



Balancing Quarantine and Self-Distancing Measures in Adaptive Epidemic Networks

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Abstract

We study the relative importance of two key control measures for epidemic spreading: endogenous social self-distancing and exogenous imposed quarantine. We use the framework of adaptive networks, moment-closure, and ordinary differential equations to introduce new model types of susceptible-infected-recovered (SIR) dynamics. First, we compare computationally expensive, adaptive network simulations with their corresponding computationally efficient ODE equivalents and find excellent agreement. Second, we discover that there exists a critical curve in parameter space for the epidemic threshold, which suggests a mutual compensation effect between the two mitigation strategies: as long as social distancing and quarantine measures are both sufficiently strong, large outbreaks are prevented. Third, we study the total number of infected and the maximum peak during large outbreaks using a combination of analytical estimates and numerical simulations. Also for large outbreaks we find a similar compensation mechanism as for the epidemic threshold. This means that if there is little incentive for social distancing in a population, drastic quarantining is required, and vice versa. Both pure scenarios are unrealistic in practice. The new models show that only a combination of measures is likely to succeed to control epidemic spreading. Fourth, we analytically compute an upper bound for the total number of infected on adaptive networks, using integral estimates in combination with a moment-closure approximation on the level of an observable. Our method allows us to elegantly and quickly check and cross-validate various conjectures about the relevance of different

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network control measures. In this sense it becomes possible to adapt also other models rapidly to new epidemic challenges.

Keywords Epidemic dynamics on networks · quarantaine · social distancing

1 Introduction

The recent COVID-19 pandemic demonstrated the necessity to develop and study effective models of epidemic dynamics Anderson and May (1991). Classical epidemic models are compartmental models leading to relatively simple low-dimensional ordinary differential equations (ODEs) Brauer and van den Driessche (2008); Diekmann and Heesterbeek (2000). These minimal ODE models can be derived from first principles Kiss et al. (2017) but often suffer from strong assumptions, such as a sufficiently high link density within the network of individuals. Within the past two decades, it became apparent that viewing the structure of the contagion process via a network science approach is crucial Pastor-Satorras and Vespignani (2001); Colizza et al. (2006); Durrett (2010); House and Keeling (2011); Pastor-Satorras et al. (2015); Thurner et al. (2020). During the COVID-19 pandemic it also became apparent that there exist two major effects controlling direct epidemic spreading in humans without an available vaccine or immediate medical treatment (non-pharmaceutical interventions): exogenous quarantine measures Maier and Brockmann (2020); Kucharski et al. (2020) and endogenous social self-distancing (or social avoidance) of existing contacts Giordano et al. (2020). Formally, measures can be considered on a finer scale, such as (digital) contact tracing Ferretti et al. (2020); Kretzschmar et al. (2020). Yet, most non-pharmaceutical interventions (NPI) can be grouped into external/exogenous ones leading effectively to quarantine-type effects, and intrinsic/endogenous ones within a population that lead effectively to a social re-organization of contact networks. There have been different modeling approaches with a trend towards large-scale black-box simulations Kerr et al. (2021). Our approach is complementary in the sense of aiming at mathematically tractable models to understand the impact of different effects in more detail. In particular, we stress that although our model design is motivated by COVID, the basic rules we present below are of generic interest for a wide variety of diseases.

In this work, we are interested in developing and analyzing effective, yet tractable mathematical network epidemic models to understand how to compare and balance the effects of quarantining and social self-distancing. Motivated by the COVID-19 pandemic Thurner et al. (2020), we start from a standard susceptible-infected-recovered (SIR) model on a complex network Kiss et al. (2017). Next, we use two well-established modelling approaches. First, we add a possible quarantine state, X , of the nodes Maier and Brockmann (2020); Peak et al. (2020) together with a transition rate, κ , of infected individuals to transition to state X . Second, we use a social self-distancing rule of susceptible individuals trying to avoid contact with infected individuals leading to re-wiring of links Gross et al. (2006); Shaw and Schwartz (2008); Risau-Gusman and Zanette (2009) controlled by a re-wiring rate, w ; we keep the population size and the total number of links fixed to account for the propensity to keep

social contact. Note that re-wiring links makes the network fully adaptive Gross and Sayama (2009), i.e., there is dynamics on and of the network. The resulting model is a Markov chain on all possible node states and all possible edge configurations. It can be simulated on small to medium size networks, but becomes quickly computationally intractable on large networks. For this reason, we derive suitable ODE approximations Kiss et al. (2017) based on a moment-closure approximation Keeling (1999); Keeling et al. (1997); Gross et al. (2006); Kuehn (2016). This approximation technique can also account for the dynamically changing connectivity of the network. We obtain a hierarchy of models. A particular model is obtained after fixing a truncation level. In our analysis we focus on the second-order or pair-approximation moment-closure, which leads to a five-dimensional ODE. We compare medium-size direct network simulations with ODE simulations.

As a next step, we investigate the main questions associated with SIR-type models: (I) Does epidemic spreading happen, or does it die out immediately? (II) How big is the cumulative size of the epidemic outbreak? (III) What is the maximum size of the infected population during an epidemic? As expected, the first question (I) can be calculated directly using local analysis and we can express the epidemic threshold as a function of the re-wiring rate w and the quarantine transition rate κ in the approximating ODEs. Questions (II)-(III) are harder to address as network structure effects preclude the application of classical methods from mathematical epidemiology to calculate the SIR outbreak size and/or the maximum peak. We develop a new technical tool by viewing the outbreak size as a global observable and applying moment-closure methods and integral estimates on the level of this observable. This technique leads to an upper bound on the global outbreak size within suitable parameter regimes, which we cross-validate numerically.

From a public health perspective, we find that large parameter regimes in the (w, κ) -plane show a linear, or almost linear relationship regarding the effects of quarantine versus social self-distancing leading to a bounded triangular region, within which the epidemic cannot be avoided, controlled, or contained efficiently. This shows that there is a balancing effect between strong quarantining and social self-distancing, i.e., the weakening of one measure necessitates the strengthening of the other and vice versa. On the one hand, this is an intuitive result, on the other, it arises without any major assumptions in broad parameter regimes in a complex fully-adaptive network epidemic model. In addition, it can be observed in network- and ODE simulations, and can be analytically treated both, locally and globally via nonlinear dynamics techniques. Hence, it seems advisable, when reducing or observing the reduction of mitigation measures, i.e., a decrease of w and/or κ , to avoid entering the dangerous triangle region as there is no easy way to exit it by a simple small change.

Our contributions are threefold. First, on the level of modeling, we derive a new class of models combining two existing approaches, namely quarantining Maier and Brockmann (2020) and social self-distancing Gross et al. (2006). For this new class of models, we employ numerical simulations, moment closure, and bifurcation theory techniques. Second, we develop a new technical tool extending existing moment closure methods Keeling et al. (1997); Gross et al. (2006); Kuehn (2016) to the integral level of observables, that could have major implications even beyond epidemic

modeling. Third, we derive clear applicable conclusions, most prominently the parametrically approximately linear relation for the effectiveness of exogenous versus endogenous NPIs in epidemic spreading.

The paper is structured as follows: In Sect. 2 we provide the detailed mathematical model for adaptive social self-distancing models with quarantine including moment-closure via pair approximation; in the appendix we develop more complicated higher-order moment-closure models. In Sect. 3 we present our main analytical and numerical results, including a new global moment-closure viewpoint for global observables. In Sect. 4, we provide a summary and outlook of how our approach can be extended to a wider set of applications.

