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A MULTI-ASPECT AGENT-BASED MODEL OF COVID-19: DISEASE DYNAMICS, CONTACT TRACING INTERVENTIONS AND SHARED SPACE-DRIVEN CONTAGIONS

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ABSTRACT

In the quest to better understand the transmission dynamics of COVID-19 and the strategies to control its impact a wide range of simulation models have been developed. Faced with a novel disease with little-known characteristics and unprecedented impacts, the need arises to model multiple aspects with very dissimilar dynamics in a consistent and formal, but also flexible and quick way to study the combined interaction of these aspects. We present an agent-based model combining kinematic movement of agents, interaction between them and their surrounding space, and centralized control over the entire population. To achieve this, we use and extend the retQSS framework to model and simulate particle systems that interact with geometries. We study different contact tracing strategies and their efficacy in reducing infections in a population going through an epidemic process driven mainly by indoor airborne contagion.

1 INTRODUCTION

The Coronavirus Disease 2019 (COVID-19) pandemic has brought humanity to a state of global alarm, reaching a worldwide scale in an unprecedentedly short time. Unlike the viruses that caused previous pandemics, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has a much higher level of infectivity.

As mass vaccination campaigns are still in progress, several non-pharmaceutical strategies have been proposed in recent months to address different aspects of the pandemic process. Among them, we can find quarantine, isolation of cases, social distancing, epidemic surveillance, etc. There are also many important questions to address to better understand and manage the disease. Some of the most frequently asked questions are: how and when to start and end confinements, how many tests should be carried out, which cohorts to vaccinate first, how many medical personnel would be needed, etc.

Virus outbreak models can be traced back to the Susceptible-Infected-Removable (SIR) model presented in (Kermack and McKendrick 1927). Hundreds of models followed pursuing similar goals, from statistical modeling to agent-based and population-based simulations. Most are used to predict possible outcomes of an entire epidemic process, while some also model possible ways to control the disease through different interventions, evaluating options to make optimal decisions. Fraser et al. (2004) presented a stochastic model to assess intervention strategies. In this work, the authors assess the application of contact tracing, concluding that its effectiveness depends on two aspects: the number of secondary infections generated by each newly infected person (known as the Reproductive Number, R) and the proportion of transmissions that occur before the onset of symptoms. This laid the foundation for many other tools and methodologies that allow users to better understand the dynamics of an epidemic and to study multiple scenarios in which

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different intervention strategies can be tested, like contact tracing, isolation, lockdowns, vaccination, etc. (Kwok et al. 2019).

Since 2020 many studies modeled SARS-CoV-2 infection spanning varied strategies to manage the epidemic. Hellewell et al. (2020) proposed a stochastic transmission model and used it to quantify the effectiveness of contact tracing techniques. They analyzed the efficacy of this strategy as a function of several model parameters such as R, the delay from symptom onset to isolation, the success probability when tracing contacts, the proportion of transmission that occurred before symptom onset, etc. Kretzschmar et al. (2020) evaluated intervention strategies using another stochastic model, highlighting the importance of reducing delays in testing and tracing to effectively control the outbreak. Wallentin et al. (2020) presented an agent-based simulation model that studied different types of lockdown scenarios. They showed that while an extreme lockdown could eliminate the virus in a few months, relaxation of isolation could lead to a second outbreak. Shoukat et al. (2020) used another agent-based model to estimate the reduction of intensive care units and hospital resources required when a population practices self-isolation and how this strategy would also delay the peak of infections.

Several intervention strategies have been proposed to reduce the impact of the pandemic. Pharmaceutical (e.g. vaccination) or non-pharmaceutical (e.g. quarantine) interventions alone are not effective, and it is accepted that a combination of these is most reasonable. The case of lockdowns is particularly complex, as the prolonged closure of businesses and schools can have serious social and economic consequences. However, some other complementary measures can mitigate these side effects while increasing the overall effectiveness of closures. Perhaps the most widely adopted of these strategies is contact tracing. *Contact Tracing* involves tracking, testing, and eventually isolating the close contacts of each confirmed positive case. Despite its appeal, this strategy is often quite expensive, especially when the number of people to track is large.

In this work, we develop an agent-based model that extends the SIR-type epidemiological dynamics with two different but mutually influencing aspects. First, we add the interaction of agents with the physical spaces through which they circulate, which may remain infected for arbitrary periods of time by hosting infected agents. In turn, infected spaces can propagate the virus to susceptible agents entering the space. This is the mechanism underlying airborne contagion via aerosols, considered to be a key factor in the spread of contagious respiratory diseases (Prather et al. 2020; Morawska and Milton 2020), reaching fatal levels in certain cases (e.g. in hospitals or choral events (Miller et al. 2021; Fears et al. 2020)). Second, we model the effects of implementing a contact tracing intervention, whereby each symptomatic agent represents an index case for its network of direct (or secondary) contacts. The latter can be reached by the government, being potentially being tested and isolated to decrease the intensity of the viral spread. We will show how the incorporation of such a feature affects the spread of the disease. Then, we will analyze the impact of contact tracing in the context of different age groups, each of which has different rates of symptomatology and mortality.

In order to deal explicitly with spatial dynamics we adopt a kinetic type of model for particles (agents) moving on a 2D space (Pulvirenti and Simonella 2020; Kuzdeuov et al. 2021).

The rest of the paper is organized as follows: In section 2 we describe a new indoor virus propagation model with contact tracing dynamics. In subsection 2.1 we show the implementation of the SEIRD(AP) model (Susceptible, Exposed, Infected -Asymptomatic or Presymptomatic-, Recovered, Dead) and in subsection 2.2 we extend it with contact tracing of different kinds. Then, in section 3 we perform simulation experiments to assess infection profiles for different combinations of infection probabilities between spaces and agents. Finally, in section 4 we analyze the reduction of spread by applying contact tracing, deciding on the convenience of tracking contacts of contacts depending on the final epidemic size.

2 A PARTICLE-BASED MODEL WITH CELL-DRIVEN DYNAMICS: CONTACT TRACING AND INFECTIONS

We present an implementation of a SIR-like model using retQSS (Santi et al. 2020), a framework for the modeling and simulation of particle systems in reticulated geometries. Particle models in retQSS are described in μ-Modelica (Bergero et al. 2012), a subset of the Modelica language Fritzson (2014), leveraging its expressive power to define spatially explicit kinetic dynamics in a compact and elegant manner. We use this platform to model agents as particles participating in an epidemic process. Agents in a population move within a virtual world represented by a grid of *GxG* cells, each of which is *CxC* in area (arbitrary units). The trajectory of an agent is modeled as a uniform rectilinear motion, with random initial position and direction, bouncing only at the borders of the grid. Finally, agents do not collide or bounce off each other. This setup provides a homogeneous mix of free agents with a uniform probability of visiting all cells in a sufficient time (provided that no external interventions are applied at the population level, nor specific mobility patterns are assigned to certain individuals).

