A Non-Markovian Model to Assess Contact Tracing for the Containment of COVID-19

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Abstract—Non-pharmaceutical interventions, such as contact tracing has been an important tool in controlling epidemic outbreaks. In this paper, we propose a non-Markovian, network-based mathematical model to assess the effectiveness of contact tracing in COVID-19 containment. To improve the reliability of the model, empirically determined distributions were incorporated for the sojourn times of the model’s states. The first-order closure approximation was used to derive an expression for the epidemic threshold. Using survey contact data collected during the 2020 fall academic semester from a university population, we determined that even four to five close contacts were sufficient to maintain the viral transmission. Additionally, our model reveals that contact tracing can be an effective outbreak mitigation measure by reducing the epidemic size by more than three-fold. Furthermore, we show that our proposed model, which accounts for the underlying complexities of the COVID-19 spreading process, performs better in short-term forecasts of case counts.

Index Terms—COVID-19, non-Markovian Models, contact Tracing, Epidemic Threshold, Weighted Contact Networks.

I. INTRODUCTION

An important aspect of COVID-19 spreading that needs to be incorporated in its mathematical modeling is non-exponential distributions of the infectious and incubation periods [1, 2]. Simulations show that different distributions of the infectious period with the same mean values lead to different epidemic curves. In addition to the states that model the disease progress and transmission among individuals, a realistic model for COVID-19 spreading should account for different management measures such as contact tracing and quarantining. This can be accomplished by introducing additional states with non-exponential sojourn times into the model. Although the traditional spreading compartment models assume exponential distributions for the transition times, there are studies that consider non-Markovian transitions in the COVID-19 spreading models [3, 4, 5, 6, 7, 8, 9, 10, 11]. Specifically in [11], the authors consider SEIR compartment model allowing non-exponential distributions for the exposed and infectious periods and derive theoretical results linking the (unobserved) parameters of the model to quantities that can be measured during the early stages of an epidemic. Also in recent years some studies have been published that analyze non-Markovian spreading over networks [12, 13, 14, 15, 16, 17, 18, 19, 20]. In [20] a Gillespie based algorithm is presented for simulating non-Markovian models. When the objective is to derive analytical results concerning non-Markovian models, both first-order [18] and second order [19] closure techniques can be used. One possible approach to study networked non-Markovian spreading is based on phase-type distributions [21]. A phase-type distribution can be used to approximate any distribution by a mixture of exponential distributions. In this approach, if a transition period between two states in a spreading model is non-exponential, it is approximated using a mixture of Markovian transitions. For instance, if a sojourn time for a state is deterministic, in other words a constant \( \tau \), we model the transition by a series of successive Markovian transitions between a large number of auxiliary states each with a mean sojourn time of \( \tau / n \), where \( n \) is the number of auxiliary states. By applying phase-type distribution to networked spreading, authors in [17] analyze the SIS spreading model with non-exponential infection and recovery times. They show that the disease-free state is globally stable if the corresponding linearized system of equations for the SIS spreading is stable.

The goal of this paper is to develop a networked non-Markovian model that is appropriate for assessing contact tracing and also to estimate the effectiveness of contact tracing using real-world data. Contact tracing is a primary public health response to infectious disease outbreaks [22, 23]. The adoption of contact tracing to contain COVID-19 met considerable challenges due to the intensive process related to manual contact tracing and the hesitancy to adopt app-based contact tracing tools [24]. Assessing the impact of contact tracing as a control measure remains of great importance. Models for assessing contact tracing have been developed for many infectious diseases before the COVID-19 pandemic. A systematic review of these models can be found in [25]. In [26], the authors derived contact patterns in the U.K. and, by simulating a model of contact tracing, investigated the efficacy of contact tracing for COVID-19. They found that controlling the spread requires tracing casual contacts in addition to close contacts, which can overwhelm the

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system. In [27], the authors also used simulations to estimate that a high proportion of cases would need to self-isolate and a high proportion of their contacts successfully be traced to contain the epidemic. Another aspect of COVID-19 that can alter the effectiveness of contact tracing is the high transmissibility of SARS-CoV-2 before and immediately after symptom onset. The prospective study reported in [28] concluded that finding and isolating symptomatic patients alone may not suffice to interrupt transmission, and more generalized measures might be required, such as social distancing. In [29], the authors use simulations to study the effectiveness of contact tracing for COVID-19 and find that the number of quarantined susceptible people increases with the increase of tracing because each confirmed case increases the number of traced contacts. However, there is an inflection point after which the number of traced contacts decrease with increased tracing because there are fewer confirmed cases.

In this paper a new networked compartmental model with compartments able to represent the disease evolution and the contact tracing process is presented in Section II. The model allows arbitrary transition time distributions so we can study effect of the states with localized sojourn time. The novel model we have proposed uniquely account for important aspects of the real-world COVID-19 transmission, including contact network, contact tracing impact and non-exponential transition times, which have not been considered together in existing studies that we are aware of. Our simulations in the paper (results shown in Figs. 5, 8(d), and 10(b)) demonstrate neglecting such aspects of the spreading process in the mathematical models can produce model outputs that deviate from observed epidemic curves. While our proposed model is more complex and requires knowledge of the contact network, we have demonstrated in Section IV-E that it performs better in forecasting short-term case counts compared to other models commonly used to study COVID-19 spreading. This is because our model provides a more precise mathematical representation of the underlying physical spreading process. Assuming non-Markovian transitions modeled using the Erlang distribution, mean field equations for such a non-Markovian model are derived, and a mathematical analysis was performed to find the epidemic threshold in Section III. The model was applied to the Kansas State University case study reported in Section IV. To estimate the effectiveness of contact tracing we built a three layer contact network by surveying the students activity and inferring the network by contact tracing and are induced by contacts with individuals in the C or D states. If a transition is not induced by any interaction, we call it nodal transition and it considers the delay between infection and quarantine (e.g., random and close contacts) and may vary in different conditions. Indeed, testing capacity could be limited, and the contact tracing strategy may only require quarantining of close contacts. In such a case, we can regard the alert state as quarantine or a subset of S but temporarily removed from the susceptible population. Moreover, we can add more states to the model and divide the confirmed (C) state into three sub-states, to differentiate the disease state of the traced contacts. However, to keep the model simple, the model only considers one C state. Since an individual induces contact tracing as long as is in the D state, after a period of time it should move to the removed (RD) state so that the contact tracing stops. Similarly, the model assumes an individual in the C state initiates contact tracing and later moves to removed state (RC). We refer to the transmission model described above as SAIDR, and Fig. 1 shows the diagram of the transitions that a person may experience in the SAIDR model. Transition from the S state to the E state is induced by contacts with individuals in the Is or Ia states. Transitions shown by dotted arrows are due to contact tracing and are induced by contacts with individuals in the C or D states. If a transition is not induced by any interaction, we call it nodal transition and it is shown by solid arrow.

In the SAIDR model, we assume the transition times are random variables drawn from appropriate distributions for the state. The traditional approach in the analysis of epidemic models assumes the distribution of transition times to be exponential, but this might not be an accurate description of all the processes considered in our spreading model. For instance, real-world data suggest transition times for the E → I process are not distributed exponentially. Some factors that have been suggested to potentially influence these transition times, specifically the incubation period (E → Is), include age, infectious dose, and physiological stressors.

