A stochastic agent-based model to evaluate COVID-19 transmission influenced by human mobility

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Abstract  The COVID-19 pandemic has created an urgent need for mathematical models that can project epidemic trends and evaluate the effectiveness of mitigation strategies. A major challenge in forecasting the transmission of COVID-19 is the accurate assessment of the multiscale human mobility and how it impacts infection through close contacts. By combining the stochastic agent-based modeling strategy and hierarchical structures of spatial containers corresponding to the notion of geographical places, this study proposes a novel model, Mob-Cov, to study the impact of human traveling behavior and individual health conditions on the disease outbreak and the probability of zero-COVID in the population. Specifically, individuals perform power law-type local movements within a container and global transport between different-level containers. It is revealed that frequent long-distance movements inside a small-level container (e.g., a road or a county) and a small population size reduce both the local crowdedness and disease transmission. It takes only half of the time to induce global disease outbreaks when the population increases from 150 to 500 (normalized unit). When the exponent $c_1$ of the long-tail distribution of distance $k$ moved in the same-level container, $p(k) \sim k^{-c_1}$, increases, the outbreak time decreases rapidly from 75 to 25 (normalized unit). In contrast, travel between large-level containers (e.g., cities and nations) facilitates global spread of the disease and outbreak. When the mean traveling distance across containers $d$ increases from 0.5 to 1 (normalized unit), the outbreak occurs almost twice as fast. Moreover, dynamic infection and recovery in the population are able to drive the bifurcation of the system to a “zero-COVID” state or to a “live with COVID” state, depending on the mobility patterns, population number and health conditions. Reducing population size and restricting global travel help achieve zero-COVID-19. Specifically, when $c_1$ is smaller than 0.2, the ratio of people with low levels of mobility is larger than 80% and the population size is smaller than 400, zero-COVID can be achieved within fewer than 1000 time steps. In summary, the Mob-Cov model considers more realistic human mobility at a wide range of spatial scales, and has been designed with equal emphasis on performance, low simulation cost, accuracy, ease of use and flexibility. It is a useful tool for researchers and politicians to apply when investigating pandemic dynamics and when planning actions against disease.
Keywords Infectious disease · COVID-19 · Agent-based modeling · Human mobility · Container model · Bifurcation

1 Introduction

COVID-19 is a highly infectious disease transmitted in several ways, including through direct physical contact, inhalation of small particles and droplets disseminated by sneezing or coughing, and droplets or virus particles entering the eyes, nose or mouth [1]. On January 30, 2020, the outbreak of COVID-19 was declared a Public Health Emergency of International Concern by the World Health Organization (WHO), which led to global recession. To date, the disease is still heavily affecting the human health and economies of most countries [2,3]. The COVID-19 pandemic has created an urgent need for models to analyze and predict the interactions between infectious diseases and human beings.

Current models for studying the spread of the pandemic and the impact of intervention strategies can be broadly divided into four types: ordinary differential equation (ODE)-based [4–6], partial differential equation (PDE)-based and stochastic differential equation (SDE)-based compartmental models [7–14], and agent-based Monte Carlo models [15–23]. The compartmental models are simple and computationally cost-effective. However, these compartmental models neglect the physical infection process [7–14], which loses accuracy in reflecting real transmission dynamics. Agent-based models consider individual differences, microscale policies and detailed actions, which can provide a comprehensive understanding of transmission dynamics as well as a flexible framework for implementing various intervention rules [15–22,24–28]. However, most agent-based models and software consider individual contacts using abstract networks and build disease propagation models that are based on these networks. The commute and contact networks are usually biased by small population samples, which are used to estimate the network topology and the weights of edges [29]. Furthermore, commute networks only reflect travel in a limited number of areas on the same spatial scale, either at the microscale containing homes, workplaces, schools and hospitals [15,16,18–20,27], or at the macroscale containing cities and countries [30–32]. The influence of realistic and multiscale human travel and physical contact on disease transmission and propagation is still largely unknown.

Thanks to the rapid development of GPS tracing techniques and the advanced statistical analysis methods, the understanding of human mobility and human travel and contact patterns has become deeper and more accurate. Recently, Alessandretti et al. [33] showed that physical space can be represented by nested containers with associated sizes, satisfying the intuitive conception of space which is hierarchical and partitioned by geographical borders. Specifically, the physical space of each individual can be represented as a hierarchy of $L$ levels ordered from the smallest to the largest. The small level contains individual positions, rooms or houses. The large level contains states or countries. At each level, the physical space is partitioned into compact containers with certain characteristic sizes. Human movements are modeled using conditional probabilities as the combination of commuting within the same containers and traveling between containers of different scales. Compared with other state-of-the-art models [34–41], the hierarchical container model provides an unbiased performance estimate and a significantly better description of human GPS traces evaluated by several indices, such as the displacement distribution and time allocation among locations [33]. Thus, these works provide new insights and opportunities for modeling and calibrating the COVID-19 infection and transmission dynamics that are influenced by human mobility and mobility-related interventions.