Yet, different motion properties such as position, velocity, and acceleration of each agent can be changed at simulation time. For example, we can define a probability P_{sp} to model a "super spreader" agent A and set its velocity (V_A^{sp}) faster than normal agents, thus increasing the likelihood of interaction (and therefore, of contagion) with other agents. Setting an agent's velocity will also be relevant to model agent isolation.

In our model, agents undergo state changes according to Communicating Finite State Machines (CFSMs) (Brand and Zafiropulo 1983) as shown in Figure 1. We define two different FSMs for an agent: the *epidemiological dynamics* FSM and the *contact tracing dynamics* FSM. We define discrete states that evolve separately although they influence each other, so that the *global state* of an agent is the composition of the *local state* of each FSM.

Cells represent delimited environments with local dynamics. Agents interact with different environments (cells) and with groups of agents within each cell. A cell, in turn, can change its state depending on the state of agents traversing it. This mechanism produces dynamically changing contact networks at two levels: local (spontaneous) networks and global (stable) patterns. With this approach, indirect influence among two agents is allowed via the cell's state (even when the cell is not shared synchronously).

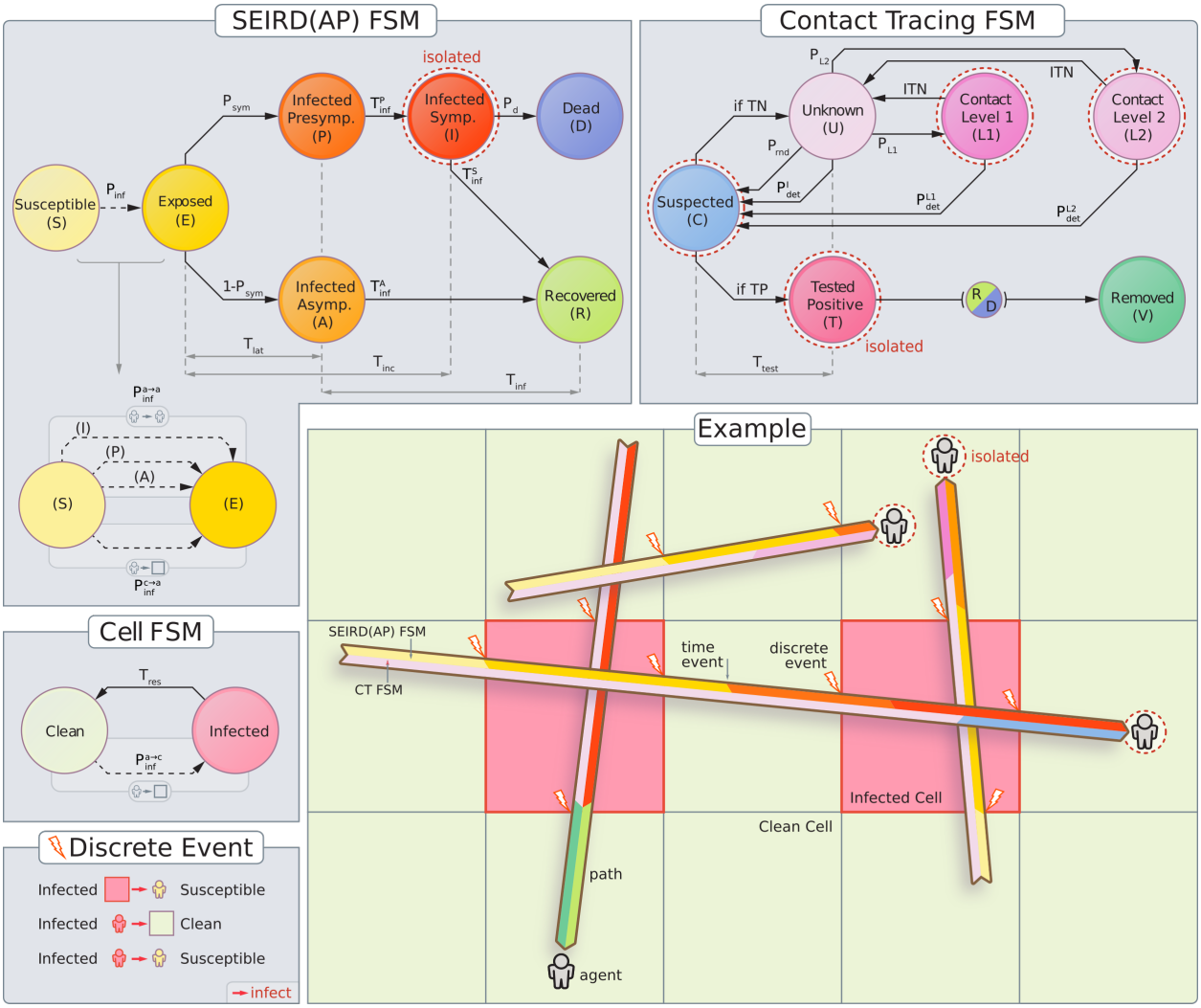
Two different types of events can produce state changes for an agent: *discrete event* and *time event*. A discrete event (or *state* event) occurs when an agent enters a new cell and triggers a set of potential contagions as a consequence of being exposed to the new *local environment*. A time event occurs when a timer elapses its period, representing typical delays of the infection (e.g. latency time) or healthcare logistics (e.g. test time). We also model three age groups for the agents (Young, Adult, and Elderly) since age is a relevant determinant for parameters such as symptomatic rate or mortality rate (Koh et al. 2021).

We will first describe the model regarding epidemiological aspects (SEIRD(AP) FSM) and then describe its extension to incorporate Contact Tracing aspects (CT FSM) highlighting the smooth process of merging novel dynamics incrementally. Finally we analyze the effects of tracking different levels (or rings) of contacts for each *index case* namely *direct contact* (Level 1) and *indirect contact* (Level 2) (also *contact of a contact*).