2 Adaptive SIR-type Network Models

Here we compare two different, yet comparable, measures present in most epidemics: social self-distancing, i.e., nodes/agents avoid infected individuals simply due to the risk of acquiring the disease themselves, and external quarantine measures, which enforce the removal of infected nodes from the population. It is evident from data that both measures have played a key role during the COVID-19 pandemic Maier and Brockmann (2020); Kucharski et al. (2020); Giordano et al. (2020). To account for the complex social structure, we start with microscopic Markov process models of susceptible-infected-recovered (SIR) dynamics on general networks with N nodes, K undirected links, and node states S , I , and R . Then we add mitigation measures to the SIR model. The well-known basic SIR rules are:

- (infection) infected I nodes infect susceptible S nodes along susceptible-infected SI links with a rate $\beta > 0$.
- (recovery/death) infected I nodes become recovered R nodes at a rate $\gamma > 0$.

One way to model social self-distancing, as proposed in Gross et al. (2006); Shaw and Schwartz (2008), is the preference of the susceptible S nodes to avoid interactions with the infected I nodes:

- (social self-distancing) SI links are re-wired at a rate $w \geq 0$ to susceptible-susceptible SS links or to susceptible-recovered SR links (with equal proportion).

The self-distancing/re-wiring rule makes the network fully adaptive Gross and Sayama (2009) and allows for very general network topologies. The rule also takes into account that links are not lost, which mirrors the desire to keep as many social connections as possible and to optimally re-wire them to mitigate risk. Note that broken social connections can be re-established in principle once both individuals are susceptible but that this process is currently modeled using uniformly at random re-wiring; see also Sect. 4 for a broader discussion of possible extensions to the model.

While it is straightforward to simulate the resulting Markov process on any given network, the simulations become prohibitively expensive for large N . It is also straightforward to use the master equation for the resulting Markov process Norris (2006) and

arrive at the following set of ODEs via standard techniques Kiss et al. (2017)

$$\begin{aligned}
 [\dot{S}] &= \frac{d}{dt}[S] = -\beta[SI], \\
 [\dot{I}] &= \frac{d}{dt}[I] = \beta[SI] - \gamma[I], \\
 [\dot{R}] &= \frac{d}{dt}[R] = \gamma[I], \\
 [\dot{SI}] &= \frac{d}{dt}[SI] = -(\beta + \gamma + w)[SI] + \beta[SSI] - \beta[ISI], \\
 [\dot{SS}] &= \frac{d}{dt}[SS] = -\beta[SSI] + w \frac{[S]}{[R]+[S]}[SI],
 \end{aligned} \tag{1}$$

where $[S] = [S](t)$, $[I] = [I](t)$, $[R] = [R](t)$, $[SI] = [SI](t)$, $[SS] = [SS](t)$, $[SSI] = [SSI](t)$ and $[ISI] = [ISI](t)$ are expectation values of the number of susceptible, infected and recovered nodes, of SI -links and SS -links and of SSI - and ISI -triplet motifs. The ordinary differential equations (ODEs) (1) represent a variation of earlier models Shaw and Schwartz (2008); Gross et al. (2006). Note that we do not allow recovered individuals to pass back into the susceptible compartment. This is a reasonable assumption when the timescale at which immunity is lost is much larger than the characteristic epidemic timescales β^{-1} , γ^{-1} and w^{-1} . This also holds to some extent for the COVID-19 pandemic, where immunity is conjectured to last on average at the order of months, whereas the infection and recovery timescales are of the order of days. Although the ODEs (1) are actually exact in the mean-field limit Kiss et al. (2017) for any graph, they are not closed as we have not written down the equations for the SSI and ISI motifs. Although these equations could be derived, they would depend on fourth-order motifs, and so on Kuehn (2016); House and Keeling (2011). To avoid studying an infinite system of ODEs, we employ a standard moment-closure pair approximation Keeling et al. (1997); Keeling (1999); Kiss et al. (2017); Gross et al. (2006), assuming that

$$[ABC] \approx m(A, B)m(B, C) \frac{[AB][BC]}{[B]} \quad \text{for } A, B, C \in \{S, I, R\},$$

where $m(A, B) = 2$ if $A = B$ and $m(A, B) = 1$ if $A \neq B$. With this closure, one obtains a system of four ODEs for the densities $\rho_I = [I]/N$ and $\rho_R = [R]/N$ of infected and recovered nodes and the per-node densities of susceptible-infected and susceptible-susceptible links:

$$\begin{aligned}
 \frac{d}{dt}\rho_I &= \beta\rho_{SI} - \gamma\rho_I, \\
 \frac{d}{dt}\rho_R &= \gamma\rho_I, \\
 \frac{d}{dt}\rho_{SI} &= -(\beta + \gamma + w)\rho_{SI} + \beta\rho_{SI} \frac{2\rho_{SS} - \rho_{SI}}{1 - \rho_I - \rho_R}, \\
 \frac{d}{dt}\rho_{SS} &= -2\beta \frac{\rho_{SI}\rho_{SS}}{1 - \rho_I - \rho_R} + w \left[\frac{1 - \rho_I - \rho_R}{1 - \rho_I} \right] \rho_{SI}.
 \end{aligned} \tag{2}$$

Here we made use of node conservation and the notation $\rho_{AB} \approx [AB]/N$ to emphasize that we are working with approximate per-node densities after moment-closure has been applied. As such, the Eq. (2) only cover the aspect of social self-distancing and

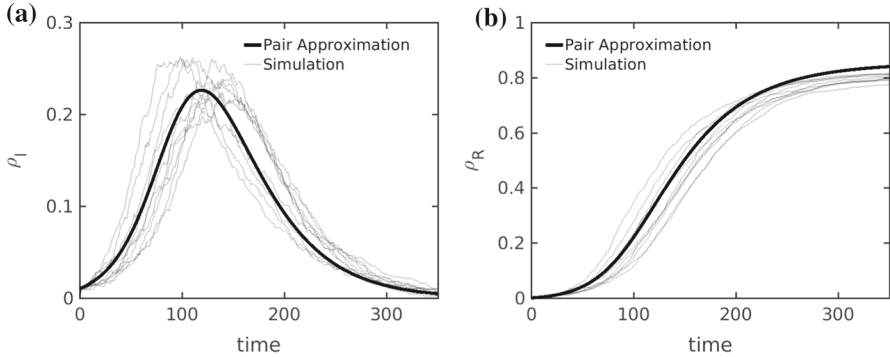


Fig. 1 Sample paths for the adaptive SIRX model (thin line) and the Pair Approximation from (4) (thick line). In **a** we depict the disease prevalence (ρ_I) and in **b** we depict the cumulative size of the recovered compartment (ρ_R). The dynamical parameters are given by a recovery rate of $\gamma = 0.025$ and an infection rate of $\beta = 0.005$. The intervention parameters for the quarantine and re-wiring rates are $\kappa = w = 0.0025$. The release rate from the quarantined compartment is $\delta = 0.001$. For the simulation we sampled from an Erdős-Rényi ensemble of size $N = 2000$ with mean degree $\mu = 15$. We initialized 1% of nodes as infected $\rho_I(0) = 0.01$ and $\rho_{SI}(0) = \mu\rho_I(0)$

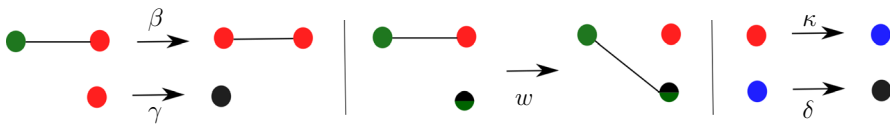


Fig. 2 Rules for the adaptive SIRX model. The two left most rules are classical SIR with infection between infected I (red) and susceptible S (green) nodes at rate β and removal/recovery to R nodes at rate γ . The middle rule is the adaptive re-wiring due to social self-distancing at rate w , where re-wiring can take place to susceptible or recovered nodes. The rightmost rules are quarantining to state X (blue) at rate κ and later removal to R at rate δ (Color figure online)

take into account the complex adaptive network structure via a second-order closure (Fig. 1).