In this study, we present a stochastic agent-based model, Mob-Cov (mobility-influenced COVID-19 transmission model), which considers hierarchical geographical mobility patterns and COVID-19 infection and recovery processes. Specifically, in the model, each person is represented by an agent that can move around freely and that has an individual-specific health condition represented by the infection and recovery rates. Assume that 0.5% of the population is infected initially. As time goes on, people surrounding the infected individuals (i.e., people in the same container with the infected people) also get infected. Sick people travels to other containers, which turns local disease transmission into a global problem and eventually induces an outbreak crisis. Next, the recovery of sick people after an infection period is included. Instead of having an outbreak, the system is able to achieve a dynamic equilibrium with the coexistence of the healthy and infected people. Furthermore, the influence of popula-
A stochastic agent-based model to evaluate COVID-19 is predicted. Overall, the Mob-Cov model and the results presented in this study provide a novel and comprehensive view of the spread of COVID-19 from a multiscale human mobility perspective, and can be valuable and helpful in evaluating and designing better interventions and mitigation measures for the pandemic.

1.1 Related work

Many mathematical and statistical models for analyzing the transmission and propagation dynamics of infectious disease have been developed in the last three years. A large proportion of the models are ODE/PDE/SDE-based compartmental models [4–14], such as the susceptible–infected–recovered (SIR) model and the susceptible–exposed–infected–recovered (SEIR) model. Combined with Bayesian statistical inference, compartmental models are widely used to (1) evaluate the progression of the pandemic, (2) infer the pandemic wave, (3) predict the expected number of cases within a time frame and (4) evaluate the effectiveness of intervention strategies using patient cases reported in different places, such as cities in China [11,12,42], Germany [9] and Italy [10]. For example, Dehning et al. [9] combined the SIR model with Bayesian parameter inference to detect the change points of disease spreading using COVID-19 case numbers measured in Germany. Various modifications of SIR/SEIR models have been made to consider different influential factors such as age, quarantine period, hosts-people transmission and climate [14,42,43]. For example, Prem et al. [42] used an age-structured SEIR model to investigate the effect of physical distancing on disease propagation. Furthermore, several reviews have also highlighted the difficulty and importance of selecting statistical methodologies to estimate parameters for even very simple compartmental models [44–47].

Agent-based models are able to overcome the limitations of traditional compartmental models which assume that the population is homogeneously mixing. Agent-based models simulate the reality that individuals (referred to as agents) interact with each other, and that these interactions are influenced by the individual’s demographics, health conditions and social environments. Therefore, although the computational cost is larger than that of compartmental models, agent-based models are usually more realistic and have a great potential for capturing the heterogeneous spatial–temporal spread of infectious diseases. Agent-based models have been used to study pandemic dynamics in Australia, Singapore, China, the USA and many other places under various interventions [15–23,49]. For example, Hoertel et al. [17] developed an agent-based disease transmission model for a social contact network. They examined the impact of lockdown period and post lockdown measures (e.g., physical distancing, mask wearing) on the cumulative disease incidence and intensive care unit(ICU)-bed occupancy. Meanwhile, several user-friendly, high-performance COVID-19 microsimulation software programs, including the OpenABM-Covid and Covasim, have been developed for researchers and public officials to easily study the COVID-19 dynamics and to inform policy decisions [19,20].

Overall, Table 1 lists the representative reviews and works of compartmental models and agent-based models for studying COVID-19 dynamics.

To capture a more realistic dynamics of COVID-19 spread, some compartmental models and most agent-based models consider individual contacts using abstract networks and build disease propagation models that are based on these networks. There are three widely used networks, i.e., microscopic commute networks for work, study and other activities [15,16,18–20], macroscopic commute networks for transport between cities and countries [29–32], and social contact networks among family members, friends, workmates, etc. [17]. In addition, some agent-based models consider individual movements in continuous physical spaces [21,22,24,25,29]. For example, Cuevas [21] studied how the combination of local random walks and long directed movements of individuals influences COVID-19 transmission. Silva et al. [22] incorporated four types of human behaviors, including walking freely, going home, going to work and going to the hospital, into the infection model, where the four walking behaviors are modeled as random walks of different velocities and directional preferences.

However, abstract contact networks reflect social connections among individuals, which are formed over a long period of time. The transient contacts between strangers and acquaintances, which can be critical to disease propagation, are largely lost. The commute networks include physical contact among individuals,
Table 1  Existing compartmental and agent-based models of COVID-19 transmission dynamics

| References                  | Model                                | Remark/conclusion                                                                 |
|-----------------------------|--------------------------------------|------------------------------------------------------------------------------------|
| Tang et al. [44] (review)   | SIR, SEIR, general compartmental model | Mechanistic models and related statistical analysis for model specification, estimation, inference and prediction. Community-level models for analyzing regional surveillance data. |
| Wang et al. [45] (review)   | SIR, SEIR                            | Basic concepts and foundation of epidemiological modeling. Evaluate epidemiological features, such as disease tendency, latent effects, susceptibility, basic reproduction numbers, asymptomatic infections, herd immunity and impact of the interventions. |
| Lin et al. [46] (review)    | SIR, SEIR, general compartmental model | Evaluate 75 mathematical and statistical models and analyze the epidemiological features. |
| Ma [47] (review)            | SIR, SEIR                            | Estimate the growth rate using maximum likelihood estimation and simple SIR, SEIR models. |
| Padmanabhan et al. [43] (spsreview) | SIR, SEIR, general compartmental model | Provide an integrated framework to unify the compartmental models. Discuss mathematical models for scenario-based analysis and active control methods. |
| Kong et al. [48] (review)   | General compartmental model          | Discuss different compartmental structures used in modeling COVID-19.              |
| Lorig et al. [49] (review)  | Agent-based model                    | Summarize 126 articles of agent-based models of COVID-19 which model transmission dynamics, disease states, human behavior and interventions. |
| Hinch et al. [19] (spssoftware) | Agent-based model (OpenABM-COVID19) | Epidemic simulation by considering multi-factors, including age-stratification, social networks, vaccination programs and digital contact tracing. |
| Kerr et al. [20] (software) | Agent-based model (Covasim)          | Epidemic simulation by considering multi-factors, including demographic information on age structure and population size, transmission networks in different social layers, physical distancing, protective equipment, vaccination, isolation, contact tracing and quarantine. |

