Infectious Disease Modelling 6 (2021) 222–231

Contents lists available at ScienceDirect

Infectious Disease Modelling

journal homepage: www.keaipublishing.com/idm

Contact tracing – Old models and new challenges

Johannes Müller [a, b, *], Mirjam Kretzschmar [c]

[a] Mathematical Institute, Technical University of Munich, Boltzmannstr. 3, 85748, Garching, Germany
[b] Institute for Computational Biology, Helmholtz Center Munich, 85764, Neuherberg, Germany
[c] Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht University, Heidelberglaan 100, 3584CX, Utrecht, the Netherlands

ARTICLE INFO

Article history:
Received 8 November 2020
Received in revised form 10 December 2020
Accepted 19 December 2020
Available online 30 December 2020
Handling editor: Yijun Lou

Keywords:
Contact tracing
Mathematical models

ABSTRACT

Contact tracing is an effective method to control emerging infectious diseases. Since the 1980's, modellers are developing a consistent theory for contact tracing, with the aim to find effective and efficient implementations, and to assess the effects of contact tracing on the spread of an infectious disease. Despite the progress made in the area, there remain important open questions. In addition, technological developments, especially in the field of molecular biology (genetic sequencing of pathogens) and modern communication (digital contact tracing), have posed new challenges for the modelling community. In the present paper, we discuss modelling approaches for contact tracing and identify some of the current challenges for the field.

1. Introduction

Emerging and re-emerging infectious diseases like SARS, Ebola, Lassa fever, Tuberculosis, and most recently COVID-19, require rapid response and targeted control measures. It is best if an immunisation of the population is possible - however, in case of emerging infectious diseases, often the necessary vaccines are not available yet, or not available in sufficient quantities. An alternative approach is to stop infection chains by non-pharmaceutical control measures, such as reducing infectious contacts by social distancing, and testing and isolating infectious individuals. Mass screening as a stand-alone control measure is effective if prevalence is high, and cheap, rapid, and reliable diagnostic tools are available. If prevalence is low, most tested persons will be uninfected such that even a small probability for a false positive test result leads to a large number of false alarms, while only few infected persons can be identified.

Contact tracing (CT) is a more focused method: Once an infected individual is diagnosed and isolated, contact persons are identified, who had potentially infectious interactions with that index case. In general, the prevalence within that group will be much higher than that in the overall population. It is effective to screen these persons, and, if necessary, to place them in quarantine or to isolate them. CT acts on several levels: *Individual level:* Infected persons are diagnosed early, are isolated, and receive medical attention. *Population level:* Transmission chains can be detected and stopped, which reduces the effective reproduction number. *Medical/scientific level:* By studying infector–infectee pairs, one can learn about who infected whom in

* Corresponding author. Mathematical Institute, Technical University of Munich, Boltzmannstr. 3, 85748, Garching, Germany.
E-mail address: johannes.mueller@mytum.de (J. Müller).
Peer review under responsibility of KeAi Communications Co., Ltd.

https://doi.org/10.1016/j.idm.2020.12.005

the outbreak. This provides information on risk factors, transmission modes, infectivity, and generation intervals of the infectious disease. This information can be central in the design of further control measures.

The main challenge in modelling CT is the individual-based character of the process. Information about the health status of single individuals and the time course of contacts between these individuals is required. A direct formulation of the process is only possible at the microscopic level. However, the main interest is at the mesoscopic and macroscopic levels: Not single individuals, but the spread of the infection in the population is the focus of interest. Model approaches need to span several scales, from the individual and its local contact network to the population. Epidemiology is part of ecology, and here we are faced with a generic challenge in ecology: From observations on the individual level we aim to understand the functioning of an ecosystem. Similar to CT, also the transmission of infection is a process occurring between individuals. For the epidemic process, it is well established how to bridge the scales. The time course of an epidemic can be readily described by mean field models as the Kermack-McKendrick model. CT, in contrast, follows the interaction given by transmission events, so can be viewed as a kind of superinfection: One could consider CT as another infection that follows the paths of the primary infection, and removes in that way infected individuals. This process superimposed on the transmission process creates a high degree of dependency between individuals. Lifting a model description for CT from the individual level to the population level involves more technical difficulties than for the case of a pure infection process.

Intuitively, CT and the transmission processes are racing each other. Starting at an index case, the infection spreads to contacts, while with CT we aim to catch up. This picture already indicates many of the properties that make a disease controllable by CT (Fraser, Riley, Anderson, & Ferguson, 2004): (a) a sufficiently large fraction of cases develops symptoms and is tested and diagnosed; (b) contacts need to be well identifiable and traceable; (c) the disease spreads slowly enough to allow CT to catch up, even if identification, testing, and quarantining of contacts comes with certain delays, and (d) diagnostic tests are able to readily identify symptomatic and asymptomatic persons.

In the first part of the review, we discuss the different modelling approaches to CT; for the sake of brevity, we only cite some publications in the main text. The interested reader is strongly advised to peruse the supplementary material (SI), where a more complete selection of papers are shortly summarised in a table. In the second part, we identify and discuss open problems and current challenges for the theory of CT.

2. Modelling approaches

Among the first papers addressing models for CT is a study by Hethcote and Yorke concerning interventions for gonorrhoea (Hethcote & Yorke, 1984). They suggest that the impact of CT is caused by a reduction in the effective transmissibility of the infection. In the same spirit (Hyman, Li, & Stanley, 2003), proposes that CT is based on the identification of infected persons by their infected contacts. In that, the “superinfection” is taken verbally, and infecteds are removed from the infectious compartment with a term that resembles the incidence term (product of discovered infecteds and prevalence). In those models, the description of CT is not based on first principles, that is, they are not derived from an individual-based stochastic model where CT can be directly formulated, but rather defined *ad hoc*: A phenomenological term (typically in an ODE) is introduced, and without deeper justification it is claimed that this term describes CT adequately. For models, that are based on first principles, three main directions can be identified:

1. Simulation models that directly simulate individuals in a large populations;
2. Pair approximation models that are extensions of mean field models incorporating information about correlations of pairs of individuals;
3. Stochastic and deterministic models that are based on a rigorous analysis of a simple branching process modelling CT.

We discuss these in the following sections, and also the phenomenological had-hoc approaches, which are relevant for practical applications.

