Kinetic and macroscopic epidemic models in presence of multiple heterogeneous populations

Andrea Medaglia and Mattia Zanella

Abstract We study the impact of contact heterogeneity on epidemic dynamics. A system characterized by multiple susceptible populations is considered. The description of the spread of an infectious disease is obtained through the study of a system of Boltzmann-type equations for the number densities of social contacts of the introduced compartments. A macroscopic system of equations characterizing observable effects of the epidemic is then derived to assess the impact of contact heterogeneity.

1 Introduction

The recent efforts to design effective non-pharmaceutical measures to mitigate the COVID-19 pandemic were based on the link between social activities and the spreading of a respiratory disease [2]. Several works in mathematical epidemiology characterized the number of contacts of the population taking into account an additional structure that is maintained for the whole dynamics. A classical example is represented by age-structured populations for which realistic contact matrices have been determined, see e.g. [1, 15, 20]. Nevertheless, recent works highlighted strong changes in contact distribution in the early phases of an epidemic, whose evolution can shape the infection dynamics, see [25]. For these reasons, in [11] it has been proposed in a simple SIR-type compartmentalization a kinetic model to couple the dynamics of an infectious disease with the contact evolution of a system of agents. At the level of observable quantities, the emerging model is characterized not only by the evolution of densities, like for classical models in compartmental epidemiology, but also by the evolution of the mean number of connections. Interestingly enough, in
the present setting models with saturated incidence rates can be easily derived with minimal assumptions [7, 17, 24]. Other recent contributions stressed were centered on the effects of the structure of contacts of agents, we mention in this direction the works [12, 16, 19].

In the present contribution, we concentrate on the influence of contact heterogeneity on the dynamics of the disease in presence of multiple susceptible populations. Each susceptible compartment can be characterized by its mean number of connections. This situation is very common when non-pharmaceutical interventions have different impacts on the population [6] or in presence of sanitary cordon measures, where a portion of the territory results highly affected by the disease. We mention recent contributions in this direction using mobility data [4, 9, 13, 18, 22].

The mathematical tools that we consider are based on kinetic theory for large interacting systems [8, 14, 21] for which we are able to derive the evolution of observable quantities from microscopic, often unobservable, dynamics. In details, we will show how heterogeneity in the contact structure plays a central role in the evolution of an epidemic. In particular, preliminary results will highlight that, in several regimes of parameters, the asymptotic number of recovered can be unexpectedly high in societies with small contact heterogeneity, compared to the ones with high contact heterogeneity. These results are coherent with the recent findings presented in [5].

In more details, the contribution is organized as follows: in Section 2 we introduce a kinetic compartmental model of interest and we briefly discuss the contact formation dynamics. A macroscopic system of equations is then derived for the coupled evolution of mass fractions and the mean number of connections. In Section 3 we present some results highlighting the impact of contact heterogeneity in the evolution of the disease. Some conclusions and research are then reported in the last Section.

2 Interplay between contact distribution and epidemic dynamics

In this section, we introduce a kinetic model to describe the spreading of an infectious disease depending on an additional variable describing the number of social contacts. Coherently with the modeling approach introduced in the recent works [10], we subdivide the total population into three main compartments: susceptible, who can contract the disease, infected infectious, who can transmit the disease and recovered, corresponding to formerly infected patients that are not infectious. Furthermore, to mimic the early effects of the epidemic, where the collective compliance to reduce the number of daily contacts is often not accepted, we subdivide the susceptible population into two main categories $S_+, S_-$ in relation to their average number of contacts, $m_{S_+}$ and $m_{S_-}$ respectively.

The contact distribution of the whole population is therefore recovered as

$$f(w, t) = f_{S_+}(w, t) + f_{S_-}(w, t) + f_I(w, t) + f_R(w, t), \quad \int_{R_+} f(w, t) dw = 1.$$
Hence, we obtain the mass fractions of population in each compartment and their momentum of order $\alpha > 0$ as

$$J(t) = \int_{\mathbb{R}^+} f_J(w, t) dw, \quad J(t)m_{\alpha,J}(t) = \int_{\mathbb{R}^+} w^\alpha f_J(w, t) dw.$$ 

Unambiguously, in the following we will indicate the mean of contact in the compartment $J$, corresponding to $\alpha = 1$, by $m_J$.