but only reflect travel in a limited number of areas on the same spatial scale. In addition, in the microscopic continuous space, studies have also shown that human mobility patterns do not follow a random walk or a scale free Lévy walk [33,37–40]. Thus, until now, it has remained a major challenge to obtain an accurate assessment of the multiscale spatial–temporal transmission of COVID-19, and the effect of mobility restrictions and the consequential dynamics are rarely understood.

1.2 Highlights of the work

This work and the proposed Mob-Cov model have the following three highlighted objectives.

- Develop a multiscale pandemic transmission model in nested geographical containers, and reveal the considerable effects of hierarchical structures and regional borders and constrictions on the dynamics of disease transmission and propagation.
- Investigate how human movements and travel at different spatial scales influence disease trans-
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Fig. 1 General working principles of the Mob-Cov model. The multiscale geographical information is represented by the hierarchical containers. People traveling between two places in the nested geographical structures is modeled using conditional probabilities. The real-world population size and GPS traces can be input into the model to acquire realistic mobility patterns. Two-state (healthy, infected) COVID-19 transmission model is developed by considering the influence of human mobility mission. Illustrate the advantages of hierarchical mobility patterns and restrictions of long-distance travel in preventing rapid spread of the disease and outbreaks, and facilitate achieving zero-COVID in a short time period.

• Provide a flexible agent-based model framework with emphasis on the influence of human mobility on epidemiological dynamics. The framework can be easily adapted to study disease progression under various interventions.

The paper is organized as follows. In Sect. 2, the mathematical foundations of the work (i.e., the nested container model of human mobility, the infection-recovery model of the pandemic) is first introduced. Then, the implementation of the Mob-Cov model and the calibration criteria of the disease dynamics are presented. Based on the model, in Sects. 3.1 and 3.2, we discuss the probability and speed of reaching a COVID-19 outbreak influenced by the microscale and long-distance movements, individual health conditions and the population number, when only the infection state is considered. In Sects. 3.3 and 3.4, when both infection and recovery states are considered, we investigate the phenomenon of bifurcating into a “live-with-COVID” state and a “zero-COVID” state influenced by human mobility and population properties. The discussion and conclusion are presented at the end.

2 The proposed Mob-Cov model

2.1 Agent-based model

To investigate the impact of human mobility on the transmission of COVID-19, we develop a stochastic agent-based Mob-Cov model. The fundamental concept of the Mob-Cov model is shown in Fig. 1. The model includes three key components: (1) a synthetic population generated with hierarchical geographical characteristics; (2) realistic human travel rules, taking into account the power law behaviors of traveling within a container and transport between containers; and (3) infection and recovery with probabilities influ-
Fig. 2 Schematics of modeling the multiscale human travel, disease transmission and recovery. (a) Human mobility on the three-level nested container structures. (b) The probability of traveling from location \( j \) to \( k \) is determined by two steps: selecting a level (e.g., selecting level 3 by \( P_{lv}(3) \)) and selecting the nested containers level by level (e.g., \( P_3(k_3), P_2(k_2), P_1(k_1) \)). (c) The COVID-19 infection and recovery rules.

2.2 Human mobility in the nested containers

The hidden geographical structure of the population is modeled using nested containers proposed by Alessandretti et al. [33]. Specifically, the physical space is assumed to contain hierarchy of \( L \) levels. Level 1 is the smallest and level \( L \) is the largest. At any level \( l \) (\( l = 1, 2, ..., L \)), the space is partitioned into \( n_l \) topologically compact containers. For \( l < L \), a container is fully included within a single parent container. Thus, each geographical location \( k \) can be uniquely identified as a sequence of containers \( (k = (k_1, k_2, ..., k_L)) \), where the child container at \( l \) level \( k_l \) is fully included in the mother container at \( l + 1 \) level \( k_{l+1} \). For example, Fig. 2a shows the schematics of a three-level hierarchical structure. At the largest level (level 3), there are 3 containers, as shown by the light orange polygons. Level 2 has 9 containers, as shown by the orange polygons. And level 3 contains 21 individual positions, represented by the black dots. The location of agent \( j \) is defined as \( j = (j_1, j_2, j_3) \), where \( j_3 \) is the ID of the left large polygons in level 3, \( j_2 \) represents the middle polygon in \( j_3 \) and \( j_1 \) is the ID of one black dot in \( j_2 \).