2.1. Individual based simulation models

Individual (or agent) based models (IBMs) are perhaps the first choice to formulate a process as complex as CT. IBMs describe the fate of every single individual in a population and their interactions. In that they explicitly incorporate a contact graph, where individuals form the nodes, and an edge connects two nodes (individuals) that might have contacts. For each edge, a stochastic process indicates the time points of contacts. The contact graph can be as simple as a complete graph where every individual may have contact with every other individual (fully connected population), a random graph as described by the configuration model, or a small world graph that reflects local and long distance contacts (Keeling & Eames, 2005; Newman, Strogatz, & Watts, 2001). Conceptual, parsimonious simulation models are used to study the influence of the contact graph structure on transmission dynamics (Kiss et al., 2006, 2008). The most detailed IBMs describe graphs that aim to represent existing societies with cities, work places, and schools (Meyers, Pourbohloul, Newman, Skowronski, & Brunham, 2005). It depends on the aim of the model how detailed the contact graph, the state of an individual, and its behaviour are formulated. In particular, CT can be easily described: Once an individual (node) is diagnosed as infected, adjacent nodes are informed/screened with a given probability, with or without a tracing delay. In one-step tracing, only direct neighbours are

included in the tracing process, while in recursive tracing an identified infected neighbour becomes a new index case for the next step of tracing. As such a model is constructed algorithmically, there is almost no limit to the degree of detail that can be included. However, very detailed models are often faced with the problem of lack of data for an appropriate parametrization.

In any case, the advantages of individual-based models for CT are three-fold: First, they are simple to formulate and to communicate; second, they allow to directly represent CT in a realistic way and in that foster and guide the development of more abstract, analytical models; and third, they provide sufficient detail, such that results can be used in public health decision making.

IBMs are frequently used to investigate specific infectious diseases, and to extract relevant information about the effectiveness of intervention strategies. Contact tracing is commonly performed to improve case finding for sexually transmitted diseases as gonorrhoea and chlamydia; for these diseases, contacts can be clearly defined - recent sex partners - and a large fraction of infections are asymptomatic, such that contact tracing greatly enhances the possibility of finding and treating infected persons. Also, the time scale of transmission of these infections is sufficiently slow to enable finding and treating contacts on a faster time scale than the generation interval. Finally, as sexual partnerships are often long lasting, treating contacts may prevent re-infections of the index case (Althaus, Heijne, Herzog, Roellin, & Low, 2012; Ghani, Swinton, & Garnett, 1997; Kretzschmar et al., 1996, 2009, 2012; Turner et al., 2006). Several IBMs investigate Tuberculosis (Kasaie, Andrews, Kelton, & Dowdy, 2014; Mellor, Currie, & Corbett, 2011; Tian et al., 2011, 2013) (see also the review article (Begun, Newall, Marks, & Wood, 2013) and references therein), Smallpox (Porco et al., 2004), also in connection with bioterrorism (Eichner, 2003), measles (Liu et al., 2015), and Ebola (Shahtori, Ferdousi, Scoglio, & Sahneh, 2018). Also, the effect of CT on control of SARS has been simulated by various IBMs, see the e.g. (Klinkenberg, Fraser, & Heesterbeek, 2006; Lloyd-Smith, Galvani, & Getz, 2003), and particularly the review article (Kwok et al., 2019) and references therein. Peak et al. (Peak, Childs, Grad, & Buckee, 2017) compare effectiveness of CT for several infections, including Ebola, influenza, and SARS. In (Armbruster and Brandeau, 2007a, 2007b), CT is analysed from a public health economics point of view. The effect of CT on the COVID-19 pandemic was investigated based on IBMs in quite a number of papers (see SI for references).

2.2. Pair approximation models

For IBMs it is hard if not impossible to obtain analytical results, while many tools are available for deterministic models. Mean field equations are a well established, heuristic approach to reformulate IBMs as described above in terms of ordinary differential equations (ODE's). Instead of counting the number of individuals of a given type (e.g., S, I, and R), the expected relative frequencies are addressed by the ODE model. In order to obtain the ODE, the population frequencies are represented by a "typical" individual, and the interactions with other individuals are replaced by an averaged interaction. That precisely is the way to derive a deterministic model as the Kermack-McKendrick (or SIR) model from a stochastic process for an infection. The success of the SIR model to describe the time course of real-world epidemics is an *a posteriori* justification for simple deterministic mean field models. In many cases, mean field models allow for a deeper understanding of the underlying mechanism, and for powerful predictions (Keeling, 1999).

In averaging interactions, all information about correlations are lost in the transition from an IBM to a mean field model. In that, a mean field model is insufficient to appropriately describe the infectious process on an inhomogeneous contact graph, particularly if the contact graph is strongly locally clustered: Is this the case, the neighbour of an infected individual often already is infected, and the spread of infection slows down. In the 1990's, mainly driven by Japanese (Sato, Matsuda, & Sasaki, 1994) and British (Keeling, 1999; Keeling, Rand, & Morris, 1997) groups, an improved mean field approximation was developed, the pair approximation. Here, not only expectations but also the correlations are formulated in an ODE model. That is, not only the expected number of individuals in state *S*, *I*, or *R*, say, but also the expected number of edges connecting e.g. *I* with *I* or *S* with *I* individuals are followed in the system of ODE's. As the model incorporates information about correlation, it can – up to a certain degree – mimic the slow down of the spread caused by spatial correlations. The disadvantage of this approach is that it requires many more equations than the simple mean field model – instead of one equation per state, we now need one equation for each pairwise combination of states. Recent developments allow even an exact analysis of an SIR-process on trees by the "message passing method", a subtle further development of the pair approximation (Wilkinson, Ball, & Sharkey, 2017).

Concerning the modelling of CT, pair approximation keeps a central piece of information that is dismissed by the mean field approximation: We know how likely it is that a neighbour of an infected individual is infected. In that, it is possible to remove infected neighbours of an index case (Eames & Keeling, 2002; Huerta & Tsimring, 2002). This idea has been discussed in a series of papers (Eames & Keeling, 2003; House and Keeling, 2010, 2011; Keeling & Eames, 2005; Tsimring & Huerta, 2003). The comparison with Monte Carlo simulations of the stochastic process indicates that the results are valid especially for large, homogeneous graphs. Most of these papers investigate the effectiveness of CT in different contact graph structures. In (House & Keeling, 2010) it is found that CT is more effective in clustered than in homogeneous populations. In (Eames, 2007) recursive and one-step tracing is compared, and "targeted CT", that is, CT focusing on a risk group, is analysed. Recursive and targeted CT were found to be particularly effective.

Models for CT based on pair approximation are mostly rather conceptual models that allow addressing fundamental questions as the influence of the contact graph, in contrast with models used to quantitatively predict the effect of CT on the spread of real-world infections. One of few exceptions is (Clarke, White, & Turner, 2012), where pair approximation is applied to predict the impact of CT on chlamydia prevalence. A simpler approach, in which only pairs are taken into account, which

may form and dissolve, is used in (Heijne, Althaus, Herzog, Kretzschmar, & Low, 2011) to investigate the impact of screening and CT, again addressing the prevalence of chlamydia.