Moreover, allowing non-exponential distributions for auxiliary processes such as D → RD, increases the degrees of freedom in the model. Hence, here we allow the nodal transitions to have non-exponential distributions. An exponential distribution
is specified by the rate parameter $\lambda$, and the assumption that a transition time is exponentially distributed implies for any infinitesimal period of time $dt$ the transition happens with a constant probability $\lambda dt$, regardless of the age of the process. Indeed, such a constant rate of transition is a suitable approximation for the transmission process. Therefore, for the transmission process $S \rightarrow E$, the model assumes the transition times are exponentially distributed as long as the infecting individual stays infected. Additionally, if a susceptible person has several infectious contacts, the model assumes the infecting processes are independent. Similarly, in the contact tracing modeling, we use exponential distribution for the transition times of processes that change state of an individual to the C or A states. The table below lists the non-Markovian and Markovian transitions in the SAIDR spreading model.

| non-Markovian | A $\rightarrow$ S, $E$ $\rightarrow$ Is and Ia |
|---------------|-----------------------------------------------|
| Markovian     | S $\rightarrow$ A, S $\rightarrow$ E, Ia $\rightarrow$ C |

Finally, to model the interactions that result in virus transmission or lead to contact tracing, we use an undirected network where the nodes represent individuals in the population and the links denote the contacts among them. Consider a network $G = (\mathcal{V}, E, W_{inf}, W_{tr})$, with $\mathcal{V}$ representing the set of nodes, $E$ the set of links between nodes, and $W_{inf}, W_{tr} : \mathcal{E} \rightarrow [0, 1]$ are weight functions defined over the links. We use the weight functions $W_{inf}, W_{tr}$ to quantify heterogeneity of the contacts in relation to virus transmission probability or contact tracing probability, respectively. In other words, for each contact, we modify the rate of exponential distributions for the infecting and contact tracing processes multiplying the rates by the contact weights. This allows us to define various types of contacts with different probabilities for virus transmission or contact tracing.

### III. Mathematical Analysis of the Model

In this section we develop a set of differential equations that approximately describes the behavior of the transmission model discussed in Section II. Indeed, there is an extensive body of research concerning Markovian spreading unfolding over networks [30], [31], [32], [33], [34], [35], [36], and it is shown the exact mathematical treatment of such a system is not tractable because we need to follow the joint state of all the nodes. Therefore, a mean-field type approximation, which assumes statistical independence of neighboring nodes’ states, is often employed to study network spreading models. Here, we limit the non-exponential random variables in the SAIDR spreading model to the Erlang distributions. This enables us to use a characteristic of Erlang distribution to cast our spreading model into a Markovian model, for which we can develop an N-intertwined set of differential equations [30]. The Erlang distribution is a two-parameter family of continuous probability distributions with the density function

$$f(t|k, \lambda) = \frac{\lambda^k t^{k-1} e^{-\lambda t}}{(k-1)!} \quad \text{for } t \in [0, \infty),$$

where the shape parameter $k$ is a positive integer, and $\lambda > 0$ is called the rate parameter. From the equation above, we can see that the exponential distribution is a special case of Erlang distribution with $k = 1$, and the mean and variance of Erlang distribution are $k/\lambda$ and $k/\lambda^2$, respectively. Since the Erlang distribution has two parameters, we can adjust them to obtain a density function with a mean and a variance close to desired values. However, the shape parameter $k$ is an integer and it is not always possible to have an Erlang distribution with an exact mean and variance as the desired values. In Fig. 3 we have plotted possible values for mean and variance of the Erlang distribution around three different values of the mean. We can see when the variance is smaller than half of the mean there is an Erlang distribution with mean and variance very close to any target values. But when the variance become larger, fixing the variance for an Erlang distribution may introduce an error of around half a day in the value mean. Hence, we use the Erlang distribution as an approximation for the nodal transitions in our spreading model.

One important characteristic of the Erlang distribution can be stated as follows: distribution of sum of $k$ independent random variables, each having an exponential distribution with rate $\lambda$, is Erlang distribution with the shape parameter $k$ and the rate

![Diagram of transitions in the SAIDR spreading model](image-url)

Fig. 1. Diagram of transitions in the SAIDR spreading model. An individual can be in one of the following 10 states: susceptible (S), susceptible alert (A), infected but not infectious (E), infectious presymptomatic or symptomatic (Is), infectious asymptomatic (Ia), detected symptomatic cases (D) that trigger contact tracing, confirmed cases (C) through contact tracing, and removed states (RD), (RU), (RC) that do not cause new infections or contact tracing. The arrow type in the diagram represents the transition mechanism. Dotted and dashed arrows show transitions that are due to the individual’s contacts, and solid arrows denote spontaneous transitions that are not forced by interactions. In the diagram, $r_{inf}^{-1}$ is the node i transition rate from the S state to the E state and $r_{inf}^{-1} = \sum_{n \in \{j,k\}} W_{inf}^{-1} \beta_n \mathbf{1}_{inf}(n) + \beta_n \mathbf{1}_{inf}(n))$, where $\mathbf{1}_{inf}(n)$ is indicator function and is one when the neighbor node is in the state Is and zero otherwise. Here we assume undirected weighted contact network where the infection transmission rates $\beta_n$, $\beta_i$ are modified by multiplying them with the contact weights, $W_{inf}$. Similarly, $r_{tr}^{-1}$ is the contact tracing rate for node i, and is affected by the contact weights $W_{tr}$.
is not tractable when the number of nodes is large. But if we use mean-field approximation [30], that assumes statistical independence of neighboring nodes’ states, we obtain a set of $M \times N$ equations, which we can solve computationally. This approximation assumes at any time, the joint probability of finding nodes $i$ and $j$ in the states $s^i$ and $s^j$ can be written as the multiplication of marginal probabilities, $Pr(s^i) \times Pr(s^j)$.

To formulate the approximate behavior of the Markovian model depicted in Fig. 2, we use dynamic variables, $S_i(t), A_i^j(t), \ldots, A_{kA}^j(t), E_i^1(t), \ldots, E_{kE}^1(t), \ldots, RU^i(t)$, which represent the probabilities of finding node $i$ in the corresponding states. If $r_i^1$ and $r_i^2$ denote the rates for the transmission and contact tracing processes exerted on node $i$, we have

$$r_i^1 = \sum_j W_{i1}^{1,j} \left( \beta_k \sum_k I_k^j + \beta_B \sum_k I_a_k^j \right),$$

$$r_i^2 = \sum_j W_{i2}^{2,j} \lambda_{tr} \left( \sum_k C_k^j + \sum_k D_k^j \right), \quad (2)$$

where $\lambda_{tr}$ is the rate for the contact tracing process, and we have assumed symptomatic and asymptomatic infectious states have different infectiousness which is reflected in the infectious rates parameters, $\beta_k, \beta_B$. Finally, using the joint state independence approximation, we can write the following set of equations for all the nodes in the network

$$\dot{S}_i(t) = -r_i^1 S_i^i - r_i^2 T_i S_i^i + A_{kA}^i \lambda_A$$

$$\dot{A}_i^j(t) = r_i^1 S_i^j - \lambda_A A_i^j$$

$$\dot{A}_k^j(t) = \lambda_A (A_{kA}^j - A_k^j) \quad \text{for} \quad k = 2, \ldots, k_A$$

$$\dot{E}_i^1(t) = r_i^1 S_i^j - \lambda_E E_i^1 - r_i^2 T_i E_i^1$$

$$\dot{E}_k^j(t) = \lambda_E (E_{kE}^j - E_k^j) - r_i^2 T_i E_k^j \quad \text{for} \quad k = 2, \ldots, k_E$$