2.1 The SEIRD(AP) model

Different SIR-like models have been proposed as extensions of the basic SIR structure. In this work we present the SEIRD(AP) model. It includes the Exposed state in addition to the Susceptible, Infected, and Recovered states. Also, it distinguishes between three types of infected contagious states: Asymptomatic (A), Presymptomatic (P), and Infected symptomatic (I). Finally, the model also sets a probability for the agent to die during the symptomatic (I) state. Agents start as Susceptible except for a number I_0 which starts as Infected symptomatic. When Infected agents move, they can infect the cells they pass through and also other agents in those cells. We define three different contagion probabilities for binomial trials, depending on the agent's state: P_{cont}^P for Presymptomatic, P_{cont}^I for Symptomatic and P_{cont}^A for Asymptomatic. If the trial

succeeds, the infection event is triggered. Otherwise, the agent does not infect at all in this cell. Infection consists of two steps: i) the agent infects other agents with probability $P_{inf}^{a \to a}$, computed individually against each agent in the cell, ii) the agent infects the cell as it passes through it with probability $P_{inf}^{a \to c}$. A cell remains infected for T_{res} units of residual time, then restoring to a Clean state. If an agent infects an already infected cell, T_{res} starts over.



$P_{inf}^{a \to a}$	Infection probability (agent to agent)	P_{det}^{I}	Detection probability (symptomatic)	P_d	Probability of death	P_{rnd}	Random detection probability
$P_{inf}^{a \to c}$	Infection probability (agent to cell)	T_{inf}^{P}	Presymptomatic infection time	P_{L1}	Probability of L1 contact success	T_{test}	Test delay time
$P_{inf}^{c \to a}$	Infection probability (cell to agent)	T_{inf}^{I}	Symptomatic infection time	P_{L2}	Probability of L2 contact success	TP	Tested positive
P_{sym}	Symptomatic probability	T_{inf}^{A}	Asymptomatic infection time	P_{det}^{L1}	Detection probability (contact level 1)	TN	Tested negative
T_{inc}	Incubation time	T_{res}	Cell residual infection time	P_{det}^{L2}	Detection probability (contact level 2)	ITN	Index case tested negative

Figure 1: Finite State Machines defining the main dynamics of the model: SEIRD(AP), Contact Tracing and Cells. Illustrative example with 4 agents (1 recovered, 3 infected) and 15 cells.

When a Susceptible agent enters an Infected cell it can get infected with a cell-to-agent probability $P_{inf}^{c \to a}$, evolving to the Exposed (E) state. After a latency period (T_{lat}) it becomes infected (with ability to infect) in two possible conditions: infected Asymptomatic (A) or infected Presymptomatic (P) depending on a binomial distribution. We define this probability depending on the age group (P_{sym}^{Y} , P_{sym}^{A}, P_{sym}^{E} for Young, Adult and Elderly agents respectively). Presymptomatic agents evolve into the Infected symptomatic

state (I) after a period $T^P_{inf} = T_{inc} - T_{lat}$, where T_{inc} is the incubation time (time since exposure to onset of symptoms).

When an agent enters state (I), there is a detection probability P^I_{det} that reflects how likely it is that the agent will come into contact with the health system and be isolated for a period T_{isol}. An isolated agent remains static and cannot infect other agents. When its infection period T^S_{inf} elapses, the agent may evolve to Recovered (R) or Dead (D) according to a probability of death that depends on the age group: P^Y_d, P^A_d and P^E_d. On the other hand, if the infected agent is Asymptomatic, it recovers after a period T^A_{inf}.

In Listing 1 we show an excerpt of the initialization phase in the SEIRD(AP) model (μ-Modelica language), using the retQSS particle-geometry framework, where position and velocity in the 2D plane are defined by Newtonian laws of motion. The initial conditions of the algorithm are defined and arrays are used to store characteristic time periods (time arrays) for the agents and cells, defining when *time events* occur, and triggering specific state changes according to Figure 1. All cells are initialized in a Clean state, and all agents are initialized as Susceptible, except for an amount I_0 of Exposed agents for whom we set their latency time T_{lat}.

Listing 1: Initialization of the SEIRD(AP) model (code excerpt)

```
model SEIRD(AP)
equation  // Agent kinetic dynamics
  for i in 1:N loop  // Derivatives of position and velocity in the x-y plane
    der(x[i]) = vx[i]; der(y[i]) = vy[i];
    der(vx[i]) = 0; der(vy[i]) = 0; // No acceleration
  end for;
initial algorithm // system initial conditions
  geometry_gridSetUp(G,C) // Size of the grid and cells
  for i in 1:CELLS loop   // Initialize the grid of cells
    cellInfectionFinishTime[i] = 0
    cellStatus[i] = CLEAN
  end for;
  for i in 1:N loop // Set random initial positions and velocities
    (x[i],y[i]) = randomXYPoint(G);
    (vx[i],vy[i]) = randomXYVelocity(V,P_SS,V_SS);
  end for;
  for i in 1:N loop // Set time arrays for SEIRD(AP) state change
    if i <= I_0 then // Initialize as Infected the first I_0 agents
      latencyStartTime[i] := 0;
    else
      latencyStartTime[i] := ∞;
    end if;
    infectionStartTime[i] := ∞;
    symptomsStartTime[i] := ∞;
    infectionFinishTime[i] := ∞;
    agentStatus[i] = SUSCEPTIBLE;
    setAgeGroupProperties(i,P_young,P_adult,P^Y_sym,P^A_sym,P^E_sym,P^Y_d,P^A_d,P^E_d);
  end for;
```

The main SEIRD(AP) dynamics are shown in Listing 2. The **when** clause in Modelica declares event-driven behavior checking for a logical condition involving both discrete and continuous variables. If combined with the **time** variable we can define time-driven events (i.e. time events) to check for time conditions that trigger state changes in the FSMs. The **particle_nextCrossingTime** function provided by retQSS evaluates the next time an agent crosses from one cell to another. The **onNextCross** function triggers the infection dynamics when the traveling agent enters the cell: i) if infected (states A, P, or I) it could infect the cell, ii) if infected, it could infect other agents (in state S), and iii) if susceptible, it could get infected by an infected cell.

This function could also return values that change the agent's time array or direction (if it needs to bounce on a border). Newly infected agents start their latency time (in their time arrays) and change state from Susceptible to Exposed.

Other functions like **onLatencyStart**, **onInfectionStart**, **onSymptomsStart** and **onInfectionEnd** define the state changes of an agent during its infection process and return updated values for future events.