2.3. Models based on branching processes

At the onset of an outbreak spreading in a homogeneous population, it is possible to replace the epidemic process by a birth-death process of independent individuals, where "birth" means a new infection and "death" recovery. Particularly the interaction of two infectious individuals is unlikely and negligible in a large, homogeneous population (Ball & Donnelly, 1995). The process generates the tree of infection: nodes are the infected individuals, and a directed edge connects infector with infectee. As recovered individuals are removed, we rather obtain a forest than a tree. As in IBMs, it is now possible to model CT directly on top of this process. If a member of the tree is diagnosed, the adjacent nodes will be screened and if infected will be isolated. It is possible to rigorously analyse this stochastic process (Ball, Knock, & O'Neill, 2011; Müller, Kretzschmar, & Dietz, 2000): CT mainly affects the removal rate of infected individuals. That is, in order to address the stochastic process, the probability to be infectious at a given time after infection is determined. This probability is the central function that allows to readily determine the effective reproduction number, or the doubling time of an infection. Various aspects of CT can be investigated in this context, as the effect of a latent period (Ball et al., 2011; Müller et al., 2000), estimation of the tracing probability from data (Blum & Tran, 2010; Müller & Hösel, 2007), or the effect of a tracing delay (Ball, Knock, & O'Neill, 2015; Müller & Koopmann, 2016). Strictly spoken, the analysis and the results are valid only for the onset (or during the decay before extinction) of the outbreak, if direct contacts between infecteds are unlikely to happen. However, using heuristic arguments, the removal rate can be approximated also in the case of high prevalence, and a modified mean field equation has been proposed (Müller et al., 2000).

Similar to pair approximation, the approach is not suited for a complex contact graph structure with small homogeneous clusters that only weakly interact. It is interesting that the central idea for the analysis of CT on the one hand, and the message passing methods used in recent versions of pair approximation on the other hand, bear a remarkable similarity. In (Okolie & Müller, 2020), the branching process analysis is generalised from homogeneous populations to populations with a prescribed contact graph, and ideas are discussed how to merge the branching process analysis and the pair approximation.

Several recent papers (Barlow, 2020; Baumgarten & Bornholdt, 2020; Kojaku, Hébert-Dufresne, & Ahn, 2020) investigate branching process models from a different point of view, via the consideration of generating functions for the degree distribution of neighbours of randomly chosen infected individuals. In that, they extend the classical percolation based analysis of epidemics on random graphs to CT. Particularly (Barlow, 2020) is able to determine the probability of extinction, a quantity that is difficult to determine analytically as CT is able to eliminate complete clusters of outbreaks, but that is of practical importance.

Brown et al. (Browne, Gulbudak, & Webb, 2015) developed a sophisticated ODE-approximation of the branching-process structure and applied that to Ebola. Becker et al. (Becker, Glass, Li, & Aldis, 2005) also proposed a simplified version of the model and investigated the SARS epidemic in a deterministic model with household-structure. Kretzschmar et al. (Kretzschmar, Van den Hof, Wallinga, & Van Wijngaarden, 2004) used a branching process to model ring vaccination for smallpox. This model was recently developed further to investigate the effectiveness of CT for COVID-19 (Kretzschmar et al., 2020a, 2020b). Based on a branching-process formulation for CT, Tanaka (Tanaka, Yamaguchi, & Sakamoto, 2020) analysed data for COVID-19 to estimate the fraction of asymptomatic cases.

2.4. Phenomenological approaches

Phenomenological approaches are not rooted in an analysis of stochastic models that obviously formulate CT in an adequate way, but typically ODEs are complemented by linear or nonlinear terms with the claim that these terms represent CT. The advantage of these models is their simplicity – they are ODE models with a rather simple structure (concerning CT) and can be readily analysed or simulated. In that, these models are suited to address real world epidemics. The difficult part is the interpretation of the results (again concerning CT), as the clear connection with first principles is not obvious.

Many of these models are applied to the HIV infection, where mostly a mass action term is used to represent CT (Agarwal & Bhadauria, 2012; Clémençon, Tran, & de Arazoza, 2008; de Arazoza & Lounes, 2002; Hyman et al., 2003; Hyman et al., 2003, 2003; Naresh, Tripathi, & Sharma, 2011), but sometimes also simply a linear term (Hsieh, Wang, de Arazoza, & Lounes, 2010). Hsieh et al. (Hsieh, de Arazoza, Lounes, & Joanes, 2005) compare predictions of different ways of modelling CT - linear, mass action, and a saturation function - with data. Interestingly enough, the authors conclude that a mass action term for CT is inferior to a linear term or a saturation function. In a similar spirit, Clarke et al. (Clarke, White, & Turner, 2013) adjust a power law term for CT based on simulations from an IBM. Also models for Chlamydia (Heffernan & Dunningham, 2009), Tuberculosis (Aparicio & Hernández, 2006), Smallpox (Kaplan, Craft, & Wein, 2002), Ebola (Berge, Tassé, Tenkam, & Lubuma, 2018), and models for COVID-19 (Giordano et al., 2020; Lunz, Batt, & Ruess, 2020) are based on this phenomenological modelling approach.

Fraser et al. (Fraser et al., 2004) proposed an influential idea with a more profound connection to first principles. The model is based on age-since-infection, and addresses approximations for the reduction of secondary cases due to CT. In that, it is similar to the branching-process analysis, but CT is formulated as a linear effect. In the article, characteristics in the timing between onset of symptoms and infectivity are identified that make an infection controllable by CT. Chen et al. (Chen, Chang,

& Liao, 2006) took up that approach to analyse a model for the SARS infection and Ferretti et al. (Ferretti et al., 2020) applied the model to the COVID-19 epidemic in Italy (find more in the SI).

3. Challenges

The efforts of the last 40 years to develop a toolbox for CT have delivered a considerable amount of modelling approaches and results. There is a general agreement about a fundamental model structure, that can be readily realised in IBMs, and there are various ways to analyse the stochastic process either approximately or rigorously – at least, if we stick to simple models. Many handy *ad-hoc* models are published. Nevertheless, some of the central questions are still open.

3.1. Classical questions

Some questions and problems have been debated for quite a while. We pick a few of these "classical questions" which we consider as interesting and/or of practical need, for discussion here.

Modelling CT. Obviously, a multitude of models are in use. Some models, as the stochastic IBMs, directly simulate CT. In an IBM, it is easy to incorporate CT appropriately. Stochastic simulation models have the advantage that their outcomes are easy to interpret, but the disadvantage that they cannot be analysed analytically. To obtain insight into the parameter dependence of CT and to develop general rules for CT is not straightforward or may be impossible, if we exclusively rely on IBMs.

