

Resonance between contact patterns and disease progression shapes epidemic spread

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The spread of a contagious disease clearly is affected by the contact patterns of infected individuals. But it remains unclear how individual contact patterns interact with the infectious and non-infectious stages through which a disease progresses over time. Here, we investigate this interplay of contact patterns and disease progression using real-world physical proximity data from the Copenhagen Networks Study. We find from the data that the number of encounters following a potential infection event varies significantly over time — with clear daily and weekly variations of encounter probability. These variations can accelerate or slow down the spread of infectious diseases, depending on how they are aligned with the infectious periods. Remarkably, the resulting resonance strongly depends on the latent period, which, although non-infectious and often neglected, is key to the degree of alignment of the infectious period with the weekly modulation. We demonstrate that the resonance effect can be reproduced by a generative process with a time-dependent encounter rate that reflects the daily and weekly modulation found in the data. Including this non-Markovian generative process into a well-mixed, mean-field spreading model, we show that resonances between contact patterns and disease progression can change the basic reproduction number considerably — in our case by up to 20%. Surprisingly, a change in latent period can thereby make epidemic spread stronger even if the individual infectiousness is unaltered.

As contagious diseases are passed on through contacts, disease spread is naturally shaped by details of the spatial and temporal contact behavior of those involved. If infected individuals do not have any sufficiently close and long contacts while being infectious, the disease will not be passed on. If instead they happen to be at several gatherings with many close-by encounters, there will likely be many secondary infections. It is hence evident that epidemic spread is affected by both, spatial features (with whom and how close) and temporal features (how often and how long) of contact behavior. To develop a faithful description of epidemic spreading processes, these basic empirical facts need to be taken into account.

The vast majority of studies in epidemic modelling, including most of the recent works in response to the Coronavirus pandemic, rely on a description based on compartmental models such as Susceptible, Infected, Recovered (SIR) and extensions thereof [1]. These models successfully capture the general mechanism and overall behavior of disease spread as described by spatio-temporal averages, but naturally fail to incorporate the effect of contact behavior. On the other hand, agent-based models do incorporate such contact behavior explicitly [2, 3], but they hence depend on the availability of actual temporal contact data [4, 5], good surrogate data [6], or approximations of the underlying spatio-temporal features of contact behavior [7].

Approaches used to uncover salient spatio-temporal features of human contact behaviour range from direct experiments [8, 9] to analyses of interaction data that can be related to human contact behavior [e.g., mobility data [10]]. There is mounting evidence that human con-

tact networks feature a high level of heterogeneity across individuals. Examples include the high variability of the influence of individuals [11] or strong clustering of either nodes [12] or links [13] that indicate strong interactions in local neighborhoods. In addition, interactions were shown to cluster in time in so-called bursts [14, 15]. Although the time lags between interactions range from minutes to years, the temporal patterns are often rather predictable [16] and feature a high degree of regularity [8]. This spurs the hope that generic aspects of contact patterns can be reduced to simple statistical descriptions.

Here, we analyze real-world physical proximity data to extract signature effects of the contact behavior on disease spread without the need to rely on compartmental or agent-based models with their inherent assumptions. In particular, we develop a statistical description of individuals interacting with an unspecified community that allows us to identify resonance effects between statistical contact patterns and disease progression. Depending on the precise alignment of the infectious period with regions of statistically high or low encounter rates, the resulting number of potentially infectious encounters does not only depend on the infectious period, but remarkably also on the latent period (the time before becoming infectious) — an effect which is omitted in common disease models. We show that this resonance effect can be explained by weekly modulations in the contact pattern statistics and that this resonance shapes epidemic spread.

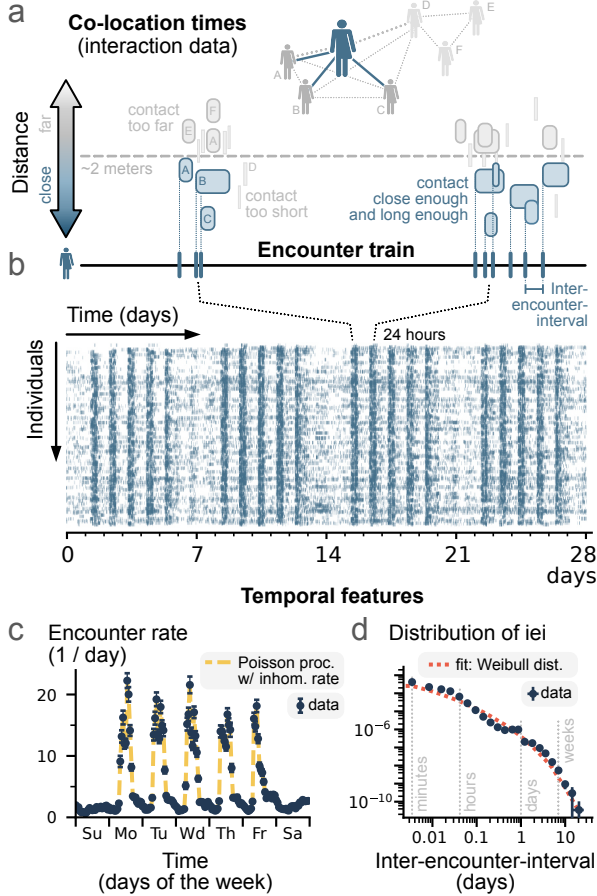


Figure 1. Real-world contact data can be represented as encounter trains. **a:** Co-location times correspond to time stamps at which a person is in contact with other people. Here we consider a disease for which contacts need to be closer than 2 meters and longer than 15 minutes to entail an appreciable transmission probability. **b:** In the encounter train, every time point corresponds to the start time of a contact that satisfies those conditions. **c:** Observables derived from the ensemble of encounter trains of all individuals reveal characteristic temporal features: The daily encounter rate, averaged across individuals and weeks, shows a clear day-night cycle and a dependence on the day of the week. **d:** The distribution of inter-encounter intervals (IEI), aggregated across all trains, shows that encounters are clustered in time, but also span multiple orders of magnitude. It can be well described by a Weibull distribution with shape parameter 0.36 and scale parameter 3029.69.

RESULTS

We base our analysis on data about physical proximity among university students (Fig. 1a) collected in the Copenhagen Networks Study (CNS) [17] and complement our results with additional data from SocioPatterns [18] in the Supplementary Information. To filter the data for sufficiently close and long contacts (see Methods), we only include contacts that are closer than

2 meters and longer than 15 minutes, which is in line with common guidelines used by regulators in the context of the COVID-19 pandemic but suitable choices for other pathogens will vary. From the remaining contact events, we extract for each individual a list of their contact starting times (encounters). These *encounter trains* are a point-process-like representation that forms the basis for our statistical analysis of individual contact patterns.

The collection of encounter trains reveals inhomogeneous contact patterns with a clear time-dependent structure (Fig. 1b). When averaging across trains and weeks of the experiment, we find this supported through a time-dependent encounter rate (Fig. 1c) — a feature that can be well reproduced by an inhomogeneous Poisson process. The collection of encounter trains further reveals strong temporal clustering — contact bursts — which becomes apparent through the distribution of inter-encounter intervals (Fig. 1d). This clustering can be well described by a Weibull distribution, with a high probability for short inter-encounter intervals (creating bursts) that slowly decays for long inter-encounter intervals (separating bursts). These temporal features are consistent with previous observations [14, 15, 19] and may be summarised as reoccurring, inhomogeneous contact patterns with local contact bursts.

In addition to these direct temporal features of contact patterns, we find strong variability across and within participants. This can be seen from the encounter statistics (Fig. 2a,b): The distribution of encounters *per train* can be modeled by an exponential, which indicates large variability across participants (Fig. 2a). This is a sign of heterogeneity between individuals — with many individuals who have low encounter rates and few individuals who have high encounter rates — and is consistent with previously observed non-Gaussian degree distributions of contact networks [17, 20]. The distribution of encounters *per day* again shows an exponential tail but it also shows a dominant peak at zero (Fig. 2b). This is a sign of variability over time — with many individuals having no valid encounters throughout complete days but some individuals occasionally aggregating large numbers of encounters. Depending on these aspects of encounter statistics and, in particular, on whether such aggregates of encounters fall into an individuals' infectious period (or not), contact patterns can profoundly affect disease spread.