Traveling in the nested containers follows a two-stage process: first deciding which level to travel at, second selecting a container at that level, and then a container within the chosen container and so on down to the lowest level. Specifically, suppose an agent is at position \( j = (j_1, j_2, ..., j_L) \), the probability for the individual to travel to position \( k = (k_1, k_2, ..., k_L) \) can be expressed as (Fig. 2b)
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\[ P(j \rightarrow k) = P_l(l) \prod_{i \leq j} P_i(k_i) \]  

where \( P_l(l) \) is the probability of the agent choosing level \( l \). The agent stays in the same container if the container level is larger than \( l \). In 1, 2, ..., \( l \) levels, the agent switches to different containers. Thus, \( k_{l+1} = j_{l+1}, k_{l+2} = j_{l+2}, ..., k_L = j_L \), but \( k_0 \neq j_0, k_1 \neq j_1, ..., k_l \neq j_l \). \( P_l(l) \) follows an exponential distribution with a parameter \( d \), which is expressed as

\[ P_l(l) = e^{-d l}, \quad \forall \ l \in [1, L] \]  

where \( l \) is an integer representing the level. Equation (2) indicates that switching between large-level containers (e.g., between countries) is less frequent than switching between small-level containers (e.g., between houses or streets).

\( P_i(k_i) \) is the probability of choose container \( k_i \) at level \( i \). \( P_i(k_i) \) follows a long-tail distribution with parameters \( c_0 \) and \( c_1 \), which is expressed as

\[ P_i(k) = k^{-(c_0+c_1 i)}, \quad \forall \ i \in [1, l] \]  

where \( i \) is the level, \( k \) is the container position. Equation (3) suggests that the travel probability decreases over the distance, especially at a large level \( i \). This indicates that, for example, traveling to a distant country is more scarcer than going to a remote road. But compared with the widely used exponential distribution, the long-tail distribution allows the happening of long-distance movements at all levels.

Equations (1)–(3) calculate the probability of switching from location \( j \) to location \( k \) if the agent takes a movement. We consider the fact that people do not travel at every time moment but need to take a rest or stay at a place for a while. Thus, the probability for an agent to take a movement is modeled with a probability \( P_m \), which is uniformly distributed between 0.2 and 0.4. A portion of people with low movement tendency (i.e., low mobility population) is further considered. The ratio of the low mobility people in the population \( r_{lm} \) is a model parameter. The probability for the low mobility people to take a movement is only \( P_m = 0.03 \).

2.3 Infection and recovery

If two agents \( i \) and \( j \) (\( i \) is infected and \( j \) is healthy) are in the same level-2 container (level-1 containers are the individual positions), infection occurs with a probability \( P_{ij} \) (Fig. 2c). If there is more than one infected agent, the infection probability of a healthy agent \( j \) is calculated as \( P_j = 1 - \prod_i (1 - P_{ij}) \), for all infected agents \( i \) in the same level-2 container as \( j \). For the high-risk population, \( P_{ij} \) follows a uniform distribution between 0.1 and 0.3. For the ratio of low-risk population, \( P_{ij} = 0.02 \). The low-risk people ratio in the population \( r_{li} \) is a model parameter. Note that, \( P_{ij} \) is chosen according to previous studies \([20,21]\). Kerr et al. \([20]\) set \( P_{ij} = 0.05 \) for household-level transmission. Cuevas \([21]\) set \( P_{ij} = 0.2 \).

After being infected for a time period \( T_{inf} \), the individual \( i \) is able to recover to a healthy state with a probability \( R_i \) (Fig. 2c).

2.4 Computational procedure

The Mob-Cov model is implemented as an iterative scheme. The flowchart is shown in Fig. 3 and the pseudocode is shown below. In the pseudocode, \( L \) is the number of levels. \( N_{cont} = (n_1, n_2, ..., n_L) \) is the number of containers in each level. \( c_0, c_1 \) and \( d \) are parameters that determine the mobility, as discussed in Sect. 2.2. \( N_p \) is the number of agents. \( N_{infb} \) is the number of initially infected agents. \( T_{inf} \) is the infection time period before the agent has a probability of recovery. \( r_{lm} \) is the ratio of the population with low mobility. \( r_{li} \) is the ratio of the population with low infection risk. NestedCont is the nested container structure, which is a nested random dictionary implemented in Python. \( P_{mi} \) is the probability of movement for agent \( i \). \( P_{ij} \) is the probability of agent \( j \) getting infected by agent \( i \). \( R_i \) is the recovery rate of agent \( i \). If agents \( i \) and \( j \) are in the same level-2 container, \( j \) is defined as the neighboring agent of \( i \).

In the simulation, the maximum iteration number is set as 2000, which is enough for the system to reach equilibrium. The number of agents is 1000. The number of initially infected people is 5. For each experiment, 50 simulations are performed, and the mean and variance of the results (including the outbreak time, ratio of the infected population during the outbreak, ratio of the infected people and zero-COVID ratio) are calculated. The simulation was performed using Python 3.8 using the SciPy (scipy.org) ecosystem. It uses NumPy (numpy.org) and Pandas (pandas.pydata.org) for fast numerical computing; and Matplotlib (matplotlib.org) and Plotly (plotly.com) for plotting. A single-core desktop computer having an Intel i9-10900K (3.7 GHz CPU processor), 16 GB RAM and 200 GB storage was used.
Fig. 3 The flowchart of the Mob-Cov model. \( rd \) is a random number uniformly distributed between 0 and 1. \( t \) is the current simulation time. \( \bar{\tilde{t}}_i \) is the infection time of agent \( i \). \( P_{mi} \), \( P_{ij}(l) \), \( P_i \), \( R_i \), and \( T_{inf} \) are model parameters discussed in Sect. 2.