Other approaches, as the pair approximation models or models based on branching processes, use first principles to develop the model structure. Also these models are able to reflect CT in an appropriate manner. Even if approximations are used to derive simplified mathematical structures, it is straightforward to check their accuracy (compare the analytical results to simulations of the original models). In that, these models are appealing. However, in some cases, the derivation is rather technical and not straightforward to communicate. This class of models are well suited for theoretical considerations. For practical purposes, simpler model structures seem to be desirable.

At that point, the phenomenological approach comes in. Here, the models are mostly compartmental (ODE) models, as often used in ecology and epidemiology. These models can be readily analysed and simulated. In that, practical applications of such simple models are easily possible, which is the strength of the phenomenological approach. The drawback is the fact that they are not rooted in first principles. It is hard to assess whether the model structure appropriately reflects reality. E.g., if CT is formulated as a linear term, this formulation is contradicting the observation that CT is based on correlations and dependencies between index cases and their infectees/infector (as a rule, dependencies are expressed by nonlinear terms). Another aspect is the parametrization of the models: Some central parameters can be obtained by observations on the micro scale; e.g., the probability of detecting a contact can be estimated using the distribution of detected cases per index case. As phenomenological approaches dismiss the micro-scale and directly jump to the macro-scale, information that is available on the micro-scale is hard to incorporate.

Easy to use models based on first principles that are well accepted and approved by the modelling community are necessary but not yet in sight.

Backward/Forward tracing. Even in one of the very first papers about CT, the seminal work by Hethcote and Yorke (Hethcote & Yorke, 1984), the distinction between backward and forward tracing is mentioned. Backward tracing means that the infector is detected by an infectee who becomes an index case, while in forward tracing the infector is the index case, while the infectee is detected. Since publication of this first article, the relative importance of backward- and forward tracing is under discussion. An individual only has one infector, but in general several infectees. This fact might indicate that forward tracing is more important. On the other hand, if we randomly select an individual in a natural contact graph, the neighbour of this individual will on average have more contacts than an average individual. This finding, also called the "friendship paradox" in the context of social networks, can be found back in epidemiological data, e.g. about influenza (Christakis & Fowler, 2010). This observation indicates that backward tracing also is of importance, as most likely we will find persons who have many contacts and already had the chance to infect many of them. This is especially central for CT in sexually transmitted infections, which are often circulating in highly connected core groups, or for CT in the context of super-spreader events.

Contact structure. Realistic social network graphs represent different, overlapping circles: Family, school, workplace, neighbourhood, and so on. We find clusters that are weakly connected. This contact structure affects the impact of CT (Kiss et al., 2008), and also is a serious modelling challenge, as triangles will be frequently present but are difficult to handle. Not only the graph structure *per se*, but also the timing of contacts is more subtle than often formulated in models. For example, school children share a classroom, and in that, all children have simultaneously contact in case of airborne infections. In contrast, most models assume that contacts occur independently and uncorrelated on different edges of the contact graph. Even more, the contact graph itself is dynamic - some edges will be dissolved and new edges are realised (e.g., if a person changes the workplace). Depending on the epidemiological parameters (for example, HIV has a long infectious period) and the time scale of the epidemic, these mechanisms might have a notable effect.

Super-spreaders. In super-spreader events, many individuals are infected by a single person. The significance of these events became particularly clear in the paper by Llyod-Smith et al. (Lloyd-Smith, Schreiber, Kopp, & Getz, 2005). Super-spreading can have several reasons: Heterogeneity in the immune response (some persons with a high pathogen load are

highly infectious), heterogeneity in the contact graph (commercial sex-workers are candidates for super-spreaders), or the dynamics in the contact process (an asymptomatic SARS-CoV-2 infected person practices in a choir).

The literature reports contradicting results concerning CT and super-spreaders. E.g., Klinkenberg et al. (Klinkenberg et al., 2006) argue that backward tracing is readily able to identify super-spreaders, s.t. a combination of backward- and forward tracing is very efficient. In (Okolie & Müller, 2020), quantitative comparisons (based on analytic results for a branching-process model for CT on random tees) indicate that the effect of CT decreases with the variance of the degree distribution, indicating that CT is less efficient for super-spreaders. Kiss et al. (2008) point out that the performance of CT depends on the contact graph structure (assortative or disassortative). Müller & Hösel (2020) claim that CT is based on different mechanisms in the case of synchronous super-spreader events (e.g., a party) than in normal situations. Obviously, the mechanism that generates super-spreader events, the epidemiological time course (latency/incubation period), the timing of contacts, and the structure of the contact graph are intertwined with CT. There is a need to investigate the effect of super-spreaders on CT more in depth.

Endemic equilibrium. CT can also be performed if the infectious process is at its endemic equilibrium. In this case, each infected individual on average has only one infector and one infectee ($R_{eff} = 1$). Why does CT pay in that situation? For sexually transmitted infections (STI), it is clear that the partner is at high risk, and a couple should rather be considered as a single entity. Partner notification in faithful pairs is a simple case of CT. It is more interesting to note that for some STIs asymptomatic persons can be infectious for a long time (several months). In that case, particularly the tracing of asymptomatic persons may be the main advantage. CT is a method to find these highly infectious persons with possibly many contacts, who are difficult to localise otherwise.

Effort required for CT. We believe that CT is a highly effective control measure, if incidence and prevalence are low. In contrast, for a high prevalence, simple screening will be better suited. Armbruster et al. investigated and compared these two control measures in a series of papers (Armbruster and Brandeau, 2007a, 2007b, 2010). Another control measure, which combines elements of CT and of screening, is clustering, which exploits the heterogeneity of contact structure: Data, e.g. obtained by CT, allow to identify subgroups with a high prevalence, which will subsequently be screened. This procedure saves resources and could be particularly suitable in a medium range of prevalence.

However, the question is more complex. Even in a situation of low prevalence, the resources required to maintain an adequate level of CT might exceed the existing capacities. For example, during the early phase of the SARS-CoV-2 epidemic, Public Health Units in New Zealand were able to handle only less than 100 index cases per day, a number, that was exceeded already in March 2020 (Verrall, 2020). Similarly, a report about an Ebola outbreak in Sierra Leone indicated an overload of the public health system even at low case numbers (Olu et al., 2016). Only few models take the limited capacity of the public health system into account (Hellewell et al., 2020; Kaplan et al., 2003). It is a largely neglected problem of practical importance to predict the number of tracing teams necessary to control infections chains. This number will heavily depend on the infection (airborne infections might require a higher effort than sexually transmitted diseases), and on the timing of latent and incubation period (Fraser et al., 2004; Klinkenberg et al., 2006). Furthermore, the available tools (how reliable are tests, the efficiency of transport and communication system e.g. in developing countries, digital CT (Braithwaite, Callender, Bullock, & Aldridge, 2020; Kretzschmar et al., 2020b), etc.) play a role. To develop realistic and reliable prediction and planing tools for the necessary resources is a challenging task.

