An interaction-based contagion model over temporal networks demonstrates that reducing temporal network density reduces total infection rate

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Abstract

Contacts’ temporal ordering and dynamics, such as their order and timing, are crucial for understanding the transmission of infectious diseases. Using path-preserving temporal networks, we evaluate the effect of spatial pods (social distancing pods) and temporal pods (meetings’ rate reduction) on the spread of the disease. We use our interaction-driven contagion model, instantiated for COVID-19, over history-maintaining random temporal networks as well as over real-world contacts. We find that temporal pods significantly reduce the overall number of infected individuals and slow the spread of the disease. This result is robust under changing initial conditions, such as initial patients’ numbers and locations. Social distancing (spatial) pods perform well only at the initial phase of the disease, i.e., with a minimal number of initial patients. Using real-life contact information and extending our interaction-driven model to consider the exposures, we demonstrate the beneficial effect of reducing the temporal density on overall infection rates. We further show that slow-spreading
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pathogens spread almost as fast-spreading pathogens in dense topologies. Our results show that given the same transmission level, there is a decrease in the disease’s rate and spread in less dense networks. Thus, reducing the rate of encounters is more effective than social distancing.

**Keywords:** COVID-19, Spatial pods, Temporal pods, Temporal density, Infection rate

1 Introduction

The SARS-CoV-2 coronavirus disease 2019 (COVID-19) has created a global crisis. In response, governments and local authorities employed Non-Pharmaceutical Interventions (NPIs) [1] to limit mobility, social interactions, and gathering to slow - or even contain - the spread of the virus [2, 3].

One social distancing solution is the creation of spatial distancing pods. In Israel, for example, to avoid school lockdowns [4], social distancing pods were employed in schools [5]. Each class was split into two disjoint sub-groups, each considered a separate pod. Due to a lack of staff and infrastructure, each class’s pods arrived at school on alternate days, thus creating both a temporal division and a spatial one.

Here, we set to quantify the different effects of temporal and spatial divisions on the spread of infection in a community.

Figure 1 demonstrates the distinction between spatial and temporal pods for the case of two pods. The original temporal network is presented on the upper panel. Spatial pods preserve the same number of interactions by keeping the same edges per time window but use a different network topology. Temporal pods are created by spreading the edges over more extended periods, reducing temporal density yet preserving the original network dynamics, i.e., all interactions between all nodes.

Intuitively, separating people into smaller groups, as is the case with spatial pods, reduces the probability of infection as the number of different people exposed to decreases. The case of temporal pods is different. Temporal pods require limiting the number of meetings per day. Scheduling all needed meetings might entail prolonging the number of days people interact (for example, lengthening a conference to allow all planned meetings to occur). On its face value, one might conjecture that increasing the number of days people meet might increase the chance of getting infected. Thus, it is not clear which would be the preferred strategy to decrease the spread of the disease.

We use an interaction-driven model over both temporal random networks and real-world interactions to research this question. The interaction-driven temporal contagion model allows for nodal states such as Susceptible, Exposed, Infected, and Recovered while considering the probability of infection at the end of each time window, thus enabling a latent Exposed state during that time window.
Fig. 1: The two mechanisms for reducing daily interactions. The original temporal network (upper panel) is split into two spatial pods (middle panel) and two temporal pods (lower panel). Spatial pods preserve the same number of edges per time window, but the network is partitioned into two disjoint groups that are active during all days. Temporal pods, depicted in the lower panel, are created by spreading the edges over longer periods, reducing temporal density, yet preserving the original full network dynamics and topological structure. The darker nodes are used to mark the nodes participating in the temporal pod during that time window.

To generate temporal random networks with temporal ordering, we implemented the algorithm suggested by Zhang et al. in [6] that generates a series of temporal random networks with continuous-time network histories. We demonstrate that when researching temporal random networks, it is crucial to consider the density at each time window, termed temporal density, and set it to a determined, stationary value (see Figure 2).