Mob-Cov model

**Input:** \( L, N_{cont}, c_0, c_1, d, N_p, N_{inf0}, T_{inf} \)

**Initialise:**

- NestedCont (nested container structure)
- agent position \( X_i \) (\( i = 1, 2, ..., N_p \))
- infected agent IDs
- \( P_{mi} \) (mobility) for each agent \( i \)
- \( P_{ij} \) (infection) for agents \( i \) and \( j \) (\( i \neq j \))
- \( R_i \) (recovery) for each agent \( i \)

**while** \((\text{iter} < \text{MaxIteration}) \) or (stop criterion) \**do**

**for each agent \( i \) do**

- if \((\text{rand} \leq P_{mi}) \) then
  - Update \( X_i \) using Eqs. (1)-(3)
  **end**

**end**

**for each infected agent \( i \) do**

**for each neighboring healthy agent \( j \) do**

- if \((\text{rand} \leq P_{ij}) \) then
  - Update agent \( j \) as infected
  **end**

- if \((\text{Infection time} > T_{inf}) \) and \((\text{rand} \leq R_i) \) then
  - Update agent \( i \) as healthy
  **end**

**end**

**end**

**Post process results and visualization**

2.5 Outbreak endpoint

To evaluate the speed of the spread of the disease in the population, the outbreak endpoint is calculated. The rational for using the outbreak endpoint instead of the time when all the agents are infected is that there are always a few individuals who have truly low infectious probability \( P_{ij} \). The time it takes for the whole population to become infected is heavily biased by the last few individuals, and therefore cannot directly and accurately reflect the system properties. Therefore, an outbreak endpoint that considers the effects of both the ratio of the infected population and the infection rate is used in this study [18,50].

The detection of the outbreak endpoint is similar to finding the knee point in system engineering. The knee point usually represents the “right decision point”, after which the system properties are no longer significantly influenced by the variation in the variable [50]. Thus, in this study, we follow the previously proposed methods and calculate the maximum point of the cost function. The maximum point is defined as the outbreak endpoint of the system. The cost function is expressed as [18]

\[
J(t) = \frac{N_{inf}(t)}{N_p} - \frac{t}{T_{max}}
\]

where \( N_{inf} \) is the number of infected agents at time step \( t \), \( N_p \) is the total population number, and \( T_{max} \) is the total time (maximum iteration number), which is defined as the time when 90\% of the population became infected plus 100 extra time steps.

It has been suggested that the cost function \( J \) can only have one global maximum point, which corresponds to the outbreak endpoint [18]. Therefore, the outbreak time can be calculated as
\[ T_b = \arg_{t \in [1, T_{\text{max}}]} \max J(t) \] (5)

Figure 4 shows a simulation example of the infection rate over time and the determination of the outbreak endpoint. Figure 4a shows that the number of newly infected people is the largest during the first 70 time steps. After 70 time steps, the infection rates gradually decrease to values close to zero. As expected, the proportion of infected people continues to increase over time, but the speed of increasing slows down (Fig. 4b). By finding the maximum point of the cost function (Fig. 4c), the outbreak point can be identified when \( t \) is approximately 70, which corresponds to an infection ratio approximately 80%. The results suggest that the first 70 time steps are the most critical period when the number of infected people increases rapidly. After approximately 80% of the population becomes infected, the infection dynamics slows down. It can take a very long time (longer than 2000 time steps) for the whole population to become infected.

It should also be noted that the outbreak happens when only the infection of individuals is included in the model. After considering the recovery of sick people, the system is likely to reach a dynamic equilibrium with both infected and healthy populations, instead of reaching the outbreak endpoint.

3 Results

3.1 The dynamics of the spread of COVID-19

Figure 5a shows a simulation result for the population distribution and the spread of COVID-19. In the simulation, the number of levels is \( L = 5 \). The mobility parameters are \( d = 1.5 \), \( c_0 = 0.8 \) and \( c_1 = 0.35 \). The number of containers in each level is \( N_{\text{cont}} = (8, 4, 9, 9, 250) \). Containers in level 5 are represented by colorful and transparent polygons. Individuals in different level-5 containers are represented by dots labeled with different colors. Figure 5b is the probability of selecting a container level, \( P_l(t) \). The gray bars are the simulation results, which fit well with the analytical expression in Eq. (2). Figure 5c is the probability of selecting a container in level 2, \( p_2(k) \). The simulation results represented by the black dots fit well to the analytical expression in Eq. (3).

At the beginning, only five individuals (four in the blue container and one in the red container) were infected. Due to the high population density in the blue container, the disease quickly spreads in one cluster at time \( t_0 \). Many infected people then travels to the nearby cluster in the same blue container, accelerating local transmission. Meanwhile, the spread of the disease is relatively slow in the red container due to the low population density. Afterward, one or two infected people travel between the level-5 containers and reach the gray and brown areas, indicating that the disease is transmitted globally. After a further local spread at time \( t_2 \), a disease outbreak occurs at \( t_3 \).

Overall, the spread of the disease is a multiscale dynamic process influenced by the probability of switching between levels (Fig. 5b) and the movement distance (Fig. 5c). The results indicate that the total population density and the initial number of infected people do not need to be truly large. When the disease starts from a high-density local population cluster (e.g., a crowded market), a small probability of traveling between large-level containers (e.g., traveling across cities or countries) can lead to a rapid global spread and outbreak. The local-to-global spread of the disease heavily relies on the long-distance movements between containers. Thus, accurately modeling both the short walks and long sojourns using the hierarchical container structure in the Mob-Cov model ensures the precise prediction of the disease dynamics.