To understand in more detail the effect of contact patterns on the spread of a particular disease, we assume that individuals can only be infected at recorded encounters (Fig. 2c). Whereas this neglects infections from outside the data set, it ensures that the temporal features of contact behavior are properly accounted for (see SM, Fig. S4 for additional controls). Once infected, an individual is assumed to be in an exposed state (not yet infectious), from which they progress into the infectious state after a latent period T_{lat} . After a further time T_{inf} ,

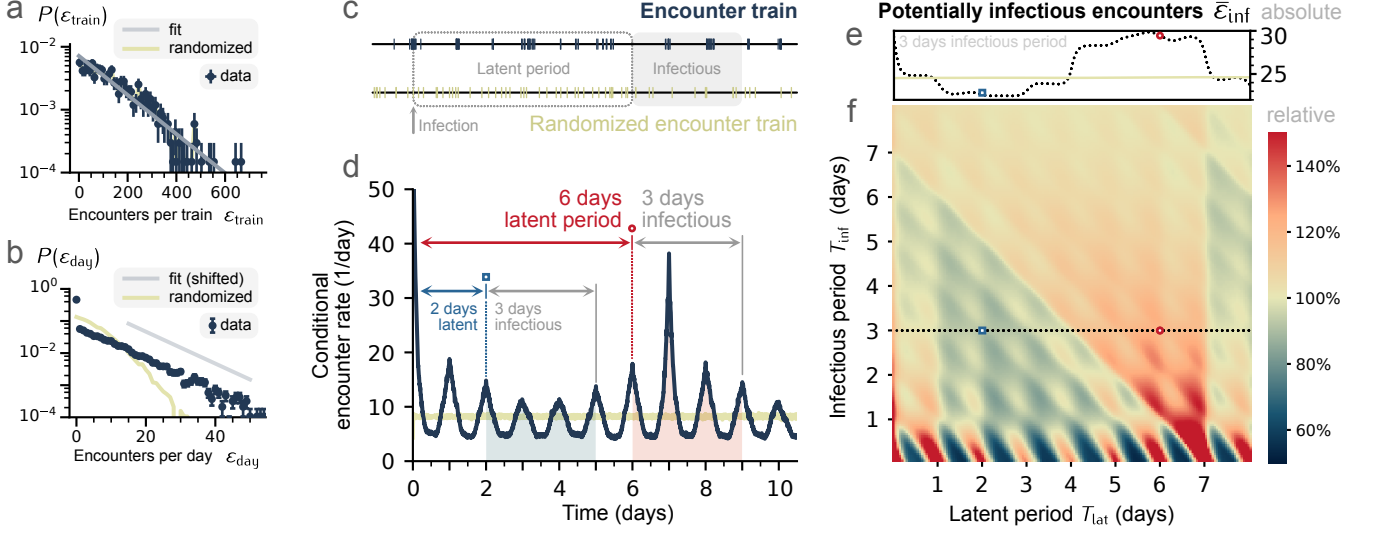


Figure 2. **The number of potentially infectious encounters is determined by a resonance between the time course of the disease progression and the temporal features of contact patterns.** **a, b:** Encounters are clustered in time, which is reflected in (exponential) probability distributions of encounters on the scale of trains and days, ϵ_{train} and ϵ_{day} . **c:** Resonance between disease progression and contact behavior: After an infection, encounters during the latent period are not infectious, but those during the infectious period are *potentially infectious*. Due to the temporal clustering at reoccurring times, the number of contacts that fall into the infectious period varies — depending on the time of the initial infection. This dependence is lost if the temporal features are weakened through randomization (marked yellow in all panels). Note that the randomization preserves the variability across individuals but destroys the temporal structure per individual (c.f. panels a & b: randomizing only suppresses large clusters in a). **d:** The *conditional encounter rate* describes the daily number of encounters a person has, on average, given that an encounter happened at time 0. The area under the curve yields the number of *potentially infectious encounters* (shaded regions for the two points marked in e, f). After randomization (yellow line), this area remains constant independently of the latent period. The peak at 7 days indicates that contact patterns statistically reoccur on a weekly basis. **e, f:** The number of potentially infectious encounters ϵ_{inf} depends on the combination of the latent and the infectious period. The difference to the randomized data (yellow, 100%) reflects both, the daily structure that creates the oscillations along the bottom, for short infectious periods, and the weekly structure that is responsible for the transition across the diagonal when infectious period and latent period sum up to seven days. **e:** Cut plane through panel f for a 3-day infectious period that shows ϵ_{inf} in absolute numbers.

the individual recovers and is no longer infectious. Since the probability of infection upon encounter depends on many factors (such as viral load, duration of contact, environmental conditions, hygiene measures), many of which are either out of control or scope of this study, we concentrate our analysis on the number of *potentially* infectious encounters ϵ_{inf} — the number of all encounters that occurred during the infectious period.

The number of potentially infectious encounters depends on both latent and infectious periods

To investigate the interplay of latent and infectious periods, we first consider a deterministic disease progression, where every infection results in the same latent period (T_{lat}) and the same infectious period (T_{inf}), with a fixed total disease duration of $T_{\text{total}} = T_{\text{lat}} + T_{\text{inf}}$ (Fig. 2c). In this case, we can enumerate all possible disease onsets for different combinations of latent and infectious periods (Fig. 2e-f) and compare the resulting mean number

of potentially infectious encounters $\bar{\epsilon}_{\text{inf}}$ to that for randomized encounter trains (yellow lines). In particular, we randomize the encounter times within each train but preserve the total number of encounters per train (Fig. 2a). These randomized encounter trains serve as a reference by removing temporal features, while leaving the variability across individuals intact.

How the latent period affects $\bar{\epsilon}_{\text{inf}}$ is demonstrated on the example of infectious diseases with a 3-day infectious period (Fig. 2e). We observe that a latent period of 1–4 days results in a lower $\bar{\epsilon}_{\text{inf}}$, compared to randomized encounter trains, whereas a latent period of 4–7 days leads to a higher $\bar{\epsilon}_{\text{inf}}$. Because the randomized trains exhibit no dependence on the latent period (the yellow line is almost constant), we conclude that temporal features of contact patterns are indeed responsible for the periodic modulation of $\bar{\epsilon}_{\text{inf}}$.

Clearly, the periodic modulation of $\bar{\epsilon}_{\text{inf}}$ depends on our chosen example and $\bar{\epsilon}_{\text{inf}}$ changes with the infectious period (Fig. 2f). To compensate for the trivial increase of $\bar{\epsilon}_{\text{inf}}$ with an increase in the infectious period T_{inf} , we con-

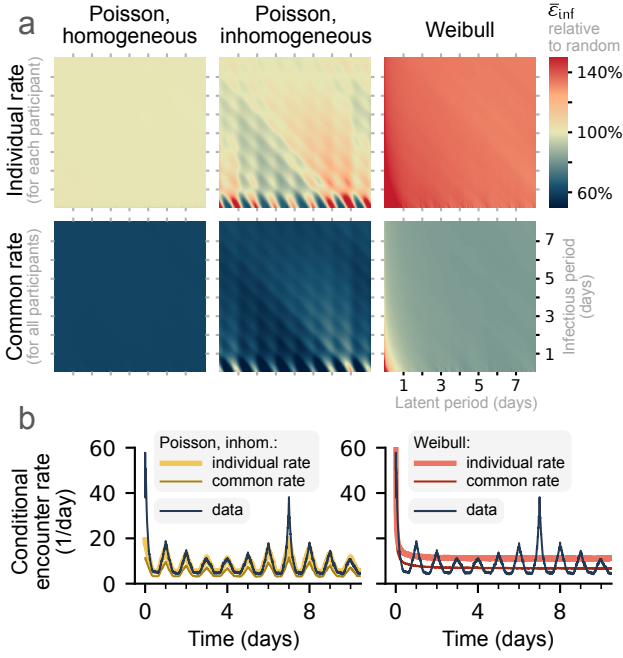


Figure 3. Every spatio-temporal feature has a unique impact on the number of potentially infectious encounters. To showcase the effect of individual features, we generated surrogate data. **a, top row:** For the surrogate data, the generating processes (columns) have an individual, unique rate for every participant, matching the data. **Bottom row:** The processes generating data for every participant share a common rate that matches the mean rate across participants in the data. Thus, the variability across individuals is lost and $\bar{\epsilon}_{\text{inf}}$ is systematically lower than in the top row. **Left column:** Surrogates from a homogeneous Poisson process lack reoccurring contact patterns and contact bursts. **Center column:** Surrogates from an inhomogeneous Poisson process with a rate matching the weekday-dependent rate of the data, c.f. Fig. 1c reproduce reoccurring contact patterns but lack contact bursts. **Right column:** Surrogates from a renewal process (with Weibull-distributed inter-encounter times, c.f. Fig. 1d), lack reoccurring contact patterns but reproduce contact bursts. **b:** The conditional encounter rate reflects the spatio-temporal features. Variability across individuals increases the conditional encounter rate (individual vs. common rate of the generating processes). Reoccurring contact patterns cause daily and weekly periodicity (Poisson, inhom.), while contact bursts cause a steep peak around zero (Weibull).

sider the relative change of $\bar{\epsilon}_{\text{inf}}$ compared to randomized trains for which $\bar{\epsilon}_{\text{inf}}$ depends on T_{inf} but not on T_{lat} . In the $(T_{\text{lat}}, T_{\text{inf}})$ plane, one can clearly see periodic modulations between regions of low (blue) and high (red) $\bar{\epsilon}_{\text{inf}}$. However, the regions smear out with increasing T_{inf} , and a few noteworthy transitions can be found for certain parameter combinations: One transition occurs when a total duration of $T_{\text{lat}} + T_{\text{inf}} = 7$ days is exceeded (large triangles, spanning almost the entire figure). Another transition occurs at $T_{\text{lat}} = 7$ days. Most prominently,

along the line of small $T_{\text{inf}} < 1$ day, we find a daily modulation between very high and low estimates, an effect that diminishes with increasing T_{inf} .

The unexpected dependence of $\bar{\epsilon}_{\text{inf}}$ on the latent period can be explained by a resonance between contact patterns and disease progression

To understand how changes of the latent period result in periodic modulations of $\bar{\epsilon}_{\text{inf}}$, we determined the *conditional encounter rate* as the average rate of encounters conditioned on an initial encounter at time 0 (Fig. 2d, see Methods). This rate shows how likely encounters are, given an encounter occurred a certain time ago. We find a strong peak around 0, indicating that immediately after an encounter there is a high chance for more encounters (contact bursts). We also find periodic modulations between high and low encounter rates, both, on the scale of days and weeks (note the peak at 7 days). The modulations indicate statistically reoccurring contact patterns: The 7-day peak, for instance, can be explained by the pattern of regular but inhomogeneous encounter rates across weekdays (Fig. 1c). These regular contact patterns are again lost for randomized encounter trains (yellow line).