### 2.1 Formation of the contact distribution

Coherently with [11], we can define a process of contact formation based on microscopic transitions for the variation of contacts of a single agent. At aggregate level, the evolution of the distributions $f_J$, $J \in \{S, I, R\}$, can be obtained through a Boltzmann-type equation. As shown in the aforementioned work, to obtain an explicit formulation of the large time distribution, it is possible to derive the following Fokker-Planck-type equation

$$\frac{\partial}{\partial t} f_J(w, t) = \frac{\lambda_J}{2} \frac{\partial}{\partial w} \left[ \left( \frac{w}{m_J} - 1 \right) f_J \right] + \frac{\sigma_J^2}{2} \frac{\partial^2}{\partial w^2} (w f_J(w, t)) \tag{1}$$

with $\lambda_J > 0$, $\sigma_J^2 > 0$ and $m_J > 0$ the mean number of contacts.

The emerging large time contact distributions $f_J^\infty(w)$, $J \in \{S, I, R\}$ of (1) can be explicitly computed and are of Gamma-type [23] coherently with experimental results in [2]. In particular, if $\mu_J = \lambda_J/\sigma_J^2 > 0$, we have

$$f_J^\infty(w) = \left( \frac{\mu_J}{m_J} \right)^{\mu_J} \frac{1}{\Gamma(\mu_J)} w^{\mu_J - 1} \exp \left\{ -\frac{\mu_J}{m_J} w \right\}, \tag{2}$$

whose momenta of order $\alpha$ are

$$\int_{\mathbb{R}^+} w^\alpha f_J^\infty(w) dw = \left( \frac{m_J}{\mu_J} \right)^\alpha \frac{\Gamma(\mu_J + \alpha)}{\Gamma(\mu_J)} = c_{\alpha,J} m_J^\alpha, \tag{3}$$

where $c_{J,\alpha} = \left( \frac{1}{\mu_J} \right)^\alpha \frac{\Gamma(\mu_J + \alpha)}{\Gamma(\mu_J)}$. Since $f_J^\infty$ is a Gamma distribution we also have

$$c_{\alpha+1,J} = \frac{\mu_J + \alpha}{\mu_J} c_{\alpha,J},$$

and $m_{\alpha+1,J} = m_{\alpha,J} \frac{\alpha + \mu_J}{\mu_J} m_J$. We remark that (2) is explicitly dependent on the positive parameter $\mu_J = \lambda_J/\sigma_J^2$ that measures the contact heterogeneity of a population in terms of the variance of the distribution of social contacts. More precisely,
small values of $\mu_f$ correspond to a larger heterogeneity of the individuals in terms of social contacts.

### 2.2 The kinetic model

The resulting system of kinetic equations is given by

\[
\begin{align*}
\partial_t f_{S+}(w, t) &= -K(f_{S+}, f_I)(w, t) + \frac{1}{\epsilon}Q_{S+}(f_{S+})(w, t) \\
\partial_t f_{S-}(w, t) &= -K(f_{S-}, f_I)(w, t) + \frac{1}{\epsilon}Q_{S-}(f_{S-})(w, t) \\
\partial_t f_I(w, t) &= K(f_{S+} + f_{S-}, f_I)(w, t) - \gamma f_I(w, t) + \frac{1}{\epsilon}Q_I(f_I)(w, t) \\
\partial_t f_R(w, t) &= \gamma f_I(w, t) + \frac{1}{\epsilon}Q_R(f_R)(w, t),
\end{align*}
\]

where $\epsilon > 0$ and $\gamma > 0$ is the recovery rate. The infection transmission is taken into account by the operator

\[K(g, f_I)(w, t) = g(w, t) \int_{S_w} \kappa(w, w_s) f_I(w_s, t) dw_s, \quad g = f_{S+}, f_{S-},\]

with $\kappa(w, w_s) > 0$ expressing the dependency of the disease transmission by the number of contacts and such that $\kappa(0, y) = \kappa(x, 0) = 0$. In [10] it has been proposed as possible example

\[\kappa(x, y) = \beta x^{\alpha_1} y^{\alpha_2}, \quad \alpha_1, \alpha_2 > 0.\]

The operators $Q_J(f_J), J \in \{S_+, I, R\}$ characterize the thermalization of the distributions $f_J(w, t)$ and as discussed in Section 2.1 are given by

\[Q_J(f_J)(w, t) = \frac{\lambda_J}{2} \partial_w \left[ \left( \frac{w}{m_J} - 1 \right) f_J \right] + \frac{\sigma_J^2}{2} \partial_w^2 \left( w f_J(w, t) \right),\]

that are mass and momentum preserving.

