Simulating virus diffusion in networks with quantum computers

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We propose to use quantum mechanical tools to simulate epidemic processes in a network. We first show a systematic way to map virus population distributions to spin-lattice configurations. Then, noticing that diffusion is a classical thermal dynamic process, we can map it to the dynamics of an effective (parametrized) Hamiltonian of a quantum thermal system. To demonstrate the rationality of the Hamiltonian, we provide numerical and analytic analyses of the evolution behaviour of the Hamiltonian. We prove that the evolution could be well described by a classical stochastic Markov process, which is consistent with the well-known epidemiological susceptible and infectious model. A practical method to determine the parameters of the thermal dynamic Hamiltonian from epidemiological inputs is exhibited. As an example, we simulate the transmission process of SARS-Cov-2 variant Omicron with a given community network.

I. INTRODUCTION

The simulation of population diffusion in networks is a significant and widely studied problem. In epidemiology, modelling virus transmission processes in complex networks lies at the core of our understanding about infectious diseases [1]. At the same time, the spreading of information, computer virus, and advertisement have a similar transmission effect to the epidemiological virus. Thus the effect of computer safeguarding [2], viral marketing [3] can be evaluated via population diffusion. Since many population diffusion phenomena could be similarly treated as an epidemic metaphor, we focus on virus diffusion processes in this article, which also aligns with the urgent need for understanding the pandemic of SARS-Cov-2 [4].

A continuous time virus diffusion process in a network is conventionally described by the Markov chain theory [5–7]. On the other hand, the Markov chain theory is widely used in understanding stochastic processes of physical thermal dynamics and statistical physics [8, 9], which are described fundamentally by quantum mechanics with an effective Hamiltonian. Here we use the term effective to describe that the Hamiltonian is a good approximation to a more fundamental theory. For example, Hubbard model is widely used to study the strong correlation effect in electron-nucleon systems [10], and it is a good approximation to describe more fundamental and complicated electron-electron Coulomb interaction. Back to epidemiology, regardless of the complicated underlying biological and ecological mechanism, one may ask whether we can find an effective Hamiltonian to describe the virus transmission, of which the evolution follows Schrödinger equation. Equipped with this effective Hamiltonian, we expect that the classical Markov chain would naturally emerge.

Simulating virus diffusion with the Markov approach is exact. However, the number of elements in the infinitesimal generator of the Markov chain is exponentially large. Thus the analysis is limited to very small graphs [1]. For this reason, analyzing the evolution behaviour of virus diffusion in large networks relies on simplified approaches such as the mean-field [11] and generating function approach [1]. On the other hand, if we have an effective Hamiltonian describing virus diffusion, we can simulate this Hamiltonian on quantum computers, deriving unquenched and dynamic properties of virus diffusion beyond the mean-field and generating function approach.

Modelling virus transmission, particularly the transmission of SARS-Cov-2 with quantum mechanical tools, has been investigated in previous works [12, 13]. However, a clear connection between the spin system and virus interactions and an explicit analysis of the evolution behaviour of the effective Hamiltonian still remains open questions. Here, we propose a thermal dynamic spin Hamiltonian to simulate virus transmission. We provide both theoretical analyses using perturbation theories and numerical studies of the evolution based on the work by Terhal and DiVincenzo [14]. The evolution of the proposed Hamiltonian matches well with the classical Markov process and resembles the epidemiological susceptible, infectious (SI) model. The results demonstrate the rationality of simulating the diffusion process with quantum mechanical processes.

As an example, we implement our model to describe the transmission of SARS-Cov-2 variant Omicron [15]. We utilize secondary attack rate (SAR) [16] and basic reproduction number (BRN) [17, 18] as epidemiological inputs to determine the parameters of the proposed effective Hamiltonian. Then, we use the Hamiltonian to make predictions about more complicated situations. Our numerical simulations are mostly done on the quantum simulator, which shows the possibility of carrying out the simulation on modern realistic quantum computers.

This article is organized as follows. In section II, we
review the phenomenological SI model to describe the virus transmission within local contact structures and introduce the parameters concerned in epidemiology. In section III, we map the virus population distribution onto a spin-lattice system, and the thermal dynamic Hamiltonian simulating the diffusion process is proposed. In section IV, according to numerical and perturbative analyses, we show that the evolution behaviour of the proposed Hamiltonian coincides with the SI model. A practical approach to determining the Hamiltonian parameters is also exhibited. Finally, we discuss some limitations and possible extensions of the model in section V.

II. PHENOMENOLOGY

In epidemiology, the phenomenological susceptible and infectious (SI) model is usually used to predict the evolution of an epidemic [13, 19]. The human population in the model is divided into two compartments where S represents the susceptible population and I represents the infected population. These two quantities are related by two coupled first-order differential equations and can be solved with some given initial conditions. For example, the susceptible population and the infected population are related by

\[
\frac{dS}{dt} = -\beta \kappa IS,
\]

where \(\beta\) is the contact transmission risk, and \(\kappa\) is the average number of contacts between each individual in the two parts of the people. The contact transmission risk characterizes the transmissibility of the virus itself. On the other hand, the average number of contacts reflects the strength of human association, which is influenced by, for example, geographical distance or whether people wear masks. For the more generalized problem of population diffusion of another object, \(\beta\) could characterize the diffusive ability of the object, and \(\kappa\) characterizes the environmental factors.

We consider a more microscopic and short-term description compared with the SI model. In the SI model, \(S\) and \(I\) represent the total number of the corresponding population. Here, we assign each community a number \(i\), representing the community’s susceptible population. We consider the initial time evolution of SI dynamics such that the exposed individuals have been infected by the disease but cannot yet transmit it. Thus, we ignore the secondary virus transmission between the exposed population and the susceptible one, i.e., the effective infectious population \((I)\) in Eq. (1) can be regarded as a constant. Such simplification leads to differential equations

\[
\frac{dS_j}{dt} = -\beta \sum_{i \in I} \kappa_{ij} I_i S_j, \quad \forall j \in S,
\]

where \(I\) denotes the set of index patients, \(S\) denotes the set of communities (or the index patient’s household, of which members are all assumed susceptible), and \(\kappa_{ij}\) is the averaged number of contacts in unit time between the patient \(i\) and the nearby susceptible population in the community \(j\). In our simulation, we will assign each index patient with one spin so that \(I_i = 1\) for all infectious sites. Since \(I_i\) is independent of time, this differential equation can be solved explicitly

\[
P_j(t) \equiv \frac{S_j(t)}{S_j(0)} = \exp \left\{ -\beta \sum_{i \in I} \kappa_{ij} I_i t \right\}, \quad j \in S,
\]

where \(P_j(t)\) represents the percentage of population at site \(j\) surviving from infection after time \(t\). Note that this percentage can also be interpreted as the possibilities of survival for each individual at site \(j\). Thus according to the above formula, we expect an exponential decay behaviour on the survival probability of each susceptible community. The decay rate is a function of \(\beta\) and \(\kappa_{ij}\), which denote the property of the virus itself and the contact pattern between the index patients and their nearby household members or communities. We will see their correspondence to the parameters in the Hamiltonian proposed in the next section.

III. THERMAL DYNAMIC MODEL

We aim to simulate the population diffusion on a 2-dimensional plane. The plane is discretized into 2-dimensional lattice sites \(\Lambda\), for which sites represent the possible habitats of the object. In case of virus transmission, one site could represent a particular person, a group of specific people, or a community. We assign one spin at each site and map it to one qubit. The two eigenvectors of the Pauli-Z operator represent two extreme cases

All infected \(\Leftrightarrow |1\rangle\), \ All susceptible \(\Leftrightarrow |0\rangle\) .

The ket notation \(|(\cdot)\rangle\) represents a quantum state. We are interested in the survival possibility of each site, which can be measured by introducing an observable \(Z_i\) for each lattice site \(i \in \Lambda\). This observable is diagonal in the \([0]\,, [1]\) basis

\[
Z_i |0\rangle = |0\rangle \quad Z_i |1\rangle = -|1\rangle.
\]

Thus the survival possibility \(P_i\) can be estimated utilizing the expectation value of \(Z_i\)

\[
P_i(t) = \frac{1 + \langle Z_i \rangle_t}{2},
\]

where the expectation value \(\langle Z_i \rangle_t\) depends on the system’s state at time \(t\). The state is described by density operator \(\rho(t)\) and the expectation value is measured by

\[
\langle Z_i \rangle_t = \text{Tr}(\rho(t)Z_i).
\]

Additionally, we can estimate the infection probability at site \(i\) as \(1 - P_i(t)\).
The problem of population diffusion is a classical problem, which is very different from the quantum mechanical one, e.g., a classical initial state cannot evolve to a superposition like $|\text{infected}\rangle + |\text{susceptible}\rangle$, yet allowed in quantum mechanics. In Appendix B, following Ref. [14], we explicitly distinguish the evolution’s classical and pure quantum parts. We proved that in case: (1) The system is accompanied by a weakly coupled heat bath. (2) The initial state is the system’s energy eigenstate, and the measured operator is a linear combination of the projectors onto the system’s energy eigenstate. Then the measured expectation values will be dominated by the evolution’s classical part. For this reason, we can introduce quantum mechanics to simulate the classical diffusion problem.

We use quantum evolution to simulate the dynamical process of diffusion. The evolution Hamiltonian is constructed as follows. First, as mentioned above, the measured operator should be the projector onto the system’s Hamiltonian eigenstates. In simulation of population diffusion, we measure the operator $Z_i$ that can be decomposed into projector $Z_i = |0\rangle \langle 0| - |1\rangle \langle 1|$. It inspires us to introduce the Ising-type system Hamiltonian (tensor product symbols $\otimes$ will be omitted throughout)

$$H_s = -\sum_{i \in I} \sum_{j \in S} \gamma_{ij} Z_i^s Z_j^s,$$

where $\gamma_{ij} > 0$ represents the inter-system coupling strength of the pair $(i, j)$. It should characterize the contact strength between the two sites, thus corresponding to $\kappa_{ij}$ introduced in the previous section. Because we do not consider secondary transmission, couplings among susceptible sites are ignored. This Hamiltonian corresponds to a ferromagnetic spin system. Its two degenerate ground states have all spins aligned to the same directions, i.e., all $|0\rangle$ or all $|1\rangle$. These ground states naturally correspond to the final states of virus transmission after an infinitely long dissipative process. Note that if no index patient is introduced, the final state should be all $|0\rangle$. Otherwise, the final state should be all $|1\rangle$, which means all the people are infected.