Tracing probability. In order to monitor the effect of CT, it is desirable to estimate the tracing probability, that is, the fraction of identifiable contacts among all infectious contacts. While data for the number of detected cases are available, the number of missed cases is usually unknown. In (Blum & Tran, 2010; Müller & Hösel, 2007), some statistical methods are developed to estimate the tracing probability (a maximum-likelihood estimator in (Müller & Hösel, 2007), and an approximate Bayesian computation/ABC method in (Blum & Tran, 2010)). A related problem is the estimation of the abundance of asymptomatic cases from tracing data. Dyson et al. (2017) propose a method based on household models for Yaws, a disabling bacterial infection, while Tanaka (Tanaka et al., 2020) aims to estimate the percentage of asymptomatic cases for COVID-19 using the branching process approach for CT. However, these questions are rather neglected by the recent literature and deserve a deeper investigation.

3.2. CT and genetic sequence data

In recent years, genetic sequencing of pathogen DNA became rather cheap, and genetic sequence data are readily available. Methods of population genetics are used to, e.g., estimate the prevalence of an infection (Frost & Volz, 2013). Genetic sequence data is also used to identify clusters of infections, and even to identify and refine transmission trees (infector/infectee relations) within a cluster (Pasquale et al., 2020). However, the combination of data from CT and from sequencing techniques is hardly exploited by now. It is natural to ask what epidemiology could gain from that combination.

Clearly, from samples of infector-infectee pairs, the mutation rate of the pathogen can be estimated. Moreover, as an infection event forms a bottle neck for the population of the pathogen, the time since infection can be estimated, and in that, it is possible to narrow down the time of the infectious contact. A comparison of different infector/infectee pairs might help to sharpen the estimations for the prevalence.

The usage of genetic sequence data together with methods from population genetics is rather young, and many powerful methods — as the SMC (McVean & Cardin, 2005) — are rather recent developments. We expect that useful tools become available in the near future.

3.3. Digital CT

The idea to use data from mobile phones to trace contacts is rather recent. First practical attempts to use digital sensors (RFID chips) to observe contacts and to investigate an empirically validated contact network in relatively small communities (school, hospital, conference) reach back to 2010 (Isella et al., 2011; Salathe et al., 2010; Stehlé et al., 2011). Soon it became clear that risk evaluation based on mobile phone data is possible and useful. Perhaps the first paper that considered modelling digital CT (DCT) was an IBM simulation study in 2014 (Farrahi, Emonet, & Cebrian, 2014). There is almost no practical experience with DCT, only semi-automated CT supported by digital techniques has been used in the Ebola outbreak 2014–2016 (see review article (Braithwaite et al., 2020) and quotations therein). Several manuscripts in the context of SARS-CoV-2 also address DCT (see the review article (Braithwaite et al., 2020) and SI).

DCT could help to improve some crucial shortcomings of classical CT. The advantages of DCT are the rapid identification of infectious contacts, and the possibility to also rapidly inform contactees. In comparison with conventional CT, DCT has the potential to strongly reduce the tracing delay, and to increase the tracing probability. In that, infections may become controllable that were not controllable before. However, these potential benefits come with a number of technical, social, medical, and practical challenges. Contacts identified by mobile devices need to correlate with infectious contacts. A rapid and cheap test for the infection is necessary, as the number of persons to test will be much higher than in conventional CT. Aspects of privacy and data protection are crucial elements of DCT. Apps need to be accepted in the society. The concept of DCT, its strengths and its weaknesses have to be communicated clearly, in a way that citizens understand and accept DCT. The system can only work if a large part of the population participates in DCT.

In the present note, we rather focus on new modelling challenges posed by DCT, and less on the social and technical aspects, though all of these points are intertwined.

Correlation between individuals. DCT promises to overcome some of the logistic problems of CT and to allow for identification of a huge number of contacts, even if a high number of infected individuals are present. The number of reported contacts per day and person in Europe is in the magnitude of 10 (Mossong et al., 2008), with a large standard deviation (which is in the same range as the average number of contacts). If we have a tracing window of one week, we easily estimate 70 contact persons per index case (with a high variance). Depending on the nature of the infection, we can (and should!) immediately inform not only the direct contactees but also the contactees of the contactees (second level tracing). In that, we easily arrive at 500 direct and indirect contactees per index case. From a practical perspective, it is of utmost importance to identify features of an infection that imply the necessity of first- second- or even higher-level tracing.

The difference to the conventional procedure is the possibility for rapid and immediate information of direct and indirect contactees, without waiting for medical tests and diagnoses. Even in a moderate outbreak, it is likely that the groups of contactees related to different index cases will overlap. These effects tremendously complicate the analysis of the models. A clear challenge is the identification of techniques that allow for the analysis of this situation.

High number of contactees/practical protocol. As discussed above, DCT can identify a high number of contracts and inform them about direct or indirect infectious contacts. From practical considerations, a reduction of that number is desirable. Technical devices may give a score to each contact, and estimate the probability of infection. That might help, but it also might be the case that scores are not reliable.

We are faced with a serious practical problem: If all casual contacts are reported, many persons might receive repeatedly a warning. In this case fatigue sets in and warnings are no longer taken seriously. DCT becomes useless. A way to escape this mechanism is the choice of smaller subgroups from the set of contactees. One possibility could be simply a random sub-set. More effective is a classification of contactees in different risk groups, e.g. according to the number of recent contacts. Particularly, asymptomatic persons that spread the infection for a longer time or super-spreaders will appear among repeatedly identified contactees.

Modelling approaches need to clarify the number of warnings a person receives under realistic conditions. In consequence, different strategies of reducing the number of contactees to be informed have to be defined. Models allow to estimate the impact of these strategies, and to filter out reasonable policies for DCT.

DCT induce heterogeneity in the population. The population is divided in a subpopulation with, and a subpopulation without a tracing device. In that, contacts within the DCT-subpopulation are readily identified, while contacts between the two subpopulations or among the non-DCT-subpopulation can only be identified by conventional CT. It is likely that acceptance of a tracing device correlates with social factors as education, economic status, political or religious orientation. In that, connected subgroups appear that escape the detection by DCT. These subgroups might form a reservoir for the infection, from which importation to the general population may occur. The fraction of the population equipped with DCT-equipment alone is not decisive, but also its distribution in a heterogeneous population. However, even for a population without other heterogeneities, the division into two subgroups might lead to unforeseen effects. This aspect is a new one that deserves deeper investigation.