Our results demonstrate that temporal pods produce a more robust reduction in the spread of the disease compared with spatial pods. Spatial pods are ineffective, especially when the number of initial patients increases. In this case, due to the prevalence of infectious people in the population, each spatial pod may contain at least one of them. In the case of temporal pods, big changes in the initial parameters (number and location of patient zero) do not translate to big changes in the results, implying that temporal pods effectively reduce the spread of the disease regardless of initial conditions.

To better understand the effect of temporal pods in a real-life community, we use real-world encounters data from the Copenhagen Networks Study.
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(CNS) dataset [7, 8]. The advantages of working with real-life temporal interactions are numerous, as human interactions are bursty, temporal, highly contextual and networked [9–13].

The CNS dataset contains, on top of the contact information, the duration of the encounters. The spreading of contagious respiratory diseases, such as severe acute respiratory syndrome (SARS) and COVID-19, is an exposure-based process that correlates with the duration of the exposure [14–18]. Hence, we expand our interaction-based model to include the duration of interactions and consider this duration as correlated with the transmitted viral load. This enables us to create a probabilistic model that does not consider interactions of different lengths equally and allows us to follow the spread of the disease on a real contact network while considering the duration of encounters. Our results show that reducing the daily temporal density reduces the spread of the disease, although spatial order was maintained by lengthening the number of days during which people meet.

We continue to examine the effect of density on virality by considering pathogens with different minimal exposure duration needed for contagion [14, 19–21]. We find that reducing the temporal density lowers the ability of pathogens to infect, as infected people have fewer opportunities to infect before they are removed. However, in the original dense network, slow-spreading pathogens, that require ten times the exposure of the fastest pathogen, are almost as contagious.

Our results demonstrate that reducing the daily temporal density of encounters significantly affects the spread of the disease.

2 Methods

2.1 Temporal network density

Given a network of interactions, its density is defined as the ratio between the number of existing edges and possible edges in the network (i.e., the probability of a random sampling of an edge).

$$\text{Density}(G) = \frac{\text{actual edges}}{\text{possible edges}} = \frac{m}{n \cdot (n-1)/2}$$ (1)

Where $m = |E|$ is the number of edges and $n = |V|$ number of nodes in the graph $G(V, E)$ representing the network.

An impediment to modeling contagious disease over temporal random networks is the fallacy of the average, as described in Figure 2. In the example, both temporal networks have the same average density. However, in the one in Figure 2a the majority of the population is infected, while in the scenario depicted in Figure 2b the infection may die out before infecting the population. One may consider both Figure 2a and its opposite case, in which there are no contacts when people are infectious, and contacts occur when recovered. Both cases have the same average density but very different outcomes. To
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Fig. 2: The falsehood of daily average density. (a) Unbounded variance allows for significantly different number of daily interactions, resulting in significantly different daily (temporal) density. (b) The variance in the number of daily interactions is bounded, hence the average density is indicative of the daily (temporal) density.

accurately assess the progress of the disease, there is a need to nullify changes in the temporal density that may decrease or increase contagion and create temporal networks with stationary densities, as depicted in Figure 2b.

2.2 Temporal random networks modeling

We first examine the effect of spatial and temporal pods on temporal random networks. Zhang et al. [6] suggest a temporal random network with changing dynamics that follow a Markov process, allowing for a continuous-time network history (see Section A.1 for details). Using this model allows for a fundamental unit of analysis that is the entire network history while keeping a stationary density. Further, we chose the parameters s.t. to allow for stationary density (see Section A.1 for details).
To ensure that no anomalies in one graph created an unusual effect, we used a mix of randomly generated graphs. We then conducted a Two-sample Kolmogorov–Smirnov test on each pair of graphs and determined that for each couple, we cannot reject the null hypothesis that both were drawn from the same probability distribution, with 95% significance [22].