Secondly, as explained above, we hope the evolution is dissipative on the system. Also, notice that the diffusion process is a thermal dynamic process accompanied by entropy growth. While if we only evolve an isolated system according to Schrödinger equation, the evolution is reversible without entropy growth. Thus we introduce a heat bath and interaction between the bath and the system. The complete Hamiltonian reads

$$H = H_s \otimes I_{\text{bath}} + I_{\text{sys}} \otimes H_b + \lambda H_{sb},$$

where $H_s$ is the Hamiltonian of the target system, $H_b$ is the Hamiltonian of the heat bath, and $H_{sb}$ describes the interaction between the system and the bath. The interaction strength is characterized by a system-bath coupling strength $\lambda$. $I_{\text{bath}}$ and $I_{\text{sys}}$ are identity operators on the corresponding Hilbert space. This Hamiltonian is inspired by the one introduced in [14], where the authors have shown that if we have an infinitely large heat bath with temperature $T$, the system will evolve to the Gibbs state

$$\rho_{\text{sys}} = \frac{e^{-H_s/(k_B T)}}{Z}, Z = \text{Tr}(e^{-H_s/(k_B T)})$$

after an infinitely long time. The Gibbs state has the same temperature $T$ as the heat bath. Here $k_B$ is the Boltzmann constant.

Considering the diffusion model, we can determine the temperature. Note that if we take $T \to 0$, the Gibbs state (10) is reduced to the ground state of system Hamiltonian $H_s$. As mentioned above, the ground state is what we expect after evolving infinitely long time. Thus in the evolution simulation, the bath temperature will be set to zero. In the numerical simulation, it is realized by resetting the bath’s spins to its ground state after a specific time interval.

It is left to choose an explicit form of the bath Hamiltonian and the interaction. Theoretically, they could be arbitrary, leading to the system’s thermalization. So they are chosen mainly considering the simplicity of computation and analysis. The bath Hamiltonian is determined as follows. We hope the thermalization for the system Hamiltonian is as fast as possible. It can be shown that the most rapid thermalization can be achieved if the energy differences in the system have the corresponding ones in the heat bath, as mentioned in [14], also see Appendix C. It is physically reasonable. In that case, the energy emitted or absorbed by the system have the corresponding sink or source in the heat bath, so the system and bath resonate and interact intensively. Thus a natural choice of the bath Hamiltonian has the same form as the system Hamiltonian

$$H_b = -\sum_{i \in I} \sum_{j \in S} \alpha_{ij} Z_i^b Z_j^b,$$

where $Z_i^b$ is the Pauli-Z operator on the bath Hilbert space and $\alpha_{ij} > 0$ is a positive real number. With this form, the most rapid thermalization can be achieved by setting $\gamma_{ij} = \alpha_{ij}$. Then, we hope the bath coupling reflects the contact transmission risk $\beta$ of a given kind of virus, which should be uniform across all spins. Thus all the bath couplings should be identical, which leads to

$$H_b = -\alpha \sum_{i \in I} \sum_{j \in S} Z_i^b Z_j^b,$$

Finally, we determine the form of the interaction Hamiltonian. We hope the interaction Hamiltonian is local and uniform for all spin sites. With locality, the Hamiltonian can be efficiently simulated on current quantum devices. The uniformity requirement is for the same reason as the one on the bath Hamiltonian. Thus we choose the interaction Hamiltonian reads

$$H_{sb} = -\sum_{i \in I \cup S} X_i^s X_i^b,$$
heat bath, and the subscripts
ysis with the Hamiltonian introduced in the last section.
the communities for more complicated cases.
parameters, we can compute the survival possibilities of
the community environment. After determining the free
actions are local and thus can be simulated efficiently
number of elements in the set. All the spin inter-
where \( X_i^s, X_i^b \) are Pauli-X operators acting on Hilbert
space of the system site \( i \) and the bath site \( i \), respectively.
Pauli-X flips the computational basis \( |0\rangle \leftrightarrow |1\rangle \).

In summary, we propose a thermal dynamic Hamiltonian to simulate the virus transmission. The Hamiltonian reads

\[
H = - \sum_{i \in I} \sum_{j \in S} \gamma_{ij} Z_i^s Z_j^s - \lambda \sum_{i \in I \cup S} X_i^s X_i^b - \alpha \sum_{j \in S} Z_j^b Z_j^b,
\]

(14)

where \( \gamma_{ij}, \lambda, \alpha \) are all positive real numbers. The superscripts \( s, b \) denote the Hilbert space of the system or heat bath, and the subscripts \( i, j \) denote the infectious and susceptible site, respectively. A visual demonstration of spins and their connection with Pauli operators is shown in figure 1. The total number of qubits required to simulate this problem is \( 2 \times (|I| + |S|) \), where \(| \cdot |\) denotes the number of elements in the set. All the spin interactions are local and thus can be simulated efficiently on near-term quantum computers. The free parameters \( \gamma_{ij}, \lambda, \alpha \) are determined by the properties of the virus and the community environment. After determining the free parameters, we can compute the survival possibilities of the communities for more complicated cases.

IV. NUMERICAL SIMULATION AND PARAMETERS DETERMINATION

We have numerical simulation and perturbative analysis with the Hamiltonian introduced in the last section.

The numerical simulation is carried out on the quantum simulator using the Qiskit SDK [20], and we use the Trotter decomposition to decompose the Hamiltonian evolution into several Trotter steps. Each Trotter step evolves a small time duration denoted by \( \delta t \). See more details in Appendix A. The infectious and susceptible sites are distinguished in the initial state by assigning \(|1\rangle\) to the former and \(|0\rangle\) to the latter. Additionally, as the index patients are always infectious during the evolution, we reset the index patients to \(|1\rangle\) once after a specific time interval \( \Delta t \). The time interval \( \Delta t = 1 \) is adopted in all the numerical experiments.

Before simulating the full Hamiltonian Eq. (14), we focus on cases where only one index patient is introduced. We rewrite the Hamiltonian as

\[
H = - \sum_{j > 0} \gamma_{0j} Z_0^s Z_j^s - \lambda \sum_{j > 0} X_j^s X_j^b - \alpha \sum_{j > 0} Z_j^b Z_j^b,
\]

(15)

where we allocate site 0 to the patient. Numerically, we find the estimated survival probabilities \( \hat{P}_j(t) \) have single-exponential decay behaviour depending on time \( t \) at all the susceptible sites, like the curves shown in figure 3a, which plot survival probabilities of site \( j = 1 \). This behaviour is consistent with phenomenological prediction in Eq. (3). The exponential decay behaviour is common when we talk about thermalization, such as the longitudinal and transverse relaxation time \( T_1, T_2 \) [21] describing the lifetime of single qubits. The behaviour is also supported by simplifying the quantum evolution into a stochastic process as written in Appendix C. With these observations, we define the thermalization rate \( \Gamma_j \) of site \( j \)

\[
\Gamma_j \equiv - \frac{\partial \hat{P}_j(t)}{\partial t} \bigg|_{t=0}.
\]

(16)

Note that in case the survival probability has single-exponential behaviour, we have \( \hat{P}_j(t) = e^{-\Gamma_j t} \). In the application of virus transmission, the thermalization rate is also called infection rate. This thermalization rate is determined by the inter-system coupling \( \gamma_{ij} \) and system-bath coupling \( \lambda \). For example, In Appendix C, we show that in case there is one infectious site and one susceptible site \( j \), the thermalization rate of the susceptible site is given by

\[
\Gamma_j \approx \lambda^2 \Delta t \left( \frac{\sin((\gamma_{0j} - \alpha)\Delta t)}{(\gamma_{0j} - \alpha)\Delta t} \right)^2,
\]

(17)

where \( \gamma_{0j} \) is the inter-system coupling between the index patient and site \( j \). \( \Delta t \) is the resetting time interval.

The inter-bath coupling \( \alpha \) can be determined utilizing the perturbative analysis. We hope the inter-system coupling \( \gamma_{ij} \) reflects the average number of contacts between
the index patient and the neighbours. Specifically, we hope the infection rate is infinitely slow as $\gamma_{ij} \to 0$. According to the perturbative analysis, it can be achieved by setting $\alpha \Delta t = k \pi, k = 1, 2, \ldots$, which holds up to order $O(\lambda^2)$ (see more details in Appendix C). For the monotonicity between the map of $\gamma_{ij}$ and the infection rate, we choose $k = 1$. In the numerical simulations, we further adopt $\Delta t = 1$ so that $\alpha = \pi$.

According to the above analysis, the system-bath coupling $\lambda$ and inter-system coupling $\gamma_{ij}$ can be determined given some phenomenological inputs. The contact transmission risk $\beta$ and the average number of contacts $\kappa_{ij}$ seem to be the best choice due to their resemblance to $\lambda$ and $\gamma_{ij}$. However, these two phenomenological inputs are still hard to be investigated in practice. Instead, as the infected population can be calculated numerically equipped with the Hamiltonian, we utilize secondary attack rate and basic reproduction number (defined in the following subsections). These two inputs are more available in the epidemiological investigation than $\beta$ and $\kappa_{ij}$. The following subsections demonstrate how they determine the system-bath and inter-system coupling. As an example, we use data describing the transmissibility of SARS-Cov-2 variant Omicron in numerical simulations.

### A. Determination of the system-bath coupling

The secondary attack rate (SAR) is defined as the number of non-index susceptible household members with a positive test result within seven days after the sample date of the index case, divided by the total number of non-index household members [16]. Thus it is the household infection probability $1 - \bar{P}_{ij}$ ($t = 7$). In reference [16], the authors reported SAR for Omicron variants of SARS-Cov-2 in Norwegian households where $\text{SAR}_{\text{Omicron}} = 25.1\%$. We use this data to determine the system-bath coupling in the thermal dynamic Hamiltonian.