For instance, when an agent reaches its infectionStartTime, onInfectionStart function is called, changing the agent state from Exposed to Presymptomatic or Asymptomatic, depending on its symptomatic probability

(which depends on age: P^Y_{sym}, P^A_{sym}, P^E_{sym} for Young, Adult, and Elderly). The Modelica code resembles closely the formal FSM definition in Figure 1.

Listing 2: SEIRD(AP) epidemiological dynamics. Main algorithm (code excerpt)

```
algorithm
  for i in 1:N loop // Iterate over each agent
    when time > particle_nextCrossingTime(i,x[i],y[i],vx[i],vy[i]) then // Agent enters a new cell -> Update
        properties
      (latencyStartTime,cellInfectionFinishTime,updateVx,updateVy) = onNextCross(time,i,P^P_cont,P^I_cont,P^A_cont,P^{a->a}_inf,P^{a->c}_inf,P^{c->a}_inf,T_res
          );
      if updateVx then // If the agent reached a border, make it bounce
        reinit(vx[i],-vx[i]);
      elseif updateVy then
        reinit(vy[i],-vy[i]);
      end if;
    end when;
    when time > latencyStartTime[i] then // Agent exposed -> Change to Exposed state
      infectionStartTime[i] = onLatencyStart(time,i,T_lat);
    end when;
    when time > infectionStartTime[i] then // Change to Asymptomatic or Presymptomatic
      (infectionFinishTime[i],symptomsStartTime[i]) = onInfectionStart(time,i,T^A_inf,T^P_inf);
    end when;
    when time > symptomsStartTime[i] then // Change to Symptomatic (only from Presymptomatic)
      infectionFinishTime[i] = onSymptomsStart(time,i,T^S_inf,P^S_det);
      if not shouldMove(i) then
        reinit(vx[i],0); reinit(vy[i],0);
      end if;
    end when;
    when time > infectionFinishTime[i] then // Change to Recovered (from Symptomatic or Asymptomatic)
      onInfectionEnd(time,i);
      if shouldMove(i) then
        (ux,uy) := randomXYVector(DEFAULT_VELOCITY);
        reinit(vx[i],ux); reinit(vy[i],uy);
      end if;
    end when;
  end for;
  for i in 1:CELLS loop // Iterate over cells to clean if infection time expired
    when time > cellInfectionFinishTime[i] then
      cellStatus[i] = CLEAN;
    end when;
  end for;
```

2.2 Including Contact Tracing Dynamics

Each pair of agents that share a cell become mutual contacts and are included in each other's contact list which lasts for a limited amount of time. As shown in Figure 1, according to the Contact Tracing FSM, agents start in Unknown state which means that nothing is known about the conditions of the agents. When an Unknown agent becomes Symptomatic there is a probability (P^I_{det}) of detection. Now, according to the contact tracing intervention strategy, when it is detected, it changes its state to Suspected in the Contact Tracing FSM. Immediately, a maximum number of contacts C_{L1} (Level 1 contacts) are queued up to be contacted and, after a tracing time T_{L1}, they are successfully contacted with a probability P_{L1}. This reflects the chances to contact them by calling or messaging. Each contacted agent changes its state to Contact Level 1 and the process is repeated for a maximum number of contacts C_{L2} (Level 2 contacts) after a time T_{L2} and with probability P_{L2}. In addition, when agents are in L1 and L2 states, also have another probability of detection (P^{L1}_{det}) and (P^{L2}_{det}), modeling the effect of taking medical care and self isolate.

Note that these probabilities are defined independently of the P^I_{det}. Agents can also change from Unknown to Suspected with a random detection probability P_{rnd} (this only happens if they are not in an Infected state). Once agents in Suspected state are tested (after a test delay time T_{test}), they can change to Tested Positive state or again to Unknown depending on the state in the SEIRD(AP) state machine. If the agent changes to Tested Positive it is automatically isolated during a time T_{isol}. If an agent gets Infected and then gets Recovered or Dead in the SEIRD(AP) FSM, it is set to the Removed (V) state in the Contact Tracing FSM. The implementation of Contact Tracing intervention strategy is shown in Listings 3 and 4.

Listing 3: Adding Contact Tracing dynamics. Extension of the initialization model in Listing 2. Newly added code highlighted in red. Previous code not shown (placeholders in blue)

```
equation
  (same code as before) // Define derivatives of the position and velocity
initial algorithm
  (same code as before) // Initialize the grid of cells
  (same code as before) // Set random positions and velocities
  (same code as before) // Set time arrays for SEIRD(AP) state change
  for i in 1:N loop     // Set time arrays for Contact Tracing state change
    testResultTime[i] := ∞;
    level1ContactTime[i] := ∞;
    level2ContactTime[i] := ∞;
    isolationFinishTime[i] := ∞;
    agentTrackingStatus[i] = UNKNOWN;
  end for;
```

Listing 4: Adding Contact Tracing dynamics. Extension of the main algorithm in Listing 3. Newly added code highlighted in red. Previous code not shown (placeholders in blue)

```
algorithm
  for i in 1:N loop
    (same code as before) // Update properties when an agent enters a new cell
    (same code as before) // Change to Exposed state
    (same code as before) // Change to Asymptomatic or Presymptomatic
    (same code as before) // Change to Recovered (from Symptomatic or Asymptomatic)
    when time > symptomsStartTime[i] then // Change to Symptomatic (only from Presymptomatic)
      (infectionFinishTime[i], testResultTime[i], level1ContactTime) := onSymptomsStart(time,i,T_inf^S,P_det^S,P_det^L1,P_det^L2,T_test);
      if not shouldMove(i) then
        reinit(vx[i],0); reinit(vy[i],0);
      end if;
    end when;
    when time > testResultTime[i] then // Test delay ends, change to Tested Positive or Unknown depending on test
        result
      isolationFinishTime[i] := onTestResult(time,i,TESTED_POSITIVE_ISOLATION_TIME);
      if shouldMove(i) then            // If test result is positive, start moving again
        (ux,uy) := randomXYVector(DEFAULT_VELOCITY);
        reinit(vx[i],ux); reinit(vy[i],uy);
      end if;
    end when;
    when time > level1ContactTime[i] then  // Contact level 1 is isolated
      (isolationFinishTime[i],level2ContactTime) := onLevel1Contact(time,i,T_isol);
      reinit(vx[i],0); reinit(vy[i],0);
    end when;
    when time > level2ContactTime[i] then  // Contact level 2 is isolated
      isolationFinishTime[i] := onLevel2Contact(time,i,T_isol);
      reinit(vx[i],0); reinit(vy[i],0);
    end when;
    when time > isolationFinishTime[i] then // When isolation ends, start moving again
      release := onIsolationFinish(time,i);
      if shouldMove(i) then
        (ux,uy) := randomXYVector(V);
        reinit(vx[i],ux); reinit(vy[i],uy);
      end if;
    end when;
  end for;
```

Time delays and probabilities mentioned above are parameters of the model and their values are presented in Table 1 (Appendix A). We fixed some parameters and swept others to analyze scenarios.