If we interpret the initial encounter as an infection, then the conditional encounter rate implies periods of statistically high and low encounters after infection. For a particular disease progression (with given latent and infectious period) we can thus obtain an estimate of the potentially infectious encounters $\bar{\epsilon}_{\text{inf}}$ by integrating the conditional encounter rate within the infectious period (Fig. 2d, shaded areas). We demonstrate this for two disease realizations with the same infectious period ($T_{\text{inf}} = 3$ days) but different latent periods: The blue area ($T_{\text{lat}} = 2$ days) is much smaller than the red area ($T_{\text{lat}} = 6$ days), because in the latter case, the infectious period covers the 7-day peak. In other words, the disease progression resonates with a statistically high number of encounters.

To develop a deeper understanding of the period modulations in $\bar{\epsilon}_{\text{inf}}$, we distinguish two main timescales that we find in the conditional encounter rate: days and weeks. Let us first consider short infectious periods on a scale below one day. Because of the daily modulation in the conditional encounter rate, small changes in the latent period can align the short infectious period either with a local (1-day) minimum or maximum. This results in a resonance on the scale of days, and is reflected in the daily modulation of $\bar{\epsilon}_{\text{inf}}$ (Fig. 2f, blue and red regions near the bottom). This effect is due to the day-night cycle affecting participants' routines, and it diminishes as the infectious period increases, because multiple peaks of the conditional encounter rate get covered.

When considering longer infectious periods on a scale

of multiple days, we find that changes in the latent period may cause the infectious period to cover the 7-day peak in the conditional encounter rate (or not). As a consequence, combinations of latent and infectious period with a total duration below 7 days result in low $\bar{\varepsilon}_{\text{inf}}$ (large blue triangle), whereas a total duration above 7 days — but with a latent period below 7 days — results in high $\bar{\varepsilon}_{\text{inf}}$ (large red triangle). For deterministic disease progression in general, if T_{inf} is smaller than a given periodic structure in the conditional encounter rate, changes of T_{lat} will determine whether or not T_{inf} covers (local) extrema, and $\bar{\varepsilon}_{\text{inf}}$ will alternate depending on T_{lat} .

Resonance effects can be reproduced for inhomogeneous Poisson processes with distributed mean rates

To better understand the resonance effect, we isolate the influence of particular features of human contact behavior. We focus on how particular temporal features (reoccurring contact patterns and contact bursts) affect the mean number of potentially infectious encounters $\bar{\varepsilon}_{\text{inf}}$ and the conditional encounter rate. We again use $\bar{\varepsilon}_{\text{inf}}$ obtained from randomized encounter trains, where temporal features have been destroyed, as a baseline. To match this baseline, we first generate contact trains as homogeneous Poisson processes (Fig. 3a, left column). As anticipated, $\bar{\varepsilon}_{\text{inf}}$ does not depend on T_{lat} .

However, we find a dependence of $\bar{\varepsilon}_{\text{inf}}$ on the details of how we constrained the rate of the Poisson process: When processes share a common rate across individuals (bottom), $\bar{\varepsilon}_{\text{inf}}$ is about 40% lower than when every process has an individual rate (top). In both cases, the rates were constrained by data, either to the mean encounter rate across individuals (bottom) or directly to the underlying distribution (top). This shows that $\bar{\varepsilon}_{\text{inf}}$ only matches the data when the variability of encounter rates across individuals is captured.

To study how resonance effects depend on reoccurring contact patterns, we generate inhomogeneous Poisson processes with a rate that is again constrained by the data and, in addition, time-dependent (cf. Fig. 1c). Following our experience from homogeneous Poisson processes, we generate the processes once with common encounter rates (bottom) and once with individual encounter rates (top, see Methods). The resulting color maps of $\bar{\varepsilon}_{\text{inf}}$ show the characteristic resonance patterns and the conditional encounter rate exhibits periodic modulations on the scale of days and weeks (Fig. 3), where — as expected — the model with individual encounter rates is in better agreement with the data.

To study how resonance effects depend on contact bursts, as suggested by the measured distribution of inter-encounter intervals (Fig. 1d), we generate renewal processes with inter-encounter intervals drawn from the

Weibull distribution. Here, we incorporate the variability across individuals by adapting the scale parameter of the Weibull distribution while preserving the shape (see Methods). The resulting color maps reveal that the latent period affects $\bar{\varepsilon}_{\text{inf}}$ predominantly for low values, as only the initial peak in the conditional encounter rate of the data is reproduced by the Weibull renewal process. This peak corresponds to the short-term behavior upon initial contact. However, since inter-encounter times are uncorrelated random samples, the Weibull renewal process only captures this short-term behavior and does not reproduce the characteristic resonance patterns. We conclude that the contact bursts are responsible for the initial peak in the conditional encounter rate and predominantly affect the leftmost part of the color maps, whereas the reoccurring contact patterns shape the periodic structure of the conditional encounter rate and, thereby, the overall pattern of the color maps.

Resonance effects remain relevant also when individual disease progression is more variable

So far, we have focused on deterministic disease progression, where latent and infectious periods had a precise duration. For a more realistic view, we want to allow for the latent and infectious periods to vary from case to case (Fig. 4). To that end, we draw the periods T_{lat} and T_{inf} from a gamma distribution, but we keep the mean duration fixed. The case-to-case variability is then parameterized through the dispersion parameter k (Fig. 4a).

By adjusting k , the gamma distribution can interpolate between delta-distributed periods ($k \rightarrow \infty$), which coincide with deterministic progression as in Fig. 2, and exponentially distributed periods ($k = 1$), as commonly assumed in computational epidemiology for mathematical tractability [7]. On the one hand, delta-distributed periods seem like a convenient but unrealistic simplification. On the other hand, exponentially distributed periods seem more realistic, but they imply an artificially high probability of short durations, which in turn leads to realizations where the infectious period either starts shortly upon infection or has close-to-zero duration (example traces in Fig. 4a). Because a realistic distribution is difficult to infer from epidemiological data, we argue for a dispersion parameter *somewhere* in between delta and exponential.

To investigate how case-to-case variability affects the number of potentially infectious encounters ε_{inf} , we consider the probability distribution $P(\varepsilon_{\text{inf}})$ (Fig. 4c), and revisit the two examples already presented in Fig. 2. For the delta-disease ($k \rightarrow \infty$, top), the red ($T_{\text{inf}} = 3$ days, $T_{\text{lat}} = 6$ days) and blue ($T_{\text{inf}} = 3$ days, $T_{\text{lat}} = 2$ days) distributions exhibit a peak at zero, they are broad, and have a long tail. Comparing red and blue, we find that

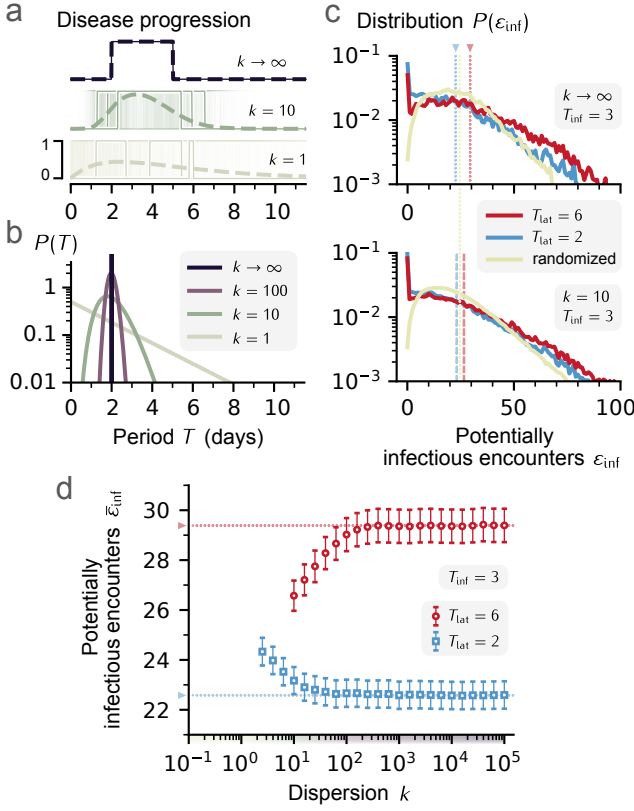


Figure 4. **Resonance effects remain relevant also when disease progression varies from person to person.** **a, b:** To include person-to-person variability, we draw both latent and infectious periods from gamma distributions characterized by the dispersion parameter k , which interpolates between exponential ($k = 1$) and delta ($k \rightarrow \infty$) distributions. For small k , T_{lat} and T_{inf} differ in duration from realization to realization — across individuals, the probability to be infectious at a given time is smeared out. As k increases, the periods vary less around their expected value and the disease progression eventually becomes deterministic (c.f. Fig. 2). **c:** Probability distributions of potentially infectious encounters ϵ_{inf} for $k \rightarrow \infty$ (top) and $k = 10$ (bottom). When randomizing trains, the probability of zero-infectious encounters is suppressed. **d:** Mean number of potentially infectious encounters $\bar{\epsilon}_{\text{inf}}$ as a function of k for the two examples from Fig. 2. For $k \rightarrow \infty$, we recover the result for deterministic disease progression (dashed lines), where the latent period induces a notable difference between the two examples. For smaller k , the resonance effects remain relevant but the difference decreases, see also Supplementary Information Fig. S3.

the latent period determines the height of the peak at zero as well as the position of the bulk distribution, and, thereby, determines the mean number of potentially infectious encounters (dashed vertical lines). The mean values clearly differ. Again comparing with the randomized encounter trains (yellow line), we find no peak at zero, a shorter tail, and no dependence on the latent period (the respective randomized lines fall on top of each other). Importantly, a peak at zero implies that the in-

fectured individual does not pass on the infection, so that the disease becomes more likely to die out if case numbers are low.