Now we consider what exactly lattice geometry is needed to estimate SAR numerically. SAR is a macroscopic quantity that is the statistical results from thousands of different community environments. To utilize

Figure 2. The map and population of a typical community environment. The community environment has five sites: one for the index patient (red dot, site 0), one for the patient’s household members (orange plate, site 1), and three for communities (rectangles, site 2, 3, 4). The population in each community is assumed to be uniformly distributed.

Figure 3. Single-exponential decay behaviour of the survival probability and the determination of system-bath coupling. (a) Numerical result for survival probabilities of the household site 1 as a function of time, where the time is in the day unit, under different system-bath coupling $\lambda$. The curves exhibit single-exponential decay $\exp(-\Gamma t)$, where $\Gamma$ is the infection rate. The simulation is carried out under two lattice size: $|I| = 1, |S| = 4$ and $|I| = 1, |S| = 1$. Their convergence shows the lattice-size independence of the survival probabilities. The vertical black-dashed line labels the seventh day. (b) The infection rate $\Gamma$ of the household site as a function of coupling $\lambda$, under log-log plot. $\Gamma$ can be well fitted by a straight line with the slop $1.955(45) \simeq 2$. It indicates the quadratic behaviour $\Gamma \simeq \lambda^2$. With the input $\text{SAR}_{\text{Omicron}} = 25.1\%$, we find $\lambda = 0.201(16)$. The number in the parentheses is the fitting error of the last two displayed digits.
the statistical result, we need to simulate a typical community environment that can represent the majority. In our simulation, we assume a typical community environment consists of five sites: one for the index patient (at site 0), one for the patient’s household members (at site 1), and three for communities (at sites 2, 3, 4). So that we have $|I| = 1, |S| = 4$, and their geographical distribution is shown in figure 2. In an actual application, the typical community environment should be large enough so that people outside the environment are almost impossible to get infected.

We estimate the survival probability of the household site. Notice that the susceptible household members are the most intimate with the index patient. Thus the infection rate of the household site 1 should be the largest among the other sites. As mentioned in the last section, this corresponds to setting inter-system coupling $\gamma_{01} = \alpha = \pi$ so that the system and bath resonate. Then, by tuning system-bath coupling $\lambda$, we can find a suitable value that satisfies the requirement of the input SAR. The numerical simulation is shown in figure 3. The quantum evolution is carried out using first-order Trotter decomposition with $\delta t = 0.01$ (see more details in Appendix A). The expectation value is measured by running the quantum circuit 4096 times for each data point, and the error bar is the statistical error. In the upper panel, the survival probabilities explicitly show the single-exponential decay behaviour for various values of $\lambda$. As SAR requires, the vertical black-dashed line labels the seventh day after introducing the index patient. We plot the infection rate in the lower panel as a function of $\lambda$. Results in figure 3 have been checked using the fourth-order Runge-Kutta method as shown in Appendix E.

In the simulation with lattice size $|I| = 1, |S| = 4$, the other three inter-system couplings $\gamma_{0j}, j = 2, 3, 4$ are chosen randomly from $(0, \pi]$. We can choose these three couplings arbitrarily because, numerically, we find these three couplings have no impact on the survival probabilities of the household site. The survival probabilities have no obvious change even if only one infectious site and one household site are simulated ($|I| = 1, |S| = 1$), as shown in figure 3a, i.e., The survival probabilities have no dependence on the lattice size. This independence behaviour simplifies the perturbative analysis as we can apply the results from the system of $|I| = 1, |S| = 1$ to that of $|I| = 1, |S| = 4$. Regarding the virus transmission, the behaviour is reasonable as the household survival probabilities should not depend on the community environment.

To determine the system-bath coupling from the input SAR, in figure 3b, we find the household infection rate and the system-bath coupling are related by

$$\Gamma \simeq \lambda^2. \quad (18)$$

Here we use the symbol $\simeq$, because the exact relation should be extracted from the fitting result given in figure 3b. This relation is consistent with our perturbative result for system $|I| = 1, |S| = 1$ as shown in Eq. (17), by adopting the resetting time $\Delta t = 1$ and $\gamma_{01} = \alpha$. On the other hand, the input SAR would also indicate a phenomenological infection rate

$$\Gamma_{\text{SAR}} \equiv -\frac{1}{t} \ln(1 - \text{SAR}) \bigg|_{t=7} \quad (19)$$

according to the single-exponential behaviour of survival probability. Thus we can solve for $\lambda$ utilizing the fitting result in figure 3b by requiring $\Gamma = \Gamma_{\text{SAR}}$. Thus determined the system-bath coupling $\lambda = 0.201(16)$ according to the input $\text{SAR}_{\text{Omicron}} = 25.1\%$.

The determined system-bath coupling has a phenomenological interpretation. As only one index patient was introduced, the SI model would predict the infection rate of the household site 1. Utilizing Eq. (3) and the definition of infection rate Eq. (16), one finds

$$\Gamma_{\text{SAR}} = \beta \kappa_{01} \quad (20)$$

where $\kappa_{01}$ denote the average number of contacts between the infectious site 0 and the household site 1. Thus we find

$$\lambda^2 \simeq \beta \kappa_{01}. \quad (21)$$

It indicates that the system-bath coupling should be larger for higher contact transmission risk and a larger average number of contacts between the index patient and the household members.

### B. Inter-system coupling and contact pattern

We turn to determine the inter-system coupling $\gamma_{ij}$ by Basic Reproduction Number (BRN), usually denoted by $R_0$. BRN is the average number of susceptible populations generated by one contagious person [17]. It is an essential epidemiological indicator to characterize the virus transmissibility. If $R_0 < 1$, the virus will decline and eventually disappear. If $R_0 = 1$, it will stay alive but will not be an epidemic. If $R_0 > 1$, it will cause an epidemic or even a pandemic. In [22], the author collected the data from 1 November 2021 to 9 February 2022 and estimated that the average BRN of Omicron is around 9.5. Additionally, we assume the index patient get non-contagious as soon as the patient experience symptoms. Thus we treat $R_0$ as the total number of infected population by one index patient after incubation. The incubation period of Omicron is around four days, according to [23].

The contact pattern over the communities determines the inter-system coupling. Their relationship is not straightforward. Notice that as the infection rate is closely connected with the numerical results and has a clear phenomenological interpretation, it can be regarded as a bridge to connect the inter-system coupling and the contact pattern. We first find the relationship between the infection rate and the contact pattern.
Next, we find the relationship between the infection rate $\Gamma_j$ and the inter-system coupling $\gamma_{0j}$. Because the infection rate is independent of the lattice size, as explained in the previous subsection, we only need to consider a system consisting of two spin sites $|I| = 1, |S| = 1$. Thus we can take the relationship according to the perturbative formula (17) as a good approximation. This formula has been checked numerically in system $|I| = 1, |S| = 1$, see figure 4. It plots infection rate as a function of inter-system coupling $\gamma$. In this numerical simulation, we take system-bath coupling $\lambda = 0.201$ derived previously. We see that $\Gamma$ is peaked at $\gamma = \pi$ and the curve is well fitted by a sinc-function with fitted parameters $\lambda = 0.1990(5)$ and $\Delta t = 1.014(5)$. They are consistent with perturbative formula predicting $\lambda = 0.201$ and $\Delta t = 1$. It is shown explicitly that as $\gamma \to 0$, the infection rate becomes very small (yet not tend to zero for tiny $\gamma$ as we discuss later). At the point $\gamma = 0$, we find the survival probability does not have single-exponential decay behaviour. It is because, at $\gamma = 0$, the Ising type Hamiltonian (8) has a transition from ferromagnetism to paramagnetism, leading to a singularity in the Hamiltonian evolution (see Appendix C for more details). This singularity does not need to be bothered because if a spin is regarded as having no coupling with the system concerned, we can exclude the spin out of the system.

In the subplot of figure 4, we plot the behaviour of infection rates as inter-system couplings are small. There is a plateau as inter-system coupling tends to zero. It indicates that the thermal dynamic model may not be able to simulate the evolution with a low infection rate. This problem is discussed in detail in Appendix D, where a solution utilizing time rescaling is provided.

Combining Eq. (17) Eq. (22) and Eq. (21), we find the phenomenological interpretation of inter-system coupling $\gamma$:

$$
\left( \frac{\sin(\gamma - \alpha)}{\gamma - \alpha} \right)^2 \approx \frac{\int S_j d^2r \exp \left(-\frac{|\vec{r} - \vec{r}_0|^2}{2\sigma^2} \right)}{\int S_j d^2r}. \tag{23}
$$

Here we have chosen $\Delta t = 1$. Thus the deviation $|\gamma - \alpha|$ indicates the distance of the community to the index patient. For the monotonicity of the map $\sigma \to \gamma$, we choose $\gamma$ within the domain $(0, \alpha]$.

C. Determination of the inter-system coupling and simulation for more index patients

We briefly summarize the strategy for the Determination of the inter-system coupling. According to Eq. (23), determining inter-system couplings is equivalent to determining the distance scale $\sigma$. Given a detailed map of the typical community environment, the location of the index patient, and the population at each site, we can try several values of $\sigma$, which gives several sets of inter-system couplings $\{\gamma_{0j}\}$. Thus the total number of
We carry out more numerical experiments based on the determined Hamiltonian. First, we check the total number of infection populations with the communities setting 2. As shown in figure (6). We find the total number of the infected population is 9.58(20) on the fourth day, which is consistent with the requirement of the BRN of Omicron. In the figure, we also use the fourth-order Runge-Kutta method to carry out the Hamiltonian simulation. The fourth-order Runge-Kutta method has less systematic error than the quantum simulator. We see that the evaluated survival probabilities from the quantum simulator are consistent with those of the fourth-order Runge-Kutta method within the error of statistics. The details of the Runge-Kutta method are shown in Appendix E.