We take into account quarantine effects, such as in the modelling of COVID-19 in Maier and Brockmann (2020). There, network structure was not considered. Quarantine effects lead to certain features of epidemic spreading that cannot be captured by classical SIR models. We denote the quarantined compartment by X . The rules we use are:

- (quarantine) infected I nodes are quarantined into a state X at a rate $\kappa \geq 0$.
- (recovery of X) quarantined nodes are released into the recovered compartment R at rate $\delta > 0$.

In particular, we consider quarantining and rewiring only for the infected compartment. This is a simplification of the present model and is not pursued like this in many real contact tracing efforts. The expected release time from the X -compartment is $\langle T \rangle = 1/\delta$, since the rates are Poissonian. However, δ does not have an effect on the amount of nodes in the infected compartment in this model and for any positive δ the amount of nodes in the recovered compartment for $t \rightarrow \infty$ is also independent of δ . All the rules are summarized graphically in Fig. 2. With these quarantine rules, we

obtain the non-closed moment equations:

$$\begin{aligned}
 \frac{d}{dt}[S] &= -\beta[SI], \\
 \frac{d}{dt}[I] &= \beta[SI] - (\gamma + \kappa)[I], \\
 \frac{d}{dt}[R] &= \gamma[I] + \delta[X], \\
 \frac{d}{dt}[X] &= \kappa[I] - \delta[X], \\
 \frac{d}{dt}[SI] &= -(\beta + \gamma + w)[SI] + \beta[SSI] - \beta[ISI] - \kappa[SI], \\
 \frac{d}{dt}[SS] &= -\beta[SSI] + w \frac{[S]}{[R]+[S]}[SI].
 \end{aligned} \tag{3}$$

More complicated variants of the rules are discussed in

appendix A. We see that the parameters appear linearly in the equations, so that any one of them can be used to re-scale the time, e.g. $t \mapsto \gamma t$. This leaves four effective dynamical parameters β , w , κ and δ . The last one does not affect the infected compartment or the recovered compartment at infinity and is therefore not part of the subsequent analysis.

Using a moment-closure pair approximation, we get the closed system

$$\begin{aligned}
 \frac{d}{dt}\rho_S &= -\beta\rho_{SI}, \\
 \frac{d}{dt}\rho_I &= \beta\rho_{SI} - (\kappa + \gamma)\rho_I, \\
 \frac{d}{dt}\rho_R &= \gamma\rho_I + \delta(1 - \rho_S - \rho_I - \rho_R), \\
 \frac{d}{dt}\rho_{SI} &= -(\beta + \gamma + w + \kappa)\rho_{SI} + \beta\rho_{SI} \frac{2\rho_{SS} - \rho_{SI}}{\rho_S}, \\
 \frac{d}{dt}\rho_{SS} &= -2\beta \frac{\rho_{SI}\rho_{SS}}{\rho_S} + w \frac{\rho_S}{\rho_S + \rho_R} \rho_{SI},
 \end{aligned} \tag{4}$$

that allows us to compare the effects of social self-distancing and quarantine. We compare this model with full network simulations in Fig. 1. The results show excellent agreement for the vast majority of sample runs for a large part of the parameter space, when $w, \kappa > 0$; see also

appendix B for additional comparisons, where even for the singular cases $w = 0$ or $\kappa = 0$ excellent agreement is observed.

3 Results

In contrast to SIS or SIRS, an epidemic eventually dies out for a standard SIR model. Hence, three questions arise:

- (I) Given an initial density of infected $I(0)$ sufficiently close to the disease-free state, does the epidemic spread, or does it die out almost immediately?
- (II) How big is the cumulative size of the epidemic outbreak R_∞ (we use r_∞ for the corresponding density)? R_∞ measures the total number of nodes at $t \rightarrow \infty$ in the recovered compartment R (respectively r_∞ is the corresponding density).

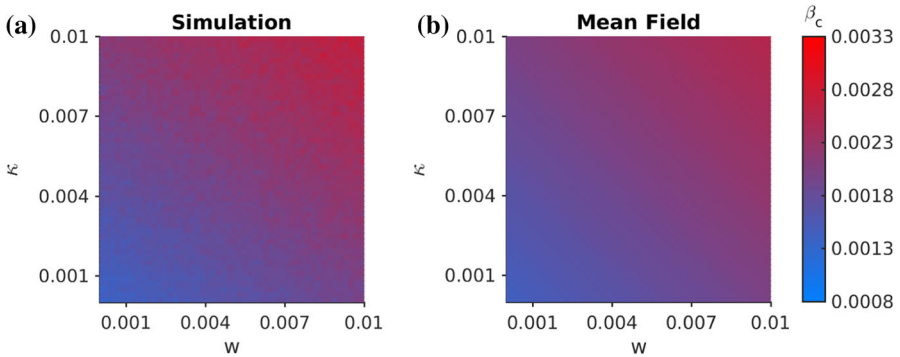


Fig. 3 Comparison of the critical infection rate. We depict the rate β at which the epidemics surpasses a threshold of $r_\infty = 0.05$, which we take as a proxy for the critical point β_c . We compare the simulations (left) with the mean-field analysis from the pair approximation (right). The parameters are as before, except for the size $N = 500$

(III) What is the maximum size of the epidemic, $[\hat{I}] := \max_t I(t)$, i.e., what is the height of the highest peak?

To answer (I), the local calculation near the disease-free state is relatively simple if we have a closed ODE model. For example, consider the adaptive SIR model without quarantining (2) and use the disease-free state ρ_* with

$$\rho_I = \rho_R = \rho_{SI} = 0 \quad \text{and} \quad \rho_{SS} = \mu/2,$$

i.e., also all links are of type SS . Here μ is the average degree of the network, which equals $2K/N$ for a network with K edges. Linearizing the vector field at ρ_* , we find that for

$$\beta < \beta_c^{\text{adp}} = \frac{\gamma + w}{\mu - 1}, \tag{5}$$

an epidemic dies out exponentially fast. A very similar calculation for the full model (4) reveals:

$$\beta < \beta_c^{\text{qurt+adp}} = \frac{\gamma + w + \kappa}{\mu - 1}, \tag{6}$$

as the critical threshold for the infection rate. We see that on a local level near ρ_* , the effects of self-distancing and quarantine are comparable as the rates of both processes lower the critical threshold in a linear way.