4. Conclusion

The joint effort of modelling and theoretical investigation of CT has borne many fruits, as the estimation of the impact of CT on the spread of infections, or methods to assess the influence of the tracing delay. Nevertheless, questions of practical importance still deserve our attention. New challenges, e.g. due to developments in genetic sequencing of pathogens and DCT,

have arisen. Simulation models promise first answers on a rapid time scale, but it will take some time until these new aspects are fully understood, and this understanding is translated into practice.

Declaration of competing interest

The authors declare no conflict of interest.

Acknowledgements

This review article is supported by a grant from the Deutsche Forschungsgemeinschaft (DFG) through TUM International Graduate School of Science and Engineering (IGSSE), GSC 81, within the project GENOMIE QADOP (JM). MK was supported by two grants from The Netherlands Organisation for Health Research and Development (ZonMw), grant number 10430022010001, and grant number 91216062, and by the H2020 project 101003480 (CORESMA).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.idm.2020.12.005.

References

Agarwal, M., & Bhadauria, A. S. (2012). Modeling H1N1 flu epidemic with contact tracing and quarantine. *International Journal of Biomathematics*, 1250038, 05.
Althaus, C. L., Heijne, J. C., Herzog, S. A., Roellin, A., & Low, N. (2012). Individual and population level effects of partner notification for Chlamydia trachomatis. *PloS One, 7*.
Aparicio, J. P., & Hernández, J. C. (2006). Preventive treatment of tuberculosis through contact tracing. *Contemporary Mathematics, 410*, 17–29.
de Arazoza, H., & Lounes, R. (2002). A non-linear model for a sexually transmitted disease with contact tracing. *IMA Journal of Maths with Applications in Medicine & Biology, 19*, 221–234.
Armbruster, B., & Brandeau, M. L. (2007a). Contact tracing to control infectious disease: When enough is enough. *Health Care Management Science, 10*, 341–355.
Armbruster, B., & Brandeau, M. L. (2007b). Optimal mix of screening and contact tracing for endemic diseases. *Mathematical Biosciences, 209*, 386–402.
Armbruster, B., & Brandeau, M. L. (2010). Cost-effective control of chronic viral diseases: Finding the optimal level of screening and contact tracing. *Mathematical Biosciences, 224*, 35–42.
Ball, F., & Donnelly, P. (1995). Strong approximations for epidemic models. *Stochastic Processes and their Applications, 55*, 1–21.
Ball, F. G., Knock, E. S., & O'Neill, P. D. (2011). Threshold behaviour of emerging epidemics featuring contact tracing. *Advances in Applied Probability, 43*, 1048–1065.
Ball, F. G., Knock, E. S., & O'Neill, P. D. (2015). Stochastic epidemic models featuring contact tracing with delays. *Mathematical Biosciences, 266*, 23–35.
Barlow, M. T. (2020). *A branching process with contact tracing*. arXiv preprint.
Baumgarten, L., & Bornholdt, S. (2020). *Epidemics with asymptomatic transmission: Sub-critical phase from recursive contact tracing*. arXiv preprint.
Becker, N. G., Glass, K., Li, Z., & Aldis, G. K. (2005). Controlling emerging infectious diseases like SARS. *Mathematical Biosciences, 193*, 205–221.
Begun, M., Newall, A. T., Marks, G. B., & Wood, J. G. (2013). Contact tracing of tuberculosis: A systematic review of transmission modelling studies. *PloS One, 8*, Article e72470.
Berge, T., Tassé, A. J. O., Tenkam, H. M., & Lubuma, J. (2018). Mathematical modeling of contact tracing as a control strategy of Ebola virus disease. *International Journal of Biomathematics, 11*, 1850093.
Blum, M. G. B., & Tran, V. C. (2010). HIV with contact tracing: A case study in approximate bayesian computation. *Biostatistics, 11*, 644–660.
Braithwaite, I., Callender, T., Bullock, M., & Aldridge, R. W. (2020). Automated and partly automated contact tracing: A systematic review to inform the control of COVID-19. *Lancet Digit Health, 2*(11), E607–E621.
Browne, C., Gulbudak, H., & Webb, G. (2015). Modeling contact tracing in outbreaks with application to Ebola. *Journal of Theoretical Biology, 384*, 33–49.
Chen, S. C., Chang, C. F., & Liao, C. M. (2006). Predictive models of control strategies involved in containing indoor airborne infections. *Indoor Air, 16*, 469–481.
Christakis, N. A., & Fowler, J. H. (2010). Social network sensors for early detection of contagious outbreaks. *PloS One, 5*, Article e12948.
Clarke, J., White, K. A. J., & Turner, K. (2012). Exploring short-term responses to changes in the control strategy for Chlamydia trachomatis. *Computational and Mathematical Methods in Medicine, 2012*, 1–10.
Clarke, J., White, K. A. J., & Turner, K. (2013). Approximating optimal controls for networks when there are combinations of population-level and targeted measures available: Chlamydia infection as a case-study. *Bulletin of Mathematical Biology, 75*, 1747–1777.
Clémençon, S., Tran, V. C., & de Arazoza, H. (2008). A stochastic SIR model with contact-tracing: Large population limits and statistical inference. *Journal of Biological Dynamics, 2*, 392–414.
Dyson, L., Marks, M., Crook, O. M., Sokana, O., Solomon, A. W., Bishop, A., et al. (2017). Targeted treatment of Yaws with household contact tracing: How much do we miss? *American Journal of Epidemiology, 187*, 837–844.
Eames, K. (2007). Contact tracing strategies in heterogeneous populations. *Epidemiology and Infection, 135*, 443–454.
Eames, K. T. D., & Keeling, M. J. (2002). Modeling dynamic and network heterogeneities in the spread of sexually transmitted diseases. *Proceedings of the National Academy of Sciences, 99*, 13330–13335.
Eames, K. T., & Keeling, M. J. (2003). Contact tracing and disease control. *Series B: Biological Sciences, 270*, 2565–2571.
Eichner, M. (2003). Case isolation and contact tracing can prevent the spread of smallpox. *American Journal of Epidemiology, 158*, 118–128.
Farrahi, K., Emonet, R., & Cebrian, M. (2014). Epidemic contact tracing via communication traces. *PloS One, 9*, Article e95133.
Ferretti, L., Wymant, C., Kendall, M., Zhao, L., Nurtay, A., Abeler-Dörner, L., et al. (2020). Quantifying SARS-CoV-2 transmission suggests epidemic control with digital contact tracing. *Science, 368*, Article eabb6936.
Fraser, C., Riley, S., Anderson, R. M., & Ferguson, N. M. (2004). Factors that make an infectious disease outbreak controllable. *Proceedings of the National Academy of Sciences, 101*, 6146–6151.
Frost, S. D. W., & Volz, E. M. (2013). Modelling tree shape and structure in viral phylodynamics. *Philosophical Transactions of the Royal Society B: Biological Sciences, 368*, 20120208.
Ghani, A. C., Swinton, J., & Garnett, G. P. (1997). The role of sexual partnership networks in the epidemiology of gonorrhea. *Sexually Transmitted Diseases, 24*, 45–56.