3 SIMULATION OF A SHARED ROOM-DRIVEN CONTAGION PROCESS

We analyze the emergence of contagion patterns as a disease spreads due to sharing closed (or semi-open) spaces, leveraging the cell-mediated contagion dynamics described in the previous section. This scenario is consistent with the latest evidence suggesting the strong influence of airborne contagions in SARS-CoV-2 (Prather et al. 2020; Morawska and Milton 2020).

We study the effect of varying the infection probabilities $P_{inf}^{a\to a}$ (agent-to-agent), $P_{inf}^{a\to c}$ (agent-to-cell) and $P_{inf}^{c\to a}$ (cell-to-agent) described before. Collisions between particles are not considered in our model, as agent-to-agent interactions are triggered in a 1-to-many scheme each time a new agent enters a cell. Higher values of $P_{inf}^{a\to a}$ depict situations when individuals are less likely to wear face masks or obey social

distancing rules. Higher values of $P_{inf}^{a\to c}$ or $P_{inf}^{c\to a}$ depict cases of poorly ventilated rooms. In Figure 2, we show the results a parameter sweeping experiments of these three probabilities, presented as heatmaps for the reproduction number R in each scenario (panels **a**, **b**, **c**, and **d**).

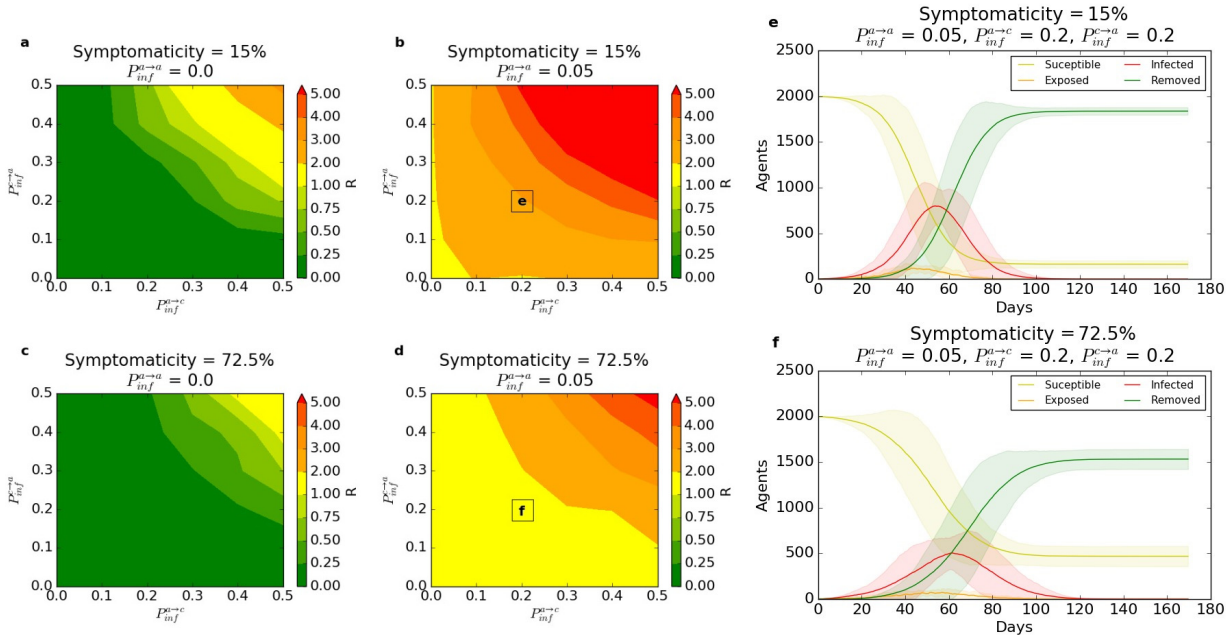


Figure 2: Average Reproductive Number R for scenarios with different probabilities of infection (panels **a** to **d**), and temporal evolution of selected compartments for specific scenarios (panels **e** and **f**).

In each simulation we calculate the overall average R as follows: first, we calculate the number of secondary infections produced by each agent during its infective period. Then we take the average of these values over all agents for the full simulation time. In turn, each point in the heatmap is calculated as the average of 10 repetitions of the stochastic simulation with the same model parameters.

The asymptomatic infection rate is still under debate. Many studies have attempted to estimate it and the variability is very high. Studies on contact tracing estimate an average asymptomatic rate of 20% (Syangtan et al. 2021), while others reported results of more than 50% (Byambasuren et al. 2020) and up to 80% (Hu et al. 2020). For this reason, we decided to simulate both low and high asymptomatic rates.

We show on each row two scenarios with different proportions of symptomatic agents: 15% (low) and 72.5% (high). These percentages are the result of the proportion of Young, Adult, and Elderly agents in the population and their respective symptomatic probabilities. For this experiment, we do not sweep the age group probabilities, and set them as $P_{young} = 0.25$ and $P_{adult} = 0.5$. As agents not showing any symptoms do not self-isolate, it is expected that a high proportion of asymptomatic agents would cause the infection to spread faster. In the bottom row, we tested a high proportion of symptomatic agents (72.5%) where the infection consistently spreads at lower rates (smaller R values for the same parameters, as compared to the top row). In general, when these two parameters increase, the average R also increases.

In the first column we assigned $P_{inf}^{a\to a} = 0$ (i.e. contagion is purely airborne, driven by infected cells) and we still found scenarios with $R > 1$. This suggests that even if agents do not infect each other directly, we can still find an epidemic process purely mediated by the concentration of aerosols in shared spaces. In the second column an agent-to-agent infection probability $P_{inf}^{a\to a} = 0.05$ is considered. As expected, this increased the average R consistently. Surprisingly, we could not assign values above $P_{inf}^{a\to a} = 0.1$ without reaching unrealistically high R values (not shown in the figure).