3.2 Factors influencing disease outbreak

The spread and outbreak of COVID-19 is influenced by various complex factors, such as the biological characteristics of the virus, health and immune conditions, population density, environmental conditions and policies. Using the Mob-Cov model, we mainly decipher the impact of microscopic and universal human mobility and individual health conditions on the spread of the disease and on outbreak (Fig. 6).

For mobility, the results show that the distance moved and the probability of traveling between large-level containers have a significant influence on the spread of the disease and the onset of the outbreak. When long-distance movements of the same level occur more frequently, which corresponds to a smaller value of \( c_1 \), the local crowdedness is reduced. Thus, it takes a longer time to reach outbreak status (Fig. 6b). Infected people are likely to be distributed in every container and the ratio of the infected population can be rela-
Fig. 4 The evolution of the infection process. (a) The rate of infection (the number of new infected people) over time. (b) The infection ratio of the population over time. (c) The cost function $J(t)$ over time. The outbreak endpoint is the global maximum point of the cost function, labeled in red. (Color figure online)

Fig. 5 Visual results of the population mobility and the spread of the disease. (a) Distribution of the health and infected individuals at four time steps. In the simulation, $L = 5$, and there are 8 containers at level 5, which are represented by the colorful polygons. Individuals in the 8 containers are represented by the colorful dots. Infected individuals are labeled as red. (b) Probability of switching to level $l$ ($l \in [1, 5]$). The gray bars are the simulation results. The dotted line is calculated using Eq. (2). (c) Probability of moving to container $k$ at level 2. The black dots are the simulation results, and the black line is calculated using Eq. (3). (Color figure online)

The rate of infection is relatively small when the outbreak happens (Fig. 6a). When individuals are likely to travel between large-level containers (e.g., cities and countries), which corresponds to a small value of $d$, the global transmission of the disease is enhanced. The time it takes for the onset of the outbreak becomes shorter. At the outbreak endpoint, more than 80% of the population has been infected. Restricting travel between large-level containers helps slow down the spread of the disease and reduce the size of the infected population (Fig. 6c, d). The ratio of the population with low mobility to the total population does not have a significant influence on the number of infected people when the outbreak happens (Fig. 6e). However, if most people prefer to stay in the same place rather than go elsewhere, the time it takes to reach the outbreak endpoint increases from 100 time steps to more than 600 time steps. In addition, the variation in the outbreak time is large when the mobility of most people is low, because the outbreak time now heavily relies on the random initial distribution of the population (Fig. 6f).
As for the individual health conditions, if a large percentage of the population has a low infection risk (e.g., most people get a COVID-19 vaccination), the speed of disease spread is reduced (Fig. 6h). The ratio of low-risk people does not have a significant influence on the infected population size at the outbreak endpoint. Approximately 70% of people were infected during the outbreak (Fig. 6g). The total population size also does not have a significant impact on the ratio of the infected population (Fig. 6i). However, when the population size is large, the local crowdedness increases the infection probability for individuals and accelerates the spread of the disease (Fig. 6i).

Finally, we find that the hierarchical structure of the containers has a large impact on the ratio of the infected population and on the outbreak time. If the hidden network of human travel contains many levels, the speed of the disease spread is slower and the outbreak time is larger (Fig. 6i). In addition, fewer people became infected at the outbreak endpoint (Fig. 6k). The results suggest that multilayer geographical barriers and borders that restrict human mobility can help reduce the spread of the disease. For example, in areas of high population density, the spread of COVID-19 tends to be fast. By zoning and adding multilevel physical borders, the transmission of the disease may be effectively reduced.

Note that, a few previous works have studied the influence of population number, individual traveling tendencies and individual health conditions on disease propagation dynamics as well [15, 51, 52]. For example, Chang et al. [15] showed that the incidence of infection decreases after applying interventions including isolation, home quarantine and international travel restrictions. Thus, the prediction of our Mob-Cov model (Fig. 5c–f) is well aligned with previous findings. Song et al. [51] found that if vaccine efficacy remains high, which means that more people are at low infection risk, infection and reinfection rates are reduced, which aligns with our results (Fig. 5g, h). Wang et al. [52]
showed that increasing the population number in a community leads to faster transmission of COVID-19. The results in Fig. 5i, j are in agreement with their conclusions. The power law distribution of traveling distance (i.e., parameter \( c_1 \)) and the container levels are unique properties of the hierarchical container model of human mobility, which has not been investigated thus far.

### 3.3 Bifurcation and state transition

When recovery from infection is considered, the system is no longer going to reach the outbreak endpoint. In contrast, depending on the infection rate, recovery speed and rate, and many other factors, two possible states may occur: (1) the “zero-COVID” state, which means that all infected people have recovered; and (2) the “living with COVID” state, during which the system reaches an equilibrium state and the number of newly infected people is equal to the number of newly recovered people. In this section, the influential factors on the transition between the “zero-COVID” state and the “living with COVID” state are investigated.