$$\dot{I}_s^j(t) = p_{hi} \lambda_E E_{sE}^j - \lambda_A I_k^j - r_i^2 T_i I_k^j$$

$$\dot{I}_k^j(t) = \lambda_{is} (I_{k1}^j - I_k^j) - r_i^2 T_i I_k^j \quad \text{for} \quad k = 2, \ldots, k_A$$

$$\dot{I}_a^j(t) = p_{hi} \lambda_E E_{aE}^j - \lambda_{ai} I_k^j - r_i^2 T_i I_k^j$$

$$\dot{I}_{kA}^j(t) = \lambda_{ia} (I_{k1A}^j - I_k^j) - r_i^2 T_i I_k^j \quad \text{for} \quad k = 2, \ldots, k_A$$

$$\dot{D}_i^j(t) = \lambda_D (D_{i1}^j - D_i^j) \quad \text{for} \quad i = 2, \ldots, k_D$$

$$\dot{C}_i^j(t) = r_i^1 T_i \left( \sum_k E_k^j + \sum_k I_k^j + \sum_k I_a_k^j \right) - \lambda_C C_i^j$$

$$\dot{C}_k^j(t) = \lambda_C (C_{k1}^j - C_k^j) \quad \text{for} \quad k = 2, \ldots, k_C$$

This characteristic implies if the random transition time of a process follows Erlang($k, \lambda$) distribution, the process can be modeled as $k$ successive transitions between $k + 1$ auxiliary states where each transition time is exponentially distributed with the rate $\lambda$. Therefore, if a nodal transition time in the spreading model of Fig. 1 has an Erlang type distribution, it is possible to replace the nodal transition with a set of successive Markovian transitions and obtain an equivalent spreading model. Fig. 2 shows a spreading model where all the transition times are exponentially distributed and it is equivalent of the original model in Fig. 1. Indeed, even if the distribution of a transition time is not Erlang, we still can use phase-type distribution method to obtain a mixture of Markovian transitions that is approximately equivalent to the original transition [21], and the spreading model equations and analysis presented in the rest of this paper will not change dramatically. However, we limit the distribution of nodal transition times to Erlang distribution because it incorporates a family of density functions with different means and variances.

Considering the Markovian spreading model in Fig. 2, let $s^i$ represents state of node $i$ among $M$ possible states in the model, and $S = [s^1, s^2, \ldots, s^N]$ represents the joint state of all the individuals in the population. Based on the nodal description of the processes, we deduce the joint state is a continuous-time Markov chain over a space consisting of $M^N$ possible network states. Therefore, the exact mathematical treatment of the system
\[
\begin{align*}
\dot{R}C_i(t) &= \lambda C_i C_{kC} \\
\dot{R}D_i(t) &= \lambda_D D_i \\
\dot{R}U_i(t) &= \lambda U_i A_i \\
\end{align*}
\]  

(3)

The system of nonlinear ordinary differential equations (ODEs) above, describes evolution of nodal states’ probability. We can use this set of equations to study behavior of the spreading processes or to estimate the model parameters. In addition, stability analysis of disease-free state of the system leads to derivation of epidemic threshold which establishes a condition that determines whether an initial infected population will vanish or has the possibility to grow. To derive the epidemic threshold the first step is to linearize the nonlinear system given by (3) about the disease-free steady state. In the disease-free state, the probability of finding nodes in any state other than the susceptible state is zero. After linearization we arrive at an independent subsystem that describes production of new infections. Since the variables in this subsystem drive other variables in the larger system, stability of this subsystem determines stability of the larger system. This subsystem can be written as follows:

\[
\begin{align*}
\dot{E}_i(t) &= r_i - \lambda_E E_i \\
\dot{I}_s^i(t) &= \mu \lambda_E E_i - \lambda_I I_s^i \\
\dot{I}_k^i(t) &= \lambda_I (I_s^{i-1} - I_k^i) \\
\dot{I}_a^i(t) &= \mu \lambda_E E_i - \lambda_I I_a^i \\
\end{align*}
\]

(4)

**Theorem 1:** Assuming the infection weight matrix \( W_{inf} \) is irreducible, the disease-free steady state is a locally stable fixed point of the dynamical system 3 if the following condition holds:

\[
(\beta_{ls}\mu \lambda_E \lambda_{bs}^{1} + \beta_{la}\mu \lambda_E \lambda_{as}^{1}) \rho(W_{inf}) < 1,
\]

(5)

where \( \rho(W_{inf}) \) denotes the spectral radius of the weight matrix \( W_{inf} \).

To prove Theorem 1 and derive the threshold condition in (5), we first rewrite the linearized subsystem 4 in a matrix form where the infection processes, represented by a matrix \( \Delta \) defined below, are separated from other transitions. To this end, we define the square matrix \( \Delta \) as the Kronecker product of the network weight matrix \( W_{inf} \) and a matrix \( \delta \) that represents the rates and the driving nodal states in the infection process,

\[
\Delta \equiv W_{inf} \otimes \delta = \\
\begin{pmatrix}
W_{inf}^{1,1} \delta & \cdots & W_{inf}^{1,N} \delta \\
\vdots & \ddots & \vdots \\
W_{inf}^{N,1} \delta & \cdots & W_{inf}^{N,N} \delta \\
\end{pmatrix}
\]

(6)

where \( 0_{k_1,k_2} \) and \( J_{k_1,k_2} \) are \( k_1 \times k_2 \) matrices of zeros and ones,

\[
0_{k_1,k_2} \equiv \begin{pmatrix} 0 & \cdots & 0 \\ \vdots & \ddots & \vdots \\ 0 & \cdots & 0 \end{pmatrix}_{k_1 \times k_2}, \\
J_{k_1,k_2} \equiv \begin{pmatrix} 1 & \cdots & 1 \\ \vdots & \ddots & \vdots \\ 1 & \cdots & 1 \end{pmatrix}_{k_1 \times k_2}
\]

(7)

We also define a matrix \( \Sigma \) that represents non-infecting processes in (4). The matrix \( \Sigma \) is a block diagonal matrix and the Kronecker product of the identity matrix of size \( N \) and a matrix \( \sigma \) that represents nodal transitions,

\[
\Sigma \equiv I_N \otimes \sigma = \\
\begin{pmatrix}
\sigma & 0 & \cdots & 0 \\
0 & \sigma & \cdots & 0 \\
\vdots & \ddots & \ddots & \vdots \\
0 & 0 & \cdots & \sigma \\
\end{pmatrix}_{N \times N}
\]

(8)

where the matrices \( H, G \) used in the definition of \( \sigma \) have the following structures:

\[
H_k \equiv \\
\begin{pmatrix}
-1 & 0 & \cdots & 0 \\
1 & -1 & \cdots & 0 \\
\vdots & \ddots & \ddots & \vdots \\
0 & 1 & \cdots & -1 \\
\end{pmatrix}_{k \times k}, \\
G_{k_1,k_2} \equiv \begin{pmatrix} 0 & 1 \\ \vdots & \ddots \\ 0 & \cdots & 0 \end{pmatrix}_{k_1 \times k_2}
\]

(9)

In the \( H \) matrix, the diagonal elements are \(-1\), the lower diagonal elements are \(1\) and the rest of elements are zero. In the \( G \) matrix, the element in the upper-right corner is \(1\) and the rest of elements are zero.