Finally, in order to find suitable values for these parameters, we looked for configurations with average values of R between 2.5 and 3.0. As this range can be met by several combinations of parameter values, we decided to choose the lowest possible values. From now on we will assume $P_{inf}^{a\to a} = 0.05$, $P_{inf}^{c\to a} = 0.2$, $P_{inf}^{a\to c} = 0.2$. As an example, panels **e** and **f** in Figure 2 show the time evolution of Susceptible, Exposed, Infected (all types) and Removed (Recovered and Dead) states of the SEIRD(AP) model for the chosen set of default parameters (highlighted in panels **b** and **d**).

4 SIMULATION OF CONTACT TRACING STRATEGIES AND DIFFERENT AGE GROUPS

We analyze contact tracing and isolation strategies on top of the SEIRD(AP) dynamics studied in the previous section. We analyze two different aspects of these interventions: i) the maximum number of contacts to trace for each suspected index case, both at Level 1 (C_{L1}, direct contact) and Level 2 (C_{L2}, contact of a contact) and ii) the delay it takes for an agent to isolate since it is contacted, also both at Level 1 (T_{L1}) and Level 2 (T_{L2}). We performed parameter sweeping experiments to estimate the resources needed for this intervention strategy, such as the number of calls per day required to effectively decrease the reproductive number R, or the number of laboratory tests needed to process the samples fast enough.

As shown in Figure 3, we evaluated the simulation outcomes in terms of how many agents were infected at the end of the experiment when the outbreak ends. This is often referred to as the Final Epidemic Size (FES) and is presented as a percentage of the total population. We simulated three scenarios, setting different proportions of Young, Adult, and Elderly agents, and studied how this structure impacts in the efficiency of the contact tracing strategy. The scenarios are defined as *young*: $(P_{young}, P_{adult}) = (0.5, 0.25)$, *adult*: $(P_{young}, P_{adult}) = (0.25, 0.5)$ and *elderly*: $(P_{young}, P_{adult}) = (0.25, 0.25)$. Only two parameters differ between age groups: symptomatic probability and probability of death. As expected, FES is consistently higher for a younger population structure because of their lower symptomatic probability. The larger the proportion of asymptomatic agents, the lower the number of isolations, which results in higher FES values.

The results do not differ greatly between Adult and Elderly populations, since their asymptomatic probabilities are similar. According to the scenarios analyzed, we can conclude that L2 contacts (contacts of contacts) should not be reached because investing resources in this mechanism is not efficient, leading to consistently higher values of FES (implying more hospitalized and deceased agents). Even when trying to reach only one L2 contact for each L1 contact (panels **d**, **e**, and **f**) the process ends with a higher proportion of infections as compared to the strategy where no L2 contacts are considered (panels **a**, **b**, and **c**).

5 DISCUSSION

Lockdowns as the only type of control strategy are a very traumatic type of intervention from the economic point of view (Ahir et al. 2020; Bai et al. 2020). Therefore, it becomes necessary to develop complementary strategies to slow the spread of the virus. Even when agents are committed to abiding by preventive measures (e.g. using facemasks and maintaining social distancing) the effects of aerosols in poorly ventilated rooms can still lead to strong contagion processes. We verified this effect in our model, supporting the increasing evidence that points to the transmission of COVID-19 occurring mostly in indoor environments via aerosols. Contact tracing is also considered a key type of intervention strategy, though its efficiency decreases noticeably when contact delays increase (Hellewell et al. 2020; Kretzschmar et al. 2020).

We show this inverse relation happens when FES is less than 40%. But for FES higher than 50%, the contact tracing delay plays a lesser role. We think this could be caused by the effect of cell-to-particle contagions, as the viral load of an agent that infected a cell (via aerosols) can last beyond the moment where the agent is detected and isolated. Further analysis is needed about the relation between contact tracing and cell-mediated contagion.

On the other hand, we conclude that the number of contacts traced per index case is a key parameter to optimize the strategy. Consistently with other studies (He et al. 2020; Moghadas et al. 2020) we verified that asymptomatic and presymptomatic transmission are key factors fueling the spread of the virus.