2.2.1 Reducing spatial and temporal density in random networks

We were interested in measuring the effect on spreading processes when reducing generated random graphs’ temporal and spatial density. Each generated graph consists of 10000 time windows, denoted by \( \tau \). Initially, each time window consists of 288 \times 5-minute intervals, as in a day. We reduced temporal and spatial densities as follows.

**Spatial** density reduction was achieved by simulating graphs consisting of completely disconnected sub-graphs, with the randomly chosen initial patients (patients zero) distributed between the sub-graphs at random. To create each spatial reduction, the sub-graphs were randomly chosen from a pool of graphs of the respective size and density (500 nodes each for a spatial reduction to two sub-graphs, for example).

**Temporal** density was reduced by splitting each time window \( i, i \in [1..10000] \) that consists of \( \tau_i = 288 \times 5 \)-minute intervals by 2, 3, 4 and so on. For instance, when divided by two, time window \( i_1 \) would now consist of \( \tau_{i_1} = 144 \times 5 \)-minute intervals, the second half turning into the consecutive time window \( i_2 \), creating a dataset twice as long while preserving the original network’s dynamics such as the order of the interactions and its topology.

Spatial pods preserve the graph’s temporal density by keeping the same amount of edges per time window but using different network topologies. Temporal pods spread the edges over longer periods. This reduces temporal density but preserves the original network dynamics. For example, to create \( k = 2 \) spatial pods, that is, two disconnected subgraphs, we generated two random networks, each is half the size of the original graph in terms of nodes and edges for each day. However, for the temporal pods, we maintained the original temporal interactions and their order but split each day \( \tau \) into \( k = 2 \) consecutive pseudo-days, namely \( \tau_1, \tau_2 \), each containing the corresponding half of the original day and hence half the temporal density.

2.3 A temporal interaction-driven contagion model

At each time window \( n \) of duration \( \tau \) with \( K_n \) interacting nodes, the probability of a node \( i, i \in K_n \) to become exposed is calculated as the complement of the probability of not being exposed in any of the encounters during that time
window with infectious nodes, as follows:

\[ P_i(S \rightarrow E) = 1 - \prod_{i,I} (1 - P_{max}) \] (2)

Where \( k_{i,I}^n \) is the subset of infected nodes in time window \( n \) that interacted with node \( i \) during that time window and thus are probable to expose it to the infection, and \( P_{max} \) is the probability of being infected in maximum exposure.

2.4 Real-life encounters: considering encounters duration heterogeneity

We model the CNS social network of interactions (see Section A.2) \( \Gamma \) as a sequence of \( T \) consecutive undirected weighted temporal graphs \( \{G_\tau \in \Gamma, \tau \in T\} \) where each temporal snapshot graph \( G_\tau = (V_\tau, E_\tau) \) denotes the subset of interacting nodes \( V_\tau \) during the \( \tau \) temporal window and the weighted edges \( E_\tau \) the interactions during this time. Each edge is a distinct interaction. Edge weight corresponds to the duration of the interaction (measured in seconds or minutes).

2.4.1 Temporal interaction-driven contagion model with various exposure levels

We model the probability of infection at each interaction with an infected individual as a Sigmoid function of the length of the interaction, where after crossing a lower threshold of minimal time for infection, the probability increases exponentially with the duration up to a maximal duration after which the probability stays stable. This is inline with epidemic understanding of a SARS-like airborne disease [14, 19].

In practice, at each encounter during a time window \( \tau \), there is a probability for a node \( i \) to get exposed and infected that is calculated as follows. Let \( d_{i,k} \) be a non-zero value for the strength of edges that enter the focal node \( i \) from infected node \( k \), where \( k \in K \), the set of infected nodes that \( i \) encounters.

\[ \forall k \in K, d_{i,k} = \begin{cases} d_{i,k} & d_{i,k} \geq D_{min} \\ P_e & d_{i,k} < D_{min} \end{cases} \] (3)

Where \( D_{min} \) is the minimum exposure required for infection, i.e., the minimal interaction duration needed for infection. If the interaction is shorter than \( D_{min} \), we set the edge’s strength to a minimal probability that will not zero the equation but reduce the probability of being infected due to this encounter.