Using the matrices \( \Delta \) and \( \Sigma \), now we can rewrite the linear system 4 as

\[
\frac{dX}{dt} = (\Delta + \Sigma) X
\]

\[
X = [x^1, \ldots, x^N]^T \\
x^i = [E^i_k, I_s^i, \ldots, I_k^i, I_a^i, \ldots, I_{ka}^i]
\]

(9)

To derive the threshold condition in (5), we analyze stability of the linear system (9). The stability of steady state is determined by the spectral bound of the square matrix \( \Delta + \Sigma \), defined as

\[
s(\Delta + \Sigma) = \sup \{ \Re(\alpha) | \alpha \in \eta(\Delta + \Sigma) \}
\]

where \( \eta(\Delta + \Sigma) \) denotes the set of eigenvalues of \( \Delta + \Sigma \). The linear system is exponentially stable if and only if the real parts of the eigenvalues are negative (i.e., if \( s(\Delta + \Sigma) < 0 \)). To find the condition that leads to a negative spectral bound we apply theorem A.1 from reference [37], which, for the sake of completeness, we state as the following theorem.

**Theorem 2:** If \( X \) is a positive matrix and \( Y \) is a positive off-diagonal matrix with \( s(Y) < 0 \), then

\[
\text{sign}(s(X + Y)) = \text{sign}(\rho(-XY^{-1}) - 1)
\]

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where $\rho(.)$ and $\text{sign}(.)$ denote spectral radius and sign functions, respectively.

We note that in reference [37], positive matrices are defined as non-zero matrices with all entries non-negative; and a matrix is defined as positive off-diagonal if all entries are non-negative except possibly those on the diagonal.

**Proof of Theorem 1:** To prove Theorem 1, we use the result of Theorem 2 to find a condition that guarantees $s(\Delta + \Sigma)$ is a negative number. To apply Theorem 2, we first investigate if the matrices $\Delta$ and $\Sigma$ satisfy the condition stated in that theorem.

From the definition of $\Delta$ and $\Sigma$ in the (6), and (8), we can see $\Delta$ is a positive matrix and $\Sigma$ is positive off-diagonal. Moreover, since $\Sigma$ is positive off-diagonal lemma 6.12 in [38] shows that $s(\Sigma) < 0$ if and only if $\Sigma$ is invertible and $-\Sigma^{-1}$ is a positive matrix. Indeed, we can directly calculate $-\Sigma^{-1}$ as follows

$$-\Sigma^{-1} = I_N \otimes (-\sigma^{-1})$$

$$-\sigma^{-1} = \begin{pmatrix} \lambda_{E}^{-1} F_{kE} & 0_{kE,k_h} & 0_{kE,k_i} \\ p_h \lambda_{E}^{-1} J_{k_h,k_E} & \lambda_{i}^{-1} F_{k_i} & 0_{k_i,k_h} \\ p_h \lambda_{E}^{-1} J_{k_h,k_i} & \lambda_{k}^{-1} F_{k_h} & 0_{k_h,k_h} \end{pmatrix}$$

where the matrices $0$ and $J$ are defined in (7), and $F_k$ is a lower triangular matrix of size $k$, with non-zero entries equal 1

$$F_k = \begin{pmatrix} 1 & 0 & \cdots \\ 1 & 1 & \cdots \\ \vdots & \vdots & \ddots \end{pmatrix}_{k \times k}$$

(11)

It is clear that $-\Sigma^{-1}$ is a positive matrix. Therefore we can use Theorem 2 to conclude that

$$s(\Delta + \Sigma) < 0$$

if and only if $\rho(-\Delta \Sigma^{-1}) < 1$.

Finally to prove Theorem 1 we will obtain an expression for $\rho(-\Delta \Sigma^{-1})$. Using the properties of the Kronecker product we can write

$$\eta(-\Delta \Sigma^{-1}) = \eta(W_{\inf} \otimes (-\sigma^{-1}))$$

$$= \{ \alpha_i \gamma_j \mid \alpha_i \in \eta(W_{\inf}), \gamma_j \in \eta(-\sigma^{-1}) \},$$

(12)

where $\eta(.)$ denotes set of eigenvalues. After calculating the matrix $(-\sigma^{-1})$, we can see only the first row is non-zero

$$-\sigma^{-1} = \begin{pmatrix} \beta_{i} p_h k_h \lambda_{i}^{-1} + \beta_{a} p_h k_a \lambda_{a}^{-1} & \cdots & \beta_{i} \lambda_{i}^{-1} \\ 0 & \cdots & 0 \\ \vdots & \ddots & \vdots \\ 0 & \cdots & 0 \end{pmatrix}$$

and therefore the eigenvalues of the matrix $-\sigma^{-1}$ are $\beta_{i} p_h k_h \lambda_{i}^{-1} + \beta_{a} p_h k_a \lambda_{a}^{-1}$ and 0. In addition, since we assume $W_{\inf}$ is an irreducible non-negative weight matrix, Perron–Frobenius theorem shows $W_{\inf}$ has a positive eigenvalue, denoted by $\rho(W_{\inf})$, and absolute value of any other eigenvalue of $W_{\inf}$ is strictly smaller than $\rho(W_{\inf})$. Combining these results about

the eigenvalues of $W_{\inf}$ and $-\delta \sigma^{-1}$ with (12), we find that

$$\rho(-\Delta \Sigma^{-1}) = (\beta_{i} p_h k_h \lambda_{i}^{-1} + \beta_{a} p_h k_a \lambda_{a}^{-1}) \rho(W_{\inf})$$

(13)

which leads to the proof of Theorem 1.

The right-hand side of (13) is the multiplication of $\rho(W_{\inf})$ by the probability of generating a new infected case by an infected individual. In general, $\rho(W_{\inf})$ depends on the network structure, but it is possible to show $\rho(W_{\inf}) < \max_{i,j} W_{ij}$. In other words, $\rho(W_{\inf})$ is smaller than the largest weighted node degree in the network. Thus the right-hand side of (13) is bounded by the maximum expected secondary infection generated by and an infected individual in the network, and if this value is smaller than one the epidemic dies out exponentially.

To numerically investigate the threshold condition, we computed prevalence of the exposed state (E) through time for a spreading unfolding on the largest component of the coauthorship network [39]. We calculated the prevalence of a state as the average of nodes’ probabilities. Fig. 4(a) shows the prevalence of exposed state for different values of $\rho$ when we assumed no contact tracing. Fig. 4(b) shows similar prevalence when there is contact tracing. In both figures, we can see the exposed state exponentially dies out when the spreading is below the threshold. Fig. 4(c) and (d) show similar plots for a random scale-free network and a random Erdős–Rényi network, respectively.

To summarize, in Fig. 4, we have plotted the exposed state prevalence in SAIIRD process unfolding on different network structures with node numbers ranging from 400 to 2000. For each network, we have plotted the prevalence for different values of the threshold parameter $\rho$. We can see regardless of contact tracing implementation and network structure, if the value of $\rho$ is smaller than 1 then the epidemic dies out. This is consistent with
Theorem 1. However, we need to clarify that Theorem 1 does not address the cases where $\rho$ is greater than 1. For instance, from Fig. 4(a) and (b), we can see for $\rho = 1.6$, which is greater than 1, the initial prevalence of the exposed state dies out exponentially if contact tracing is applied. However, when contact tracing is not implemented, the exposed state prevalence grows, and the epidemic dies out only when the susceptible population diminishes.