We can simulate the model having more than one infectious site utilizing the thermal dynamic Hamiltonian 14. Here, the inter-bath coupling $\alpha$ should differ from the model having one infectious site. Because according to the phenomenological Eq. (3), when a susceptible site interacts with multi-infectious sites, the infection rate should be a linear summation of infection rates from each infectious site. To fulfill this requirement as good as possible, we adopt the bath coupling $\alpha = \pi/|I|$ (see Eq. (C44). Here we take $\Delta t = 1$). However, the linearity requirement of the infection rate can not be satisfied exactly. Because the infection rate $\Gamma$ as a function of inter-system coupling $\gamma$ is non-linear (see more details in Appendix C, especially Eq. (C43)). Thus the evaluated infection probabilities are consistent with phenomenology only qualitatively. In figure 7, we plot the infection probabilities of each community on different days, introducing one and two index patients, respectively. We see that the infection probabilities in the case of introducing two patients are larger than in introducing one. The inter-bath coupling is $\alpha = \pi/2$ for the former case and $\alpha = \pi$ for the latter. We take the system-bath coupling $\lambda = 0.201$ for both cases. We assume that all the index patients have the same activity range derived previously. Thus the inter-system couplings can be derived utilizing the method mentioned above.
Figure 7. Simulation of infection probabilities on different days, with one (upper row) and two (lower row) index patients introduced. The red points label the position of the index patients. The rectangles denote the communities. We assume all the patients’ activity ranges follow the same Gaussian distribution with \( \sigma = 65 \). Each community’s population and geometry settings follow figure 2. We see the infection rates rise by introducing one more patient.

V. DISCUSSION

In this work, we proposed a thermal dynamic model to describe the dynamic virus transmission process in networks. We analyzed the evolution behaviour of the Hamiltonian perturbatively and numerically using the quantum simulator. We proved that the quantum evolution reduced to a classical Markovian process and the behaviour resembles a simplified SI model. Our work demonstrates the possibility of simulating classical virus transmission with quantum computers. Thus the whole Markovian evolution with an exponential large infinitesimal generator can be computed in principle.

As a demonstration, we have made some simplifications and assumptions in the thermal dynamic model as follows

- We consider the virus transmission among communities in the early period. Thus the recovered, deceased and immune populations are not considered. For the same reason, secondary transmission among susceptible sites is also ignored.

- All the populations are assumed equally susceptible. Thus all the spin sites have identical contact transmission risks, and the contact transmission risk does not depend on time.

- We assume the populations in all the communities are static and uniformly distributed.

- We assume the index patient’s activity range follows isotropic 2-d Gaussian distribution. Thus, the average number of contacts between the infectious and susceptible sites depends only on their geographical distance.

We have the Hamiltonian simulation on small spin-lattice systems with these assumptions, which helps to determine the parameters in the Hamiltonian and then carry out simulations in more complicated cases, such as introducing one more patient.

The thermal dynamic Hamiltonian could have many variants to simulate the virus transmission. For example, the index patient may often commute between his working place and home. Thus the Hamiltonian could depend on time to simulate a more extended displacement of the index patient. In the same way, the time-dependent Hamiltonian could consider the mobility of the population in the communities. The Hamiltonian can also be modified to describe the process including secondary transmission among the susceptible sites. However, naively adding Pauli-\(ZZ\) interactions between every two susceptible sites is not a good strategy. In reality, if we add contact between two susceptible sites, the infection rates of both communities should get more prominent. It is not the case if we simulate the Hamiltonian with full \(ZZ\) couplings, where we find both infection rates getting less prominent. It is because in the Ising-type system Hamiltonian \(H_s = -\sum_{ij} \gamma_{ij} Z_i Z_j\), the two configurations \(|0\rangle\) and \(|1\rangle\) are symmetric in energy. However, in reality, index patients increase the infection rate of nearby communities, whereas the non-index population do not decrease the infection rate of nearby communities correspondingly. This asymmetry was broken by the resetting operation on the infectious sites in our numerical simulation but is unsuitable for susceptible sites. Thus, to simulate secondary transmission, the symmetry between \(|0\rangle\) and \(|1\rangle\) has to be broken by, for example, introducing local magnetic terms into the Hamiltonian. The magnetic terms explicitly break \(|0\rangle\) and \(|1\rangle\) symmetry at the cost of more parameters to be determined. The more complicated simulation is left for further work.

The problem of non-linearity of the infection rate can also be solved by introducing alternative system-bath interaction Hamiltonian. A proper system-bath interaction Hamiltonian would make \(O(\lambda)\) term in the evolution ex-
pansion non-vanish so that the linearity of the infection rate could be preserved.

The thermal dynamic Hamiltonian (14) is interesting in its own right. It is simple enough that we can analyze its evolution according to perturbation theory. It could provide some insights for interpreting the classical diffusion process of a lattice system accompanied by a heat bath from the point of view of quantum mechanics.

VI. ACKNOWLEDGEMENTS

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Appendix A: Preliminaries on quantum simulation

In this appendix, we introduce the details of our numerical simulation and some mathematical notations used in the following appendixes. Quantum simulation gets well known due to Richard Feynman [25]. It encodes a quantum system on several quantum bits (or qubits). Qubits are the basic building block of modern quantum computers. One qubit comprises a 2-dimensional Hilbert space that can be represented with computational basis $|0\rangle$ and $|1\rangle$, as we used in the main text. $N_q$ qubits comprise a $2^{N_q}$ dimensional Hilbert space following the principle of tensor product of many-body quantum systems. We denote this Hilbert space as $\mathcal{H}$.

We briefly introduce how a quantum system evolves on a universal quantum computer. To encode a quantum system on qubits, we need to find a Hamiltonian $H$ to evolve a quantum state according to the Schrödinger equation

$$|\psi(t)\rangle = e^{-iHt}|\psi(0)\rangle. \quad (A1)$$

Here we assume $H$ is independent on time. The Hamiltonian is written as a linear combination of multi-qubit Pauli operators

$$H = \sum_m h_m \sigma_m, \quad (A2)$$

where each $\sigma_m$ is a tensor product of single-qubit Pauli operators $\sigma_m = \sigma_m^1 \ldots \sigma_m^{N_q}$, $\sigma_m \in \{I, X, Y, Z\}$ and $h_m$ is a real number. However, universal quantum computers cannot evolve this Hamiltonian as a whole. Instead, the evolution can be decomposed into several basic evolution blocks. The basic evolution blocks used in our numerical simulation are summarized below. Here we choose the basic gate as two-qubit CNOT gate [21].

\begin{align}
&e^{-i\frac{\theta}{2}ZZ} = e^{-i\frac{\theta}{2}Z} \\
&e^{-i\frac{\theta}{2}XX} = H e^{-i\frac{\theta}{2}Z} H \\
\end{align}

With these evolution blocks, the Schrödinger evolution can be carried out using the first-order Trotter decomposition (or higher order Suzuki-Trotter decomposition [26] for higher accuracy)

$$e^{-iHt} = \left(\prod_m e^{-i\delta t h_m \sigma_m}\right)^N + \mathcal{O}(q^2/N), \quad (A5)$$

where $N$ is the total number of Trotter steps to evolve for time $t$, $\delta t \equiv t/N$ denotes the time length of one Trotter step. The systematic error due to Trotter decomposition can be controlled by increasing $N$ for a given evolution time $t$. In our numerical simulations, we choose Trotter step $\delta t = 0.01$ if not specified.

The Schrödinger equation describes an isolated system’s evolution. For a non-isolated system’s evolution, the description is more complicated. Specifically, we consider the system accompanied by a heat bath. Their state as a whole is described by density operator $\rho_{sb}$, which is a non-negative, trace-1 linear operator [27]. The system is also described by density operator $\rho_s$ in Hilbert space...
\( \mathcal{H} \), which is given by
\[
\rho_s = \text{Tr}_b(\rho_{sb}), \tag{A6}
\]
where \( \text{Tr}_b \) is the partial trace operation over the bath Hilbert space.

Assume the whole state \( \rho_{sb} \) still follows the Schrödinger equation. The system’s evolution alone is a thermal dynamic evolution, which can be regarded as a map from one density operator to another. Generally, such a map is a Completely Positive and Trace Preserving (CPTP) map that can be represented by
\[
S(\rho_s) = \sum_i A_i^\dagger \rho_s A_i. \tag{A7}
\]
We also call this map a quantum channel. As a quantum channel is also a linear map on the space of density operator, we can simplify this representation by introducing Hilbert-Schmidt space denoted by \( \mathcal{B}(\mathcal{H}) \), where each density operator is represented by a \( 2^{2N} \) column vector
\[
\rho_s \rightarrow |\rho_s\rangle. \tag{A8}
\]
Hilbert-Schmidt space is equipped with an inner product \( \langle \rho_1 | \rho_2 \rangle = \text{Tr} \left( \rho_1^\dagger \rho_2 \right) \), which possesses the physical meaning of the possibility of measuring state \( \rho_1 \) given state \( \rho_2 \). We call the column vector \( |B\rangle \) superket and the dual vector \( \langle A | \) superbra. The superket can be expanded with an orthogonal basis which is induced by an orthogonal basis \( \{|m\rangle\} \) (like the energy eigenstates) in Hilbert space \( \mathcal{H} \)
\[
|\phi^{(0)}_{nm}\rangle \equiv |n\rangle \langle m|). \tag{A9}
\]
The meaning of superscript \( (0) \) will get clear later in Appendix B. Thus Eq. (A7) can be represented by
\[
\langle \phi^{(0)}_{nm} | S | \rho_s \rangle = \sum_{kl} S_{nm:kl} \rho_s_{kl},
\]
\[
S_{nm:kl} = \sum_i A_{nk}^i A_{ml}^i, \tag{A10}
\]
where \( \rho_s_{kl} = \langle k | \rho_s | l \rangle \), \( A_{nk}^i = \langle n | A^i | k \rangle \) and \( * \) denote complex conjugate. Here we use the definition of inner product in Hilbert-Schmidt space, i.e., \( \langle \phi^{(0)}_{nm} | S | \rho_s \rangle = \text{Tr}(m \langle n | S(\rho_s)) = \langle n | S(\rho_s) | m \rangle. \)

Another key element in our simulation is resetting operation, which can also be described using the language of density operator and partial trace. Consider a system composed by subsystems A and B. It is described by a density operator \( \rho \). If system A is reset to a pure state \( |k'_A\rangle \), the whole system’s density operator becomes
\[
\rho' = |k'_A\rangle \langle k'_A| \otimes \rho^B \tag{A11}
\]
where \( \rho^B = \text{Tr}_A \rho \) is the reduced density operator on the Hilbert space of the subsystem B.