These results are

consistent with the numerics. Figure 3 shows the rate β at which we record an epidemic outbreak/growth for direct network simulation, as well as for numerical integration of the mean-field ODEs. The linear level-set structured in the (w, κ) -diagram expected from (6) is clearly visible on the network simulation and the ODE integration levels. This answers questions (I), and means that a combination of measures is

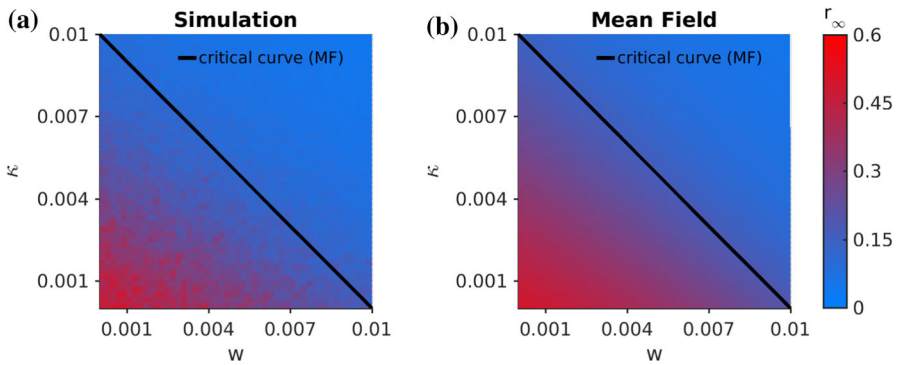


Fig. 4 Comparison of the overall size of the epidemic r_∞ for a fixed $\beta = 0.0025$ in the (κ, w) -parameter plane. **a** Simulations. **b** Mean-field analysis from the pair approximation. We also indicate the critical curve, as calculated from (6). Again $N = 500$, $\mu = 15$, $\gamma = 0.025$, $\delta = 0.01$ and $\rho_I(0) = 0.01$

particularly effective to contain a disease early on. Since it is unrealistic to assume that social self-distancing happens effectively in the situation of a new disease, our SIRX model suggests that one has to compensate and focus more on quarantine measures of infected individuals in the early phase.

However, the local structure near the disease-free state only yields a partial picture of SIR models. In fact, one often does observe epidemic outbreaks for SIR-type dynamics. For this case, we study r_∞ to answer our second question (II). Figure 4 shows r_∞ for a range of values near the epidemic transition in the (w, κ) -plane. We compare numerical simulations that estimate $\langle \lim_{t \rightarrow \infty} R(t)/N \rangle$ with the pair approximation $\rho_{R,\infty} := \lim_{t \rightarrow \infty} \rho_R(t)$ of Eq. (4).

The numerical results indicate another linear relation between the parameters w and κ . In general, it is impossible to get an exact formula for the cumulative size of the epidemic outbreak for an arbitrary model for SIR-type dynamics on complex networks. Yet, we can arrive at an implicit formula starting with in our adaptive SIRX model (3). We denote the expected final number of recovered individuals by $R_\infty := \lim_{t \rightarrow \infty} [R](t)$ and write it as

$$R_\infty = R_\infty - [R](0) = \int_0^\infty [\dot{R}] dt,$$

where we used $[R](0) = 0$ and we have omitted the argument t of the last integrand for brevity. Now we use the differential equation for $[R]$ and insert it to get

$$R_\infty = \gamma \int_0^\infty [I] dt + \delta \int_0^\infty [X] dt.$$

We obtained two integrals, which would suffice to calculate r_∞ . Using the same idea as for $[R]$, we find for $[I]$ and $[X]$ that

$$0 - [I](0) = \int_0^\infty [\dot{I}] dt = \beta \int_0^\infty [SI] dt - (\kappa + \gamma) \int_0^\infty [I] dt$$

$$0 = \int_0^\infty [\dot{X}] dt = \kappa \int_0^\infty [I] dt - \delta \int_0^\infty [X] dt,$$

as there cannot be any quarantined infected nodes in the beginning or at the end of the epidemic. Using the ODE for $[I]$, we get

$$R_\infty = \gamma \int_0^\infty [I] dt + \kappa \int_0^\infty [I] dt = \beta \int_0^\infty [SI] dt + [I](0).$$

Several crucial observations are evident in the formula. The procedure generically does not terminate on the infinite network level as in generic cases, we expect that all motifs could eventually occur. This means that without further assumptions only an infinite series expression for R_∞ is obtained or we could obtain upper or lower bounds. Still, the infinite series and particularly upper bounds are very informative as they display the influence of the different parameters and link them to higher-order network motifs. Indeed, at the next step, the expected number of links comes into play. We get

$$\begin{aligned} 0 - [SI](0) &= \int_0^\infty [S\dot{I}] dt \\ &= -(\beta + \gamma + w + \kappa) \int_0^\infty [SI] dt + \beta \int_0^\infty [SSI] dt - \beta \int_0^\infty [ISI] dt, \end{aligned}$$

and we obtain

$$R_\infty = [I](0) + \frac{\beta}{\beta + \gamma + w + \kappa} [SI](0) + \frac{\beta^2}{\beta + \gamma + w + \kappa} \int_0^\infty ([SSI] - [ISI]) dt. \tag{7}$$

The infinite sum will yield new motif terms involving infected nodes at every step at the time $t = 0$. This demonstrates the importance of the network structure. For example, a highly connected first cluster of infected nodes yields a large number of SI -links and thereby a large final outbreak size. We could continue this procedure to obtain an infinite series formally but this does not give any concrete quantitative approximations. Instead, we aim for an upper bound of the total number of infected/recovered.

A new technical step is that we directly impose the moment-closure pair approximation *directly on (7)*, using the approximate densities $\rho_I, \rho_R, \rho_{SI}$ and ρ_{SS} of the closed equations (4). We get an approximation for r_∞ in terms of $\rho_{R,\infty} := \lim_{t \rightarrow \infty} \rho_R(t)$,

$$\begin{aligned} r_\infty \approx \rho_{R,\infty} &= \rho_I(0) + \frac{\beta}{\beta + \gamma + w + \kappa} \rho_{SI}(0) \\ &+ \frac{\beta}{\beta + \gamma + w + \kappa} \int_0^\infty \beta \frac{\rho_{SI}}{\rho_S} (2\rho_{SS} - \rho_{SI}) dt. \end{aligned} \tag{8}$$

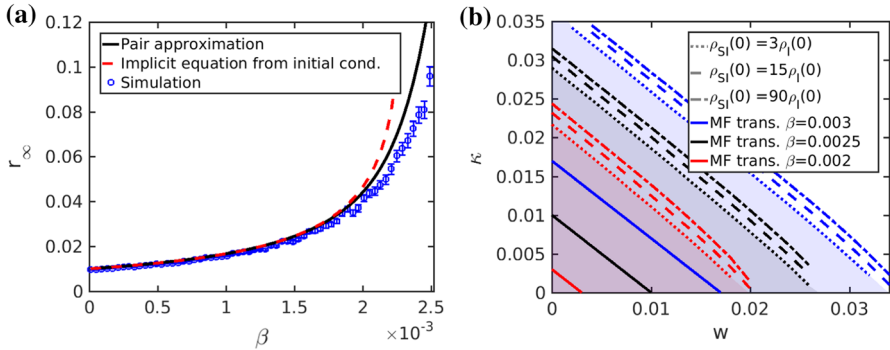


Fig. 5 Approximations of r_∞ . In **a** we show three approximations of r_∞ for the adaptive SIRX model via the Pair Approximation (4), the implicit Eq. (9) and via repeated simulation of the stochastic dynamics. The implicit equation is an inequality in r_∞ and depends on the initial conditions, which are here chosen in agreement with the other approximations, namely $\rho_I(0) = 0.01$ and $\rho_{SI}(0) = \mu\rho_I(0)$. The inequality is achieved by a Hölder bound, which requires a positivity condition on $\dot{\rho}_{SI} - 2\dot{\rho}_{SS} \geq 0$ at all times. In **b** we show the shaded regions where the positivity condition holds for a range of infection rate $\beta = 0.003$ (blue, upper set of lines), $\beta = 0.0025$ (black, middle set of lines) and $\beta = 0.002$ (red, lower set of lines). For each infection rate we show the boundary for a set of initial conditions to illustrate the dependence on $\rho_I(0)$ and $\rho_{SI}(0)$. For the initial SI -link density we choose a mean-field scenario (dashed line) with $\rho_{SI}(0) = \mu\rho_S(0)$, a scenario (dash-dotted line) with dis-proportionally many initial SI -links $\rho_{SI}(0) = 6\mu\rho_S(0)$ and a scenario (dotted) with very few SI -links $\rho_{SI}(0) = (\mu/5)\rho_S(0)$. The mean-field transition lines (solid lines) are seen at the respective infection rates in the respective colours. They all lie within the positivity region. All parameters are as before, in particular $\mu = 15$ (Color figure online)