Changing to the non-deterministic disease progression ($k = 10$, Fig. 4c bottom), the distribution from randomized data is barely affected. However, the red and blue distributions are more similar to each other, but also broader and smoother than for the the delta-disease. This can be explained by gamma-distributed periods acting as a smoothing kernel along both dimensions of Fig. 2f, where variability in the infectious period directly affects $\bar{\epsilon}_{\text{inf}}$, while variability in the latent period affects $\bar{\epsilon}_{\text{inf}}$ through the resonance effect. Consequently, we expect that with decreasing dispersion parameter k , the mean number of potentially infectious encounters becomes independent of the mean latent period. Indeed, when considering $\bar{\epsilon}_{\text{inf}}$ as a function of k , we find that the estimates for our two examples approach each other, as k decreases (Fig. 4d).

Note that our analysis has a lower bound in k once realizations of disease progression (latent + infectious period) cannot find sufficiently many initial encounters to fit into the finite duration of the experiment (4 weeks for CNS). However, using other examples with smaller latent and infectious periods (where we can acquire enough statistics), we show that the two extreme cases meet for $k \approx 1$ (see Supplementary Information Fig. S3).

Resonance effects shape epidemic spread

To demonstrate how the described resonance effect shapes epidemic spread, we devise a mean-field spreading process with resonance-driven infections (Fig. 5a). Here, the epidemic starts from a single infected individual. For each infected individual, we generate encounters according to the conditional encounter rate (resonance included) or according to a randomized control (resonance neglected). Importantly, infected individuals undergo deterministic disease progression, where during the infectious period secondary infections are created from encounters with a constant probability (see Methods). Because of the constant probability of infection, the mean number of secondary infections is proportional to $\bar{\epsilon}_{\text{inf}}$ and thereby shaped by resonance.

However, the pace of epidemic spread is not solely governed by the number of secondary infections. Apart from a larger number of secondary infections, a shorter time from initial infection to secondary infections also accelerates the spread. Since this generation time is directly influenced by the latent period, we expect the latent period to determine the absolute pace of spread. We find this supported in Fig. 5b, where shorter latent periods result in a faster increase of daily new cases, compared to longer latent periods. However, when comparing the mean-field model with and without resonance effects, we

find that resonances can increase ($T_{\text{lat}} = 2$ days) or decrease ($T_{\text{lat}} = 6$ days) the pace of the spread by up to 20%.

The modulation of epidemic spread due to resonance can be understood through the expected number of secondary infections (Fig. 5c). The mean number of secondary infections per individual (the basic reproduction number R_0) is — by construction — proportional to $\bar{\varepsilon}_{\text{inf}}$; hence, it shows the same modulation as a function of the latent period. These modulations of R_0 explain the different pace of spread with and without resonance (Fig. 5b, solid vs. opaque lines) since R_0 is proportional to the pace of epidemic spread at a given generation time.

It is illustrative to additionally consider a naive estimate R_{naive} with the false assumption of a fixed generation time. Whereas such an estimate does not directly reflect R_0 , it quantifies the pace of epidemic spread that can be compared across different latent periods (Fig. 5b, blue vs. red). Indeed, it confirms the expectation that the spread is slower for longer latent periods. In addition, we find resonances to cause modulations of R_{naive} (shaded regions, red vs. blue). This highlights that changes in the latent period dominate the pace of disease spread while resonance effects shape it.

DISCUSSION

In summary, we analyzed physical proximity data of university students from the Copenhagen Networks Study and found that contact patterns can resonate with disease progression (Fig. 2). These resonances arise from periods of statistically high and low encounter rates, which can be attributed to reoccurring contact patterns and variability in the encounter rates across individuals (Fig. 3). Surprisingly, contact bursts seem less relevant for disease models with a latent period.

The temporal structure of contact patterns shapes epidemic spread in several ways. We found that, compared to randomized data, it makes infection chains more likely to die out when case numbers are low (Fig. 4c), which affects the robustness of the spreading process. This robustness, however, is only relevant for low case numbers and does not directly determine the pace of epidemic spread. In this regard, we showed that beyond the trivial dependence on the generation time the pace of epidemic spread can be substantially increased or decreased by resonance effects (Fig. 5). Using our mean-field model, we were able to separate the two effects. This emphasizes the importance of the temporal structure of contact patterns for assessing the robustness and the pace of epidemic spread.

Our individual-based perspective enables us to discover so-far unexplored effects of contact patterns on disease progression based on the following assumptions. For one, we assume encounter trains to be independent, neglecting

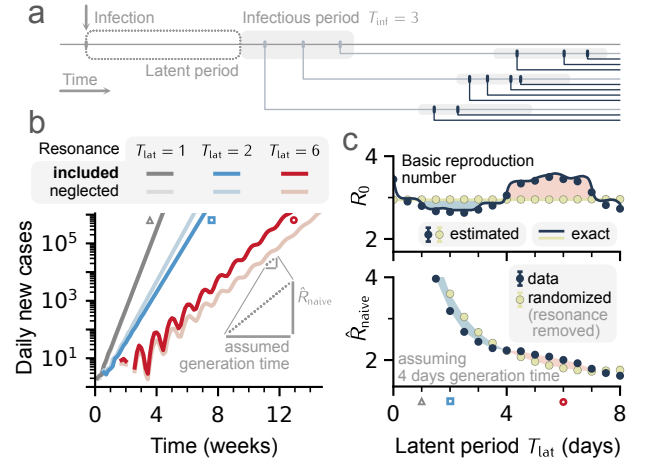


Figure 5. Temporal resonance shapes mean-field epidemic spread. **a:** Sketch of the mean-field model with subsequent infections due to one initial infection, assuming a basic reproductive number of $R_0=3$. **b:** Daily new cases (logscale) for disease progression with $T_{\text{inf}} = 3$ days and different latent periods T_{lat} . When temporal features are destroyed through randomization, case numbers are biased (faint lines). **c:** The bias corresponds to the difference of R (shaded regions) when derived from the original data with temporal features (blue) compared to the randomized data (yellow). Note the difference between the basic reproduction number R_0 (which is the true expectation value of the number of secondary infections) and a naive estimate of the reproduction number from case numbers $\hat{R}_{\text{naive}}$ (which falsely assumes a fixed generation time).

correlations due to the temporal network, which implies that individuals interact with a mean field and restricts our analysis to an individual-based perspective. Moreover, we treat multiple encounters with the same individual inside the infectious interval as independent contributions. This has negligible effects on our conclusions of how contact patterns affect disease spread as long as disease transmission has a sufficiently low probability, such that multiple encounters merely increase the probability of transmission. Further, we share a common assumption in computational epidemiology and consider binary disease states (infectious or not), which neglects time-dependent viral loads. Therefore, it is consistent to also neglect the duration of contacts (otherwise, the probability of infection would be proportional to the integral of the viral load over the contact duration). Altogether, we arrive at our point-like representation of encounter trains that allows us to create generative models and surrogate data, exposing the origin of resonance effects between disease progression and contact patterns.

We develop our results at the example of data from the Copenhagen Networks Study, but we expect our conclusions to apply to a broad range of settings of human interaction. To confirm our main findings, we repeated our analysis with a complementary data set [9] from sociopat-

terns.org, which was recorded differently (using "near field chips"), in a different country (France) and for a different social group (office workers). Since we find consistent results (Supplementary Information, Fig. S2), we expect that resonance effects appear in various contexts that give rise to reoccurring contact patterns.

Our results emphasize the relevance of including temporal features when modelling disease spread. To reproduce those temporal features of contact behavior, we had to introduce non-Markovian contact dynamics in the form of inhomogeneous encounter rates or Weibull-distributed inter-encounter intervals. In fact, the latter was previously used to study the effect of contact bursts on disease spread [21]. It was found that Weibull-distributed inter-encounter intervals have a drastic effect on the epidemic threshold for disease models without a latent period, which can be attributed to the frequency of small inter-encounter intervals [22] and is consistent with our observation of a peak near zero in the conditional encounter rate (Fig. 2). Our results suggest that considering the more general case of non-zero latent periods causes shifts in the epidemic threshold that depend on the latent period. As such, this motivates future work to study resonance effects in complex networks.

In addition, we also considered non-deterministic disease progression in the form of gamma-distributed latent and infectious periods (Fig. 4), which highlights the complex interplay of temporal features from both contact behavior and disease progression. We found that, as disease realizations become less deterministic, the impact of reoccurring contact patterns on the number of potentially infectious encounters is smeared out. Importantly, the vanishing impact of resonances also occurs for exponentially distributed latent and infectious periods. While for exponential disease progression it was shown that non-Markovian spreading dynamics can be mapped onto effective Markovian models [23], such a mapping is elusive for non-exponential disease progression. This implies, however, that resonance effects as well as so-far unknown effects that could derive from non-Markovian contact behavior are likely not captured by traditional models of exponential disease progression.