Resetting can be realized in a quantum circuit if a perfect measurement of a subsystem is possible. Assume \( \{|k_A\}\) is a complete basis in the Hilbert space of system A. Performing a projection measurement \( P_k = |k_A\rangle \langle k_A| I_B \) on the initial density operator \( \rho \), the outcome is
\[
\rho(k) = |k_A\rangle \langle k_A| \otimes \rho^B, \tag{A12}
\]
with the probability
\[
p(k) = \text{Tr} \left( \rho^A |k_A\rangle \langle k_A| \right), \tag{A13}
\]
where \( \rho^A = \text{Tr}_B \rho \) is the reduced density operator on the Hilbert space of system A. After the measurement, according to the measurement result, we transform the outcome state \( |k_A\rangle \) unitarily to \( |k'_A\rangle \) using the quantum gate while keeping \( \rho^B \) unchanged. The final state is
\[
\rho' = |k'_A\rangle \langle k'_A| \otimes \rho^B, \tag{A14}
\]
which is the same as the partial trace outcome (A11).

A typical example is when subsystem A is a single qubit and \( |k'_A\rangle = |0\rangle_A \). The resetting operation of this case can be represented using a quantum circuit
\[
A \quad \overline{\quad} \quad B \quad (0) \tag{A15}
\]
where the first gate is a measurement in \( \{|0\rangle, |1\rangle\} \) basis, and the second gate is an X-gate if the measurement outcome is \( |1\rangle \) while doing nothing if the outcome is \( |0\rangle \).

Appendix B: Perturbative evolution expansion

In this appendix, we briefly review the general formalism of the time evolution of a system with a weakly coupled heat bath. Thus, developed perturbative evolution expansion helps build the relationship between classical diffusion and quantum evolution, which will be discussed in the next appendix. We use the Hamiltonian introduced in [14]
\[
H = H_s \otimes I_{\text{bath}} + I_{\text{sys}} \otimes H_b + \lambda H_{sb}. \tag{B1}
\]
In our simulation, the coupling term \( H_{sb} \) can be formally written as
\[
H_{sb} = \sum_i S^i \otimes B^i. \tag{B2}
\]
The whole system follows the exact Schrödinger evolution
\[
\rho_s \otimes \rho_b \rightarrow e^{-iHt} \rho_s \otimes \rho_b e^{iHt}, \tag{B3}
\]
where \( \rho_s \) and \( \rho_b \) are the system’s and bath’s initial density operators, respectively. \( \beta \) is the inverse temperature of the heat bath. The heat bath is set to be Gibbs state
\[
\rho_b = \frac{e^{-\beta H_b}}{\text{Tr}(e^{-\beta H_b})}. \tag{B4}
\]
Theoretically, thermal evolution requires an infinitely large heat bath, which requires an infinitely large number of qubits. To reduce the computational resource, we couple each system qubit with one bath qubit and reset it to its Gibbs state once after a specific time interval $\Delta t_b$. In our numerical simulation, the time interval is equal to the time interval of resetting the infectious site $\Delta t$. The resetting operation is a good approximation to mimic an infinitely large heat bath and is experimentally realizable on modern quantum devices. In our simulation, since we take $\beta \to \infty$, the Gibbs state reduces to the ground state of $H_b$.

We aim to analyze how systems evolve under the evolution Eq. (B3). The evolution of the system density operator is derived by tracing out the bath Hilbert space

$$S_{\lambda,t}(\rho_s) \equiv \text{Tr}_b(e^{-iH_t\rho_s} \otimes \rho_b e^{iH_t}) \quad \text{(B5)}$$

where $S_{\lambda,t}$ is a CPTP map acting on the initial system density operator $\rho_s$. In reference [14], the authors show that this CPTP map can be treated perturbatively with the small parameter $\lambda$. The map can be expanded by

$$S_{\lambda,t} = S_{t}^{(0)} + \lambda S_{t}^{(1)} + \lambda^2 S_{t}^{(2)} + \lambda^3 S_{t}^{(3)} + \mathcal{O}(\lambda^4) \quad \text{(B6)}$$

where $S_{t}^{(0)}$ is the evolution of the system alone without the coupling of the heat bath $S_{t}^{(0)} = S_{\lambda=0,t}$. In that case, the system’s evolution can be solved exactly by observing

$$S_{t}^{(0)} |\phi_{nm}^{(0)}\rangle = e^{-i(E_n - E_m)t} \langle m | e^{iH_{s}t} | n \rangle |\phi_{nm}^{(0)}\rangle \quad \text{(B7)}$$

where $|n\rangle$ is the eigenstate of the system’s Hamiltonian with eigenvalue $E_n$ satisfying

$$H_s |m\rangle = E_m |m\rangle \quad \text{(B8)}$$

**Classical and pure quantum evolution**

In this subsection, to support our simulation of the classical diffusion process on the quantum computer, we prove that in case: (1) The system-bath coupling $\lambda$ is small. (2) The initial state is the energy eigenstate of the system. (3) The measured operator is a linear combination of the projectors onto the system’s energy states; then, the measured expectation values will be dominated by the classical part of the evolution. Note that due to the linearity of the quantum channel, the latter two conditions are reduced to that the initial state and the measured operators are all like $|n\rangle \langle n| = |\phi_{nm}^{(0)}\rangle \langle \phi_{nm}^{(0)}|$. First, we distinguish between evolution’s classical and pure quantum parts. They can be distinguished explicitly in the zeroth order of the evolution expansion (B6). Note that $S_{t}^{(0)}$ is diagonal under the basis $|\phi_{nm}^{(0)}\rangle$ according to

$$S_{t}^{(0)} = \sum_{mn} e^{-i(E_n - E_m)t} |\phi_{nm}^{(0)}\rangle \langle \phi_{nm}^{(0)}| \quad \text{(B9)}$$

with

$$S_t^{c(0)} \equiv \sum_{m} |\phi_{m}^{(0)}\rangle \langle \phi_{m}^{(0)}|, \quad S_t^{q(0)} \equiv \sum_{m \neq n} \mu_{mn} |\phi_{mn}^{(0)}\rangle \langle \phi_{mn}^{(0)}| \quad \text{(B10)}$$

where we denote $|\phi_{m}^{(0)}\rangle = |\phi_{m}^{(0)}\rangle$ and $\mu_{mn} = e^{-i(E_n - E_m)t}$. If we assume a non-degenerate energy spectrum of the system, the phase accumulation in $S_t^{q(0)}$ can be regarded as a pure quantum effect. It will vanish under an infinitely long time average. The classical part $S_t^{c(0)}$ leaves the energy eigenstates $|n\rangle \langle n| = |\phi_{m}^{(0)}\rangle$ unchanged. If we treat each $|\phi_{m}^{(0)}\rangle$ as a classical state, $S_t^{c(0)}$ can also be regarded as a classical trivial stochastic matrix under the energy eigenbasis, satisfying normalisation condition

$$\sum_{\ell} \langle \phi_{\ell}^{(0)} | S_{t}^{c(0)} | \phi_{k}^{(0)} \rangle = 1, \quad \forall k. \quad \text{(B11)}$$

Apply the above analysis to the population diffusion process. We choose the system Hamiltonian as in Eq. (8), of which eigenstates are tensor products of $|0\rangle$ and $|1\rangle$. These eigenstates are the initial states as well as the final states in which we are interested. A classical trivial stochastic matrix $S_{t}^{c(0)}$ leaves an input eigenstate unchanged. Thus, Population does not diffuse if the coupling between system and bath vanishes. As we see in the numerical simulations, the thermalization rate is zero if $\lambda = 0$.

Consider the higher order correction of $S_{\lambda,t}$. As shown in the next section, if we take the explicit form of the thermal dynamic Hamiltonian, the odd-order corrections $S_{t}^{(2k+1)}$ of $S_{\lambda,t}$ vanish. Thus the leading order $S_{t}^{(1)}$ have no contribution to the thermal evolution and the next leading order $S_{t}^{(2)}$ will bring out correction to eigenvalues and eigenvectors in Eq. (B10). The correction can be evaluated similarly to the formal perturbative correction to the eigenvalues and eigenvectors for Hermitian Hamiltonian, as introduced in many quantum mechanics textbook [27]. One needs to notice that as the eigenvalues in $S_{t}^{(0)}$ are degenerate while in $S_{t}^{(2k)}$ are not, we need to carry out degenerate perturbation and non-degenerate one respectively on these two parts. The degenerate perturbation selects the true bases in the degenerate subspace by solving a secular equation regarding $S_{t}^{(2)}$. The selected true bases are superpositions of the original bases

$$|\phi_{m}^{(0)}\rangle \equiv \sum_{n} V_{mn} |\phi_{n}^{(0)}\rangle \quad \text{(B12)}$$

where $V$ represents a unitary transformation. Consider the second-order correction to the Eq. (B10). The result reads
where
\[
|\phi_{nm}\rangle = |\phi_{n0}\rangle + \lambda^2 \sum_l |\tilde{\phi}_l^{(0)}\rangle \frac{S_{l,0,0}^{(2)} |\phi_{00}\rangle}{\mu_{nl} - 1} + \lambda^2 \sum_{n' \neq m'} |\phi_{n'0}\rangle \frac{S_{n'0,m}^{(2)} |\phi_{0m}\rangle}{\mu_{nm} - \mu_{n'm'}} + O(\lambda^4),
\]
(13)

In a classical stochastic process, the input and output states are all classical states, denoted by |\phi_l^{(0)}\rangle and |\phi_l^{(0)}\rangle respectively and l \neq k. Thus after the quantum evolution for time t under system-bath coupling \(\lambda\), the probability of measuring |\phi_l^{(0)}\rangle receiving contributions from the classical part and the pure quantum part at the leading order is
\[
\begin{aligned}
\langle\langle \phi_l^{(0)} | S_{\lambda,t}^{(0)} | \phi_k^{(0)} \rangle \rangle &= O(\lambda^2), \\
\langle\langle \phi_l^{(0)} | S_{\lambda,t}^{\infty} | \phi_k^{(0)} \rangle \rangle &= O(\lambda^4).
\end{aligned}
\]
(14)

We find \(\langle\langle \phi_l^{(0)} | S_{\lambda,t}^{(0)} | \phi_k^{(0)} \rangle \rangle \ll \langle\langle \phi_l^{(0)} | S_{\lambda,t}^{(0)} | \phi_k^{(0)} \rangle \rangle\) for a small enough \(\lambda\). It shows that the classical evolution dominates the result in numerical simulation. The \(O(\lambda^2)\) dependence of the evolution has been explicitly shown in our numerical simulation (see figure 3). In the next appendix, we will focus on calculating the classical part of the evolution.