Each time-window, \( \tau \), the probability of node \( i \) to get infected as the complement of the probability of not being infected in any of its encounters with infectious nodes in that time window:

\[ P_i(S \rightarrow E, \tau) = 1 - \prod_k^K (1 - \min\left(\frac{d_{i,k}}{D_{max}}, 1\right) \cdot P_{max}) \] (4)
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Where $P_i(S \to E, \tau)$ is the probability of node $i$ in state $Susceptible$ to transition from state $Susceptible$ to state $Exposed$; $d_{i,k}$ is the duration of the $k_{th}$ interaction of node $i$; $P_{\text{max}}$ is the probability of being infected given a maximal [i.e., continuous] exposure to the infecting agent; and $D_{\text{max}}$ is a normalization factor that denotes the [minimal] duration of the exposure for which the probability of infection is maximal. $P_{\text{max}}$ represents, for example, the fact that some of the population might already be protected for various reasons against the infecting agent, and thus, typically, $P_{\text{max}} < 1$.

The literature further shows that COVID-19 variants, such as Alpha and Delta, are associated with different exposure levels of transmitted viral load [20, 21]. Thus, the virality of such pathogens is correlated in our modeling with the minimum exposure latency, $D_{\text{min}}$. We use it to determine the effect of density on virality by measuring the virality of variants that correspond to various $D_{\text{min}}$ values.

3 Results

3.1 Random networks with changing spatial and temporal pods

In this experiment, the density of randomly generated graphs is reduced. There are 10,000 time windows of 1000 nodes each. A time window duration is denoted by $\tau$. Initially, each time window consists of $288 \times 5$-minute intervals, as in a day. We reduced temporal and spatial densities as described in Section 2.2.1. The experiments were performed with two randomly selected initial patients and ten randomly selected initial patients.

Figure 3 depicts the results of the experiments for spatial and temporal density reduction by showing the percentage of the population infected with the interaction-driven model in each scenario. Figures 3a, 3c correspond to reduced spatial density obtained by separation to disjoint groups, with two and ten initial number of randomly selected patients zero, respectively. Figures 3b, 3d correspond to reduced temporal density, with two and ten initial number of randomly selected patients zero, respectively.

In Figures 3a and 3b we see that when $k = 2$ with two patients zero yields, on average, less infection in the case of the spatial pods than in the temporal ones. In the case of spatial pods, it is probably correlated with the random placement of patients zero. If both are in one of the spatial pods, the other pod stays safe. If the initial patients are randomly placed one at each spatial pod, then the probability of the majority of the population being infected is high. These considerations explain well the large variance for $k = 2$ in Figure 3a. As the number of spatial pods increases, the chance of having an infectious member in one of the nodes decreases, and the solution of spatial pods works well. Temporal pods seem to be at least as good if not better for $k \geq 3$ temporal pods. Unlike spatial pods, the number of meetings is limited, but there is no separation between groups. Halving the temporal density and hence the number of daily meetings has little effect, as the initial network is quite dense.
Fig. 3: Percentage of population infected with an interaction-driven model over temporal random networks with spatial and temporal pods. Left panels correspond to reduced spatial density obtained by separation to disjoint groups (x-axis denotes the number of groups). Right panels correspond to the relative reduction in the temporal density (x-axis denotes the factor by which the spatial or temporal density was reduced). Upper panel depicts infection with two randomly selected patients zero, lower panel depicts infection with ten randomly selected patients zero. Each boxplot depicts the results of 200 corresponding simulator iterations over the 10000 temporal networks. Thus each graph is the result of 2000 iterations.
Temporal graph density

Fig. 4: Daily temporal density histogram of the CNS social networks. X-axis counts the number of days with the same temporal density, y-axis denotes the temporal density percentage: (a) Original temporal network; (b) Reduced density - CNS with half the original temporal density and twice days; (c) Reduced density - CNS with one-third the original temporal density and therefore three times the number of days.

