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# Preface

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The insights provided by including contact networks in the study of the spread of infectious diseases have been long recognized by those working on sexually transmitted infections (STIs). For such diseases, contact tracing is an important strategy for their control and it renders detailed contact networks (see, for instance, [5]). Indeed, some of the findings that researchers on STIs obtained in the 1970s and 1980s such as, for instance, the high impact of contact heterogeneity [1], the disproportionate contribution of the so-called core groups to the spread of STIs [17], or even the expression for the basic reproduction number $R_0$ in populations with a given distribution of contact rates (degree distribution) [2], have been rediscovered since 2000 as network theory became widely adopted in modelling the spread of infections in human populations and computer networks. This has been specially notorious since the occurrence of the SARS epidemic in the south of China in 2003. This epidemic provided documented examples of super-spreading events [13] in respiratory-based diseases and it showed that neglecting social contact heterogeneity leads to wrong estimates of the early epidemic growth [7]. Since then, network epidemiology has become an important research topic in mathematical biology because it leads to better models and predictions while posing challenging questions in both stochastic and deterministic modelling approaches [3].

The papers selected for this volume cover a wide range of modelling issues that are currently addressed by researchers in the field. The topics of the articles presented span some methodologically motivated ones to others related to aspects human behaviour, spatial networks, and real-life epidemics.

Miller and Kiss [9] review some of the current approaches and challenges in the field. In particular, the authors focus on three classes of models, namely, pairwise models, effective degree models, and edge-based compartmental models (EBCM). A hierarchy among different models is established according to the assumptions used to derive the mathematically simpler model. A main challenge for the authors is to develop a modelling framework that circumvents the hypothesis of independence among the disease states of the neighbours of a node, a central issue if one wants to deal with clustered networks and with models for SIS-type epidemic spread.

Another approach to the study of epidemic dynamics is based on the derivation of the master equation for the probability of having $k$ infectious nodes at time $t$ in a population under the chosen modelling assumptions. Nagy, Kiss and Simon [10] adopt this approach and extend the classical formulation for fully connected networks to include infection rates that depend on the topology of the network. This

improved version of the master equation relies, for SIS epidemics, on a theoretical approximation of the average number of links between susceptible and infectious nodes at time $t$.

Rattana, Miller, and Kiss [11] consider EBCM and pairwise models to describe SIR epidemics on weighted networks. The distribution of weights among the links is assumed to be either random or degree-dependent. In both cases, expressions for the basic reproduction number $R_0$ and for the final epidemic size are derived from EBCM, which are more amenable to computation than pairwise models. Finally, the equivalence between both modelling approaches is numerically shown by comparing them with stochastic simulations of the epidemic on networks generated according to the configuration model, and a formal proof of such model equivalence is sketched.

Another way to associate weights to links is to assign to each individual a risk factor drawn from a probability distribution and, then, define a combined risk behaviour of the individuals involved in a potential contact as an increasing function of the individual risk factors. This approach is adopted by Brandonjić [12] who considers the random connection of individuals located at points on $\mathbf{R}^2$ generated according to a Poisson point process with intensity $\lambda$. The probability of connecting a pair of individuals is assumed to be equal to the product of a non-increasing function of their Euclidean distance times their combined risk behaviour. Under this assumption it is proved that, above a critical intensity $\lambda_c$, there exists an infinite component in the weighted random connection model.

A classic problem in network epidemiology is the study of the impact of the network architecture on the epidemic dynamics. This is the aim of the paper [16] by Szabó-Solticzky and Simon who study the epidemic spread in a class of 3-regular cycle graphs. These graphs are circles of $N$ nodes (labelled from 1 to $N$) where, for a fixed $d$, node $i$ ($i = 1, \ldots, d, 1 + 2d, \ldots, 1 + 3d$, etc.) is connected to its two nearest neighbours and to the node $i + d$ in such a way that every node has exactly degree 3. The resulting graph has $N/(2d)$ subgraphs of size $2d$ lying along a circle. The authors obtain an analytic estimation of the passing time of the infection through each subgraph and derive a model for the epidemic propagation in the whole cycle graph.

The 2003 SARS epidemic, as well as other epidemics like the 2009 H1N1 influenza A, vividly examplified the discontinuous nature of the geographical advance of epidemics in humans at a global scale. Their dynamics have been recently described in terms of metapopulations by several authors. Juher and Mañosa [6] analyse the epidemic threshold for an SIS-epidemic occurring on heterogeneous metapopulations whose architecture is statistically described in terms of complex networks. Moreover, extending previous models, the individual contact rate in each local population is assumed to be a non-decreasing function of its size. Their analysis is based on the characterization of the spectrum of the connectivity matrix for correlated (assortative or disassortative) networks.

