011. doi:10.1007/978-3-642-21449-3{_}2.

[62] World Health Organization. Infection prevention and control: Contact tracing, 5 2017. URL: https://www.who.int/news-room/q-a-detail/contact-tracing.

[63] World Health Organization. *Managing epidemics*. World Health Organization, 2018. URL: https://www.who.int/emergencies/diseases/managing-epidemics/en/.

[64] World Health Organization. Coronavirus disease 2019 (COVID-19) Situation Report 94. Technical report, World Health Organization, 4 2020. URL: https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports.

[65] World Health Organization (WHO). Increase effectiveness, maximize benefits and minimize harm Screening programmes: a short guide. Technical report, World Health Organization, Copenhagen, 2020. URL: https://apps.who.int/iris/bitstream/handle/10665/330829/9789289054782-eng.pdf.