However, reducing the density to one-third of the original one, i.e., each day now "spreads" over three days with respect to the number of daily meetings, results in a clear reduction in the average number of infections. Only 53% of the population is infected, with a variance that is limited to ±10%. When the daily density has further reduced, the disease is under control, with less than 25% of the population infected, in the vast majority of the iterations.

Temporal pods produce a more robust reduction in the spread of the disease compared with spatial pods. The results are more defined when the number of initial patients increases. Due to their large number, each spatial pod has a considerable chance of containing at least one of the patients zero in the system. Thus, on average, the majority of the population is infected in our experiment (over 60%) regardless of the number of spatial pods (i.e., $k \in [2..10]$). However, temporal pods give a robust result of reducing the spreading of the disease regardless of the number of patients zero. Big changes in initial parameters (number and location of the initial patients) do not translate to big changes in the result for temporal pods, as evident when comparing Figures 3c and 3d.

Lowering meeting rates reduces the spread of the disease in the population. The stretching over several days of the meetings designed to occur on a single day introduces a trade-off: a meeting that was supposed to occur with someone who was, on the meeting’s intended day, not infected, may, after the delay, be a meeting with an infectious person. Alternatively, a delayed meeting may enable the person to recover or isolate at home. Our results indicate that this trade-off results, on average, in reducing the spread of the disease.

3.2 Real-world networks with changing temporal density

Moving from a random network to real-world encounters, we measure the effect of changing the temporal density on the spread of the disease over complex, real-world network. The CNS temporal network, devised from proximity
data, is amongst the detailed available contact data collected during the pre-pandemic era (see Section 2.4 and Section A.2). Its daily temporal density is depicted in Figure 4a. Over two-thirds (∼ 64%) of the days have a temporal density of 0.2 or lower. That is, the overall number of interactions is at most 20% of the possible number of interactions. A quarter of the days have a temporal density of 25%, and the remaining 11% days are very dense, up to half the maximal possible density in which everybody meets everybody.

In this experiment, we reduce temporal density by splitting each day to \( k = [2, 3, \ldots, 10] \) pseudo days with \( 1/k \) the interactions of the original day. Thus, for example, a network with half the density will be depicted as twice as long, pseudo-days wise. Figures 4b and 4c show the temporal density distribution during these pseudo-days.

We examine our temporal interaction-driven contagion model, as described in section 2.4.1 over the network, with a randomly chosen initial contagious patient zero. Each point results from 200 iterations with a randomly chosen initial patient zero. We regard each pseudo-day as a regular day, with a lower temporal density. In each of the encounters with infected individuals for which the minimal exposure time for infection is met or exceeded, the probability for infection also depends on \( P_{\text{max}} \), as described in Equation 4. To enable a visual comparison, we show the same number of days, or pseudo-days, in the results depicted in Figure 5. When the interaction-driven contagion model is run over the original network, seen in figure 5a, it takes less than 12 days to infect 80% of the population, and by the end of the 29 days timeline, over 90% of the population was infected. However, when the temporal density was reduced, as in Figures 5b and 5c, the spread is slower, and the overall number of infected individuals is lower, to 80% for the network with half the initial density and to 50% for the network with one third the original density.

### 3.2.1 The effect of density on virality

We continue to examine the effect of density on virality. We correlate the speed of the variant with the minimal amount of time needed for infection, \( D_{\text{min}} \). Figure 6 depicts the distribution of daily meetings length in the CNS dataset averaged over all days. The vast majority of the meetings are short, with a few meetings lasting longer than 200 minutes.