From now on we will omit time dependency. Integrating both sides of (4) we get

\[
\begin{align*}
\frac{dS_+}{dt} &= -\beta m_{\alpha_1} S_+ m_{\alpha_2} I(t) S_+ I \\
\frac{dS_-}{dt} &= -\beta m_{\alpha_1} S_- m_{\alpha_2} I(t) S_- I \\
\frac{dI}{dt} &= \beta \left[ S_+ m_{\alpha_1} S_+ + S_- m_{\alpha_1} S_- (t) \right] m_{\alpha_2} I(t) - \gamma I \\
\frac{dR}{dt} &= \gamma I
\end{align*}
\]
that is not closed like classical compartmental modeling since it depends on the evolution of local mean values $m_f(t)$. A possible way to obtain a closed system of equations is obtained by resorting to a limit procedure that is classical in statistical physics. Indeed, for $\epsilon \ll 1$ the distribution functions $f_j(w, t)$ collapse to Gamma-type densities with mass fractions $J(t)$ and local mean values $m_f(t)$. After multiplication by $w$ we get

$$
\frac{d(S_m S_i)}{dt} = -\beta m_{a_1 S_i} m_{a_2 I} S_i I,
\frac{d(S_m S_i)}{dt} = -\beta m_{a_1 S_i} m_{a_2 I} S_i I,
\frac{d(m_I)}{dt} = \beta (m_{a_1 S_i} S_i + m_{a_1 S_i S_m}) m_{a_2 I} I - \gamma m_I I
\frac{d(R m_i)}{dt} = \gamma m_I I
$$

Hence, in view of (3) we obtain the following closed system for the evolution of the mass fractions and mean connections in each compartment

$$
\begin{align*}
\frac{dS}{dt} &= -\beta c_{a_1 S} c_{a_2 I} m_{S} m_{a_2 S} I S_i I, \\
\frac{dS}{dt} &= -\beta c_{a_1 S} c_{a_2 I} m_{S} m_{a_2 S} I S_i I, \\
\frac{dI}{dt} &= \beta c_{a_2 I} \left[ c_{a_1 S} m_{S} m_{a_1} + c_{a_1 S} m_{S} m_{a_1} \right] m_{a_2 I} I(t) - \gamma I(t) \\
\frac{dR}{dt} &= \gamma I(t)
\end{align*}
$$

and

$$
\begin{align*}
\frac{dm_{S_i}}{dt} &= -\frac{\beta \alpha_2}{\mu_S} c_{a_1 S} c_{a_2 I} m_{S} m_{a_1} m_{a_2} I, \\
\frac{dm_{S_i}}{dt} &= -\frac{\beta \alpha_2}{\mu_S} c_{a_1 S} c_{a_2 I} m_{S} m_{a_1} m_{a_2} I, \\
\frac{dm_I}{dt} &= \beta c_{a_2 I} m_{I} \left[ c_{a_1 S} m_{S} m_{a_1} \left( \frac{\alpha_1 + \mu_S}{\mu_S} m_{S_i} - m_I \right) \\
&\quad + c_{a_1 S} m_{S} m_{a_1} \left( \frac{\alpha_1 + \mu_S}{\mu_S} m_{S_i} - m_I \right) \right] \\
\frac{dm_R}{dt} &= \gamma (m_I - m_R) \frac{I}{R}.
\end{align*}
$$

We observe that the obtained social SIR model with generalized interaction forces reduces to the one obtained in [10] in the case of a unique susceptible population with the choice $\alpha_1 = \alpha_2 = 1$. 