The threshold condition we have derived, only depends on the network structure, infection transmission rates, probability of becoming a symptomatic case, and expected infectious periods for the symptomatic and asymptomatic states. This is clear from (13), if we notice that $k_0 \lambda_s^{-1}$ and $k_0 \lambda_a^{-1}$ are the expected values for the corresponding Erlang distributions. Although the variances for the infectious period distributions do not change the threshold condition, they affect the prevalence of the states as shown in Fig. 5. To generate this figure, we calculated the prevalence curves for two spreading processes unfolding on the network of the previous example. In the first process, we assumed the symptomatic and asymptomatic infectious periods are distributed exponentially with expected values of four and six, respectively. For the second process, we changed the exponential distributions to Erlang distributions with similar expected values but variance of 2.

IV. CASE STUDY

In this section the we apply the SAIDR spreading model to COVID-19 transmission among Kansas State University (K-State) students in Manhattan, Kansas. This model accounts for symptomatic and asymptomatic transmission in COVID-19 spreading and also includes transitions and states that model non-pharmaceutical interventions. Therefore, it can be used to evaluate the effectiveness of different strategies for reducing the virus spread or predicting the epidemic size. However, this requires information about the network structure and other model parameters. We performed a survey among individuals associated with the university and the survey results were used to build a random contact network. Assuming this contact network for the population, we use weekly positive COVID-19 cases to estimate unknown model parameters such as the transmission rate $\beta_{ls}$. Later, we use the estimated parameters to study the effectiveness of contact tracing among the students.

A. Contact Network

To build a weighted contact network we sent an online survey to all Kansas State University students and staff via the university webmail system in December 2020. This survey asked about participation in social interactions during the 2020 fall semester (August 2020-December2020). During this semester, some of the classes were held in-person and some held online. Students were present in Manhattan, Kansas, during this time and a mask mandate was in effect in all the facilities related to the university and in community public spaces.

We asked questions about housing status, age and role in the university, as well as the number of close contacts such as roommates, family members, or coworkers. In another question we also asked for the number of people they regularly meet in close proximity for a total contact duration of less than four hours per week (such as friends). The responses to this question were used to build a second layer of contacts that can be traced but have lower transmission probability. To find the level of social interaction we asked the respondents to specify average number of visits per week to different public locations such as bars, restaurants, coffee shops and stores. We also asked about the frequency of their participation in social events such as religious or sports events. For each public location type, the respondents also indicated duration of the visits and number of people with whom they interact in close proximity. Detailed information about the survey can be found in the Appendix.

After processing the data, based on their housing type and role in K-State, we divided the respondents into six groups. These groups are: (1) graduate students (2) factually and staff, (3) undergraduate students living in off campus apartment or houses, (4) undergraduate students living in fraternities or houses (Greek houses), (5) and (6) undergraduate students using two available on-campus housing options. The rationale for this division is that the respondent age and housing type possibly leads to different levels of social interaction and number of close contacts. Using the survey data, we calculated the following parameters for each group $g$

- $V_1^g$, average number of close contacts
- $V_2^g$, average number of people met for total duration of less than four hours per week

and for the three types of public spaces which are (1) bars, restaurants, and coffee shops, (2) stores and services, and (3) social events

- $P_l^g$, proportion of individuals in group $g$ who visits public space type $l$,
- $H_l^g$, average weekly hours they spent in these locations,
- $n_l^g$, average number of people they encounter

Values of these parameters are presented in Table 1.

Using these parameters, we built three layers of networks for the whole population comprising the groups mentioned before and an additional group which is the rest of the town population. Since we conducted the survey only among the individuals associated with university, we did not have the parameter values for the general public not associated with the university. Hence, we extended the parameters extracted for the group of faculty and staff to that additional group. Population of different

---

Fig. 5. Effect of infectious period distributions on the E and R states’ prevalences. For the curves tagged with “Non-exponential transition time”, we used Erlang distributions with the same expected values as the the curves tagged with “Exponential transition time” but with different variances.
groups that we assumed in our calculations are given in the table below.

| group # | 1     | 2     | 3     | 4     | 5     | 6     |
|--------|-------|-------|-------|-------|-------|-------|
| $\nu^g_0$ | 3.5   | 3.5   | 5.8   | 14.3  | 4.5   | 3.5   |
| $\nu^g_1$ | 3.5   | 2.3   | 4.9   | 10.7  | 5.1   | 4.2   |
| location 1 |       |       |       |       |       |       |
| $p^g_i$ | 0.5   | 0.4   | 0.6   | 0.73  | 0.53  | 0.5   |
| $H^g_i$ | 1.76  | 0.7   | 3.25  | 4.46  | 2.25  | 1.5   |
| $n_i$ | 4     | 2.7   | 6     | 5.3   | 5.3   | 3     |
| location 2 |       |       |       |       |       |       |
| $p^g_i$ | 0.91  | 0.92  | 0.89  | 0.81  | 0.75  | 0.91  |
| $H^g_i$ | 2     | 1.7   | 2.6   | 2.1   | 1.5   | 3     |
| $n_i$ | 4.5   | 4.2   | 4.9   | 4.3   | 4.7   | 4.7   |
| location 3 |       |       |       |       |       |       |
| $p^g_i$ | 0.3   | 0.26  | 0.45  | 0.72  | 0.56  | 0.33  |
| $H^g_i$ | 0.85  | 0.7   | 1.72  | 4.3   | 3     | 2.37  |
| $n_i$ | 6     | 5.5   | 8     | 14    | 8.6   | 4.9   |

For layer one, $L_1$, which is the layer of close contacts, consider clusters of $\nu^g_0 + 1$ individuals within the main groups. We assume among $\nu^g_0$ close contacts for each individual, a fraction $F$ of the contacts happens within the cluster the individual belongs to, and the remaining contacts are randomly established using configuration model. To set up the random links we divided the population into three subpopulations: (a) group 1, (b) group 2 and (c) groups 3, 4, 5, 6. Then we established the random links only among the individuals belonging to the same subpopulation.

For layer two, $L_2$, we used configuration model where the node degree of an individual in group $g$ was set to $\nu^g_1$. We assumed undergraduate students have only links with other undergraduate students and graduate students with other graduate students. To decrease the number of model parameters, we express daily infection transmission rate in this layer in terms of transmission rate through the close contacts of layer $L_1$, which we denote by $\beta$. If we assume the effective daily contact duration in $L_1$ is only eight hours and compare that with weekly duration of links we divided the population into three subpopulations: (a) group 1, (b) group 2 and (c) groups 3, 4, 5, 6. Then we established the random links only among the individuals belonging to the same subpopulation.

For layer three, $L_3$, which represents interaction through public spaces, we considered a complete graph over the whole population and weighted the link between any two nodes $i$ and $j$ by

$$\sum_{l=1}^{3} P_l g(i) g(j) \frac{P_l g(i) g(j)}{\sum_{k=1}^{N} P_l g(k) g(k)} H^g_l H^g_j C_l$$

In this expression, $l$ enumerates three different public spaces we mentioned before, $g(i)$ represents group assignment of node $i$ and $N$ is the total population. $C_l$ is a coefficient that when is multiplied by $H^g_l H^g_j \beta$, gives an estimate of infection transmission rate between nodes $i$ and $j$ in public space $l$, assuming $\beta$ is transmission rate for a close contact link in the layer $L_1$. Indeed, if $T_l$ is the total hours per week that the public space is active $H^g_l H^g_j \beta / (7T_l)$ is daily expected hours that nodes $i$ and $j$ overlap in such a location. Moreover, if we assume the effective daily duration of close contact is eight hours, $H^g_l H^g_j \beta / (7 \times 8T_l)$ gives an estimate of daily infection transmission rate between nodes $i$ and $j$ in the public space $l$. Here, we use $T_1 = 35$, $T_2 = 70$, $T_3 = 15$.