We vary the minimal duration that correlates for infection, \( D_{\text{min}} \) in the range \([5, 120]\) by five minutes at each iteration. \( D_{\text{min}} = 5 \) indicates a variant that is transmissible even in all meetings, while \( D_{\text{min}} = 120 \) corresponds to a variant that is transmissible only if exposure is very long. In each of the encounters with infected individuals for which the minimal exposure time for infection is met or exceeded, the probability for infection also depends on \( P_{\text{max}} \), as described in Equation 4.

Figure 7 depicts the virality of pathogens requiring different minimal exposure values for contagion, corresponding to various \( D_{\text{min}} \) values over, the CNS dataset. For each \( D_{\text{min}} \) value we performed 200 iterations with a random placement of patient zero. The total infection rate is presented as a box-plot form...
of the distribution of the results measured over the 200 iterations for each of the \( D_{\text{min}} \) values. We then experimented on the CNS dataset with half the daily density and third the daily density (Figure 4 above details the distribution of daily temporal densities for each of the networks). Figure 7a shows the results for the different pathogens on the original network. Faster pathogens that need less exposure time for transmission infect most of the population. Slower pathogens that require higher exposure time to infect are less successful. However, as can be seen in Figures 7b and 7c, reducing the temporal density lowers the ability of pathogens to infect, as infected people have fewer opportunities to infect before they are removed. When the temporal density of the network is halved, fast pathogens can infect the majority of the population. However, we see that there were hardly any infected in some of the iterations, which means that patient zero had only a few encounters in which the disease was not transmitted. In cases where the minimal exposure time required for infection is large, we see that the reduction in daily temporal density has a high effect. For example, to infect at least 50% of the population in the network with half the daily density, a pathogen needs to infect in 0.7 the exposure duration of a pathogen in the original network. Moreover, in the original dense network, slow-spreading pathogens that require ten times the exposure of the fastest pathogen are almost as contagious.

4 Discussion

Temporal networks enable the research of disease progression while following possible routes the disease follows. This research departs from an aggregated view into a temporal path-respecting one and follows the spread of the disease over the timeline of interactions. We show that when researching temporal networks, the daily temporal density can change significantly, and hence the average density of a network can conceal significant variance between the days. Hence, temporal density should be considered. We then generate temporal random networks with stationary temporal density to compare the spread of the disease under various policies.

Spatial, social distancing pods, were beneficial for a low number of initial patients, which may correspond to the low transmissible epidemic or the early days of a more contagion one. Thus, applying social distancing pods is probably beneficial only at the early stages of a pandemic or for slowly transmitted diseases. In our simulations, the spatial pods are separated. In real life, however, such separation is often not feasible over time, which will further reduce the effectiveness of this solution. Spatial pods, however, can be used as a model for nonrandom mixing in populations. For example, in cases where similar people group together. Reports of nonrandom mixing in disease spreading exist for the measles outbreak in Chicago in the 1980s, and the Swine flu in Fort Dix in 1976 [23]. In these cases, the nonrandom mixing between the groups resulted in only one of the groups getting the disease. Nonrandom mixing patterns and their effect on spatial and temporal pods are part of our future work plan.
Our interaction-driven contagion model enables the consideration of meetings’ duration. We model the duration as a proxy for the transmitted viral load [14, 15]. We demonstrate the effect of reduction in temporal density on the spread of the disease over real-world contextual data. We show that reducing the daily temporal density (while preserving all encounters and their order) reduces the spread of the disease. Our modeling enables us to consider pathogens that differ in their transmissibility. We further show that slow-spreading pathogens spread almost as fast-spreading pathogens in dense topologies.

In future research, we plan to investigate the effect of different temporal density trends on the progressions of the disease while considering different modular structures.

5 Conclusion

Social distancing pods were the go-to solution in many countries as a way to reduce the spread of the disease. Our results, exploiting an interaction-based contagion model, indicate that this solution is effective only at the first stages of a disease or low-transmissible disease.

Our key result shows that reducing the frequency of daily encounters, which we refer to as temporal density, is very effective for high virality situations, and a drastic reduction in the daily number of interactions in a community can be highly effective in controlling the spread of the disease.