Giordano, G., Blanchini, F., Bruno, R., Colaneri, P., Di Filippo, A., Di Matteo, A., et al. (2020). Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy. *Nature Medicine*.
Heffernan, C., & Dunningham, J. A. (2009). Simplifying mathematical modelling to test intervention strategies for Chlamydia. *Journal of Public Health and Epidemiology, 1*, 022–030.
Heijne, J. C., Althaus, C. L., Herzog, S. A., Kretzschmar, M., & Low, N. (2011). The role of reinfection and partner notification in the efficacy of Chlamydia screening programs. *The Journal of Infectious Diseases, 203*, 372–377.
Hellewell, J., Abbott, S., Gimma, A., Bosse, N. I., Jarvis, C. I., Russell, T. W., et al. (2020). Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts. *The Lancet Global Health, 8*, e488–e496.
Hethcote, H. W., & Yorke, J. A. (1984). Gonorrhea transmission dynamics and control. *Lecture notes in biomathematics, 56*.
House, T., & Keeling, M. J. (2010). The impact of contact tracing in clustered populations. *PLoS Computational Biology, 6*, Article e1000721.
House, T., & Keeling, M. J. (2011). Insights from unifying modern approximations to infections on networks. *Journal of The Royal Society Interface, 8*, 67–73.
Hsieh, Y. H., de Arazoza, H., Lounes, R., & Joanes, J. (2005). A class of methods for HIV contact tracing in Cuba: Implications for intervention and treatment. In *Deterministic and stochastic models of AIDS epidemics and HIV infections with intervention* (pp. 77–92). World Scientific.
Hsieh, Y. H., Wang, Y. S., de Arazoza, H., & Lounes, R. (2010). Modeling secondary level of HIV contact tracing: Its impact on HIV intervention in Cuba. *BMC Infectious Diseases, 10*.
Huerta, R., & Tsimring, L. S. (2002). Contact tracing and epidemics control in social networks. *Physical Review E - Statistical Physics, Plasmas, Fluids, and Related Interdisciplinary Topics, 66*.
Hyman, J. M., Li, J., & Stanley, E. A. (2003). Modeling the impact of random screening and contact tracing in reducing the spread of HIV. *Mathematical Biosciences, 181*, 17–54.
Isella, L., Romano, M., Barrat, A., Cattuto, C., Colizza, V., den Broeck, W. V., et al. (2011). Close encounters in a pediatric ward: Measuring face-to-face proximity and mixing patterns with wearable sensors. *PloS One, 6*, Article e17144.
Kaplan, E. H., Craft, D. L., & Wein, L. M. (2002). Emergency response to a smallpox attack: The case for mass vaccination. *Proceedings of the National Academy of Sciences, 99*, 10935–10940.
Kaplan, E. H., Craft, D. L., & Wein, L. M. (2003 sep). Analyzing bioterror response logistics: The case of smallpox. *Mathematical Biosciences, 185*(1), 33–72.
Kasaie, P., Andrews, J. R., Kelton, W. D., & Dowdy, D. W. (2014). Timing of tuberculosis transmission and the impact of household contact tracing. An agent-based simulation model. *American Journal of Respiratory and Critical Care Medicine, 189*, 845–852.
Keeling, M. J. (1999). Correlation equations for endemic diseases: Externally imposed and internally generated heterogeneity. *Series B: Biological Sciences, 266*, 953–960.
Keeling, M. J., & Eames, K. T. (2005). Networks and epidemic models. *Journal of The Royal Society Interface, 2*, 295–307.
Keeling, M., Rand, D., & Morris, A. (1997). Correlation models for childhood epidemics. *Series B: Biological Sciences, 264*, 1149–1156.
Kiss, I. Z., Green, D. M., & Kao, R. R. (2006). Infectious disease control using contact tracing in random and scale-free networks. *Journal of The Royal Society Interface, 3*, 55–62.
Kiss, I. Z., Green, D. M., & Kao, R. R. (2008). The effect of network mixing patterns on epidemic dynamics and the efficacy of disease contact tracing. *Journal of The Royal Society Interface, 5*, 791–799.
Klinkenberg, D., Fraser, C., & Heesterbeek, H. (2006). The effectiveness of contact tracing in emerging epidemics. *PloS One, 1*, e12.
Kojaku, S., Hébert-Dufresne, L., & Ahn, Y. Y. (2020). *The effectiveness of contact tracing in heterogeneous networks*. arXiv preprint.
Kretzschmar, M. E., Rozhnova, G., Bootsma, M. C. J., van Boven, M., van de Wijgert, J. H. H. M., & Bonten, M. J. M. (2020). Impact of delays on effectiveness of contact tracing strategies for COVID-19: A modelling study. *The Lancet Public Health, 5*, e452–e459.
Kretzschmar, M. E., Rozhnova, G., & van Boven, M. E. (2020). *Isolation and contact tracing can tip the scale to containment of COVID-19 in populations with social distancing*. medRxiv preprint.
Kretzschmar, M., Satterwhite, C., Leichliter, J., & Berman, S. (2012). Effects of screening and partner notification on Chlamydia positivity in the United States: A modeling study. *Sexually Transmitted Diseases, 39*, 325–331.
Kretzschmar, M., Turner, K. M. E., Barton, P. M., Edmunds, W. J., & Low, N. (2009). Predicting the population impact of chlamydia screening programmes: Comparative mathematical modelling study. *Sexually Transmitted Infections, 85*, 359–366.
Kretzschmar, M., van Duynhoven, Y. T., & Severijnen, A. J. (1996). Modeling prevention strategies for Gonorrhea and Chlamydia using stochastic network simulations. *American Journal of Epidemiology, 144*, 306–317.
Kretzschmar, M., Van den Hof, S., Wallinga, J., & Van Wijngaarden, J. (2004). Ring vaccination and smallpox control. *Emerging Infectious Diseases, 10*, 832.
Kwok, K. O., Tang, A., Wei, V. W. I., Park, W. H., Yeoh, E. K., & Riley, S. (2019). Epidemic models of contact tracing: Systematic review of transmission studies of severe acute respiratory syndrome and Middle East respiratory syndrome. *Computational and Structural Biotechnology Journal, 17*, 186–194.
Liu, F., Enanoria, W. T. A., Zipprich, J., Blumberg, S., Harriman, K., Ackley, S. F., et al. (2015). The role of vaccination coverage, individual behaviors, and the public health response in the control of measles epidemics: An agent-based simulation for California. *BMC Public Health, 15*.
Lloyd-Smith, J. O., Galvani, A. P., & Getz, W. M. (2003). Curtailing transmission of severe acute respiratory syndrome within a community and its hospital. *Series B: Biological Sciences, 270*, 1979–1989.
Lloyd-Smith, J. O., Schreiber, S. J., Kopp, P. E., & Getz, W. M. (2005). Superspreading and the effect of individual variation on disease emergence. *Nature, 438*, 355–359.
Lunz, D., Batt, G., & Ruess, J. (2020). *To isolate, or not to isolate: A theoretical framework for disease control via contact tracing*. medRxiv preprint.
McVean, G. A. T., & Cardin, N. J. (2005). Approximating the coalescent with recombination. *Philosophical Transactions of the Royal Society B: Biological Sciences, 360*, 1387–1393.
Mellor, G. R., Currie, C. S. M., & Corbett, E. L. (2011). Incorporating household structure into a discrete-event simulation model of tuberculosis and HIV. *ACM Transactions on Modeling and Computer Simulation, 21*, 1–17.
Meyers, L. A., Pourbohloul, B., Newman, M. E. J., Skowronski, D. M., & Brunham, R. C. (2005). Network theory and SARS: Predicting outbreak diversity. *Journal of Theoretical Biology, 232*, 71–81.
Mossong, J., Hens, N., Jit, M., Beutels, P., Auranen, K., Mikolajczyk, R., et al. (2008). Social contacts and mixing patterns relevant to the spread of infectious diseases. *PLoS Medicine, 5*, e74.
Müller, J., & Hösel, V. (2007). Estimating the tracing probability from contact history at the onset of an epidemic. *Mathematical Population Studies, 14*, 211–236.
Müller, J., & Hösel, V. (2020). *Contact tracing & super-spreaders in the branching-process model*. arXiv preprint.
Müller, J., & Koopmann, B. (2016). The effect of delay on contact tracing. *Mathematical Biosciences, 282*, 204–214.
Müller, J., Kretzschmar, M., & Dietz, K. (2000). Contact tracing in stochastic and deterministic epidemic models. *Mathematical Biosciences, 164*, 39–64.
Naresh, R., Tripathi, A., & Sharma, D. (2011). A nonlinear HIV/AIDS model with contact tracing. *Applied Mathematics and Computation, 217*, 9575–9591.
Newman, M. E., Strogatz, S. H., & Watts, D. J. (2001). Random graphs with arbitrary degree distributions and their applications. *Physical Review E - Statistical Physics, Plasmas, Fluids, and Related Interdisciplinary Topics, 64*, Article 026118.
Okolie, A., & Müller, J. (2020). Exact and approximate formulas for contact tracing on random trees. *Mathematical Biosciences, 321*, 108320.
Olu, O. O., Lamunu, M., Nanyunja, M., Dafae, F., Samba, T., Sempiira, N., et al. (2016). Contact tracing during an outbreak of Ebola virus disease in the western area districts of Sierra Leone: Lessons for future Ebola outbreak response. *Front Public Health, 4*, 130.
Pasquale, D. K., Doherty, I. A., Miller, W. C., Leone, P. A., Sampson, L. A., Ledford, S. L., et al. (2020). Factors associated with human immunodeficiency virus infections linked in genetic clusters but disconnected in partner tracing. *Sexually Transmitted Diseases, 47*, 80–87.
Peak, C. M., Childs, L. M., Grad, Y. H., & Buckee, C. O. (2017). Comparing nonpharmaceutical interventions for containing emerging epidemics. *Proceedings of the National Academy of Sciences, 114*, 4023–4028.