Using $\beta\rho_{SI} = -\dot{\rho}_S$ and applying the logarithmic derivative gives

$$\rho_{R,\infty} \approx K_0 + \frac{\beta}{\beta + \gamma + w + \kappa} \int_0^\infty -\frac{d}{dt}(\ln(\rho_S))(2\rho_{SS} - \rho_{SI}) dt,$$

where $K_0 := \rho_I(0) + \frac{\beta}{\beta + \gamma + w + \kappa} \rho_{SI}(0) > 0$. Integration by parts and $\ln(x) = -|\ln(x)|$ for $x \in (0, 1]$ yields

$$\rho_{R,\infty} = K_0 + \frac{\beta}{\beta + \gamma + w + \kappa} \left(-\ln(\rho_S)(2\rho_{SS} - \rho_{SI}) \Big|_0^\infty - \int_0^\infty |\ln(\rho_S)|(2\dot{\rho}_{SS} - \dot{\rho}_{SI}) dt. \right)$$

Next, we use $|\ln(\rho_S(t))| \geq |\ln(\rho_{S,\infty})| = -\ln(1 - \rho_{R,\infty})$ and assume that $2\dot{\rho}_{SS} - \dot{\rho}_{SI} \leq 0$ for all times (see the computations below for further justification of this assumption). Then we obtain the bound

$$\rho_{R,\infty} \leq K_0 + \frac{\beta}{\beta + \gamma + w + \kappa} \left(-\ln(\rho_S)(2\rho_{SS} - \rho_{SI}) \Big|_0^\infty + \ln(1 - \rho_{R,\infty}) \int_0^\infty (2\dot{\rho}_{SS} - \dot{\rho}_{SI}) dt \right)$$

$$\begin{aligned}
 &= K_0 + \frac{\beta}{\beta + \gamma + w + \kappa} \left(-\ln(1 - \rho_{R,\infty}) 2\rho_{SS}(\infty) + \ln(1 - \rho_I(0)) \right. \\
 &\quad \left. (2\rho_{SS}(0) - \rho_{SI}(0)) + \ln(1 - \rho_{R,\infty}) (2\rho_{SS}(\infty) - 2\rho_{SS}(0) + \rho_{SI}(0)) \right) \\
 &= K_0 + \frac{\beta}{\beta + \gamma + w + \kappa} \ln \left[\frac{1 - \rho_I(0)}{1 - \rho_{R,\infty}} \right] (2\rho_{SS}(0) - \rho_{SI}(0))
 \end{aligned}$$

Simplifying yields the desired upper bound

$$\rho_{R,\infty} \leq \rho_I(0) + \frac{\beta}{\beta + \gamma + w + \kappa} \left[\rho_{SI}(0) + (2\rho_{SS}(0) - \rho_{SI}(0)) \ln \left[\frac{1 - \rho_I(0)}{1 - \rho_{R,\infty}} \right] \right]. \tag{9}$$

The bound (9) is a transcendental inequality in $\rho_{R,\infty}$. Regarding our assumption

$$\dot{\rho}_{SI} \geq 2\dot{\rho}_{SS}, \tag{10}$$

we find that it holds numerically for a broad ranges of parameters. In Fig. 5(b) we show the domains of validity for the positivity assumption in the (w, κ) -plane for a range of infection rates and initial conditions. The assumption holds in a neighbourhood around the critical transition.

Note that our analysis is in sharp contrast to the classical three-dimensional SIR ODE model, where an exact implicit functional relation for r_∞ can be obtained. Therefore, having an upper bound available such as (9) helps us to study the parameter dependencies. The same linear combination of the two parameters w and κ appears as in the local bifurcation case near the epidemic threshold. Now however, they occur via an inverse. The same conclusions as for the local epidemic spreading near the outbreak threshold are valid: we need a linear mix of quarantine and social self-distancing to keep the total number of infected r_∞ under control. Figure 5(a) shows a comparison of the upper bound for with numerical simulations of the full network as well as simulations of the pair approximation ODEs. Both capture the main trend well that occurs when the infection rate is increased. When the total infected population is around the 10 percent level, our approximations show that employing a combination of quarantine and social-distancing might be effective in practice, while going beyond this level, a very steep increase of the total number of infected occurs.

Finally, we answer question (III) regarding the maximum peak $[\hat{I}]$ of the number of infected, i.e., $[\hat{I}]/N$ is the maximal fraction across the entire duration of the epidemic. Figure 6 shows the corresponding results comparing direct network simulations in Fig. 6(a) with mean-field approximations in Fig. 6(b). The structure of the results is familiar in the sense that a linear dependence between w and κ emerges for our studied parameter ranges. Therefore, one can conclude that using a *well-tuned* combination of quarantine and social self-distancing outside of a triangular region in (w, κ) -space is likely to be not only effective in preventing outbreaks, or reducing the total number of infected during epidemic, but also to prevent high peaks. This conclusion is also

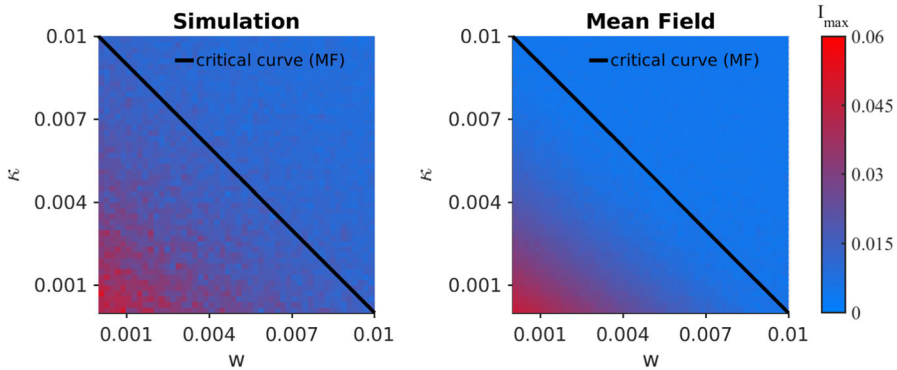


Fig. 6 Comparison of the maximal disease prevalence. **a** Simulation. **b** Mean-field from the Pair Approximation. We also indicate the critical curve, as calculated from (6). We can see here a slight deviation, which can be explained by the fact that the simulations are random processes. The average of sample path maxima over many sample paths is not the same as the maximum of the average of sample paths. The former overestimates the expectation value of ρ_I . Again $N = 500$, $\mu = 15$, $\gamma = 0.025$, $\beta = 0.0025$, $\delta = 0.01$, $\rho_I(0) = 0.01$ and $\rho_{SI}(0) = \mu\rho_I(0)$

very robust over a wide variety of parameter ranges and we provide further numerical results in Appendix C to support this claim.