First, the influence of distance moved determined by parameter \( c_1 \) is studied. When individuals tend to make long-distance movements in same-level containers (i.e., mobility parameter \( c_1 \) is smaller than 0.25), the system is able to achieve “zero-COVID”. Increasing \( c_1 \) leads to the local accumulation of people and facilitates the spread of the disease. Bifurcation occurs when \( c_1 \) becomes larger than 0.25. The transition appears to be discontinuous, and a hysteresis loop appears in the bistable region where both the “zero-COVID” state (black dots) and “living with COVID” state (orange dots) coexist. When \( c_1 \) is larger than 0.4, the percentage of infected people in the population increases to more than 30% (Fig. 7a). Figure 7b shows that the probability of zero-COVID after a long enough time rapidly decreases to zero when \( c_1 \) is larger than 0.25. We further show the variation in the ratio of infected people over time (Fig. 7c). If the system eventually reaches a “zero-COVID” state, the fluctuation curves are those labeled in pink. Otherwise, the curves are those labeled in gray. Figure 7c shows that there is a peak of the ratio of infected people at the beginning, because healthy people continue to become infected but infected people have not yet recovered. Afterward, the ratio of infected people decreases and then fluctuates between 3 and 5%.

In some cases, the ratio of infected people decreases to zero, indicating that all sick people recover.

Next, the effects of the percentage of people with low mobility in the population are evaluated. Increasing the percentage of people with low mobility reduces the spread of the disease and the ratio of infected people. Thus, the system transitions from the “living with COVID” state to a bistable region, and then to the “zero-COVID” state (Fig. 7d). The probability of achieving zero-COVID increases rapidly when the ratio of people with low mobility is larger than 60% (Fig. 7e). In addition, the variation of the infected ratio is large and the time needed for the system to reach the “zero-COVID” state is longer when the ratio of people with low mobility is 70% (Fig. 7f). This phenomenon may be caused by the stochastic locations and movements of infected people. When infected people stay in crowds for a while, it facilitates disease transmission and increases the ratio of infected people. However, if the infected people are located in a remote area, the low mobility characteristics help reduce the infection.

Finally, we also found that varying the population number leads to bifurcation, and that the bistable region with both the “zero-COVID” state and “living with COVID” state appears at an intermediate population number (i.e., approximately 400 to 500 people) (Fig. 7g). The probability of zero-COVID decreases in the bistable region (Fig. 7h). When the population size is larger than 600, the system is likely to reach equilibrium and more than 20% of the population is infected (Fig. 7g). When the population number is less than 400, all the infected people can recover from the disease in the first 1000 time steps (Fig. 7i) in most simulation cases.

In addition, the effects of infection and recovery probabilities on spread of the disease and the ratio of infected people have also been studied. As expected, increasing the infection probability leads to an increase in the ratio of infected people, while increasing the recovery probability reduces the ratio of infected people. These results are trivial and are not shown here.

### 3.4 Phase diagram and transient dynamics

When people take frequent and short local movements, or the population size is large, the spread of the disease is easier and faster. The ratio of infected people is larger than 10%. Reducing local crowdedness helps decrease
Fig. 7 Bifurcation diagrams and the transition between “living with COVID” and “zero-COVID” states. (a) The bifurcation diagram of infected population ratio influenced by mobility parameter $c_1$. Black dots and line are for “zero-COVID” state. Orange dots and line are for “living with COVID” state. Each dot is the mean result of 50 simulations. (b) The percentage of achieving “zero-COVID” influenced by $c_1$. (c) Variation of the infected population ratio over time in the two states when $c_1 = 0.25$. (d) Bifurcation diagram of infected ratio influenced by the percentage of low mobility people. Gray and light orange dots represent rare cases (1 or 2 out of 50 simulation cases). (e) The percentage of achieving “zero-COVID” influenced by the low mobility people ratio. (f) Variation of the infected ratio over time when the low mobility people ratio is 0.7. (g) Bifurcation diagram of the infected ratio influenced by the population number. (h) The percentage of achieving “zero-COVID” influenced by the population number. (i) Variation of the infected ratio over time when the population number is 400.

Fig. 8 Phase diagram of the transient zero-COVID ratio over time influenced by human mobility and population size. (a) The effect of mobility parameter $c_1$ which represents the traveling distance in the containers. (b) The effect of low mobility people ratio in the population. (c) The effect of the population number.
the ratio of infected to approximately 5%. Then, fluctuations in the system originating from random human travel, infection and recovery behaviors may lead to the disappearance of COVID-19 infection (Figs. 7c, f and i). The probability of zero-COVID-19 and the time it takes are influenced by many factors. In this section, we analyzed the phase diagrams of the ratio of infected people over time influenced by the mobility parameter $c_1$, the people with a low mobility ratio and the population number.

Figure 8a shows that the system reaches the equilibrium state quickly (i.e., either the “zero-COVID” state or the “living with COVID” state) when $c_1$ is larger than 0.4 or smaller than 0.25. When $c_1 \in [0.25, 0.4]$, the system is bistable and the probability of zero-COVID drops significantly with a small increase of $c_1$. The time needed for a thorough recovery from COVID-19 becomes longer. From Fig. 8b, it can be found that having a large portion of people with low mobility in the population (i.e., more than 30%) increases the time needed for zero-COVID. Although the recovery time is longer, the probability of reaching the “zero-COVID” state is higher. When the ratio of people with low mobility is small, the system is likely to be in the “living with COVID” equilibrium state. Moreover, the population number also influences the transient COVID-19 transmission dynamics (Fig. 8c). When the number of people in the population is small (i.e., less than 400), all people can recover from the disease within the first 500 time steps. When the number of people increases, the probability of reaching the zero-COVID state is lower and the population recovery takes a longer time. When the population contains more than 800 people, the newly infected people and the people who have recovered achieve a dynamical equilibrium. The system is in the “live with COVID” state, and more than 20% of people in the population are infected.