Porco, T. C., Holbrook, K. A., Fernyak, S. E., Portnoy, D. L., Reiter, R., & Aragón, T. J. (2004). Logistics of community smallpox control through contact tracing and ring vaccination: A stochastic network model. *BMC Public Health, 4*.
Salathe, M., Kazandjieva, M., Lee, J. W., Levis, P., Feldman, M. W., & Jones, J. H. (2010). A high-resolution human contact network for infectious disease transmission. *Proceedings of the National Academy of Sciences, 107*, 22020–22025.
Sato, K., Matsuda, H., & Sasaki, A. (1994). Pathogen invasion and host extinction in lattice structured populations. *Journal of Mathematical Biology, 32*, 251–268.
Shahtori, N. M., Ferdousi, T., Scoglio, C., & Sahneh, F. D. (2018). Quantifying the impact of early-stage contact tracing on controlling Ebola diffusion. *Mathematical Biosciences and Engineering, 15*, 1165–1180.
Stehlé, J., Voirin, N., Barrat, A., Cattuto, C., Colizza, V., Isella, L., et al. (2011). Simulation of an SEIR infectious disease model on the dynamic contact network of conference attendees. *BMC Medicine, 9*, 87.
Tanaka, T., Yamaguchi, T., & Sakamoto, Y. (2020). Estimation of the percentages of undiagnosed patients of the novel coronavirus (SARS-CoV-2) infection in Hokkaido, Japan by using birth-death process with recursive full tracing. *PloS One, 15*, Article e0241170.
Tian, Y., Alawami, F., Al-Azem, A., Osgood, N., Hoeppner, V., & Dutchyn, C. (2011). A system dynamics model of tuberculosis diffusion with respect to contact tracing investigation. In *Proceedings of the 2011 winter simulation conference (WSC)*. IEEE, Article e72470.
Tian, Y., Osgood, N. D., Al-Azem, A., & Hoeppner, V. H. (2013). Evaluating the effectiveness of contact tracing on tuberculosis outcomes in saskatchewan using individual-based modeling. *Health Education & Behavior, 40*, 98S–110S.
Tsimring, L. S., & Huerta, R. (2003). Modeling of contact tracing in social networks. *Physica A, 325*, 33–39.
Turner, K. M. E., Adams, E., Gay, N., Ghani, A., Mercer, C., & Edmunds, W. J. (2006). Developing a realistic sexual network model of chlamydia transmission in Britain. *Theoretical Biology and Medical Modelling, 3*, 3.
Verrall, A. (2020). *Rapid audit of contact tracing for COVID-19 in New Zealand*. New Zealand: University of OtagoWellington.
Wilkinson, R. R., Ball, F. G., & Sharkey, K. J. (2017). The relationships between message passing, pairwise, Kermack–McKendrick and stochastic SIR epidemic models. *Journal of Mathematical Biology, 75*, 1563–1590.