B. Model Parameters

For some of the parameters in the spreading model described in Section III, we use the values below in our calculations.

Following reference [40], we assume the proportion of infected individuals that never show symptoms is $\rho_{is} = 0.30$, and their infectiousness is lower than those who develop symptoms such that $\beta_{is} = 0.75 \beta_{ik}$.

For transition from the E state to the I states, we use $\lambda^{-1}_E = 1.255$ days, and $\kappa_E = 3$. These values lead to a distribution with the mean of 3.76 days and the standard deviation of 2.17 (Fig. 6). We chose this distribution using the data in reference [41], where authors report the distributions for the incubation period (i.e., from time exposure to exposure symptoms onsets) and the serial interval which is time between the symptoms onsets of successive cases in a chain of transmission. In fact, the reported negative serial interval implies pre-symptomatic transmission of COVID-19 during the incubation period. To estimate the E state period in our model, we assumed that, in the incubation period, infected individuals go through several stages with the same expected time, and in one of the stages they start transmitting the infection. If this stage is before the onset of symptoms, we will observe negative serial intervals. Hence, we approximated the incubation period with an Erlang distribution with $k = 4$ and $\lambda^{-1} = 1.255$ days, which has the same median and 95th percentile as the reported distribution in [41]. Next, we simulated a chain of transmission assuming transmission starts at a specific stage of incubation period with a specific transmission rate, and we recorded the distribution of the serial interval. By exploring different values for the stage that the transmission starts, and also the transmission rate, we found that the simulated distribution of the serial interval is closest to the reported one in [41], if the transmission starts at the stage four. Therefore, we used an Erlang distribution with $k = 3$ and $\lambda^{-1} = 1.255$ days for the period of E state in our model.

Considering the transition from the Ia state to the RU state, we assumed an Erlang distribution of mean six days and variance of two days. Fig. 6 shows the corresponding density function. This
value of variance implies that there is almost no transmission after day nine [42] and there is transmission in the first week when the viral load is high [42].

Here we use similar distributions for the random times of the transitions C → RC and D → RD. If we assume $k_C = k_D = 8$ and $\lambda_C^{-1} = \lambda_D^{-1} = 0.25$ days, and $\lambda_{tr} = 1.5$ day$^{-1}$, then the probability that a node in D or C state induces a contact tracing transition in its neighbor is 0.92. We calculate the contact tracing success probability as

$$
\int_0^\infty f(t|k_D, \lambda_D)(1 - e^{-\lambda_{tr}t})dt = \frac{\lambda_{tr}}{\lambda_{tr} + \lambda_D} \sum_{i=0}^{k_D-1} \left( \frac{\lambda_D}{\lambda_{tr} + \lambda_D} \right)^i.
$$

We can adjust this probability by changing $\lambda_{tr}$ or $k_C$, $k_D$, $\lambda_C$, $\lambda_D$. For instance, if we change $\lambda_{tr}$ to only 0.18 day$^{-1}$, the probability becomes 0.3. In our calculations, we assume the rate of success for contact tracing through the layer of close contacts, $L_1$ is high, and through the layer $L_2$ is lower, therefore, we chose $\lambda_{tr} = 1.5$ and 0.18 day$^{-1}$ for the layers $L_1$ and $L_2$, respectively. We need to note that the duration of staying in the C or D state cannot be long, otherwise these states may induce repetitive contact tracing in the model.

The other transition that we specify its random time distribution is $A \rightarrow S$. Here, we set the $k_A = 12$ and $\lambda_A^{-1} = 0.5833$ days. These values lead to a distribution that is concentrated between days 4 and 11 with a mean of 7 days (Fig. 6).

C. Approximate Model

Practically, we can solve the network spreading model ODEs of the system 3, even if the population is large. This system of equations is applicable for any network with an arbitrary structure. However, the three-layer network we described in Section IV-A, to some extent, is homogeneously. Specifically, the nodes that belong to a same group have similar type and number of links in the layers $L_1$ and $L_2$, and their community contacts through the layer $L_3$ are identical. Hence, we expect that the probability vectors of the nodes with a similar group assignment will be similar, and we may use one probability vector for all the nodes in a group. Within this approximation, the dimension of the dynamical system that describes the spreading process is smaller, and each group of nodes is represented by only one node. The ODEs for this system are similar to those in (3), except that the superscript $i$ for the variables now enumerate the groups and run from 1 to 6. To have a closed system of ODEs, we also need to approximate the infection and tracing rates, $r_i^j$ and $r_i^j$, in term of the probability vectors of the groups. The infection rate of a node in group $i$ can be written as

$$r_i^j = \sum_{j=1}^{6} \Omega_i^{j} \left( \beta_{ls} \sum_{k} I_{sk}^{j} + \beta_{ls} \sum_{k} I_{sk}^{j} \right),$$

where $\Omega_i^{j}$ determines the contribution of group $j$ in the infection rate of a node in group $i$ and $\beta_{ls}$ are infection transmission rates through close contacts of the layer $L_1$. Considering the definition of the network parameters in Section IV-A, $\Omega_i^{j}$ is approximated by

$$\Omega_i^{j} = \mathcal{V}_i^{j} \left( 1 - \mathcal{F} \right) \sum_{k} \mathcal{V}_i^{k} \left( 1 - \mathcal{F} \right) \omega_{i,k}^{j} N_k + \delta^{i,j} \mathcal{F} \mathcal{V}_i^{j}$$

$$+ \zeta \mathcal{V}_i^{j} \sum_{k} \mathcal{V}_i^{k} \omega_{i,k}^{j} N_k$$

$$+ \sum_{l=1}^{\mathcal{V}_i^{j}} \mathcal{P}_i^{j} \mathcal{H}_i^{j} \mathcal{H}_i^{j} \mathcal{C}_i$$

where $\delta^{i,j}$ is the Kronecker delta function, $N_i$ is the population of group $i$ and $\zeta = 4/(7 \times 8)$ is the ratio of infection transmission rate for the contacts in the layer $L_2$ to that of the layer $L_1$. Furthermore, $\omega_{i,j} = 1$, if existence of links between the nodes in groups $i$ and $j$ is allowed in the network layers $L_1$ and $L_2$, otherwise $\omega_{i,j} = 0$. The first, second and third lines in the equation above give the contribution of the network layers $L_1$, $L_2$ and $L_3$ in the infection rate, respectively. Also, the contact tracing rate for the nodes in group $i$, represented by $r_i^j$, can be approximated as

$$r_i^j = \sum_{j} \Omega_i^{j} \lambda_{tr} \left( \sum_{k} C_i^{j,k} + \sum_{k} D_i^{j,k} \right),$$

where $\lambda_{tr} = 1.5$ day$^{-1}$ is the tracing rate through the close contacts in the layer $L_1$ and

$$\Omega_i^{j} = \mathcal{V}_i^{j} \left( 1 - \mathcal{F} \right) \sum_{k} \mathcal{V}_i^{k} \left( 1 - \mathcal{F} \right) \omega_{i,k}^{j} N_k + \delta^{i,j} \mathcal{F} \mathcal{V}_i^{j}$$

$$+ \zeta \mathcal{V}_i^{j} \sum_{k} \mathcal{V}_i^{k} \omega_{i,k}^{j} N_k$$

In the equation above, $\zeta$ is the ratio of tracing rate through the layer $L_2$ to $\lambda_{tr}$, which we set at 0.18/1.5.