4 Conclusion & Outlook

In this work, we provided three contributions. First, we developed a new type of adaptive network models that include two of the most important epidemic control measures: quarantine and social self-distancing. We derived mean-field models via pair approximation; even more detailed approximation schemes are discussed in the appendix. Second, we analyzed the new model via a numerical combination of direct network simulations and mean-field ODEs, which show excellent agreement. We focused on three questions regarding (I) the epidemic threshold, (II) the total number of infected individuals, and (III) the maximum peak of the epidemic. In all three cases, we demonstrated for a broad range that the parameters controlling quarantine and social-distancing enter in a comparable linear combination to control the epidemic spread. This has the practical implication that a suitable combination of these two measures outside of a well-defined triangular region in parameter space is the best choice as one cannot expect either measure to be executed perfectly in practice. Third, on a technical level, we have shown a new way to provide estimates for the total infected population during an epidemic by using pair approximation and integral estimates directly on the level of the final infected number observable. This provides a new technical tool for broad classes of epidemic models on networks since one can now aim to employ moment-closure on many other observables directly. Yet, it remains an open conjecture, whether one can give an analytical proof of the parametrically linear trade-off relationship between quarantine and social-distancing on a global dynamical systems level. We anticipate that this problem is challenging as it would require full

control of nonlinear dynamics in phase space, which is usually difficult to achieve – even for simple dynamical systems.

Many generalizations of the presented model are possible. For example, one could try to use slightly different rules for the link dynamics allowing for link deletion Ball et al. (2019); Tufekci and Wilson (2013). Yet, we conjecture that the same analysis principles we have developed here still apply. Furthermore, it would be desirable to not only consider the mitigation of the epidemic itself but also whether quarantine and social self-distancing can help us or are detrimental to detect early-warning signs for large epidemic outbreaks O'Regan and Drake (2013); Widder and Kuehn (2016); Brett et al. (2018). This line of research has already been started in recent years for epidemics on adaptive networks Kuehn et al. (2015); Horstmeyer et al. (2018) but the interplay between pre-epidemic mitigation measures and warning signs has remained unexplored. Another important generalization would be to consider non-Markovian network epidemic models Van Mieghem and Van de Bovenkamp (2013); Sherborne et al. (2018); Clauß and Kuehn (2022). This could account for behavioral changes based upon historical NPI data or allow for re-establishing a social network that has been broken due to self-distancing.

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Data Availability No real-world has been used for this manuscript. Simulations were performed with a standard numerical ODE integrator and Gillespie network simulation algorithm. The code is available at <https://github.com/leomarlo/SIRXw>

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Appendix

A More on Moment Equations for Adaptive Epidemics with Quarantine

In this appendix, we provide more details on moment-closure schemes and the derivation of our reduced system. Beyond the pair approximation (4), refined approximation schemes are possible, which we present in this appendix in more detail.

A.1 Full Closed Quations up to Second Order Motifs

First, we write down the full closed equations up to second-order motifs, which are given by

$$\frac{d}{dt}[S] = -\beta[SI], \quad (11)$$

$$\frac{d}{dt}[I] = \beta[SI] - (\gamma + \kappa)[I], \quad (12)$$

$$\frac{d}{dt}[R] = \gamma[I] + \delta[X], \quad (13)$$

$$\frac{d}{dt}[X] = \kappa[I] - \delta[X], \quad (14)$$

$$\frac{d}{dt}[SI] = -(\beta + \gamma + w + \kappa)[SI] + \beta[SSI] - \beta[ISI] \quad (15)$$

$$\frac{d}{dt}[SS] = -\beta[SSI] + w \frac{[S]}{[R] + [S]}[SI] \quad (16)$$

$$\frac{d}{dt}[SR] = +\gamma[SI] + \delta[SX] - \beta[ISR] + w \frac{[R]}{[R] + [S]}[SI] \quad (17)$$

$$\frac{d}{dt}[SX] = -\delta[SX] - \beta[ISX] + \kappa[SI] \quad (18)$$

$$\frac{d}{dt}[II] = -2\gamma[II] + \beta[SI] + \beta[ISI] - 2\kappa[II] \quad (19)$$

$$\frac{d}{dt}[IR] = +2\gamma[II] - \gamma[IR] + \delta[IX] + \beta[ISR] - \kappa[IR] \quad (20)$$

$$\frac{d}{dt}[IX] = -\gamma[IX] - \delta[IX] + \beta[ISX] + 2\kappa[II] - \kappa[IX] \quad (21)$$

$$\frac{d}{dt}[RR] = +\gamma[IR] + \delta[RX] + \kappa[RX] \quad (22)$$

$$\frac{d}{dt}[RX] = +\gamma[IX] - \delta[RX] + 2\delta[XX] + \kappa[RI] \quad (23)$$

$$\frac{d}{dt}[XX] = -2\delta[XX] + \kappa[XI] \quad (24)$$

We see that Eqs. for the nodes (11)-(14) depend only through $[SI]$ on the equations of the links. Equation (15) depends on itself, on $[SSI]$ and on $[ISI]$. After pair approximation there is another dependence on $[SS]$ entering, so we need (16). The resulting equations are self-contained.

If we now add further equations for the other link densities we obtain Eqs. (17)-(24). We also see from Eqs. (11)-(24), that there is node and link conservation. Note that there is no dependence on the average degree. And there should not be. We could enforce it, by redefining the variables. So for instance if we have $\rho_S := [S]/N \in [0, 1]$ for the per-capita density of susceptibles in the population and $\rho_{SI} := [SI]/N \in [0, \mu/2]$ for the average number of $[SI]$ -links per node, then we could redefine $\rho_{\hat{SI}} := [SI]/L \in [0, 1]$ as an actual density (with $\rho_{\hat{SI}} = (2/\mu)\rho_{SI}$) and Eqs. (11) would then read

$\frac{d}{dt} \rho_S = -\beta(\mu/2)\rho_{SI}$. Yet, this explicit dependence on the degree is not necessary, so we have decided to employ the equations without this additional parameter.

Starting from the second-order, we can also write down the third-order equations:

$$\begin{aligned} \frac{d}{dt} [SSI] &= -r[SSI] - \kappa[SSI] - w[SSI] + w \frac{[S]}{[S] + [R]} [SI] \frac{[SI]}{[S]} \\ &\quad - \beta[SSI] - \beta[S \overset{I}{S} I] - \beta[ISSI] \\ \frac{d}{dt} [ISI] &= -2r[ISI] - 2\kappa[ISI] - 2w[ISI] - 2\beta[ISI] - \beta[I \overset{I}{S} I]. \end{aligned}$$

These equations can also be written in the alternative form via pair approximation densities as follows

$$\begin{aligned} \frac{d}{dt} \rho_{SSI} &= -r\rho_{SSI} - \kappa\rho_{SSI} - w\rho_{SSI} + w \frac{\rho_S}{\rho_S + \rho_R} \rho_{SI} \frac{\rho_{SI}}{\rho_S} - \beta\rho_{SSI} \\ &\quad - \beta\rho_{SI} \frac{\rho_{ISI}}{\rho_S} - \beta\rho_{SI} \frac{\rho_{SSI}}{\rho_S} \\ \frac{d}{dt} \rho_{ISI} &= -2r\rho_{ISI} - 2\kappa\rho_{ISI} - 2w\rho_{ISI} - 2\beta\rho_{ISI} - \beta\rho_{SI} \frac{\rho_{ISI}}{\rho_S}. \end{aligned}$$

Now the procedure to develop higher-order moment-closures could be pursued Kiss et al. (2017) but we have not found that the resulting equations add significant practical insight in our context. Instead, we are going to derive to interesting variations of the equations we have analyzed.