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Appendix A Supplementary information

A.1 Temporal random networks

We examine the effect of spatial and temporal pods on temporal random networks. Zhang et al. [6] suggest a temporal random network with changing dynamics that follow a Markov process, allowing for a continuous-time network histories moving from a static definition of a random graph with a fixed number of nodes \( n \) and edge probability \( p \) to a temporal one, \( G_{n,p} \rightarrow G_{n,p,t} \) such that \( E(G_{n,p,t}) \sim G_{n,p} \). Defining \( \lambda = \) probability per time granule of a new edge to appear and \( \mu = \) probability per time granule of an existing edge to disappear Zhang et al. [6] show that the equilibrium probability of an edge is

\[
p = \frac{\lambda}{\lambda + \mu}
\]  

(A1)

Our implementation, a Python package that we refer to as RandomDynamicGraph (RDG), (https://github.com/ScanLab-ossi/DynamicRandomGraphs), generates large-scale dynamic random graphs according to the defined density. The package focuses on massive data generation; it uses efficient math calculations, writes to file instead of in-memory when datasets are too large, and supports multi-processing.

We chose \( \lambda, \mu \) in a manner that allows for a stationary density for all \( G_{n,p,\tau} \), where \( \tau \in [1..10000] \) and \( n = 1000 \). Figure A1 depicts the densities of the resulted 10000 consecutive random graphs created with the RandomDynamicGraph package.

Figure A1 clearly shows stationary temporal density in one sample generated random network.

A.2 Mining the CNS data

The CNS social network of interactions was used in the following manner. To consider close encounters, the data was mined taking into account that the
probability for infection is inversely correlated with distance, and decreases dramatically as the distance grows [24]. The proximity information was registered as the Received Signal Strength (RSSI) function. However, it is hard to infer exact distance information from the RSSI [25–27]. Moreover, the distance itself is not a single component in calculating the chance of infection - the directions of standing, ventilation, and the environment are also significant parameters [28–30]. To simplify the model we do not consider all the above. As stronger signal correlates roughly with high proximity, we consider interactions for the RSSI $\geq -90$.

A.3 Interaction-driven temporal SEIR-like model

The SEIR-like model, depicted in Figure A2 is as follows. All nodes are initially in the Susceptible ($S$) state, when a random small set of nodes in the initial time window become infectious (i.e., patients zero). Susceptible nodes (in state $S$) that become exposed according to Equation 2 enter state Exposed ($E$) at the end of the time window $\tau$ during which they were exposed to infectious nodes. Nodes in state Exposed become Infected after a delay that corresponds to $d_{E\rightarrow I}$, where $d_{E\rightarrow I} \sim \tau \cdot N(1,1)$. Infected nodes (in state $I$) stay infectious for $d_{I\rightarrow R}$, where $d_{I\rightarrow R} \sim \tau \cdot N(10,1)$. After this delay they enter state Recovered ($R$) and are no longer infectious.
Fig. 5: Disease spread in identical time frames while reducing temporal density on the real-world CNS network: (a) Original temporal network; (b) Reduced density - network with half the original temporal density; (c) Reduced density - network with one-third the original temporal density.
Fig. 6: Distribution of daily meetings length in the CNS dataset averaged over all days
Fig. 7: The effect of density on virality for various minimal exposure times needed for contagion (different $D_{\text{min}}$ values). (a) Original temporal network; (b) Reduced density - network with half the original temporal density; (c) Reduced density - network with one-third of the original temporal density.
Fig. A1: A series of 10000 snapshots in time of a temporal random network generated with our RDG python package with stationary density

\[ 1 - P_i \]

\[ P_i \text{ (Eq. 2)} \]

\[ d \sim \tau \cdot N(1, 1) \]

\[ d \sim \tau \cdot N(10, 1) \]

Fig. A2: The state machine for each node in our interaction-driven contagion model for random temporal networks with no edge weights.