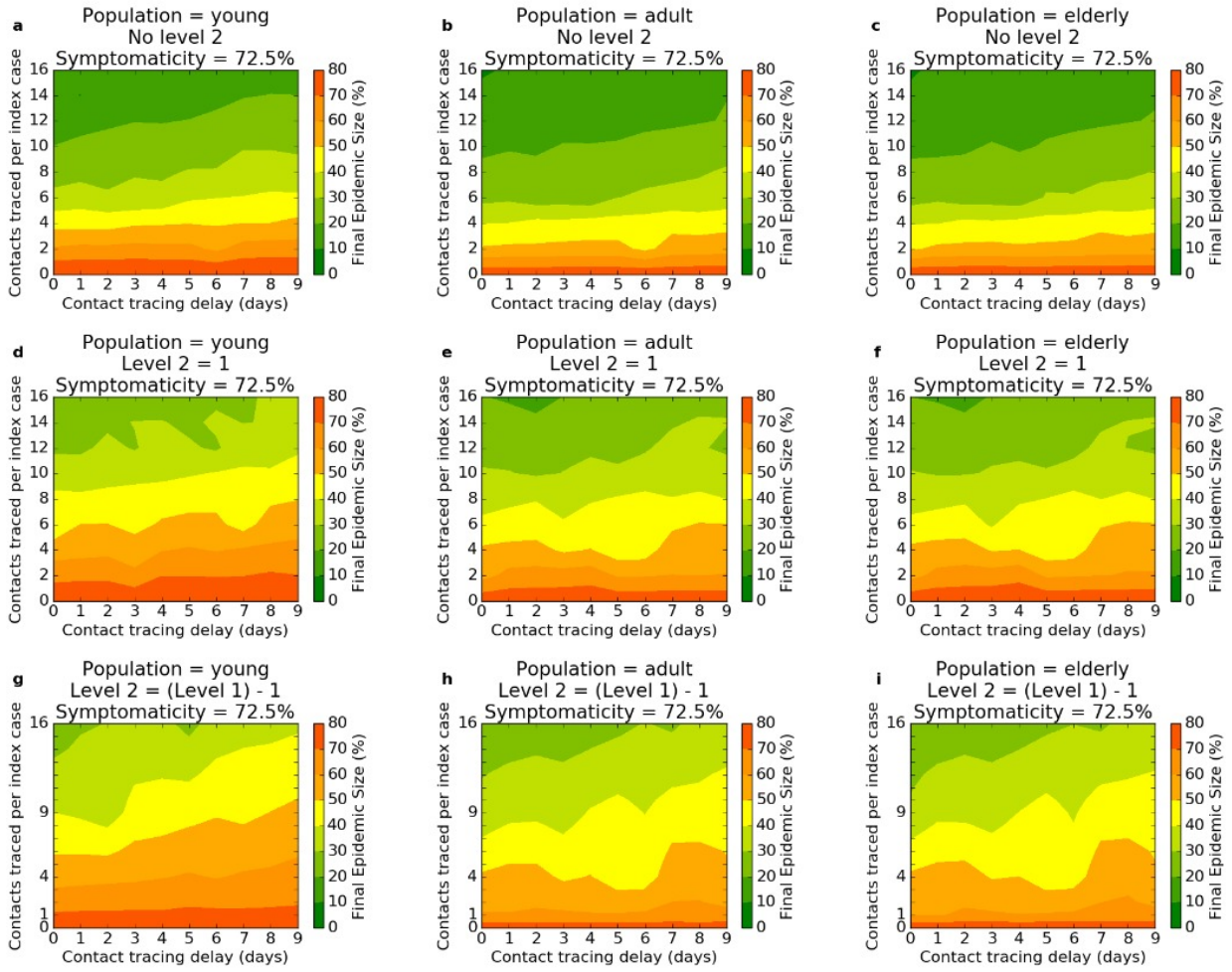


Figure 3: Final Epidemic Size (FES) for varied contact tracing strategies and different age groups.

6 CONCLUSIONS

In this work, we extended the retQSS particle-geometry modeling and simulation framework to analyze varied types of dynamics relevant to the transmission of the SARS-CoV-2 virus. We were able to build compact, easy-to-interpret specifications of hybrid dynamics combining a continuous subsystem (the kinetic motion of particles), very frequent discrete events (the cell boundary crossings), and a complex set of timed events linked to mutually influencing Finite State Machines. By adopting a Modelica-based specification, the intricacies of solving numerically all possible interactions between continuous, discrete event, and time event dynamics are hidden from the modeler, which greatly simplifies his or her tasks, facilitating interdisciplinary work on complex models. We combined diverse domains of concern, such as an epidemic process (the SEIRD(AP) model), a public policy (Contact Tracing), and the interaction of particle-like agents simulating contagion through a cell-mediated mesoscopic process. Our experimental results are consistent with other works in the literature, suggesting that the analysis performed is useful to understand effects of contact tracing strategies in epidemics driven by airborne infections.

We plan to extend this work by conducting more comprehensive parameter sweep experiments to assess the sensitivity of the model, use more specific probability distributions for intervals characteristic of the stages of infection (incubation, pre-syntomatic, etc.), and include other non-pharmaceutical interventions such as intermittent lockdowns.

A APPENDIX

Table 1: Parameters of the model.
U(a,b): Uniform distribution on the interval. [a-b]: Interval of values. [a,b,...]: Enumeration of values.
T: Type of usage (F=Fixed, S=Sweep).

Parameter	Symbol	Value	Units	T	Parameter	Symbol	Value	Units	T
Environment					Contact remaining prob. decay	D_{rem}	0.95	Factor	F
Total number of agents	N	2000	Agents	F	Test delay time	T_{test}	U(0.5,2)	Days	F
Initial infected agents	I_0	5	Agents	F	*SEIRD(AP)*				
Grid size (GxG)	G	10	Cells	F	Infection prob. (agent to agent)	$P_{inf}^{a\to a}$	[0,0.05]	Prob.	S
Cell size (CxC)	C	4	Distance	F	Infection prob. (agent to cell)	$P_{inf}^{a\to c}$	[0-0.5]	Prob.	S
Default velocity	V	1	Speed	F	Infection prob. (cell to agent)	$P_{inf}^{c\to a}$	[0-0.5]	Prob.	S
Population					Cell residual time	T_{res}	U(0.5,1)	Days	F
Super spreader prob.	P_{SS}	0.05	Prob.	F	Symp. prob. (young)	P_{sym}^{Y}	[0.0,0.5]	Prob.	S
Super spreader velocity	V_{SS}	4	Speed	F	Symp. prob. (adult)	P_{sym}^{A}	[0.2,0.8]	Prob.	S
Young prob.	P_{young}	[0.25,0.5]	Prob.	S	Symp. prob. (elderly)	P_{sym}^{E}	[0.2,0.8]	Prob.	S
Adult prob.	P_{adult}	[0.25,0.5]	Prob.	S	Contagion prob. (presymp.)	P_{cont}^{P}	0.5	Prob.	F
Contact Tracing					Contagion prob. (symp.)	P_{cont}^{I}	1.0	Prob.	F
Random detection prob.	P_{rnd}	0.01	Prob.	F	Contagion prob. (asymp.)	P_{cont}^{A}	0.5	Prob.	F
Symp. detection prob. (L1)	P_{det}^{L1}	0.9	Prob.	F	Latency time	T_{lat}	U(1,3)	Days	F
Symp. detection prob. (L2)	P_{det}^{L2}	0.9	Prob.	F	Symp. detection prob.	P_{det}^{I}	0.9	Prob.	F
Contacts traced (L1)	C_{L1}	[0-16]	Indiv.	S	Isolation time	T_{isol}	20	Days	F
Contacts traced (L1)	C_{L2}	[0-16]	Indiv.	S	Infection time (presymp.)	T_{inf}^{P}	U(4,7)	Days	F
Contact success prob. (L1)	P_{L1}	0.95	Prob.	F	Infection time (symp.)	T_{inf}^{I}	U(8,11)	Days	F
Contact success prob. (L2)	P_{L2}	0.95	Prob.	F	Infection time (asymp.)	T_{inf}^{A}	U(12,18)	Days	F
Contact delay time (L1)	T_{L1}	[0-9]	Days	S	Death prob. (young)	P_{d}^{Y}	0.0	Prob.	F
Contact delay time (L2)	T_{L2}	[0-9]	Days	S	Death prob. (adult)	P_{d}^{A}	0.005	Prob.	F
Contact remaining prob. base	P_{rem}	0.95	Prob.	F	Death prob. (elderly)	P_{d}^{E}	0.03	Prob.	F

REFERENCES

Ahir, H., N. Bloom, and D. Furceri. 2020. "Global uncertainty related to Coronavirus at Record High". *IMF Blog. https://blogs.imf.org/2020/04/04/ global-uncertainty-related-to-coronavirus-at-record-high/, accessed 4th April 2020*.