The resonance effect between contact patterns and disease progression has implications for non-pharmaceutical interventions through individual behaviour. Because we identified statistically reoccurring contact patterns as the dominant source of resonance, interventions could be designed so that contacts predominantly take place during times when individuals are likely not infectious. As an example, if a hypothetical disease had exactly two days latent period and three days infectious period, an intervention could encourage individuals to only meet people at a specific day of the week — which then have zero chance to pass on the infection. The scenario changes to one where the probability of subsequent infections can be reduced if latent and infectious period are drawn from

known distributions. This emphasizes how important it is to know the actual time-course of a disease, so that individuals can adapt their contact patterns and non-pharmaceutical interventions can be designed to actively exploit resonance effects.

Conversely, the resonance effect may be one part of the gain function in viral evolution. From an evolutionary perspective, a virus could increase its spreading by adapting the latent period. Compared to an increase of the infectious period, this could be beneficial since the latent period remains unnoticed by the host, precluding measures like self-isolation.

METHODS

Our methods are publicly available [24] and applied to open-access data-sets [9, 17].

Extracting contact behavior from real-world physical proximity data

Consider data composed of a list of co-locations (physical proximity) described by (i) timestamp, (ii) user id A, and (iii) user id B. We first sort the co-location times into unique lists for all id pairs (A,B) and (B,A). For each valid id A, we then iterate over all B and the associated list of co-location times for (A,B) to construct pairwise contacts by merging successive co-location times. We thereby construct lists of contacts for each participant A, where each contact is defined through its starting time s^i and its duration D^i , resulting in $\{(s^i, D^i)\}_A$.

From the lists of contacts, we construct a point-process-like representation for each participant that we call *encounter train* (see Fig. 1). Throughout the manuscript, an *encounter* refers to the starting time of a contact. The encounter train of participant A is the time-sorted list of all contact starting times and can formally be written as

$$T(t) = \sum_i \delta(t - s^i) \quad (1)$$

The data from the Copenhagen Networks Study [8, 17] is based on Bluetooth signals between phones of individuals that participated in the study. The published data is a list of interactions described by (i) a timestamp, (ii) user id A (iii) user id B (which can be negative if the interaction is with a device outside of the study or an empty scan), and (iv) the received signal strength indicator (RSSI, Bluetooth signal strength). The RSSI can be considered as a proxy for interaction distance, especially since all participants used the same device [8], with an RSSI ≈ -80 corresponding to a distance of about $2\text{ m} \pm 0.5\text{ m}$. We consider RSSI < -80 to indicate interactions within each time window to be further

than 1.5 m apart at all times, and exclude them. Consequently, we filter the raw data to only include those interactions that are within the study (user id $B \geq 0$) and have $RSSI \gtrsim -80$. The data set covers a duration of 28 days, with a temporal resolution is $\delta t = 5$ min, for 675 encounter trains.

Time-dependent encounter rate

Because encounter trains are a point-process-like representation, we can define an encounter rate as the number of encounters in a window of size Δt . Assuming statistical independence between weeks and between participants, we determine the time-dependent encounter rate by averaging the number of encounters in a time windows of size $\Delta t = 1$ h throughout the week (i.e. first hour of a Sunday until last hour of a Saturday) across weeks of the experiment and across participants. Statistical errors are calculated on the level of participants using standard delete- m jackknife error analysis.

Distribution of number of encounters

From the encounter trains, we can directly obtain the distribution of the number of encounters in given time window. Here, we focus on two cases. The *total number of encounters* is the number of encounters measured per encounter train and the respective distribution is obtained from the sample of trains. The *daily number of encounters* is the number of encounters of individuals during a day. The distribution of daily number of encounters is obtained by counting for each train the sum of encounters for every day in the experiment. Statistical errors are calculated on the level of participants using standard delete- m jackknife error analysis.

Inter-encounter interval (IEI)

In order to study temporal clustering and contact bursts, we determine inter-encounter intervals (IEIs). An IEI is defined as the interval between consecutive encounter times. Because each encounter train has a different number of encounter times (n_j), it contributes a different number of IEIs ($m_j = n_j - 1$). Since we are interested in the statistics of the contacts, each encounter has the same statistical weight independent of the encounter train of origin. Consequently, the distribution of IEIs is simply the distribution over all observed intervals. Statistical errors are evaluated on the level of observed intervals using delete- m_j jackknife error analysis.

Conditional encounter rate

To investigate how contact patterns interact with disease spread, it is useful to ask how many encounters an infected individual might have after they were infected. Thus, in addition to the time-dependent encounter rate, we also evaluate a time-dependent encounter rate conditioned on having a contact at time $t = 0$. This conditional encounter rate is constructed by iterating over all encounters of each encounter train. For each encounter, we construct a time-dependent encounter rate with the time resolution of the experimental data and a selected length (typically 10 days) or, if this does not fit into the remaining duration of the experiment, whichever duration is left. We then average over all these time-dependent encounter rates taking into account their different lengths. Statistical errors are calculated on the level of encounters using delete- m_j jackknife error analysis.

Randomizing trains

To obtain surrogate data (without resonance effects) that we can compare against, we randomize encounter trains that preserve person-to-person variability but destroy temporal correlations in the data: For every encounter train, we preserve the number of encounters (and, thus, person-to-person variability), however, for each encounter we draw a new random start time within the duration (and at the temporal resolution) of the experiment.

Approximating contact behavior with point process models

We consider two models that capture different temporal aspects of the individual contact behavior statistics: An inhomogeneous Poisson process and a Weibull renewal process. In both cases, we constrain the models with specific aspects of the data and create artificial encounter trains with matching duration (for CNS this is 28 days) and matching number of trains. We distinguish between models with common rates across trains and those with individual rates per train to match the empirical distribution of the data (Fig. 2b).

The *inhomogeneous Poisson process* is a Poisson process with a time-dependent rate. The rate is constrained by the week-averaged encounter rate $r(t)$ (Fig. 1c) with time resolution of the data. In our model with common rates, we generate each encounter train by thinning [25], where we start from an initial event 7 days in the past, generate subsequent events from a homogeneous Poisson process at rate $r_{\max} = \max_t[r(t)]$, and accept events at time t with the probability $r(t)/r_{\max}$. In our model

with individual rates, we do the same but rescale individual encounter rates as $r^i(t) = r(t)\varepsilon_{\text{train}}^i / \sum_i \varepsilon_{\text{train}}^i$, where $\varepsilon_{\text{train}}^i$ is the total number of encounters of the respective train from the data.

The *Weibull renewal process* is a renewal process with Weibull-distributed inter-encounter intervals with the goal to reproduce the statistics of the inter-encounter intervals (IEIs). Motivated by previous models [21], we describe the one-point statistics of IEIs as a Weibull distribution (cf. Fig. 1d) with fit parameters (shape, scale) = (0.36, 3029.69). For each train, independent realizations are generated with a renewal process that starts 7 days in the past (to ensure equilibration) and creates subsequent times by drawing a random IEI from the Weibull distribution. In our model with common rates, all renewal processes share the same parameters as above. In our model with individual rates, we adjust the mean rate of the Weibull renewal process, which is proportional to the inverse scale, by adjusting the scale parameter of the individual Weibull distribution as $\text{scale}^i = \text{scale} \sum_i \varepsilon_{\text{train}}^i / \varepsilon_{\text{train}}^i$.

Disease models

We consider a disease that progresses in three discrete states after infection, first exposed, then infectious, and recovered. Throughout the states, infectiousness is binary, either infectious or not. The duration within the exposed state is called *latent period* and the duration within the infectious state is called *infectious period*. The duration of each period is drawn from uni-variate distributions that define the class of the disease model.

We first consider *deterministic disease progression*, where the periods are drawn from delta distributions and, hence, always the same. This model would be most intuitive to a patient who expects that different stages of a disease will last for a certain time. However, it is quite different to the common Markov model of disease progression, in which the periods would be drawn from exponential distributions — as expected for Poisson processes that describe many state transitions, from radioactive decay to chemical reactions.

We further consider *non-deterministic disease progression*, where the periods are drawn from a gamma distribution with dispersion parameter k . The dispersion parameter allows us to interpolate between a delta distribution ($k \rightarrow \infty$) and an exponential distribution ($k = 1$). Clinically observed distributions of periods between disease states are neither delta distributed nor exponential distributed and may be best described by distributions with a clear peak but vanishing probability at zero, such as log-normal distributions or gamma distributions with dispersion parameters in between $(1, \infty)$.

Estimating potentially infectious encounters

Because the probability of an infection depends on many factors, we focus on all encounters that have a chance to be infectious. We therefore define potentially infectious encounters as the number of encounters during the infectious period.

For the deterministic disease progression with latent period T_{lat} and infectious period T_{inf} , we iterate over all encounters s^i across all trains, check whether the disease progression still fits into the duration T_{exp} of the experiment ($s^i + T_{\text{lat}} + T_{\text{inf}} \leq T_{\text{exp}}$), and if true count the number of subsequent encounter s^j for which $T_{\text{lat}} < s^j - s^i < T_{\text{lat}} + T_{\text{inf}}$.

For the non-deterministic disease progression with dispersion parameter k , we generate 10^6 samples of disease realizations, each of which yields one estimate of ε_{inf} . For each sample, we first draw a random realization of the disease progression ($T_{\text{lat}}^i, T_{\text{inf}}^i$). We then draw disease start times s^i from the ensemble of all encounters until we find an s^i such that the disease progression is within the remaining duration of the experiment, i.e., $s^i + T_{\text{lat}}^i + T_{\text{inf}}^i \leq T_{\text{exp}}$. Only once we have a valid disease start time s^i , we count the number of subsequent encounters within the infectious period as above. If, for any disease realization, we need to draw more than 1000 disease start times until we find a valid one, we abort the estimation for the set of parameters $(T_{\text{lat}}, T_{\text{inf}}, k)$. By this procedure to first draw and fix a random realization of the disease progression, we avoid a bias towards small periods that would occur due to the finite period of the experimental data.

