Dynamics of epidemic diseases without guaranteed immunity

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The global SARS-CoV-2 pandemic suggests a novel type of disease spread dynamics. WHO states that there is currently no evidence that people who have recovered from COVID-19 and have antibodies are immune from a second infection [1]. Conventional mathematical models consider cases for which a recovered individual either becomes susceptible again or develops an immunity. Here, we study the case where infected agents recover and only develop immunity if they are continuously infected for some time. Otherwise, they become susceptible again. We show that field theory bounds the peak of the infectious rate. Consequently, the theory’s phases characterise the disease dynamics: (i) a pandemic phase and (ii) a response regime. The model excellently describes the epidemic spread of the SARS-CoV-2 outbreak in the city of Wuhan, China. We find that only 30% of the recovered agents have developed an immunity. We anticipate our paper to influence the decision making upon balancing the economic impact and the pandemic impact on society. As long as disease controlling measures keep the disease dynamics in the “response regime”, a pan-
The rapid spread of a disease across a particular region or regions (epidemic) or the global outbreak of a disease (pandemic) \[2\] can have a detrimental effect on health systems, on local and global economies including the financial markets and the socio-economic interactions, ranging from the city to the international level. Measures to reduce the pandemic spread include curtailing interactions between infected and uninfected parts of the population, reducing infectiousness or the susceptibility of members of the public \[3\]. The two major strategies governments use to handle an outbreak are to slow down an outbreak (mitigation) or to interrupt the disease spread (suppression). Since each of those interventions bears itself significant risks for the societal and economic well-being, it is crucial to understand the effectiveness of these strategies (or any hybrid of them).

Mathematical methods provide essential input for governmental decision making that aims at controlling the outbreak. Among those are statistical methods \[4, 5\], deterministic state-space models \[6\] with its prototype developed by Kermack and McKendrick \[7\], and a variety of complex network models, e.g., \[8, 9\]. The different mathematical approaches have different objectives: A significant application of the statistical methods frequently aims at the early detection of disease outbreaks \[4\], while modelling either tries to develop a model as realistic as possible for a given outbreak or to design a simplistic model, which, however, reveals some universal truth about the outbreak dynamics.

In the simplest version, the so-called compartmental models \[7, 10\] consider the fraction of the population which is either susceptible (S), infected (I) or removed (R) from the disease network. Coupled differential equations capture the dynamics of the disease that determine the time dependence of S, I and R. Extensions add more compartments to the SIR model such as (E) exposed. For example, such an SEIR model was used in \[11\] for a description of the Ebola
outbreak in the Democratic Republic of Congo in 1995. Compartmental models have been applied to describe the recent SARS-CoV-2 outbreaks \cite{12,13,14,15,16,17}. For example, the elaborate model from Giordano at al. uses a total of 8 compartments - susceptible (S), infected (I), diagnosed (D), ailing (A), recognized (R), threatened (T), healed (H) and extinct (E) - to describe the COVID-19 epidemic in Italy. Compartmental models have been extended in order to capture stochastically unknown influences, such as changing behaviours \cite{18}. Such models were recently used to analyse the COVID-19 outbreak in Wuhan \cite{19}.

Compartmental models address global quantities such as the fraction of susceptible individuals \(S\) and assume that heuristic rate equations can describe the disease dynamics. In cases of a strongly inhomogeneous (social) network, e.g. taking into account different population densities, the above assumption seems not always be justified. In these cases, spatial disease spread patterns can be described by a stochastic network model with Monte-Carlo simulations a common choice for the simulation.

In this paper, we consider a disease dynamics for which the duration (severity) of the illness depends on the amount of exposure. Using an elementary (social) network, we are looking for universal mechanisms describing a pandemic spread. We will reveal a connection to statistical field theory, enabling us to characterise an outbreak with the tools of critical phenomena. We will discuss the impact of the findings on policies to curb an outbreak and will draw conclusions from the recent Covid19 outbreak in Hubei, China.

**Model basics:** We assume that each individual has two states \(u\) with \(u = 0\) - susceptible and \(u = 1\) - infected. Each individual interacts with four 'neighbours' of the social network. The disease spread is described as a stochastic process. At each time step (say 'day'), the probability that an individual gets infected (or recovers) depends on the status of the neighbours in the social network. Here, we only study the simple case of a homogeneous network with four neighbours.
for each site. We also consider periodic boundary conditions to minimise edge effects.