**Appendix C: Liouvillian formalism and calculation for evolution expansion**

To find an explicit form of the evolution expansion in Eq. (B6), we introduce the Liouvillian formalism [28] for the time evolution of the density operator.

In Schrödinger picture, the time evolution of the density operator is given by the von-Neumann equation
\[
\frac{d\rho(t)}{dt} = -i[H, \rho(t)] = -iL\rho(t).
\]
(1)

Here \(L\) is called the Liouville operator, the linear Lie product operator of the system Hamiltonian \(H\). If the Hamiltonian is explicitly time-independent, this equation can be integrated as
\[
\rho(t) = e^{-iLtt} \rho(0),
\]
(2)

where \(e^{-iLtt}\) is defined by its Taylor expansion
\[
e^{-iLtt} \rho(0) = \sum_k \frac{1}{k!} (-it)^k L^k \rho = e^{-iHt} \rho e^{iHt}.
\]
(3)

Thus we recovered the time evolution of the density operator as we used in Eq. (B5). The evolution operator on the system is given by
\[
S_{\lambda,t}(\rho_s) = \text{Tr}_b(e^{-iLt} \rho_s \otimes \rho_{b,\beta}).
\]
(4)

To find an explicit form of \(e^{-iLtt}\), we first decompose the Liouville operator of the system-bath Hamiltonian into two parts
\[
L = L_1 + L_2; L_1 \equiv (L_s + L_b), L_2 \equiv \lambda L_{sb},
\]
(5)

where \(L_s, L_b\) and \(L_{sb}\) are Liouville operator of \(H_s, H_b\) and \(H_{sb}\) respectively. The operator identity [28] for time-independent \(L_1, L_2\) reads
\[
e^{-iLtt} = e^{-iL_{1}tt} - i \int_{0}^{t} dt' e^{-iL_{1}(t-t')} L_2 e^{-iL_{1}tt}.
\]
(6)

The desired evolution \(e^{-iLtt}\) is shown on both sides of this identity. Since \(\lambda\) in \(L_2\) is a small quantity, iteratively taking the left hand side \(e^{-iLtt}\) into the right hand side \(e^{-iLtt}\) gives the perturbative expansion of \(e^{-iLtt}\). Taking this expansion into Eq. (4), we find the perturbative formula of \(S_{\lambda,t}\) for each order of \(\lambda\). For example, the first-order expansion reads
\[
S_{\lambda,t}^{(1)}(\rho_s) = \text{Tr}_b(-i \int_{0}^{t} dt_1 e^{-i(L_s + L_b)(t-t_1)} L_{sb} e^{-i(L_s + L_b)t_1} \rho_s \otimes \rho_{b,\beta}).
\]
(7)

We show that the odd order terms vanish if we take thermal dynamic Hamiltonian (14). Take the first order term as an example. Because \(\rho_{b,\beta}\) is the Gibbs state of \(H_b\), it commutes with \(H_b\) and \(e^{-iL_{sb}(t_1)} \rho_{b,\beta} = \rho_{b,\beta}\). Then, we take system-bath coupling Hamiltonian as in Eq. (B2). It leads to
\[
S_{\lambda,t}^{(1)}(\rho_s) = -i \text{Tr}_b \left( \int_{0}^{t} dt_1 e^{-i(L_s + L_b)(t-t_1)} \sum_l [S_l \otimes B^l, \rho_s(t_1) \otimes \rho_{b,\beta}] \right),
\]
(8)
where \( \rho_s(t_1) \) is the time evolved \( \rho_s \)
\[
\rho_s(t_1) = e^{-iH_s t_1} \rho_s e^{iH_s t_1}.
\] (C9)

Expand the commutator and use the cyclic permutation

\[
S_l^{(1)}(\rho_s) = -i \sum_i \int_0^t dt_1 e^{-iL_s(t-t_1)}
(\rho_s(t_1) - \rho_s(t_1)S_l^i) Tr_B(B^i \rho_{b,\beta}).
\] (C10)

This equation is a generalization of Eq. (2.72) in reference [14]. We take \( B^i \) as \( X_b^i \) and \( H_b \) as in Eq. (11). We see this equation vanishes by expanding the trace with \( H_b \)’s eigenstates \(|n\rangle \)

\[
Tr_B(B^i \rho_{b,\beta}) = \sum_n e^{-\beta Е_n} \langle n|X_i|n\rangle = 0,
\] (C11)

where the last equality hold since \(|n\rangle \) takes all possible configurations of bit string like \( |1011\ldots\rangle \).

Similarly, The third order correction \( S_l^{(3)}(\rho_s) \) has terms with coefficients like

\[
Tr_B(B^i(t')B^j(t'')B^i(t''')\rho_{b,\beta}),
\] (C12)

where \( S_{mn}^i = \langle m|S_i^n|n\rangle \), and \( \hat{h}^{ij}(\omega) \) is defined as a spectrum function of the heat bath

\[
\hat{h}^{ij}(\omega) = \int_{-\infty}^{+\infty} dt e^{-i\omega t} h^{ij}(t),
\] (C15)

\[
h^{ij}(t) \equiv Tr_B(B^i e^{-iH^b t} B^j e^{iH^b t} \rho_{b,\beta}).
\]

1. Application to the thermal dynamic Hamiltonian

We apply the above perturbative results to the thermal dynamic Hamiltonian (14). First, we calculate the spectrum of the heat bath taking bath Hamiltonian (11) and \( B^i = X_b^i \). With the commutation relations of Pauli operators, it gives

\[
h^{ij}(t) = \begin{cases} 
\exp\left\{-2i \sum_{\nu \in S} \alpha_{\nu}^{ij} t\right\}, & i = j \in S, \\
\exp\left\{-2i \sum_{\nu' \in S} \alpha_{\nu'}^{ij} t\right\}, & i = j \in I, \\
0, & i \neq j.
\end{cases}
\] (C16)

where \( t', t'', t''' \) denote some evolution times. The trace can be expanded similarly

\[
\sum_{nml} e^{-iE_n(t'-t''-i\beta)} e^{-iE_m(t''-t')} e^{-iE_l(t''-t'')}
\langle n|X_k|m\rangle\langle m|X_j|l\rangle\langle l|X_i|n\rangle = 0. \] (C13)

Thus the third order correction vanishes since any bit strings can not be recovered by flipping \((0 \leftrightarrow 1)\) exchange odd times. We can generalize the result to arbitrary odd order corrections, which means \( S_l^{(2k+1)}(\rho_s) \) vanishes for all non-negative integer \( k \). On the other hand, only the even order terms need to be considered.

The lowest order term \( S_l^{(2)} \) can be calculated following a similar procedure, as given in [14]. We only focus on the classical part of the evolution, which means that we are interested in the matrix elements of \( S_l^{(2)} \) between two classical states \( \langle \phi_m^{(0)} | S_l^{(2)} | \phi_n^{(0)} \rangle \). The matrix element has a physical meaning of the probability of observing the system’s final state \(|m\rangle \), given an initial state \(|n\rangle \). The matrix element can be evaluated exactly. The result is

\[
\langle \phi_m^{(0)} | S_l^{(2)} | \phi_n^{(0)} \rangle = 2 \int_{-\infty}^{+\infty} \frac{d\omega}{2\pi} \sum_{ij} \hat{h}^{ij}(\omega) \left[ S_{mn}^j S_{nm}^i \frac{1 - \cos((\omega + Е_n - Е_m)t)}{(\omega + Е_n - Е_m)^2} \right]
- \delta_{mn} \sum_l S_{ml}^j S_{ln}^i \frac{1 - \cos((\omega + Е_n - Е_l)t)}{(\omega + Е_n - Е_l)^2}, \] (C14)

where we take the zero temperature limit \( \beta \to \infty \) so that only the ground states of the heat bath contribute, i.e., the spin parallel states. The spectrum function can be derived by Fourier transformation according to Eq. (C15)

\[
\hat{h}^{ij}(\omega) = \begin{cases} 
2\pi \delta(\omega + 2\sum_{\nu \in S} \alpha_{\nu}^{ij}) & i \neq j, \\
2\pi \delta(\omega + 2\sum_{\nu' \in S} \alpha_{\nu'}^{ij}) & i = j \in S, \\
0 & i \neq j.
\end{cases}
\] (C17)