4 Discussion

The COVID-19 pandemic has presented an unprecedented challenge and an urgent need for accurate understanding and rapid prediction of disease dynamics on a wide range of scales. As an airborne disease that transmits mainly through close-contact meetings and interactions, peoples’ physical locations and movements are the most fundamental drivers of that drive the spread of COVID-19. However, due to the limited understanding of human mobility patterns, until now, how intertwined long-distance travel and local movements lead to contagion events and virus spread has remained illusive.

In this study, we developed a mobility-influenced COVID-19 transmission model (Mob-Cov) using the agent-based Monte Carlo modeling strategy and the multilevel nested spatial containers. Human mobility behavior is restricted based on a hierarchical structure of spatial containers, corresponding to geographical places from buildings, via streets and cities, to nations and continents. By merging the power law movements within containers and the macroscopic transport between containers, the human travel patterns that are generated are highly realistic, which ensures an accurate study of the transmission dynamics of COVID-19 as influenced by mobility. In addition, due to the flexibility of agent-based modeling strategy, different individual movements and health conditions are considered in the model.

Specifically, we listed a few important findings and numerical results here.

1. Long-distance travels within a small-level container (e.g., a street or a county) help alleviate the local crowdedness of people and reduce infection. For example, when $c_1$ decreases from 1 to 0.5, the outbreak time becomes three times longer.
2. Traveling between levels and large-level containers (e.g., traveling between cities and nations) converts local transmission to global disease spread, facilitating the outbreak of the disease. For example, when $d$ increase from 1 to 2, the outbreak time doubles.
3. The hierarchical geographical structures (i.e., the number of container levels) help reducing the disease transmission. When the number of container levels increases from 3 to 6, the outbreak times becomes approximately 5 times longer.
4. When the ratio of people with low mobility or low infection rate is larger, the transmission of disease is slower. When the population size is large (more crowded), the time it takes to reach outbreak is shorter. But the total infected people ratio at the outbreak point is less influenced by the ratio of people with low mobility and low infection risk and population size.
5. The infection and recovery of individuals lead to bifurcation and the appearance of two dynamic states, the “zero-COVID” state and the “live with
COVID” state. The ability to achieve a zero-COVID state, in which all the infected people have recovered, is largely influenced by human mobility and population number. In general, when the moving distance in the same container is larger ($c_1 < 0.25$), more than 80% of people has low mobility, and the population size is smaller than 400, the probability of achieving a zero-COVID state is 100%.

Overall, the Mob-Cov model can be built upon in several major directions in future works. (1) Infection in a wide range of spatial scales can be considered. Specifically, compared with previous works, infection on a contact network estimated from human GPS data mainly captures the transmission between cities and nations [30–32]. Agent-based models using random walks to describe human movements can only be used to study the microscopic actions within a building or a district [21,22,24,25]. The multilevel hierarchical container structures in Mob-Cov provide a more realistic and accurate description of individuals moving across multiple scales while maintaining a reasonable computational cost. Specifically, nested container structures of human mobility can be estimated from the real-world human GPS data using the maximum likelihood estimator [33]. After constructing the hierarchical container structures, the multistate disease propagation model can be built by defining the infection and recovery properties of individuals. Furthermore, the digital tracing of infected individuals can be input into the model to help identify high-risk areas and populations over a long period of time. (2) Various interventions can also be easily implemented and used to study the impact of policies on the control of the COVID-19 pandemic, especially regarding the effective scales of restriction in these policies. For example, the multiscale Mob-Cov model can be used to investigate the most effective level (i.e., a street or a county) to impose a mobility restriction intervention. (3) In this work, we focused on modeling the multilevel human mobility while implementing only simple rules of disease infection. It would be interesting to further build upon the Mob-Cov model, applying it to complex infection scenarios, including susceptible, exposed, infectious and recovered states. In particular, the susceptible and exposed states can be critical, since the latent period between being infected and becoming infectious has made the disease more contagious. Moreover, whether the epidemic wave changes the nested mobility networks; and how it is influenced by population characteristics (e.g., age, gender, level of urbanization) should be further investigated using our Mob-Cov model.

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**Data availability** Several examples of the data used to generate the figures as well as the description of the data are available at http://pan.dlut.edu.cn/share?id=s1nea9tz2y73. The code used to generate all the data in this manuscript is available at https://github.com/jessychen2018/Mob-Cov.

**Declarations**

**Conflict of interest** The authors declare that they have no conflict of interest.

**Appendix**

The notions variables and parameters used in the Mob-Cov model are shown in Table 2.

| Parameter/Variable | Description |
|--------------------|-------------|
| $d$                | The inverse of the mean moving distance across different levels of containers |
| $c_0$, $c_1$     | Parameters of the long-tail distribution of the moving distance in the same container |
| $L$                | Number of container levels |
| $N_{cont}$        | Number of containers in each level |
| $P_{mi}(t)$       | The probability of agent $i$ taking a movement at time step $t$ |
| $P_{ij}$          | The probability of a healthy agent $j$ being infected by agent $i$ |
| $T_{inf}$         | Infection time period |
| $T_b$             | The outbreak time of the disease |
| $R_i$             | Recovery probability of agent $i$ |
| $P_{lv}(l)$       | The probability of selecting level $l$ during a movement |
| $P_l(k_i)$        | The probability of selecting container $k_i$ in level $l$ |
| $X_i$             | Position of agent $i$ |
| $N_p$             | Number of agents |
| $N_{inf}$         | Number of initially infected agents |
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