**Immunity:** We study two closely related scenarios.

(i) There is no immunity. Every individual can be reinfected and can recover only to be susceptible again.

(ii) Individuals can be reinfected and recover. Only if individuals stay infected for $\tau$ consecutive days, they are considered *immune*.

In case (ii), the sites of immune individuals are removed from the disease network.

**Disease dynamics:** If $x$ is a site of the disease network, at every time step the state $u_x \in \{0, 1\}$ is randomly chosen with probability

$$P(u_x) = \frac{1}{N_x} \exp \{(4\beta n_x + 2h) u_x\}, \quad n_x = \sum_{y \in \langle xy \rangle} u_y,$$

(1)

where $\langle xy \rangle$ is an elementary link on the lattice joining sites $x$ and $y$ and, hence, $n$ is the number of infected neighbours, and $N_x = 1 + \exp \{4\beta n_x + 2h\}$ is the normalisation. The parameter $\beta$ describes the *contagiousness* of the disease. The parameter $h$ is linked to the probability to contract the disease from outside the network. In fact, if no-one of the network is infected ($n_x = 0, \forall x$), the probability $p$ that any individual contracts the disease, is connected to $h$ by

$$p = \frac{\exp\{2h\}}{1 + \exp\{2h\}}.$$

If the lattice contains $N$ individuals (i.e., sites), one time step is said to be completed if we have considered $N$ randomly chosen sites for the update.

**The pandemic spread as a critical phenomenon:** Scenario (ii) show the typical time evolution of an epidemic with the infections rate approaching zero for large times due to agents
recovering and an increasing number being immune. By contrast, scenario (i) has an asymptotic state independent from the initial state and described by statistical field theory. After the change of variable $z_x = 2u_x - 1$, the asymptotic state is described by the partition function of the Ising model [20 21]:

$$Z = \sum \{z_x = \pm 1\} \exp\{\beta \sum (xy) z_x z_y + H \sum z_x\}$$

with $H = h + 4\beta$, which is the well-known partition function for Ising spins $z$ in an external magnetic field $H$. The disease dynamics of scenario (i) corresponds to a Markov chain of local updates in the Ising model with Markov time identified as real time.

$$H = 0, \quad h(\beta) = -4\beta, \quad p(\beta) = \frac{1}{e^{8\beta} + 1}. \quad (2)$$

For a vanishing external field $H$, the model shows a critical behaviour with a phase transition at $\beta = \beta_c = \ln(1 + \sqrt{2})/2 \approx 0.44$. In the ordered phase for $\beta > \beta_c$, a small seed probability
Figure 2: Left: Scenario (ii): rate of infected (red bars) and rate of infected + immune (green bars) for two sets of model parameters: pandemic phase ($p = 0.05$, $\beta = 0.41$) and response regime ($p = 0.04$, $\beta = 0.38$) both for $\tau = 11$. Right: Comparison of scenario (ii) results with actual data from the COVID-19 outbreak in Hubei province.

$p > 0$ triggers an infection rate close to 100% of the population. The model is in the 'pandemic' phase. For $\beta < \beta_c$, the model is in the 'response' phase, i.e., the infection rate is in repose to the seed probability $p$, but no outbreak occurs. The asymptotic infection rate can be calculated using Markov Monte-Carlo methods. We used a modified Swendsen-Wang cluster algorithm, which performs near the phase transition [22]. Our numerical findings are summarised in figure 1, left panel. Curve 2 clearly separates both phases - the pandemic phase and the response regime. Note that the dynamics (1) corresponds to the standard heat-bath update [23]. Starting from a healthy population ($u_x = 0, \forall x$), it takes the 'thermalisation' time $t_{th}$ that the daily infection rate starts fluctuating around its asymptotic value.

**Immunity:** Let us now study scenario (ii), where individuals can develop immunity if they are infected for $\tau$ consecutive days. For $\tau > t_{th}$, the peak infection rate is that of the asymptotic state of the corresponding model (i) and, hence, inherits the classification 'pandemic' or 'response' phase. This is illustrated in figure 1, right panel, for the pandemic phase for several
values of $\tau$. Figure 2 illustrates the vastly different behaviour of the disease spread in the pandemic phase ($\beta = 0.41, p = 5\%$) and in the response regime ($\beta = 0.38, p = 4\%$). Results are for a $N = 100 \times 100$ network and $\tau = 11$. Note that the curve for 'infected+immune' ('triangle' symbol) in the pandemic phase is not monotonically increasing with time since the infected individuals can return to 'susceptible' state, i.e., not every infected individual becomes immune. Note that in the response regime ('circle' and 'square' symbol), the 'pandemic' peak is absent altogether. However, on the downside, the so-called 'herd immunity' slowly develops over time.