Thus in the matrix element of \( S_l^{(2)} \), only terms with identical \( i, j \) need to be considered. Then take \( S^i = X^i_s \). One finds \( \langle \phi_m^{(0)} | S_l^{(2)} | \phi_n^{(0)} \rangle \) does not vanish only if \((1) m = n \), where the second term of Eq. (C14) contributes. (2) \( m, n \) differ at one spin site, where the first term of Eq. (C14) contributes. Also due to \( S^i = X^i_s \), the summation \( \sum_j \) in Eq. (C14) only takes the configuration \( l \) that differ from \( m(n) \) at one spin site. With some algebra, the diagonal
matrix elements of $S^{(2)}_I$ read
\[
\langle \langle \phi^{(0)}_n | S^{(2)}_I | \phi^{(0)}_n \rangle \rangle = -2 \left( \sum_{j \in S} A_{n,j}(t) + \sum_{i \in I} B_{n,i}(t) \right),
\] (C18)
where
\[
A_{n,j}(t) = \frac{1 - \cos \left( -2 \sum_{i \in I} \alpha_{ij} + E_n - E_{X_j(n)} \right) t}{\left( -2 \sum_{i' \in S} \alpha_{ij'} + E_n - E_{X_i(n)} \right)^2},
\]
\[
B_{n,i}(t) = \frac{1 - \cos \left( -2 \sum_{j \in S} \alpha_{ij} + E_n - E_{X_i(n)} \right) t}{\left( -2 \sum_{j' \in S} \alpha_{ij'} + E_n - E_{X_j(n)} \right)^2}.
\] (C19)

Here we use $X_j(n)$ to represent the bit string $n$ flipped at site $j$. The off-diagonal elements can be derived similarly
\[
\langle \langle \phi^{(0)}_{X_j(n)} | S^{(2)}_I | \phi^{(0)}_n \rangle \rangle = 2A_{n,j}(t), \quad j \in S,
\]
\[
\langle \langle \phi^{(0)}_{X_j(n)} | S^{(2)}_I | \phi^{(0)}_n \rangle \rangle = 2B_{n,i}(t), \quad i \in I,
\] (C20)
while the other matrix elements vanish. According to the above two equations, one finds the relationship
\[
\sum_m \langle \langle \phi^{(0)}_m | S^{(2)}_I | \phi^{(0)}_n \rangle \rangle = 0.
\] (C21)

It indicates that each column of the evolution matrix $S_{\lambda,t} \simeq 1 + \lambda^2 S^{(2)}_I$ summed to 1, thus $S_{\lambda,t}$ retains a stochastic matrix in the second order perturbation expansion.

Another essential element in the Hamiltonian simulation is resetting infectious sites to $\{1\}$ once having unitary evolution $S_{\lambda,\Delta t}$ with a short time interval $\Delta t$. We first demonstrate how it behaves in the classical part of the evolution and then prove the following property of the thermal dynamic evolution. The property helps to determine the inter-bath coupling as discussed in Sec. (IV).

Property. The infection rate of susceptible site $j$ tends to 0 in the limit $\gamma_{ij} \to 0, \forall i \in I$ up to $O(\lambda^2)$, if the system-bath coupling $\sum_{i' \in I} \alpha_{ij} \Delta t = k \pi, k = 1, 2, \ldots$

Proof. Given a classical probability distribution $\{p_{m^S,m^I}\}$, the spin configuration reads
\[
|\psi\rangle \equiv \sum_{m^S,m^I} p_{m^S,m^I} |\phi^{(0)}_n\rangle
\] (C22)
\[
= \sum_{m^S,m^I} p_{m^S,m^I} |m^S_1 m^S_2 \ldots m^S_{|S|} m^I_1 m^I_2 \ldots m^I_{|I|}\rangle,
\] (C23)
where each $m^S_i$ taking values 0 or 1. Susceptible sites and infectious sites are distinguished explicitly in the second line. The resetting operation $R$ on the infectious set transforms it into
\[
R |\psi\rangle = \sum_{m^S} \left( \sum_{m^I} p_{m^S,m^I} \right) |m^S_1 m^S_2 \ldots m^S_{|S|} 1 \ldots 1\rangle.
\] (C24)

Here $\sum_{m^I} p_{m^S,m^I}$ is the probability of measuring the corresponding final state. Thus $R$ is also a stochastic matrix with unit entries at row $|m^S_1 m^S_2 \ldots m^S_{|S|} 1 \ldots 1\rangle$ and the corresponding columns $|m^I_1 m^I_2 \ldots m^I_{|I|}\rangle$.

To prove the property of zero infection rate up to $O(\lambda^2)$, first notice that if we set $\gamma_{ij} \to 0$, the energy differences $E_n - E_{X_j(n)}$ will all go to zero. Then taking $\sum_{i' \in I} \alpha_{ij} \Delta t = k \pi$, we find $A_{n,j}(\Delta t)$ vanish as indicated in Eq. (C19). If we further prove that $B_{n,i}$ has no contribution in the whole evolution involving resetting operation, then $S^{(2)}_I$ is effectively zero. Thus the system does not evolve up to $O(\lambda^2)$, and the infection rate is approximately zero. Consider the adjoint action $RS_{\lambda,\Delta t}$. The $n$-th column of $S_{\lambda,\Delta t}$
\[
S_{\lambda,\Delta t} |\phi^{(0)}_n\rangle = \sum_m |\phi^{(0)}_m\rangle \langle \langle \phi^{(0)}_m | S_{\lambda,\Delta t} | \phi^{(0)}_n \rangle \rangle = \sum_{m^S,m^I} |\phi^{(0)}_{m^S,m^I}\rangle \langle \langle \phi^{(0)}_{m^S,m^I} | S_{\lambda,\Delta t} | \phi^{(0)}_n \rangle \rangle
\] (C25)
can be regarded as a classical probability distribution as in (C23). It follows normalization condition $\sum_{m^S,m^I} \langle \langle \phi^{(0)}_{m^S,m^I} | S_{\lambda,\Delta t} | \phi^{(0)}_n \rangle \rangle = 1$ as indicated by Eq. (C21). Then the action of $R$ on Eq. (C25) is given by Eq. (C24). The probability of deriving $|m^S_1 m^S_2 \ldots m^S_{|S|} 1 \ldots 1\rangle$ is
\[
\sum_{m^I} p_{m^S,m^I} = \langle \langle \phi^{(0)}_{m^S,m^I} | S_{\lambda,\Delta t} | \phi^{(0)}_n \rangle \rangle.
\] (C26)

Note that the summation $\sum_{m^I}$ includes terms from (C18) and the second line of (C20) with all $i \in I$. Thus in the expression of the above probability, terms concerning $B_{n,i}$ cancel; in other words, they have no contribution to the whole evolution. So the property is proved. ■

The evolution matrix $S_{\lambda,\Delta t}$ resembles the classical Markov matrix describing virus spread in networks, such as the one shown in [6]. Assuming an infinitesimal generator $Q$ of the Markov chain as defined in [6], the evolution matrix thus correspond to
\[
S_{\lambda,\Delta t} \sim e^{Q \Delta t}
\] (C27)
where the superscript $T$ denotes matrix transpose.

Repeated actions of the stochastic matrix lead to exponential decay when the stochastic matrix is diagonalizable, as in the Markovian process. We will see these in the following example and find the case where the diagonalization is not permitted.

2. EXAMPLE: One infectious site and one susceptible site

As an example of the above derivation, we calculate the matrix elements of the classical part of the thermal dynamic evolution, including one infectious site and one
susceptible site. It can be used to explain the numerical data we derived in figure 3.4. The Hamiltonian of this 2-system+2-bath model reads
\[ H = -\gamma Z_0^a Z_1^a - \lambda (X_0^b X_1^b + X_1^b X_1^b) - \alpha Z_0^b Z_1^b, \]
where the infectious site is labelled by 0 and the susceptible site by 1. As explained previously, the initial state is a classical bit string. Specifically, the 1 infectious and 1 susceptible system is initialized as \( |\text{init}\rangle = |0\rangle_1 \otimes |1\rangle_0 = |01\rangle \). The survival probability of the susceptible site \( \hat{P}_1 \) is estimated by measuring the \( Z_1 \) operator. It can be written as
\[ Z_1 = |00\rangle \langle 00| + |01\rangle \langle 01| - |10\rangle \langle 10| - |11\rangle \langle 11|, \]
which are all projectors of the classical bit string. For simplicity, we represent classical configurations of bit string utilizing column vectors
\[ |\phi_{00}^{(0)}\rangle = \begin{pmatrix} 1 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad |\phi_{01}^{(0)}\rangle = \begin{pmatrix} 0 \\ 1 \\ 0 \\ 0 \end{pmatrix}, \]
\[ |\phi_{10}^{(0)}\rangle = \begin{pmatrix} 0 \\ 0 \\ 1 \\ 0 \end{pmatrix}, \quad |\phi_{11}^{(0)}\rangle = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 1 \end{pmatrix}. \]
A classical probability distribution \( \{ p_m^s, m_t \} \) can be written into a column vector, and the matrix representation of \( R \) is
\[ \begin{pmatrix} p_{00} \\ p_{01} \\ p_{10} \\ p_{11} \end{pmatrix} \rightarrow \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} + \begin{pmatrix} 0 \\ 1 \\ 0 \\ 0 \end{pmatrix} = \begin{pmatrix} p_{00} \\ p_{01} \\ p_{10} \\ p_{11} \end{pmatrix}. \]