Errors are calculated using the delete- m_j jackknife method because the relevant statistics is the ensemble of encounters which differs from train to train.

Mean-field model for epidemic spread

To study the effect of resonances on the pace of epidemic spread, we devise a mean-field model that neglects potential interactions with non-susceptible individuals in an infinite population. In our model, each infected individual generates independent encounter trains. Encounter trains are generated as inhomogeneous Poisson processes that start at the infection with a time-dependent rate given by the conditional encounter rate (Fig. 2d) to model the statistically reoccurring contact patterns. Encounters that occur during the infectious period cause secondary infections with a chosen probability p . Every secondary infection then generates a new encounter train and so on. This model can be considered as a continuous-time non-Markovian branching process.

For simplicity, we restrict our example to deterministic diseases with fixed latent and infectious periods.

Jackknife error estimation

To estimate statistical errors of our results, we use jackknife error estimation while carefully taking into account the size of the left-out data set.

The basic idea of the jackknife method is to estimate from some data $X = \{x_1, \dots, x_g\}$ the variance of an observable $\hat{O} = f(X)$ using a systematic resampling approach [26]. Jackknife samples O_j are generated by systematically leaving out data, e.g., $\hat{O}_j = f(X_{\bar{j}})$ with $X_{\bar{j}} = \{x_1, \dots, x_{j-1}, x_{j+1}, \dots, x_g\}$. Importantly, here each x_i can be a block of (differently many) data points. While typically these blocks have the same size m (delete- m jackknife), they could have different sizes m_j (delete- m_j jackknife), which will be relevant for some of our cases. From the jackknife samples, one can show that bias-reduced estimators of the mean and variance are given by [27]

$$\begin{aligned}\hat{O}_J &= \sum_{j=1}^g \frac{1}{h_j} \left(h_j \hat{O} - (h_j - 1) \hat{O}_j \right), \\ \hat{\sigma}_J^2 &= \frac{1}{g} \sum_{j=1}^g \frac{1}{h_j - 1} \left(h_j \hat{O} - (h_j - 1) \hat{O}_j - \hat{O}_J \right)^2, \quad (2)\end{aligned}$$

where $h_j = (\sum_{j=1}^g m_j)/m_j$, and $\hat{O} = f(X)$ is the naive estimate. For blocks of equal size, $m_j = m$, we have $h_j = g$ and this simplifies to

$$\begin{aligned}\hat{O}_J &= g\hat{O} - \frac{g-1}{g} \sum_{j=1}^g \hat{O}_j, \\ \hat{\sigma}_J^2 &= \frac{g-1}{g} \sum_{j=1}^g \left(\hat{O}_j - \frac{1}{g} \sum_{j=1}^g \hat{O}_j \right)^2. \quad (3)\end{aligned}$$

In our case, the data X is the set of all encounter trains and in the resampling step we leave out individual encounter trains. Since trains include differently many encounters, this can result in removing blocks of different sizes. In particular, all observables that derive from the number of encounters, e.g., $\bar{\varepsilon}_{\text{inf}}$ or $P(\varepsilon_{\text{inf}})$, require the delete- m_j analysis, (2), to estimate the statistical error. On the other hand, for observables that depend on time-binned data, e.g., the time-dependent rate, each encounter train has the same size given by the number of time bins during the recording such that the delete- m analysis, (3), is sufficient to estimate the statistical error.

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SUPPLEMENTARY INFORMATION

In the Supplementary Information, we provide additional controls to verify the robustness of our results in the main manuscript. In particular, we provide a reanalysis of our main data set where we only consider participants for which we can ensure a continuous participation in the experiment (Sec.), as well as an analysis of an additional, independent data set (Sec.). Both analyses fully support our main results. In addition, we provide complementary analyses of our main data set. These include (i) a more detailed analysis of shorter latent and infectious periods compared to the example considered in the main manuscript that allows us to reach the low-dispersion regime (Sec.), and (ii) an analysis that verifies that our conclusions remain valid for infections from outside of the study group (Sec.).

Control regarding continuous contribution of participants

In the main manuscript, we use the full published data set of the Copenhagen Networks Study [17], covering the physical proximity data of 675 participants. Upon closer inspection, there are periods both at the beginning and the end of the experiment without entries for some of these 675 participants. Since entries also occur for Bluetooth signals with unknown devices, this may indicate irregularities in the contact behavior of some of the participants, e.g., incomplete participation of individuals.

In order to make sure that our results are not affected by such boundary effects, we reanalyzed the data and included only the contact trains of those individuals for which any Bluetooth signal was recorded on both the first and last day of the study (Fig. S1). Technically, we achieved this easily by restricting our analysis to those IDs for which timestamps were recorded within the first day ($\text{timestamp} < 1 \cdot 24 \cdot 60 \cdot 60\text{s}$) and the last day ($\text{timestamp} > 27 \cdot 24 \cdot 60 \cdot 60\text{s}$), reducing the data set to 533 contact trains.

This control analysis fully supports our quantitative results from the main manuscript (Fig. S1) such that we can rule out artifacts from boundary effects of incomplete participation. In particular, we observe a matching weekly structure of the encounter rate (Fig. S1a), a matching distribution of inter-encounter intervals that can be fitted with a Weibull distribution (Fig. S1b), and a matching conditional encounter rate (Fig. S1c). Consequently, both mean potentially infectious encounters for deterministic disease progression (Fig. S1d and f) as well as for non-deterministic disease progression (Fig. S1e and g) match our main results (Figs. 2 and 3 in main manuscript).

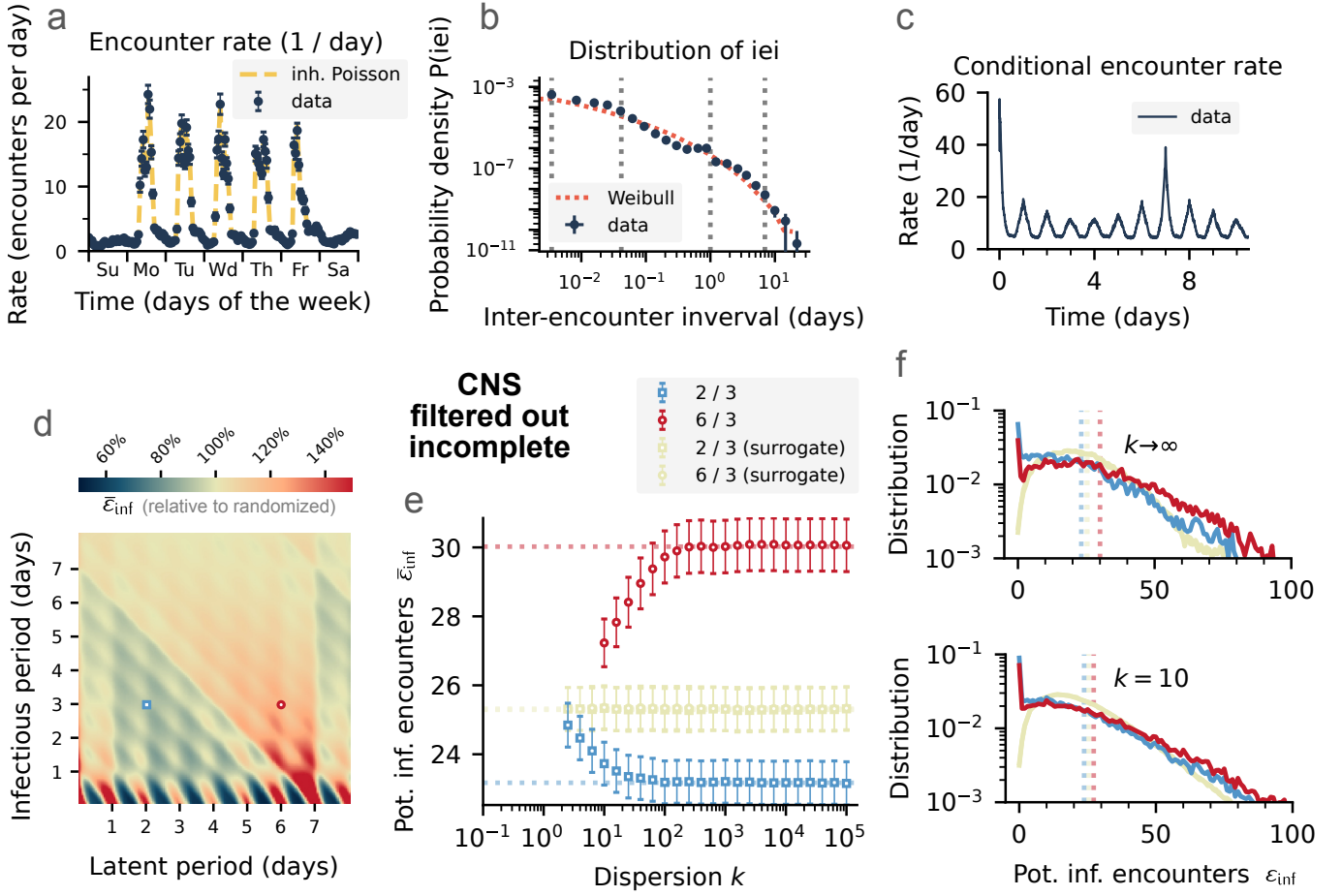


Figure S1. **Control regarding continuous contribution of participants** Here, we excluded those trains that did not have any encounters during the first or last day. This results in 533 instead of 675 trains. Panels match key figures from the main manuscript. Results are consistent. The fitted Weibull distribution has shape parameter 0.37 and scale parameter 3161.95.