**Comparison with data:** We stress that the model assumption of a homogeneous (social) network with 'four neighbours' is unrealistic. The knowledge of the underlying disease network is essential to make quantitative predictions for e.g. the critical value $\beta_c$ of the contagiousness. Here, we adopt a different approach: we assume that qualitative time evolution of bulk quantities such as the fraction of infected individuals is within the grasp of model scenario (ii) and use those as fit functions to determine the model parameters such as $\beta$, $p$ and $\tau$ by comparison with actual data.

For this study, we used data from the COVID-19 outbreak in 2020 in the Hubei province in China [24]. The data of the number of infected individuals show a jump at day 73 (on the arbitrary time scale) by 40%, which is due to a change in reporting. We assume that the same 'under-reporting' has occurred in the days before, and have corrected the data by multiplying the number of infected (and infected+recovered) by a factor 1.4 for times $t \leq 73$. Let $D(t, \tau, \beta, p)$ be the fraction of the population of infected individuals as a function of time $t$ and depending on the parameters $\tau$ (time to develop immunity), $\beta$ (contagiousness) and $p$ seed probability to get infected. We have calculated $D(t, \tau, \beta, p)$ using a $N = 250 \times 250$ lattice. We checked that the result is independent of the lattice size in the percentage range for the parameters relevant in
this study. If \( D_{\text{wuhei}}(t) \) quantifies the measured values for the number of infected in the Hubei outbreak, we want to approximate these data, i.e.,

\[
D_{\text{wuhei}}(t) \approx N_{\text{pop}} D(t - t_s, \tau, \beta, p)
\]

with a suitable choice of the parameter \( N_{\text{pop}}, t_s, \beta \) and \( p \). Since the offset of the time axis in the Hubei data is arbitrary, we have chosen the shift \( t_s \) such that the peaks of simulated data and measures data coincide. All other parameters are treated as fit parameters. Altogether, we find a good agreement with the data for:

\[
N_{\text{pop}} \approx 68k, \quad t_s \approx 50, \quad \tau \approx 21, \quad \beta \approx 0.48, \quad p \approx 3.3\%.
\]

The model data overshoot the data in the early days of the epidemic spread, which could be related to underreporting due to limited testing capabilities. It is interesting to observe that the curve of the infection rate is asymmetric: the slope of the raise at the beginning is larger than the slope of the decline after the maximum. Also, the number of infected seem to level off at a non-zero value. In the present model, this is explained as follows: with more agents being immune, it is harder for susceptible agents to be continuously infected for time greater or equal \( \tau \) and, thus, to develop immunity. We also find that only about 30% of the infected (and recovered) develop an immunity.

**Conclusions and interpretations:** A new type of stochastic disease model is proposed: agents can recover from an infection and are susceptible again. They only develop immunity if their infection lasts longer than a characteristic time \( \tau \). For \( \tau \rightarrow \infty \), the infection rate is described by statistical field theory. For finite \( \tau \), the infection rate of the field theory provides an upper bound of the infection rate of the dynamical model. This opens up the possibility to characterise the disease dynamics in the light of critical phenomena of the underlying field theory: a pandemic
spread corresponds to the ordered phase of the field theory, and the critical value for the contagiousness is that for the phase transition. The disease is in controllable response mode if the corresponding field theory is in the disordered phase.

Quantitative results, reported here, are derived with an unrealistic homogeneous disease network for which each agent interacts with four neighbours. Nevertheless, we find that the Covid19 data of the Hubei outbreak are well represented. For this case, we find that only 30% of infected develop an immunity.

The heavy tail of the decline of the number of infected, which levels off at non-zero values, is an inherent feature of the model and can be traced back to the fact that agents can be reinfected. In a network with a sizeable portion of immune agents, it is increasingly challenging to develop immunity. If these model assumptions were underpinned by medical investigations, achieving ‘herd immunity’ would be difficult. This should influence the decision to what extent efforts focus on developing a cure or a vaccine.

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