The role of human behaviour in epidemic progress is of current interest in epidemiology. Romero-Severson, Meadors and Volz [14] consider random changes in contact rates occurring at the individual level in long-lived infections, such as those caused by HIV, after a period of stable behaviour over which the contact rates remain constant. By using the generating function formalism and assuming exponentially distributed periods of stable behaviour, the authors the derive an expression for the random number of infections generated by newly-infected individuals, compute its expectation, i.e. the basic reproduction number, its variance, and the probability of an epidemic.

Sahneh *et al.* [15] study the impact of behavioural responses on the epidemic threshold by introducing the class of alerted individuals in a network epidemic model with two layers. One layer corresponds to a network of physical contacts through which infection transmission occurs. The second layer is an additional network, with the same set of nodes as the contact network, which accounts for the information dissemination about the health status of individuals.

The duration of the infectious period in most simple epidemic models is exponentially distributed. This is not the case in non-Markovian epidemic models. House [4] studies the equivalence between two non-Markovian stochastic formulations of an SIR epidemic in finite populations. He proves that the discrete heterogeneous Markovian epidemic (DHME), defined as a continuous-time Markov chain for the number of susceptible and infected individuals with degree $k$, is equivalent to the previous stochastic

formulations when the infectious periods are exponentially distributed. Finally, the deterministic limit of the DHME is obtained in the form of an ODE system.

McMahon *et al.* [8] investigate the spread of Rift Valley fever in East Africa with a multi-host network geographic epidemic model for the mosquito and host populations. Their detailed network model incorporates rainfall, land use, rule-based movement of the animal and human populations among geographic regions (nodes of the network), and the mosquito life cycle, including vertical transmission. The severity of the model epidemics is strongly correlated with the duration of the rainy season and herd immunity, created by past epidemic waves and vaccination, plays a significant role in the epidemic cycles. The model simulations indicate an effective a multi-layered mitigation strategy combines vector control, movement control of animals during an epidemic cycle, and the yearly vaccination of young animals.

We are very grateful to all the authors for their contributions, and we hope that readers will find the results contained in them useful and illuminating, as well as motivational for stimulating further research in this area.

## References


[1] R.M. Anderson. *The epidemiology if HIV infection: Variable incubation plus infectious periods and heterogeity in sexual activity.* J. R. Statist. Soc. A, 151 (1988), 66–93.

[2] R.M. Anderson, R.M. May. Infectious diseases of humans; dynamics and control, Oxford Univ. Press, Oxford, 1991.

[3] O. Diekmann, J.A.P. Heesterbeek, T. Britton, Mathematical tools for understanding infectious diseases dynamics, Princeton University Press, Princeton, 2013.

[4] T. House. *Non-Markovian stochastic epidemics in extremely heterogeneous populations.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 153–160.

[5] A.M. Jolly, J. L. Wylie. *Gonorrhoea and chlamydia core groups and sexual networks in Manitoba.* Sex. Transm. Infect. 78 (2002), i145–i151.

[6] D. Juher, V. Mañosa. *Spectral properties of the connectivity matrix and the SIS-epidemic threshold for mid-size metapopulations.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 108–120.

[7] L.A. Meyers, B. Pourbohloul, M.E.J. Newman, D.M. Skowronski, R.C. Brunham. *Network theory and SARS: predicting outbreak diversity.* J. Theor. Biol. 232 (2005), 71–81.

[8] B.H. McMahon, C.A. Manore, J.M. Hyman, M.X. LaBute, J.M. Fair. *Coupling vector-host dynamics with weather geography and mitigation measures to model Rift Valley fever in Africa.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 161–177.

[9] J.C. Miller, I.Z. Kiss. *Epidemic spread in networks: existing methods and current challenges.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 4–42.

[10] N. Nagy, I.Z. Kiss, P.L. Simon. *Approximate master equations for dynamical processes on graphs.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 43–57.

[11] P. Rattana, J.C. Miller, I.Z. Kiss. *Pairwise and edge-based models of epidemic dynamics on correlated weighted networks.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 58–81.

[12] M. Bradonjic. *Outbreak of infectious diseases through the weighted random connection model.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 82–88.

[13] S. Riley et al. *Transmission dynamics of the etiological agent of SARS in Hong Kong: Impact of public health interventions.* Science 300 (2003), 1961–1966.

[14] E.O. Romero-Severson, G.D. Meadors, E.M. Volz. *A generating function approach to HIV transmission with dynamic contact rates.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 121–135.

[15] F.D. Sahneh, F.N. Chowdhury, G. Brase, C.M. Scoglio. *Individual-based information dissemination in multilayer epidemic modeling.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 136–152.

[16] A. Szabó-Solticzky, P.L. Simon. *The effect of graph structure on epidemic spread in a class of modified cycle graphs.* Math. Model. Nat. Phenom., 9 (2014), no. 2, 89–107.

[17] J.A. Yorke, H.W. Hethcote, A. Nold. *Dynamics and control of the transmission of gonorrhea.* Sex. Transm. Dis. 5 (1978), 51–56.