Analysis of an alternative data set

To further challenge the robustness of our results, we repeated our analysis on a completely independent data set. Here, we consider contact data recorded at one of the office buildings of the French Institute for Public Health Surveillance *InVS* [20]. This data is recorded with a different technique, namely so-called near-field chips that only record signals in close proximity ($\lesssim 5$ m) and avoid to threshold the Bluetooth signal. Moreover, the temporal resolution of contacts is 20 s as opposed to 5 min in the main manuscript. In addition, the data is recorded for a different social group, namely adults within an office building. Last, the data is recorded in another country (France) by a different collaboration (SocioPatterns). The data set spans 2 weeks of recording 145 participants (two thirds of the staff agreed to participate).

The analysis of this completely independent data set provides completely consistent results to those presented in the main manuscript (Fig. S2). When comparing the results, we have to highlight that the available statistics for this data set are much smaller due to smaller duration and smaller number of participants. However, we clearly see the expected weekly structure in the encounter rate (which is here again dominated by working days because of office hours), the distribution of inter-encounter intervals that is well described by a Weibull distribution, as well as the typical conditional encounter rate with a peak at 7 days. Consequently, also the results for both deterministic and non-deterministic disease progressions are consistent with our main findings albeit confined to shorter latent and infectious periods due to the shorter experimental duration. We conclude that the additional data set fully supports our results in the main manuscript.

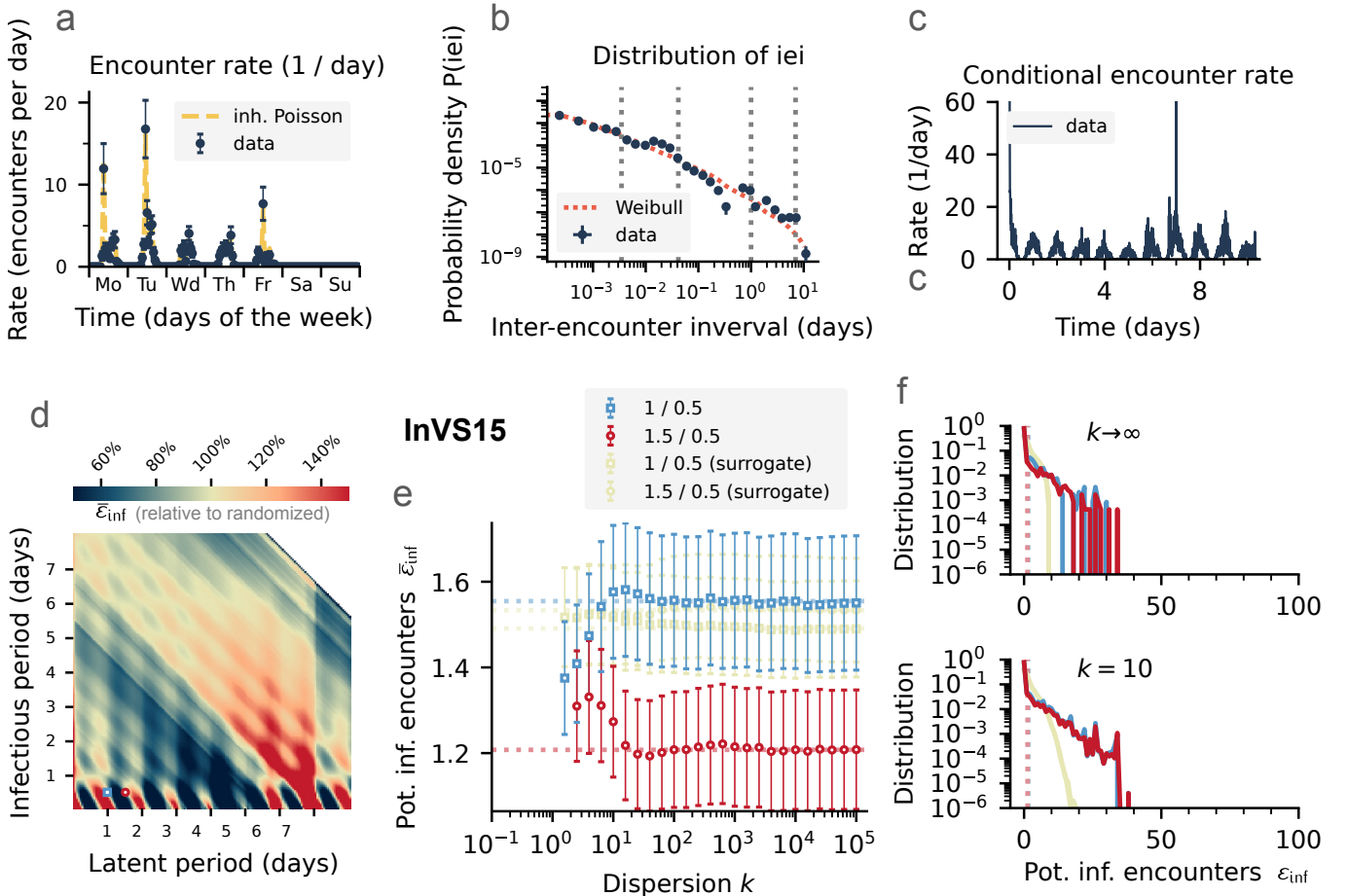


Figure S2. Main results for other data set. Because the recording lasted *only* two weeks, the duration of disease that can be sampled to estimate the dispersion were limited to the fast disease progression. The fitted Weibull distribution has shape parameter 0.22 and scale parameter 675.03.

Example with smaller latent and infectious periods provides additional insight on low-dispersion regime of non-deterministic disease progression

In the main manuscript, we repeatedly consider the example of disease progression where the expected infectious period was $T_{\text{inf}} = 3$ days and the expected latent period T_{lat} was either 2 or 6 days. We chose this example as it illustrates well the resonance effect on the scale of multiple days. However, we were not able to sample the dispersion relation for small k values (more variability across disease realizations) because the 28-day duration of the data becomes too short once the periods of disease progression are close-to exponentially distributed ($k \rightarrow 1$).

To avoid this issue (and to illustrate the resonance on shorter timescale), we here compare with another hypothetical example of a “faster” disease progression, where $T_{\text{inf}} = 0.5$ days and T_{lat} is either 1 or 1.5 days (Fig. S3). In this case, expected periods are very short so that even realizations with periods that severely exceed their expected value fit into the 28-day duration.

We find a few noteworthy aspects: First, the absolute value of potentially infectious encounters ε_{inf} is much lower for faster disease progression. This is due to the much shorter infectious period. However, the relative deviation from the randomized surrogate data is consistent between the two examples. Second, we now find that the longer latent period (1.5 vs. 1.0) leads to fewer potentially infectious encounters (red vs. blue). This is just a result of the chosen latent periods: choosing an even longer latent period would result in a respective decrease of ε_{inf} (e.g. 2.0 vs 1.5, cf. Fig. 2f main manuscript). Third, as expected, as $k \rightarrow 1$, the resonance effect vanishes and the estimates of ε_{inf} overlap for different latent periods, because individual disease realizations become very variable (cf. Fig. 4a).

We can obtain even more information from the distributions of potentially infectious encounters (Fig. S3). Here, we notice that distributions obtained from data for both slow and fast disease progression share similar features that distinguish them from distributions obtained from randomized encounter trains: Those distributions obtained from contact data features distinct peaks for zero potentially infectious encounters and heavy tails towards large number of potentially infectious encounters. The peaks at zero clearly depend on the chosen latent period and are partly responsible for the differences in the mean. However, beyond affecting the mean, these peaks at zero ε_{inf} also affect the robustness of the disease spread: Compared to randomized contact trains, more contact trains obtained from data have a much higher chance of zero secondary infections such that disease spread from a single infection is much less robust compared to randomized trains (Poisson statistics).

Beyond such robustness effects that are relevant only for low incidence, the distributions obtained from contact data are more variable than for randomized trains when compared in the high-dispersion regime. In particular, distributions obtained from contact data stretch to much higher ε_{inf} which resembles so-called super spreaders. As expected, the probability of both low and high ε_{inf} increases with decreasing dispersion because the resulting gamma distribution features increased probability for both shorter and longer infectious periods. Consequently, one may expect that for lower dispersion the distributions obtained from contact data and randomized data become more similar as confirmed by our example of short latent and infectious periods (Fig. S3, fast disease spread).