A.2 Adaptive SIR with Infinite Quarantine

Following Maier and Brockmann (2020), we consider the modification that not the links are removed but that there is a state $[X]$ which represents the quarantined state. In that state, the disease cannot be transmitted along the link. So all those links attached to that node are removed from the pool of transmittable links. Effectively this behaves like link-removal. Hence, we obtain the equations

$$\begin{aligned} \frac{d}{dt} [S] &= -\beta[SI] - \kappa_0[S] \\ \frac{d}{dt} [I] &= \beta[SI] - \gamma[I] - (\kappa_0 + \kappa)[I] \\ \frac{d}{dt} [R] &= \gamma[I] \\ \frac{d}{dt} [X] &= \kappa_0[S] + (\kappa_0 + \kappa)[I] \\ \frac{d}{dt} [SI] &= -(\beta + \gamma + w)[SI] + \beta[SSI] - \beta[ISI] - 2\kappa_0[SI] - \kappa[SI] \\ \frac{d}{dt} [SS] &= -\beta[SSI] + w * \frac{[S]}{[R] + [S]} [SI] - 2\kappa_0[SS] \end{aligned}$$

This latter model has the following density representation:

$$\begin{aligned}\frac{d}{dt}\rho_S &= -\beta\rho_{SI} - \kappa_0\rho_S \\ \frac{d}{dt}\rho_I &= \beta\rho_{SI} - \gamma\rho_I - (\kappa_0 + \kappa)\rho_I \\ \frac{d}{dt}\rho_R &= \gamma\rho_I \\ \frac{d}{dt}\rho_{SI} &\approx -(\beta + \gamma + w + 2\kappa_0 + \kappa)\rho_{SI} + \beta\rho_{SI}\frac{2\rho_{SS} - \rho_{SI}}{\rho_S} \\ \frac{d}{dt}\rho_{SS} &\approx -2\beta\frac{\rho_{SI}\rho_{SS}}{\rho_S} + w\left[\frac{\rho_S}{\rho_S + \rho_R}\right]\rho_{SI} - 2\kappa_0\rho_{SS}\end{aligned}$$

A.3 Adaptive SIR with Quarantine and a Return Rate

Lastly, let us consider, as before, that there is a quarantined compartment, but quarantine does not last forever and there is a return rate $\delta > 0$. We still allow for both, the susceptible population as well as the infected population to be quarantined at a rate κ_0 and $\kappa_0 + \kappa$ respectively. If they are healthy quarantined individuals, then they are transferred back into the susceptible compartment. If they are infected, they transition into the recovered compartment, respectively, at the aforementioned rate δ . In summary, this yields the equations

$$\begin{aligned}\frac{d}{dt}[S] &= -\beta[SI] - \kappa_0[S] + \delta[X_S] \\ \frac{d}{dt}[I] &= \beta[SI] - \gamma[I] - (\kappa_0 + \kappa)[I] \\ \frac{d}{dt}[R] &= \gamma[I] + \delta[X_I] \\ \frac{d}{dt}[X_S] &= \kappa_0[S] - \delta[X_S] \\ \frac{d}{dt}[X_I] &= (\kappa_0 + \kappa)[I] - \delta[X_I] \\ \frac{d}{dt}[SI] &= -(\beta + \gamma + w)[SI] + \beta[SSI] - \beta[ISI] - 2\kappa_0[SI] - \kappa[SI] + \delta[X_{SI}] \\ \frac{d}{dt}[SS] &= -\beta[SSI] + w * \frac{[S]}{[R] + [S]}[SI] - 2\kappa_0[SS] + \delta[X_{SS}] \\ \frac{d}{dt}[X_{SS}] &= -\delta[X_{SS}] + 2\kappa_0[SS] - \beta[X_{SI}] \\ \frac{d}{dt}[X_{SI}] &= -\delta[X_{SI}] - \gamma[X_{SI}] - (\kappa_0 + \kappa)[X_{SI}] + \kappa_0[SI]\end{aligned}$$

After the usual reduction steps, we obtain a closed system of eight ODEs for the densities

$$\begin{aligned}
\frac{d}{dt}\rho_S &= -\beta\rho_{SI} - \kappa_0\rho_S + \delta\rho_{X_s} \\
\frac{d}{dt}\rho_R &= +\gamma(1 - \rho_R - \rho_S - \rho_{X_s} - \rho_{X_i}) + \delta\rho_{X_i} \\
\frac{d}{dt}\rho_{X_s} &= +\kappa_0\rho_S - \delta\rho_{X_s} \\
\frac{d}{dt}\rho_{X_i} &= +(\kappa_0 + \kappa)(1 - \rho_S - \rho_R - \rho_{X_s} - \rho_{X_i}) - \delta\rho_{X_i} \\
\frac{d}{dt}\rho_{SI} &= -(\beta + \gamma + w + 2\kappa_0 + \kappa)\rho_{SI} + \beta\rho_{SI}\frac{2\rho_{SS} - \rho_{SI}}{\rho_S} + \delta\rho_{IX_s} \\
\frac{d}{dt}\rho_{SS} &= -2\beta\frac{\rho_{SI}\rho_{SS}}{\rho_S} + w\frac{\rho_S}{\rho_S + \rho_R}\rho_{SI} - 2\kappa_0\rho_{SS} + \delta\rho_{SX_s} \\
\frac{d}{dt}\rho_{SX_s} &= -\delta\rho_{SX_s} + 2\kappa_0\rho_{SS} - \beta\rho_{SX_s}\frac{\rho_{SI}}{\rho_S} \\
\frac{d}{dt}\rho_{IX_s} &= -\delta\rho_{IX_s} - \gamma\rho_{IX_s} - (\kappa_0 + \kappa)\rho_{IX_s} + \kappa\rho_{SI}.
\end{aligned}$$

In future work, it could be interesting to study the differences between the slight variations of adaptive social self-distancing epidemic networks with quarantine we presented in this appendix. Yet, we conjecture that the key major effects to understand the control of the disease are already displayed by our five-dimensional main system (4).

B Additional Comparisons between Network Simulations and the Moment Equations

In this appendix, we collect several additional examples of comparisons between direct network simulations and the moment-closure Eq. (4). As outlined in the main part of the paper, we are particularly interested in the parameters w controlling the re-wiring of social self-distancing and the quarantine rate κ . We have already shown a comparison in Fig. 1, where $\kappa > 0$ and $w > 0$, which showed very good agreement between network dynamics and the moment-closure approximation. To check the robustness of the approximation, we provide here also in Fig. 7 the case $w = 0$ and $\kappa > 0$, while in Fig. 8 we consider $\kappa = 0$ and $w > 0$. Even for these quite singular cases, the approximation via second-order moment-closure works extremely well.