Kinetic and macroscopic epidemic models
2.3 Saturated incidence rate

Fixing \( m_I(t) = \bar{m}_I > 0 \) from the first two equations in (6) we get

\[
\frac{dm_{S_\pm}}{dt} = -\beta_\pm(t)m_{S_\pm}^{n_1+1},
\]

being \( \beta_\pm(t) = \frac{\beta \alpha_1}{\mu_{S_\pm}} c_{a_1,S_\pm} c_{a_2,t} \bar{m}_I^{n_2} I(t) \) complemented with the initial condition \( m_{S_\pm}(0) \). The exact solution of the above equation reads

\[
m_{S_\pm}(t) = \frac{m_{S_\pm}(0)}{1 + \frac{\beta \alpha_1^2}{\mu_{S_\pm}} m_{S_\pm}^{n_1}(0)c_{a_1,S_\pm} c_{a_2,t} \bar{m}_I^{n_2} \int_0^t I(s)ds}^{1/n_1}.
\]

Hence, with the introduced assumption we get the following set of first order macroscopic equations with saturated incidence rate

\[
\begin{align*}
\frac{dS_+}{dt} &= -\beta c_{a_1,S_+,c_{a_2,t} H_+(t,I(t)) S_+,I}, \\
\frac{dS_-}{dt} &= -\beta c_{a_1,S_-,c_{a_2,t} H_-(t,I(t)) S_-,I}, \\
\frac{dI}{dt} &= \beta c_{a_1,I}(c_{a_1,S_+} H_+(t,I(t)) S_+ + c_{a_1,S_-} H_-(t,I(t)) S_-) I - \gamma I \\
\frac{dR}{dt} &= \gamma I(t),
\end{align*}
\]

(7)

where

\[
H_\pm(t,I(t)) = \frac{\bar{m}_I^{n_2} m_{S_\pm}^{n_1}(0)}{1 + \frac{\beta \alpha_1^2}{\mu_{S_\pm}} m_{S_\pm}^{n_1}(0)c_{a_1,S_\pm} c_{a_2,t} \bar{m}_I^{n_2} \int_0^t I(s)ds}.
\]

is a generalization of the classical saturated incidence rate.

To understand the influence of contact heterogeneity we divide the equations for \( S_\pm(t) \) in (7) by \( dR/dt \). Hence, in the limit \( t \to +\infty \) we have

\[
\frac{dS_\pm^\infty}{dR^\infty} = -\xi_\pm \mu_{S_\pm} \frac{S_\pm^\infty}{1 + \xi_\pm R^\infty},
\]

(8)

where \( \xi_\pm = \frac{\beta c_{a_1,S_\pm} c_{a_2,t} \bar{m}_I^{n_2} m_{S_\pm}^{n_1}(0)}{\gamma \mu_{S_\pm}} > 0 \) is a given constant. The solutions of (8) are

\[
S_\pm^\infty(R^\infty) = S_\pm(0)\left(1 + \xi_\pm R^\infty\right)^{-\mu_{S_\pm}},
\]

(9)

since \( S_\pm^\infty(R^\infty = 0) = S_\pm(0) \).

For large times we have

\[
S_+^\infty + S_-^\infty + R^\infty = 1.
\]
Taking advantage of the explicit solution (9) we can rewrite the last relation as

$$1 - R^\infty = S_+(0) \left( 1 + \xi_+ R^\infty \right)^{-\mu_{S_+}} + S_-(0) \left( 1 + \xi_- R^\infty \right)^{-\mu_{S_-}},$$

(10)

whose solution defines the dependence of $R^\infty$ by the introduced contact heterogeneity.

3 Numerical results

In this section, we present several numerical experiments for the system (5)-(6) and the system (7) with saturated incidence rate. In particular, we focus on the relation between the fraction of the recovered at the equilibrium $R^\infty$ and the coefficients $\mu_{S_\pm}$ measuring the heterogeneity of the population of the compartments $S_\pm$ in terms of the variance of the contact distribution. More specifically, small values of $\mu_{S_\pm}$ correspond to a larger heterogeneity of the individuals with respect to the social contact, since $\mu_{S_\pm} = \lambda_{S_\pm} / \sigma^2_{S_\pm}$.