To compare the approximated spreading model with the high dimension network model, we calculated the total population of students in the D, C, RD, RC states using both models assuming similar initial condition and the contact network parameter $\mathcal{F} = 0.3$. Fig. 7 shows this population for different values of $\beta_{ls}$ and $\lambda_{tr}$. In this figure, the points shown by square markers were calculated using the network spreading model equations and the line plots are the result of the approximate spreading.
D. Estimation of Effectiveness of Contact Tracing

In this section we use the reported positive COVID-19 cases to estimate the effectiveness of contact tracing among the students during the 2020 fall semester. We assume the infection spreading follows our spreading model, and the contact network and the parameters’ values are those discussed before in Sections IV-A and IV-B. Since we do not know the value of infection transmission rate $\beta_k$ and the distribution of infectious period for the symptomatic state, Is, we use a Markov chain Monte Carlo (MCMC) scheme to estimate these unknown parameters and eventually obtain an estimation for the effectiveness of the contact tracing [43], [44], [45]. Although there are some published results regarding these parameters, we believe they cannot be extended to all populations. For instance, the distribution of the symptomatic infectious period, by which we mean the period an infectious symptomatic is spreading infection before removal from the population, depends on the level of population vigilance and testing.

Here we use the Metropolis’Hastings algorithm, which is a MCMC method, to estimate the spreading model parameters. In general, this algorithm generates samples of a random variable for which we can calculate the probability ratio between the samples. Later, these samples can be used for calculating numerical approximations of functions of the random variable. To detail the algorithm, consider a k-dimensional random vector $X = (X_1, \ldots, X_k)$, with a probability distribution proportional to $f(x_1, \ldots, x_k)$. The algorithm generates a sequence of $M$ random samples $\{x^i = (x_1^i, \ldots, x_k^i)\}_{i=1}^M$ following the steps below:

1) Choose an initial sample $x^1$, and set $t = 1$
2) Generate a proposal sample $x = (x_1, \ldots, x_k)$, such that each $x_i$ is normally distributed with a mean of $x_i^t$ and a predefined standard deviation, i.e., $x_i \sim N(x_i^t, \sigma_i^2)$
3) Calculate acceptance probability, $\rho = \min(1, \frac{f(x)}{f(x^t)})$.
4) Generate a uniformly distributed random number in the interval $[0,1]$.
5) If $r \leq \rho$ accept $x$ as the next sample, $x^{t+1} = x$, otherwise set $x^{t+1} = x^t$.
6) Set $t = t + 1$, and go back to step 2.

In order to adopt this algorithm in our estimation problem, we need to define the function $f(x)$ for our problem. Let $\{o_s\}$ and $\{y_s\}$ denote an observed spreading data series and the corresponding outputs of spreading model. For a vector of spreading model parameters, $\mathbf{x}$, we assume $f(x) = S(x)^{-2}$, where $S(x)$ is the sum of absolute deviation of the spreading model outputs, $S = \sum_s |o_s - y_s|$. Using this function, the fitting algorithm generates a sequence of samples from the parameter space such that the density of samples in a region with the error $S$ is four times the density in a region with the error $2S$. In the definition of $f(x)$, we have used the sequences $\{o_s\}$ and $\{y_s\}$, which we need to specify. Since we had access to the weekly new positive COVID-19 cases among the students, we use the weekly cumulative cases from the start of the fall semester as the sequence of observed spreading data $\{o_s\}$. The corresponding sequence from the model, $\{y_s\}$, is the weekly population of students in the states D, DR, C, CR. Regarding the spreading model, we use the approximate model in Section IV-C because it is computationally fast and its outputs are similar to those calculated by the network model ODEs. Concerning the open parameters in spreading model, we assumed the parameter vector $\mathbf{x}$ consists of the following components:

- Mean and variance of the infectious period for the symptomatic state. Assuming these parameters, we find an Erlang distribution with same mean and a variance closest to the parameter value, and we use this distribution in the model.
- Transmission rate $\beta_k$, which we allow to change every 15 days. Effectively, through the course of spreading, average $\beta_k$ in every 15 days is represented by one component of $\mathbf{x}$.
- Timepoint of the first observation in the sequence $\{o_s\}$ with respect to the initial day of the model calculation. We solve the ODEs for the model with the initial $E$ state probability equal to 0.005.
- Parameters $\mathcal{F}$ which is defined in Section IV-A, and relates to the structure of the close contact network layer.

Following the procedure explained in this section, we generated sequences with 1 million samples of the open parameters. In the Bayesian inference paradigm, the generated samples estimate the full posterior distribution of the open parameters, and we can use these samples to infer the marginal distribution of the open parameters or functions of them. Using such distributions, we can obtain credible intervals for the parameters. Fig. 8(a) shows a histogram of the samples’ absolute error $S$. The output
of the spreading model for a sample with the minimum error of 330 is shown in Fig. 8(b) where we have plotted the population of students in the D, C, RD and RC states and compared it with the reported students’ positive cases.

To estimate the effectiveness of contact tracing, we set the contact tracing rate in the model to zero, $\lambda_{tr} = 0$, and for each sample of the open parameters we recalculated the total population of infected students at the day of last observation. Next, we calculated the ratio of this population to the same population of infected students at the day of last observation. Then, we divided this ratio by the factor $S(x)$, which has mean of 0.6. We have also shown the Interquartile range and the estimated median for $\beta_{Is}$ in Fig. 9(d). Although in each parameters sample, $\beta_{Is}$ could change every 15 days, the estimated distribution for $\beta_{Is}$ in this plot changes more frequently. This is because each parameter sample has different offset with respect to the first observation time point. Moreover, we can see a range of values for $\beta_{Is}$, but if we assume $\beta_{Is} = 0.1$, the factor $S(x)$ in the threshold (13) becomes 0.24. Considering the fact that the largest eigenvalue of a network is larger than its minimum node degree, we can deduce that if the individuals in a population have more than four close contacts the threshold condition for the epidemic die-out will not satisfy. This is especially significant because in the calculation we have assumed the mean of infectious period for the Is state is only 1.5 days which is very small.

In Fig. 10(a), we have shown average effective reproduction number among the students. For each student $i$ we calculate the effective reproduction number as

$$S^m(t) = \frac{\sum m(t)W_{inf}^m}{\text{students living in Greek houses}}.$$  

where $W_{inf}$ represents the total weighted contact network, $S^m(t)$ is the probability that a node $m$ is in the susceptible state at time $t$ and $(\beta_{Is}p_{Is}k_{Is}^{-1} + \beta_{Is}p_{Is}k_{Is}^{-1})$ is the probability that an infected individual spreads the virus to a susceptible individual in a homogeneously mixed population. This expression corresponds to the the classic definition of the effective reproduction number in a homogeneously mixed population. Since the number of contacts varies among different student groups, we calculated the average effective reproduction number and the results are shown in Fig. 10(a). To show heterogeneity of the effective reproduction number, in Fig. 10(b) we have compared this quantity for the students living in Greek houses and off-campus apartments.
proposed models resulted in more accurate case predictions. The errors noted in the plots are the sum of the absolute values of the prediction errors.