C Additional Parameter Robustness Results

In this appendix, we collect additional parameter robustness studies for our results presented in Sect. 3. In particular, we have selected parameters within a range roughly compatible with the COVID-19 pandemic. Although it is clear that we cannot model the epidemic dynamics exactly with our adaptive SIR model (in fact, no model will give a fully accurate description), we are primarily interested in whether make major

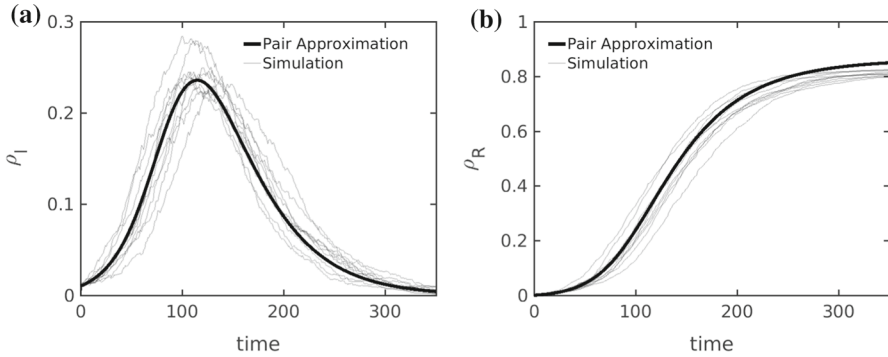


Fig. 7 Sample paths for the adaptive SIRX model (thin line) and the Pair Approximation from (4) (thick line). In **a** we depict the disease prevalence (ρ_I) and in **b** we depict the cumulative size of the recovered compartment (ρ_R). The dynamical parameters are given by a recovery rate of $\gamma = 0.025$ and an infection rate of $\beta = 0.005$. The intervention parameters for the quarantine and re-wiring rates are $\kappa = 0.0025$ and $w = 0$. The release rate from the quarantined compartment is $\delta = 0.001$. For the simulation we sampled from an Erdős-Rényi ensemble of size $N = 2000$ with mean degree $\mu = 15$. We initialized 1% of nodes as infected $\rho_I(0) = 0.01$ and $\rho_{SI}(0) = \mu\rho_I(0)$

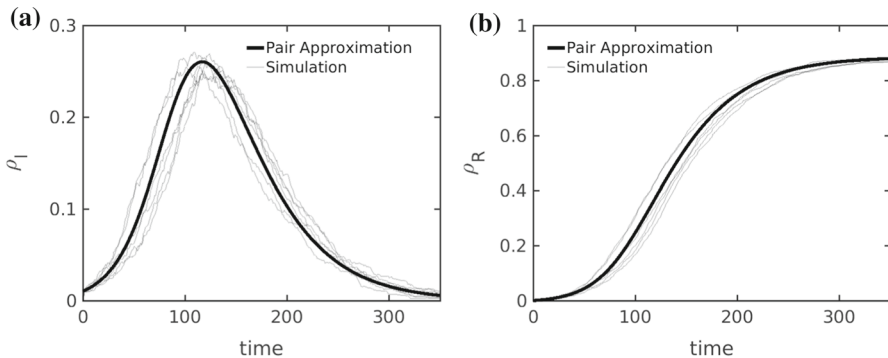


Fig. 8 Sample paths for the adaptive SIRX model (thin line) and the Pair Approximation from (4) (thick line). In **a** we depict the disease prevalence (ρ_I) and in **b** we depict the cumulative size of the recovered compartment (ρ_R). The dynamical parameters are given by a recovery rate of $\gamma = 0.025$ and an infection rate of $\beta = 0.005$. The intervention parameters for the quarantine and re-wiring rates are $\kappa = 0$ and $w = 0.0025$. The release rate from the quarantined compartment is $\delta = 0.001$. For the simulation we sampled from an Erdős-Rényi ensemble of size $N = 2000$ with mean degree $\mu = 15$. We initialized 1% of nodes as infected $\rho_I(0) = 0.01$ and $\rho_{SI}(0) = \mu\rho_I(0)$

changes in the parameters still lead to the main observed trade-off between quarantine and social self-distancing. Indeed, Figs. 9-11 confirm that the observations we have made above regarding mean-field accuracy and trade-off between the quarantine and social distancing remain even for very different sets of parameters.

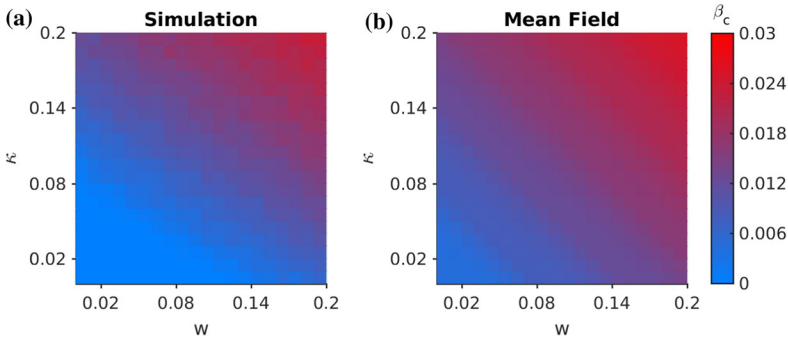


Fig. 9 We depict the rate β at which the epidemics surpasses a threshold of $r_\infty = 0.05$, which we take as an indication for the critical point β_c . We compare the simulations (left) with the mean field analysis (right). The parameters are $N = 500$, $\mu = 15$, $\gamma = 1/14$, $\delta = 1/10$ and $I(0) = 5$

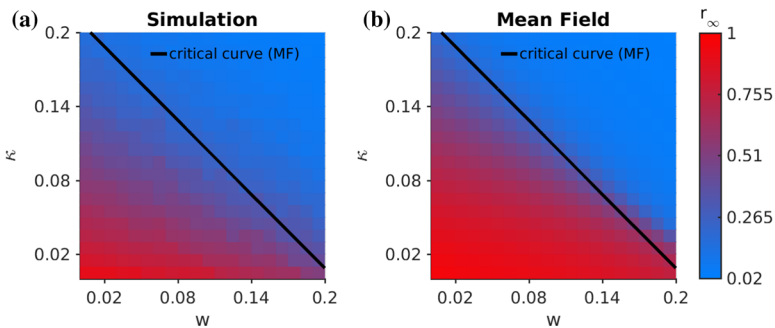


Fig. 10 We depict the final fraction of recovered individuals r_∞ at an infection rate of $\beta = 0.02$. We compare the simulations (left) with the mean field analysis (right) and indicate the critical curve. Again $N = 500$, $\mu = 15$, $\gamma = 1/40$, $\delta = 1/10$ and $I(0) = 5$

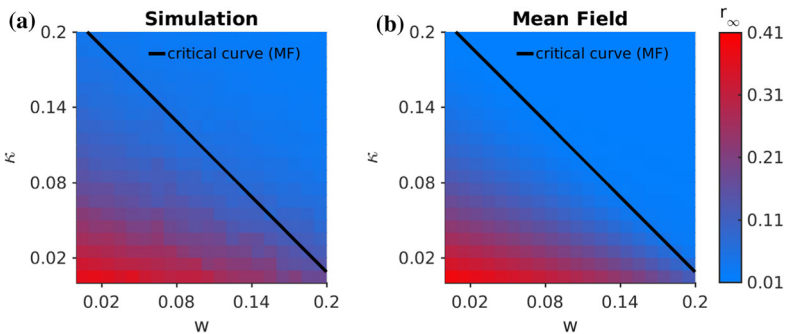


Fig. 11 We depict the maximal fraction of infected individuals $\widehat{[I]}/N$ at an infection rate of $\beta = 0.02$. We compare the simulations (left) with the mean field analysis (right) and indicate the critical curve, as calculated from (6). We see here a strong difference. This can be explained by the fact that the simulations are random processes. The average of sample path maxima over many sample paths is not the same as the maximum of the average of sample paths. The former overestimates the expectation value of ρ_I . Again $N = 500$, $\mu = 15$, $\gamma = 1/14$, $\delta = 1/10$ and $I(0) = 5$

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