Bai, H. M., A. Zaid, S. Catrin, K. Ahmed, and A. Ahmed. 2020. "The socio-economic implications of the coronavirus pandemic (COVID-19): A review". *Int. J. Surg.* 8(4):8–17.

Bergero, F., X. Floros, J. Fernández, E. Kofman, and F. E. Cellier. 2012. "Simulating Modelica models with a stand-alone quantized state systems solver". In *Proceedings of the 9th International MODELICA Conference, September 3-5, Munich, Germany*, 237–246.

Brand, D., and P. Zafiropulo. 1983, April. "On Communicating Finite-State Machines". *J. ACM* 30(2):323–342.

Byambasuren, O., M. Cardona, K. Bell, J. Clark, M.-L. McLaws, and P. Glasziou. 2020. "Estimating the extent of asymptomatic COVID-19 and its potential for community transmission: systematic review and meta-analysis". *Official Journal of the Association of Medical Microbiology and Infectious Disease Canada* 5(4):223–234.

Fears, A. C., W. B. Klimstra, P. Duprex, A. Hartman, S. C. Weaver, K. Plante, D. Mirchandani, J. Plante, P. V. Aguilar, D. Fernandez et al. 2020. "Comparative dynamic aerosol efficiencies of three emergent coronaviruses and the unusual persistence of SARS-CoV-2 in aerosol suspensions". *medRxiv. https://www.medrxiv.org/content/10.1101/2020.04.13.20063784v1, accessed 29th February 2021*.

Fraser, C., S. Riley, R. M. Anderson, and N. M. Ferguson. 2004. "Factors that make an infectious disease outbreak controllable". *Proceedings of the National Academy of Sciences* 101(16):6146–6151.

Fritzson, P. 2014. *Principles of Object Oriented Modeling and Simulation with Modelica 3.3: A Cyber-Physical Approach*. John Wiley & Sons.

He, X., E. H. Lau, P. Wu, X. Deng, J. Wang, X. Hao, Y. C. Lau, J. Y. Wong, Y. Guan, X. Tan et al. 2020. "Temporal dynamics in viral shedding and transmissibility of COVID-19". *Nature medicine* 26(5):672–675.

Hellewell, J., S. Abbott, A. Gimma, N. I. Bosse, C. I. Jarvis, T. W. Russell, J. D. Munday, A. J. Kucharski, W. J. Edmunds, F. Sun et al. 2020. "Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts". *The Lancet Global Health* 8(4):e488–e496.

Hu, Z., C. Song, C. Xu, G. Jin, Y. Chen, X. Xu, H. Ma, W. Chen, Y. Lin, Y. Zheng et al. 2020. "Clinical characteristics of 24 asymptomatic infections with COVID-19 screened among close contacts in Nanjing, China". *Science China Life Sciences* 63(5):706–711.

Kermack, W. O., and A. G. McKendrick. 1927. "A Contribution to the Mathematical Theory of Epidemics". *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character* 115(772):700–721.

Koh, H. K., A. C. Geller, and T. J. VanderWeele. 2021. "Deaths from COVID-19". *JAMA* 325(2):133–134.

Kretzschmar, M. E., G. Rozhnova, M. C. J. Bootsma, M. van Boven, J. H. H. M. van de Wijgert, and M. J. M. Bonten. 2020. "Impact of delays on effectiveness of contact tracing strategies for COVID-19: a modelling study". *The Lancet Public Health* 5(8):e452 – e459.

Kuzdeuov, A., A. Karabay, D. Baimukashev, B. Ibragimov, and H. A. Varol. 2021. "A Particle-Based COVID-19 Simulator with Contact Tracing and Testing". *IEEE Open Journal of Engineering in Medicine and Biology* 2:111–117.

Kwok, K. O., A. Tang, V. W. Wei, W. H. Park, E. K. Yeoh, and S. Riley. 2019. "Epidemic models of contact tracing: systematic review of transmission studies of severe acute respiratory syndrome and Middle East respiratory syndrome". *Computational and structural biotechnology journal* 17:186–194.

Miller, S. L., W. W. Nazaroff, J. L. Jimenez, A. Boerstra, G. Buonanno, S. J. Dancer, J. Kurnitski, L. C. Marr, L. Morawska, and C. Noakes. 2021. "Transmission of SARS-CoV-2 by inhalation of respiratory aerosol in the Skagit Valley Chorale superspreading event". *Indoor air* 31(2):314–323.

Moghadas, S. M., M. C. Fitzpatrick, P. Sah, A. Pandey, A. Shoukat, B. H. Singer, and A. P. Galvani. 2020. "The implications of silent transmission for the control of COVID-19 outbreaks". *Proceedings of the National Academy of Sciences* 117(30):17513–17515.

Morawska, L., and D. K. Milton. 2020. "It is time to address airborne transmission of coronavirus disease 2019 (COVID-19)". *Clinical Infectious Diseases* 71(9):2311–2313.

Prather, K. A., L. C. Marr, R. T. Schooley, M. A. McDiarmid, M. E. Wilson, and D. K. Milton. 2020. "Airborne transmission of SARS-CoV-2". *Science* 370(6514):303–304.

Pulvirenti, M., and S. Simonella. 2020. "A kinetic model for epidemic spread". *Mathematics and Mechanics of Complex Systems* 8(3):249–260.

Santi, L., J. Fernández, E. Kofman, and R. Castro. 2020, June. "retQSS: A Novel Methodology for Efficient Modeling and Simulation of Particle Systems in Reticulated Geometries". *arXiv e-prints*:arXiv:2006.05495. https://arxiv.org/abs/2006.05495.

Shoukat, A., C. R. Wells, J. M. Langley, B. H. Singer, A. P. Galvani, and S. M. Moghadas. 2020. "Projecting demand for critical care beds during COVID-19 outbreaks in Canada". *Cmaj* 192(19):E489–E496.

Syangtan, G., S. Bista, P. Dawadi, B. Rayamajhee, L. B. Shrestha, R. Tuladhar, and D. R. Joshi. 2021. "Asymptomatic SARS-CoV-2 carriers: a systematic review and meta-analysis". *Frontiers in public health* 8:1066.

Wallentin, G., D. Kaziyeva, and E. Reibersdorfer-Adelsberger. 2020. "COVID-19 intervention scenarios for a long-term disease management". *International journal of health policy and management* 9(12):508–516.

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