E. Short-Term Forecasting Performance

To demonstrate the advantage of our proposed model and the importance of considering the physical underlying spreading complexities in mathematical modeling, we compared its one-step and two-step forecasting performance with other COVID-19 spreading models commonly used. Specifically, we used particle filtering to forecast case numbers among Kansas State University students, as we believe the physical spreading process in that location reflects the complexities in our model. Our model outperformed the other models, on average, because it accounted for the different complexities of the underlying process. Fig. 11 shows the predictions and results of our comparison. In this figure, Model A represents our proposed model that takes into account non-exponential sojourn times and a heterogeneous contact network we built using the survey data. Model B, on the other hand, is similar to our proposed model, but it replaces non-exponential sojourn time distributions with exponential distributions of similar means and assumes a homogeneous population. Therefore, Model B considers contact tracing and asymptomatic infectious individuals. Ignoring contact tracing in Model B yields the SEIsIaR model, which is a modification of the SEIR compartment model that allows for asymptomatic infectious individuals. The SEIsIaR model is referred to as Model C in the figure. The traditional Markovian SEIR compartment model is represented as Model D, and we can observe that it yields the worst prediction error. Therefore, it is evident that incorporating various aspects of the underlying spreading process in our proposed models resulted in more accurate case predictions.

V. Conclusion

In this work, we have developed and investigated a novel non-Markovian network-based model to assess the effectiveness of contact tracing and shed light on conditions to contain a COVID-19 outbreak. Traditionally, many epidemic models assume states’ sojourn times are exponentially distributed, which is not the case in most real-world processes. For instance, using an exponential distribution for the quarantine duration in COVID-19 spreading process is not justifiable. In our study, we address such non-Markovian transitions by introducing auxiliary states into the original spreading model. Our SAIDR model also accounts for the processes that induce quarantine and contact tracing in a complex contact network. We derived the epidemic threshold condition by linearizing the mean-field equations for the networked spreading model. This threshold condition is a function of infection transmission rates, the expected value of the infectious period, and the contact network spectral radius. Although the threshold condition does not depend on the type of the infectious period distribution, our calculations show the distribution of the infectious period affects the epidemic curve. We also applied the developed model to the COVID-19 transmission among Kansas State University (K-State) students in Manhattan, Kansas. To this end, we perform a survey to develop a three-layer contact network. The first layer includes close contacts. The second layer accounts for regular contacts with a short weekly duration such as contacts with friends; and the third layer represents the random contacts in public spaces. After fitting the spreading model using the observed case data, we were able to estimate the effectiveness of contact tracing during the observation period. Our calculations show that contact tracing effectively reduced the epidemic size by more than three-fold. Furthermore, our study shows when considering homogeneous contact patterns, even limiting the number of contacts to a very small number (i.e., 4-5) per individual would not be enough to contain the COVID-19 epidemic. One limitation of our study is that we use first-order closure approximation to derive the mean-field equation and the threshold condition. In our future work, we plan to develop moment closure technics appropriate for such networked spreading model. Although our SAIDR model accounts for various aspects of COVID-19 transmission, we have not included vaccination in the model because no individual was vaccinated in the period of the case study. However, it is possible to generalize the model to include the vaccination effects by adding a new vaccinated state and specifying the corresponding transition rules. Our approach in Section IV can be applied to other locations and different variants of COVID-19, but it requires adapting the parameters to the specific location. Furthermore, to obtain the numerical results in Section IV, we use some assumptions regarding the percentage of the traced contacts and the relation between the infection rates in different contact layers. Even though the assumptions are reasonable in the context of this case study, it highlights further investigation to estimate such parameters. Overall, we have proposed a non-Markovian model for evaluating SARS-CoV-2 transmission and containment strategies. The model includes various components and states to study such spreading processes more realistically. In the model, we have introduced three removed states to track the transition history of individuals, which could be useful in fitting the model to different observation types and evaluating different measures for disease containment. We also have introduced the auxiliary states to account for the non-exponential distribution of some of the states’ sojourn times. Considering such non-Markovian transitions is in accordance with real-world data.
APPENDIX A

The questions asked in the survey of the case study presented in Section IV-A are the following.

Q1. What is your age?
Q2. Indicate your role at K-State: a) Undergraduate student, b) Graduate student, c) Faculty, d) Staff.
Q3. What is your normal housing status during the semester? a) Off-campus apartments or houses, b) Jardine apartments, c) Sororities and fraternities, d) On-Campus residence halls.

In a typical week before the Thanksgiving break,

Q4. For each of the total contact durations below, write the number of people you regularly met at a distance less than six feet: a) More than 4 hours per week (examples are roommates, family members, or coworkers), b) Between 1 to 4 hours per week (for instance, friends or classmates), c) Between 15 minutes to 1 h per week (for instance, friends or others that you might occasionally meet).
Q5. Consider your visits to Rec or any Gym or other shared exercise space. Write: a) the average number of times you visit this type of location, b) the average duration for each visit in hours (decimals will work; e.g., 0.5 hours for 30 minutes), c) the average number of people you encounter in each visit at less than 6 feet distancing.
Q6. Consider your visits to the student Union, dining centers, and coffee shops on campus. Write: a) the average number of times you visit this type of location, b) the average duration for each visit in hours, c) the average number of people you encounter in each visit at less than 6 feet distancing.
Q7. Consider your visits to bars, restaurants, and coffee shops off campus. Write: a) the average number of times you visit this type of location, b) the average duration for each visit in hours, c) the average number of people you encounter in each visit at less than 6 feet distancing.
Q8. Consider your visits to stores and other types of services off campus. Write: a) the average number of times you visit this type of location, b) the average duration for each visit in hours, c) the average number of people you encounter in each visit at less than 6 feet distancing.
Q9. Consider your participation in other types of social gatherings (examples are sport, religious, and social events). Write: a) the average number of times you visit this type of location, b) the average duration for each visit in hours, c) the average number of people you encounter in each visit at less than 6 feet distancing.

In total 2478 respondents completed the survey among which 388 respondents belong to the group one, 944 to the group two, 700 to the group three, 94 to the group four, 294 to the group five and 58 to the group six.

APPENDIX B

To show the flexibility of our model, we have applied it to county data by estimating location-specific parameters. Considered counties include the ones hosting Kansas University (KU), Oklahoma State University (OSU), and University of Wyoming (UW). These universities are located in college towns almost similar to the town in our case study. Unfortunately, we could not find the case data for the students and only county level case data are available. Moreover, to account for an unavailable contact network structure, we scaled the network built in the case study of Section IV, based on the student population and the demography of each county. Fig. 12. shows the estimated infectious rates. Although the values are not exactly the same, the infectious rates for counties hosting KU and OSU are in the estimated range for the KSU students. The main reason for the difference is the fact that we do not know the county level network structure for the counties hosting KU and OSU and we are fitting the model to the county level cases data. For the county hosting UW other reasons might play a role since the epidemic curve is very different as we can see in Fig. 13. In this figure, for each location, we have plotted the model output for a set of parameters resulting in the smallest error comparing with the cumulative cases from the start of semester. We can see the model can be fitted to the case data.

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