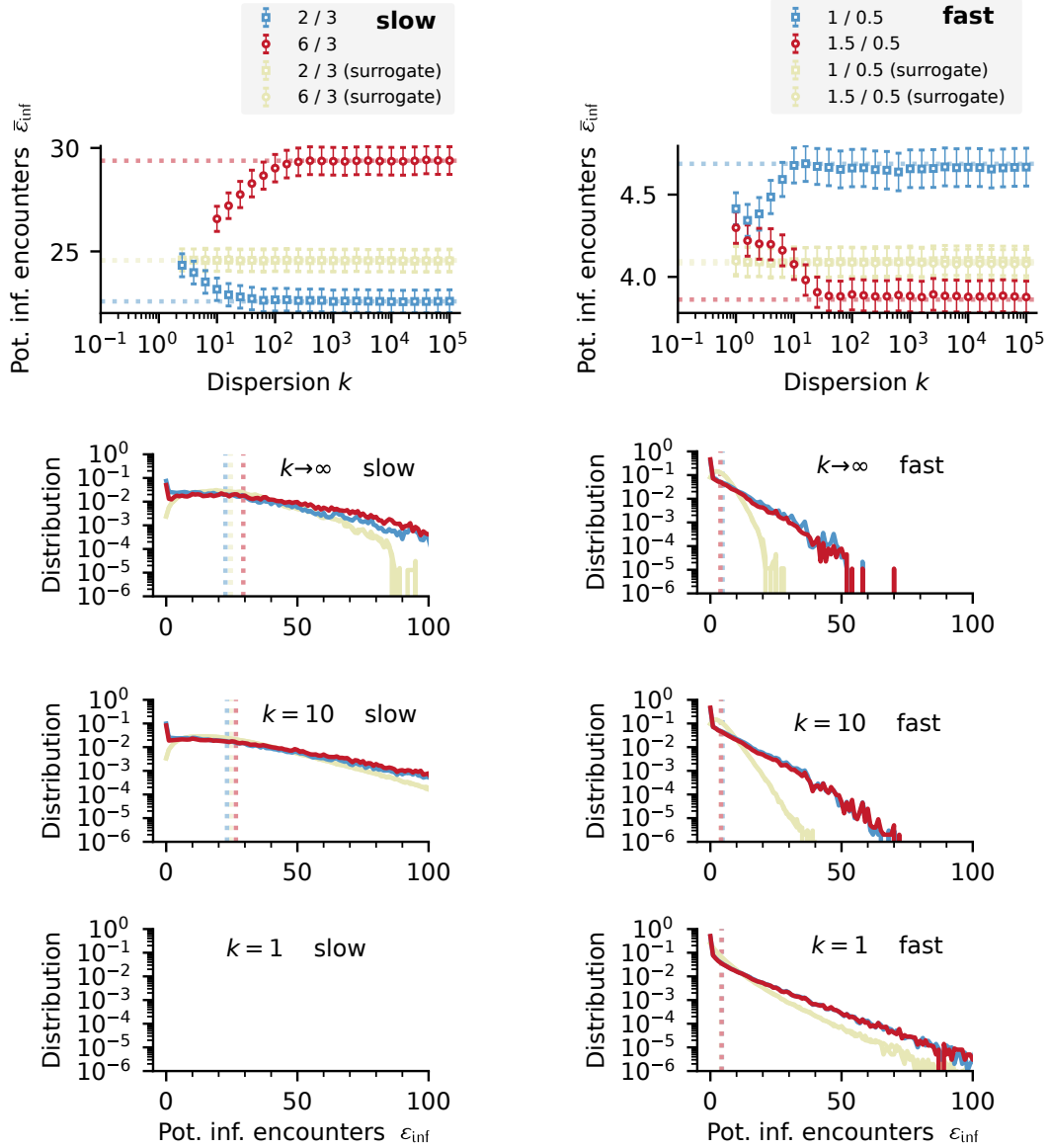


Figure S3. **Example with smaller latent and infectious periods provides additional insight on low-dispersion regime of non-deterministic disease progression.** Here, we compare the example from the main manuscript (*slow*, $T_{\text{inf}} = 3$ days and T_{lat} either 2 or 6 days) with a hypothetical *fast* disease progression ($T_{\text{inf}} = 0.5$ days and T_{lat} either 1 or 1.5 days) and show both the mean number of potentially infectious encounters as a function of the dispersion parameter k of the gamma-distributed latent and infectious period (top) as well as their distributions for selected k . Due to the finite duration of the recording, the accessible low-dispersion regime is determined by the mean latent and infectious period, because for low dispersion large periods quickly exceed the finite duration. For faster disease progression (smaller latent and infectious period), we observe resonances on smaller timescales (cf. Fig. 2 main manuscript) and in addition reach the low-dispersion regime of exponentially distributed periods ($k = 1$) commonly assumed in epidemiological simulations. As one can see more clearly for faster disease progression, the mean number of potentially infectious encounters approach each other in this low-dispersion regime, which can only be anticipated from the results for slower disease progression. This implies that resonance effects will not be present for exponentially distributed latent and infectious periods but only for more realistic non-exponentially distributed ones with higher dispersion. In addition, one can see that estimates of mean potentially infectious encounters for randomized surrogate data in both cases of slow and fast disease progression are independent of dispersion, such that randomized encounter trains destroy resonance effects.

Infections from outside the study group

To check the effect of contacts that could take place with people who were not part of the study, we investigate disease onsets that do not directly follow the contact patterns observed in the data (Fig. S4). In our analysis of the main manuscript, an infection could only originate from an encounter with another participant in the data set. Here, we keep the original encounter trains (to evaluate potentially infectious encounters) but the disease onset can occur at any time, due to a hypothetical contact with an external person. We focus on the resulting distribution and the mean of potentially infectious encounters ε_{inf} (Fig. S4).

We distinguish the following different possibilities of disease onset:

- internal:** Onsets occur as in the main manuscript only at encounters recorded in the data set. This naturally incorporates the spatio-temporal structure of encounters, in particular their temporal inhomogeneity (Fig. 1c, main manuscript) and their variability across participants (Fig. 2a, main manuscript).
- external i):** Onsets occur completely random, at random times for random participants. This neglects both temporal inhomogeneity of encounters and their variability across individuals.
- external ii):** Onsets occur at random times with probability proportional to the encounter rate (Fig. 1c, main manuscript) for random participants with probability proportional their total number of encounters (Fig. 2a, main manuscript). This incorporates both the (averaged) temporal inhomogeneity of encounters and the variability across individuals.
- external iii):** Onsets occur at random times with probability proportional to the encounter rate (Fig. 1c, main manuscript) but for uniformly random participants. This incorporates the (averaged) temporal inhomogeneity of encounters but neglects the variability across individuals.
- external iv):** Onsets occur at uniformly random times for random participants with probability proportional their total number of encounters (Fig. 2a, main manuscript). This neglects the temporal inhomogeneity of encounters but incorporates the variability across individuals.

Once an onset has been chosen, the disease progression is modeled as in the main manuscript. We stick to the examples of gamma-distributed latent and infectious periods with $k = 10$ to evaluate potentially infectious encounters. In particular, we consider the prime example from the main manuscript with $T_{\text{inf}} = 3$ days and $T_{\text{lat}} = 2/6$ days where resonances between disease progression and contact patterns causes clear differences in the number of potentially infectious encounters for internal disease onsets (cf. Fig. 4 in main manuscript).

Comparing the different version of disease onset (Fig. S4), we can attribute clear effects to both the temporal inhomogeneity of the onset time as well as the variability of onset times across individuals. Please note that in all cases the encounter statistics of the actual encounter trains did not change — all we change is the statistics of the disease onset time. Please note further that the results for different version of disease onset represent the extreme scenario where all disease onsets originate from external sources.

Comparing the distributions of ε_{inf} for fixed T_{lat} (Fig. S4a and Fig. S4c each), we notice that those distributions that best resemble the shape of internal disease onset are those where external disease onset statistics incorporate the variability across individuals (external ii and iv). This can be explained by the fact that also for internal disease onset more onsets occur for contact trains with more encounters, which in turn increases the probability of higher ε_{inf} and thereby also the mean ε_{inf} . It appears that for the overall shape of the distribution, as well as the leading order of its mean value, it is not necessary that disease onsets occur with the same temporal inhomogeneity as true encounters for the chosen infectious periods (this may change for very small infectious periods though).

Comparing further the results of specific disease onsets for different T_{lat} (Fig. S4b comparing solid vs opaque symbols), we notice that incorporating the temporal inhomogeneity into the disease onset (external disease ii and iii) seems relevant for resonance effects that cause differences in the mean of ε_{inf} for the selected latent periods. This can be explained by the observation reported in the main manuscript that reoccurring contact patterns are the main cause for resonance effects. The results for the extreme scenarios of external infections thus fully support our conclusions from the main manuscript.

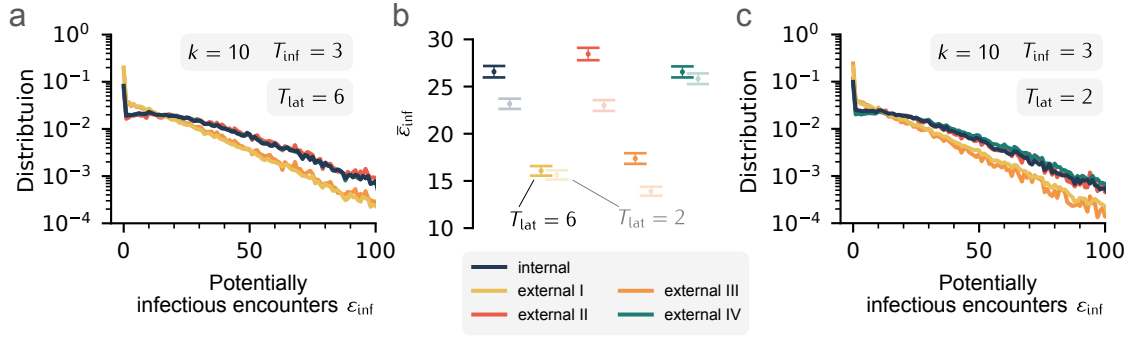


Figure S4. **Infectious encounters for external infections** In our analysis in the main manuscript, we preserved the temporal features by constraining disease onsets to available encounters. **a:** Distribution of potentially infectious encounters for non-deterministic disease progression ($k = 10$). 6 days latent period. **b:** Comparison of the mean $\bar{\varepsilon}_{\text{inf}}$ between 6 days latent period (dark) and 2 days latent period (light). **c:** Same as a), but 2 days latent period.