Since $R^\infty$ defined in (10) depends on several parameters defining the initial set-up of the contact distribution, to understand the influence of the contact heterogeneity, we fix the following values

$$m_{S_+}(0) = m_R(0) = m_I(0) = \bar{m}_I = 15, m_{S_-}(0) = 10,$$

$$S_+(0) = 0.68, S_-(0) = 0.28, I(0) = R(0) = 0.02,$$

$$\alpha_1 = \alpha_2 = 1, \gamma = 0.1.$$
Therefore, with these choices $R^\infty$ is function of $\mu_{S_-}$ and $\mu_{S_+}$ with a parametric dependence on $\beta$, that is linked to the reproduction number

$$R_0 = \frac{\beta}{\gamma} m_I(0) \left( S_+(0)m_{S_+}(0) + S_-(0)m_{S_-}(0) \right).$$

The relation between the contact structure of the agents and the spreading of the epidemics has been recently studied in literature [5, 12]. In particular, regarding the COVID-19 pandemic, it has been pointed out that a smaller heterogeneity could be associated to a larger value of the recovered at the equilibrium.

Similarly to [5], we consider first the case $R_0 = 2.5$. This choice is then compared with the case of an infectious disease characterized by $R_0 = 1.25$. In details, we solve numerically equation (10) for $R^\infty$, varying $\mu_{S_-}$ and taking different values of $R_0$. As we can observe from Figure 1, $R^\infty$ is an increasing function of both the coefficients $\mu_{S_-}$ regardless of the considered values of the reproduction number $R_0$.

A rather different behavior can be observed for the non-saturated system (5)-(6). In particular, $R^\infty$ exhibits in this case a maximum for small $\mu_{S_-}$. This means that a higher heterogeneity is linked to a larger value of the recovered at the equilibrium. As a consequence, we note also that different conditions of the heterogeneity of the social contacts could be associated to the same $R^\infty$, despite they have a distinct time evolution.

As we can easily observe from the left panel of Figure 2, $R^\infty$ in the $R_0 = 1.25$ scenario has a maximum for high conditions of heterogeneity and then it decreases as the parameters $\mu_{S_-}$ increase. On the contrary, the $R_0 = 2.5$ case in the right panel of Figure 2 shows the same trend of the system with saturated incidence rate. In more details, in Figure 3 we show the time evolution of the system (5)-(6) for $R_0 = 2.5$. We clearly see that a decreasing contact heterogeneity is associated to bigger fraction of recovered for large times.
In the end, we observe that for high values of heterogeneity parameters the model with saturated incidence rate, mimicking non-pharmaceutical protection measures such as a lockdown strategy, exhibits a lower fraction of recovered at the equilibrium than the system (5)-(6). In particular, for small $R_0$, a fixed mean of contacts in the infected compartment is able to avoid the maximum for small $\mu_S$ shown in the left panel of Figure 2.

**Conclusion and perspectives**

In this short note, we focused our attention on a kinetic compartmental model describing the spread of an infectious disease. The process of contact formation is coupled with the epidemic dynamics. We show that the presence of contact heterogeneity is central for the assessment of the evolution of a disease. The interplay between the process leading to the formation of social contacts and Maxwellian models with multiple interactions studied in [3] is currently under deeper investigation.

**Acknowledgements** This work has been written within the activities of GNFM group of INdAM (National Institute of High Mathematics). The research was partially supported by the Italian Ministry of Education, University and Research (MIUR): Dipartimenti di Eccellenza Program (2018–2022) - Dept. of Mathematics “F. Casorati”, University of Pavia.

**References**

1. Albi, G.; Pareschi, L.; Zanella, M.: Control with uncertain data of socially structured compartmental models. *J. Math. Biol.*, 82: 63 (2021).
10 Andrea Medaglia and Mattia Zanella

2. Béraud, G. et al.: The French Connection: The First Large Population-Based Contact Survey in France Relevant for the Spread of Infectious Diseases. *PLoS ONE*, **10**(7): e0133203 (2015).

3. Bobylev, A. V.; Cercignani, C.; Gamba, I.: On the self-similar asymptotics for generalized nonlinear kinetic Maxwell models. *Commun. Math. Phys.*, **291**(3): 599–644, 2009.

4. Bertaglia, G.; Boscheri, W.; Dimarco, G.; Pareschi, L.: Spatial spread of COVID-19 outbreak in Italy using multiscale kinetic transport equations with uncertainty. *Math. Biosci. Eng.*, **18**(5):7028–7059 (2021).

5. Britton, T.; Ball, F.; Trapman, P.: A mathematical model reveals the influence of population heterogeneity on herd immunity to SARS-CoV-2. *Science*, **369**:6505 (2020).