According to Eq. (C17). The spectrum functions are given by
\[ \tilde{h}^{00}(\omega) = \tilde{h}^{11}(\omega) = 2\pi \delta(\omega + 2\alpha), \quad \tilde{h}^{01}(\omega) = \tilde{h}^{10}(\omega) = 0. \]
The matrix elements of \( S_{\lambda,\Delta t} \) read
\[ S_{\lambda,\Delta t} = \left( \begin{array}{cccc} 1 - 2\lambda^2 A_0 & 0 & \lambda^2 A_1 & 0 \\ \lambda^2 A_0 & 1 - 2\lambda^2 A_1 & 0 & \lambda^2 A_0 \\ 0 & \lambda^2 A_0 & 0 & 1 - 2\lambda^2 A_1 \\ 0 & 0 & \lambda^2 A_1 & 1 - 2\lambda^2 A_0 \end{array} \right) + \mathcal{O}(\lambda^4), \]
where
\[ A_0 \equiv \frac{\sin^2((\gamma + \alpha)\Delta t)}{(\gamma + \alpha)^2}, \quad A_1 \equiv \frac{\sin^2((\gamma - \alpha)\Delta t)}{(\gamma - \alpha)^2}. \]
It can be seen explicitly that both \( S_{\lambda,\Delta t} \) and \( R \) are stochastic matrices, satisfying the relationship
\[ \sum_m (S_{\lambda,\Delta t})_{mn} = 1, \quad (S_{\lambda,\Delta t})_{mn} \geq 0, \quad \text{for small enough } \lambda. \]
The effect of staggering implementations \( RS_{\lambda,\Delta t}RS_{\lambda,\Delta t} \ldots \) on an initial state can be evaluated by assuming the following stochastic matrix’s eigen-decomposition
\[ RS_{\lambda,\Delta t} = U \left( \begin{array}{cccc} 1 & 0 & 0 & 0 \\ 0 & 1 - \lambda^2 (A_0 + A_1) & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{array} \right) U^{-1}, \]
where the columns of \( U \) are the corresponding eigenvectors of \( RS_{\lambda,\Delta t} \). The existence of the eigendecomposition will be discussed later. The only non-zero eigenvalue \( 1 - \lambda^2 (A_0 + A_1) \) leads to the single-exponential decay behaviour in the numerical simulation. The expectation value of the bit string projector \( |l\rangle \langle l| \) given an initial state \( |k\rangle \langle k| \) after evolving time \( t \) is
\[ \langle \langle \phi_{l}^{(0)}| (RS_{\lambda,\Delta t})^{t/\Delta t} |\phi_{k}^{(0)}\rangle \rangle = B_0 + B_1 [1 - \lambda^2 (A_0 + A_1)]^{t/\Delta t}, \]
where \( B_0, B_1 \) are some irrelevant \( t \)-independent constants. To match the exponential decay behaviour shown in the main text, the above formula can be reformulated as \( B_0 + B_1 e^{-\Gamma t} \). The decay constant on the exponential is the infection rate, which reads
\[ \Gamma(\gamma) = \frac{1}{\Delta t} \ln \frac{1}{1 - \lambda^2 (A_0 + A_1)} = \frac{\lambda^2 (A_0 + A_1)}{\Delta t} + \mathcal{O}(\lambda^4). \]

Compare the coefficients \( A_0 \) and \( A_1 \). Note that \( A_0 \) and \( A_1 \) are like a Dirac delta function according to the identity
\[ \delta(\omega) = \lim_{\Delta t \rightarrow \infty} \frac{\sin^2(\omega \Delta t)}{\pi \omega^2 \Delta t}. \]
Thus for \( \alpha \gg |\alpha - \gamma| \) and large \( \Delta t \), \( A_0 \) can be neglected compared with \( A_1 \). It leaves
\[ \Gamma(\gamma) \simeq \lambda^2 \Delta t \left( \frac{\sin((\gamma - \alpha)\Delta t)}{(\gamma - \alpha)\Delta t} \right)^2. \]
Thus derived sinc-function has been checked in the numerical simulation of figure 4. It shows that the infection rate has two properties that are useful in the process of Hamiltonian parameters determination. (1) If we set the system and bath Hamiltonian identical, i.e., \( \gamma = \alpha \), we find \( \Gamma \) reaching its maximum \( \lambda^2 \Delta t \). (2) If we set \( \alpha \Delta t = k\pi, k = 1, 2, \ldots \), the infection rate \( \Gamma \rightarrow 0 \) as \( \gamma \rightarrow 0 \).
Now we discuss the existence of the eigendecomposition (C36). It can be seen by calculating the determinant of $U$. One finds

$$\text{det}(U) = \frac{(1 + A_0/A_1)(1 - \lambda^2(A_0 + A_1))}{\lambda(A_0 - A_1)}. \quad (C41)$$

The determinant is singular if $A_0 = A_1$, where $U$ is not invertible and the exponential decay behaviour cannot be guaranteed. Specifically, it happens as we set the inter-system coupling $\gamma = 0$. At this point, the survival probability does not have single-exponential decay behaviour as mentioned in the previous subsection. The phenomenology if we choose $\lambda$ in the numerical simulation with the help of time rescaling.

With analogy, we can generalize the result of one infectious site and one susceptible site (C40) to multi-infectious sites and one susceptible site. According to the independence of the infectious sites and the expression for $A_{n,j}$ in Eq. (C19), the infection rate of the susceptible site $j$ is

$$\Gamma_j \simeq \lambda^2 \Delta t \left( \frac{\sin(\sum_{i' \in I}(\gamma_{i'j} - \alpha_{i'j})\Delta t)}{\sum_{i' \in I}(\gamma_{i'j} - \alpha_{i'j})\Delta t} \right)^2. \quad (C42)$$

It is shown explicitly that if we take $\sum_{i' \in I} \alpha_{i'j} \Delta t = k\pi$ and $\gamma_{i'j} \to 0$, the infection rate tends to zero up to $O(\lambda^2)$, as proved in the previous subsection. The phenomenological SI model requires the linearity of the infection rate $\Gamma_j(\sum_{i' \in I} \gamma_{i'j}) = \sum_{i' \in I} \Gamma_j(\gamma_{i'j})$. However, due to the nonlinearity of the sinc-function, Eq. (C42) can only satisfy

$$\Gamma_j\left( \sum_{i' \in I} \gamma_{i'j} \right) = \frac{\lambda^2 \Delta t}{k^2} \left( \sum_{i' \in I} \frac{\sin(\gamma_{i'j}\Delta t)}{\pi} \right)^2 + O(\gamma^3)$$

$$= \left( \sum_{i' \in I} \frac{1}{k} \sqrt{\Gamma(\gamma_{i'j})} \right)^2 + O(\gamma^3) \quad (C43)$$

where we take $\alpha \Delta t = \pi$ in $\Gamma(\gamma_{i'j})$ and $\sum_{i' \in I} \alpha_{i'j} \Delta t = k\pi$ in $\Gamma_j$. Thus, the infection rate of $j$ surrounded by multi-infectious sites qualitatively satisfies the requirement of phenomenology if we choose $k = 1$ in the numerical simulation. The requirements of $k = 1$ and inter-bath couplings’ uniformity lead to

$$\alpha_{i'j} = \frac{\pi}{|\Gamma| \Delta t} \quad (C44)$$

This setting is utilized in our numerical simulation with two index patients introduced as shown in figure 7.

Appendix D: Simulation for small infection rate with time rescaling

In the main text of figure 4b, we find a plateau in the subplot. It indicates that the evolution with a low infection rate may not be able to be simulated with the thermal dynamic Hamiltonian. On the other hand, we want to simulate the evolution with a low infection rate. Because practically, we hope to simulate virus transmission over a large-scale community environment. In that case, the infection rate is supposed to be exponentially low as distance increases, following, for example, Eq. (22). Here, we provide a way to simulate the evolution with a low infection rate with the help of time rescaling.

We notice that the perturbative prediction in the subplot of figure 4b satisfy the requirement that as $\gamma \to 0$, infection rate $\Gamma \to 0$. Thus if the perturbation hold for small enough $\gamma$, a low infection rate can be simulated. It requires that the perturbation parameter $\lambda$ be tuned smaller than inter-system coupling $\gamma$. However, naively shrinking $\lambda$ leads to an obvious variation of the physical survival probability. For example, the result violates the requirement of SAR. This violation can be solved by introducing time rescaling

$$t \to t/a, \quad (D1)$$

where $a$ is a scaling factor, $t$ is the time shown on the computer, while $t/a$ is the physical time. The infection rate is rescaled to

$$\Gamma \to \Gamma a \quad (D2)$$

to ensure the survival probability unchanged according to Eq. (16). When tuning smaller $\lambda$, $\Gamma$ will also get smaller (as $\Gamma \propto \lambda^2$). To keep physical survival probability such as SAR unchanged or, $\Gamma a$ unchanged, $a$ should get larger. Thus tuning $\lambda$ smaller is permitted at the cost of longer time simulation on the computer (to keep $t/a$ unchanged). With this freedom of rescaling, all the physical quantities are unchanged, and the parameter $\lambda$ is free to choose. The up-triangular and down-triangular curves in the subplot of figure 4b show the infection rate with the alternative $\lambda = 0.400, 0.050$, which correspond to scaling factor $a = 0.253, 16.07$ respectively. We see that the larger $a$ is taken, the lower the infection rate can be reached.

Appendix E: Numerical results using explicit fourth-order Runge-Kutta method

Numerical results in the main text are from a quantum simulator with errors from first-order Trotter decomposition and a limited number of measurements. To check if the error in quantum simulation is small enough, we use the Runge-Kutta method to carry out the Schrödinger evolution given an initial density operator $\rho(t)$. The Runge-Kutta method is a family of methods of solving partial differential equations numerically. We utilize the fourth-order Runge-Kutta method to solve Schrödinger evolution on density operator directly

$$ih \frac{\partial \rho(t)}{\partial t} = [H, \rho(t)], \quad (E1)$$

$N$-th order Runge-Kutta method accumulates error in the order of $O(h^{N+1})$ in each step, where $h$ is the step
time adopted [29]. In our numerical calculation, step time $h$ is as small as the machine precision. However, since the number of partial differential equations equals the number of entries in the density matrix, the time complexity of the method grows exponentially as the number of qubits increases.

Figure 8. The survival probability of the household site 1 as a function of time is shown in the left panel, and the infection rate $\Gamma$ of the household site as a function of system-bath coupling $\lambda$ is shown in the right panel. These numerical results are derived from the fourth-order Runge-Kutta method that can be cross-checked with those in figure 3. We find the same single exponential behaviour of survival probabilities on the household site and the same $\Gamma$ dependence on $\gamma$.

In figure 8 and 6, we use fourth-order Runge-Kutta method to check with results from first-order Trotter decomposition in figure 3 and 6 respectively. We find that results from the quantum simulator converge to those of the Runge-Kutta method within the error of statistics. It indicates that errors in the quantum simulation are well controlled.

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