6. Buonomo, B.; Della Marca, R.: Effects of information-induced behavioural changes during the COVID-19 lockdowns: The case of Italy: COVID-19 lockdowns and behavioral change. *R. Soc. Open Sci.*, **7**:201635 (2020).

7. Capasso, V.; Serio, G.: A generalization of the Kermack-McKendrick deterministic epidemic model. *Math. Biosci.*, **42**: 43–61 (1978).

8. Cercignani, C.: *The Boltzmann Equation and its Applications*, Springer Series in Applied Mathematical Sciences, Vol. **67**, Springer–Verlag, New York, NY (1988).

9. Della Marca, R.; Loy, N.; Tosin, A.: An SIR-like kinetic model tracking individuals’ viral load 2021. Preprint, doi:10.13140/RG.2.2.32046.02883.

10. Dimarco, G.; Pareschi, L.; Toscani, G.; Zanella M.: Wealth distribution under the spread of infectious diseases. *Phys. Rev. E*, **102**:022303 (2020).

11. Dolbeault, J.; Turinici, G.: Heterogeneous social interactions and the COVID-19 lockdown outcome in a multi-group SEIR model. *Math. Model. Nat. Phenom.*, **15**: Article Number 36 (2020).

12. Dutta, R.; Gomes, S.; Kalise, D.; Pacchiardi, L.: Using mobility data in the design of optimal lockdown strategies for the COVID-19 pandemic. *PLoS Comput. Biol.*, **17**(8):e1009236 (2020).

13. Furioli, G.; Pulvirenti, A.; Terraneo, E.; Toscani, G.: Fokker–Planck equations in the modelling of socio-economic phenomena. *Math. Mod. Meth. Appl. Sci.*, **27**(1): 115–158 (2017).

14. Hethcote, H.W., Modeling heterogeneous mixing in infectious disease dynamics, in Models for Infectious Human Diseases, edited by V. Isham and G. F. H. Medley, Cambridge University Press, Cambridge, UK, 215–238 (1996).

15. Loy, N.; Tosin, A.: A viral load-based model for epidemic spread on spatial networks. *Math. Biosci. Eng.*, **18**(5):5635–5663 (2021).

16. Lewis, D.: Superspreading drives the COVID pandemic - and could help to tame it. *Nature*, **590**:544–546 (2021).

17. Liu, X.; Stechlinski, P.: Infectious disease models with time-varying parameters and general nonlinear incidence rate. *Appl. Math. Model.*, **36**(5):1974–1994 (2012).

18. Loy, N.; Tosin, A.: A viral load-based model for epidemic spread on spatial networks. *Math. Biosci. Eng.*, **18**(5):5635–5663 (2021).

19. Nielsen, B.F.; Simonsen, L.; Sneppe, K.: COVID-19 Superspreading suggests mitigation by social network modulation. *Phys. Rev. Lett.*, **126**:118301 (2021).

20. Novozhilov, A.S.: On the spread of epidemics in a closed heterogeneous population. *Math. Biosci.*, **215**: 177–185 (2008).

21. Pareschi, L.; Toscani, G.: *Interacting Multiagent Systems: Kinetic Equations and Monte Carlo Methods*, Oxford University Press, Oxford (2013).

22. Salam, P. S. A.; Bock, W.; Klar, A.; Tiwari, S.: Disease contagion models coupled to crowd motion and mesh free simulation. *Math. Mod. Meth. Appl. Sci.*, **31**(6):1277–1295 (2021)

23. Toscani, G.: Entropy-type inequalities for generalized Gamma densities. *Ric. Mat.*, **70**: 35–50 (2021).

24. Zanella, M.; Bardelli, C.; Dimarco, G.; Deandrea, S.; Perotti, P.; Azzi, M.; Figini, S.; Toscani, G.: A data-driven epidemic model with social structure for understanding the COVID-19 infection on a heavily affected Italian Province. *Math. Mod. Meth. Appl. Sci.*, in press (2021).

25. Zhang, J.; Litvinova, M.; Liang, Y.; Wang, Y.; Wang, W.; Zhao, S.; Wu, Q.; Merler, S.; Viboud, C.; Vespignani, A.: Changes in contact patterns shape the dynamics of the COVID-19 outbreak in China. *Science*, **368**(6498):1481–148, 2020.