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

Esteban Lanzarotti
Lucio Santi
Rodrigo Castro

ICC-CONICET
FCEyN, UBA
Ciudad Universitaria, Pabellón 1
C1428EGA, Buenos Aires, ARGENTINA

Francisco Roslan
Leandro Groisman

Departamento de Computación
FCEyN, UBA
Ciudad Universitaria, Pabellón 1
C1428EGA, Buenos Aires, ARGENTINA

ABSTRACT

In the quest to better understand the epidemic dynamics of COVID-19 and possible strategies to mitigate its impact, a wide range of simulation models have been developed for various purposes. Faced with a novel disease with little-known characteristics and an unprecedented impact, the need arises to model multiple aspects with very dissimilar dynamics in a consistent, formal, yet flexible and quick way, in order to then study the combined interaction of these dynamics. We present an agent-based model combining kinematic movement of agents, interaction between them and their surrounding space and a top-down control over the entire population. To achieve this, we extend the retQSS framework to model and simulate particle systems interacting with geometries. In this work, we study different contact tracing strategies and their efficacy in a population undergoing an epidemic process driven mainly by airborne infections in indoor environments.

1 INTRODUCTION

The COVID-19 pandemic disease has put humanity on global alarm, reaching worldwide scale in an unprecedented short time. Unlike previous pandemics, SARS-CoV-2 surprised with a high level of infectiousness.

As the execution of a massively effective vaccination campaign is still pending, several non-pharmaceutical strategies have been proposed over the last months to address different aspects of the pandemic process. Along these, we can find quarantine, case isolation, social distancing, epidemic surveillance, etc. There are also many important questions to address in order to better understand and manage the disease. Some of the most frequently asked questions are: how and when to start and end a confinement, how many tests should be carried out, which cohorts to vaccinate first, how much medical personnel would be needed, etc. For these types of questions many efforts have been devoted to provide answers by means of simulation models to evaluate possible future scenarios.

Models of virus outbreaks date back to the SIR model presented in Kermack and McKendrick (1927). Hundreds of models followed until the present pursuing similar goals, from statistical modeling to agent-based and population-based simulations Vynnycky and White (2010). 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 new infected case (known as the Reproductive Number, R) and the proportion of transmissions that occur before symptoms onsets. These discoveries laid the foundations for many other tools and methodologies

that allow users to understand infection dynamics and test multiple scenarios where different intervention strategies can be tested, like contact tracing, isolation, lockdowns, vaccination, etc. Kwok et al. (2019).

In 2020 many studies modeled SARS-CoV-2 infection outbreaks including different ways of intervening the epidemic. Hellewell et al. (2020) proposed an stochastic transmission model and used it to quantify the effectiveness of contact tracing techniques. They analyzed the feasibility of being successful when applying this strategy as a function of several parameters of the model like the R, the delay from symptom onset to isolation, the probability of tracing contacts, the proportion of transmission that occurred before symptom onset, etc. Kretzschmar et al. (2020) assessed intervention strategies with another stochastic model highlighting the importance of testing and tracing delays to effectively control the outbreak. Wallentin et al. (2020) presented an agent based simulation model studying 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) showed another agent based model to project the intensive care units required with and without self isolation and how this strategy would lead to hospital resources usage reduction and 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 are not effective alone, and combinations of these are accepted as the most reasonable course of action. The case of lockdowns is particularly complex, as prolonged closures 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 the locks. Perhaps the most widely adopted of these strategies is contact tracing. *Contact Tracing* consists of tracking, testing and eventually isolating close contacts from each confirmed positive case. Despite its appeal, this strategy is often quite expensive, especially when the number of people to be followed is high.

In this work we develop an agent-based model that extends epidemiological SIR-like dynamics with two different but mutually influencing aspects. Firstly, we add the interaction of agents with the physical spaces through which they circulate, which may remain infected for arbitrary periods of time upon hosting infected agents. In turn, infected spaces may propagate the virus to susceptible agents entering the space. This is the mechanism behind airborne contagion through aerosols, regarded as a key factor in the spread of contagious respiratory diseases (Prather et al. (2020), Chagla et al. (2020)), in certain cases reaching fatal levels (e.g. hospitals or choral events Miller et al. (2020), Van Doremalen et al. (2020), Fears et al. (2020)). Secondly, we model the effects of implementing a contact tracing intervention, by which each symptomatic agents represents an index case for its network of direct (or secondary) contacts. The latter can be reached by the government, being potentially tested and isolated in order to diminish the intensity of the viral propagation.

We will show first how the incorporation of the aforementioned feature affects the spread of the disease. Afterwards, we will analyze the impact of contact tracing in the context of different age groups, each featuring different symptomatic and death rates.

The rest of the paper is organized as follows: In section 2 we describe a new model for indoor space-driven virus spread with contact tracing dynamics. In subsection 2.1 we display how to implement a SEIRD(AP) model and in subsection 2.2 we extend it with contact tracing of different kinds. Afterwards, in section 3 we conduct simulation experiments to assess infection profiles for different combinations of probabilities of infection between spaces and agents. Finally in section 4 we analyze the reduction of the spread by applying contact tracing, deciding on the convenience of tracing contacts of contacts in terms of the achieved final epidemic size.

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

We present an implementation of a SIR-like model using retQSS (Santi et al. (2020)), a framework for modeling and simulation of particle systems in reticulated geometries. Particle models in retQSS are

described in μ-Modelica Bergero et al. (2012), a simplified subset of the Modelica language (Fritzson (2014)), leveraging its expressive power to define spatially-explicit, kinetic-driven dynamics in a compact and elegant way. 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 having a size of *CxC* (arbitrary) units of length. The trajectory of an agent is continuous and linear, with constant speed, random initial position and direction, bouncing only at the borders of the grid. Finally, agents don't collide or bounce with each other. This setup provides an homogeneous mixture of free agents with uniform probability of visiting all cells in a sufficient amount of time (provided no external interventions are applied at population level, nor specific mobility patterns are assigned to given individuals)

Yet, different motion properties such as position, speed and acceleration for each particle can be changed at simulation time. For example, we can define a probability P_{sp} for an agent A to change its speed (V_{sp}) to model a "super spreader" moving faster than normal agents, thus increasing the likelihood of interaction (and therefore contagion) with other agents. As we will see below, this feature 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 though influencing each other, so that the *global state* of an agent is the composition of its *local state* at 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 traversing agents. 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 not sharing that cell synchronously).

Two different types of events can produce a state change in an agent: discrete and temporal. A discrete event occurs when an agent enters a new cell, triggering a set of potential contagions as a consequence of being exposed to the new *local environment*. A temporal event occurs when a timer elapses a period, representing delays of the infection (e.g. latency time) or healthcare logistics (e.g. testing time).

We also model three age groups for agents (Young, Adult and Elderly) as age is a relevant determinant for parameters such as symptomatic rate or lethality.

We will first describe the model for the epidemiological aspects (SEIRD(AP) FSM) and afterwards we 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 contacting 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. This model includes the Exposed state in addition to Susceptible, Infected and Recovered states. Also, it distinguishes between three types of contagious states: Asymptomatic (A), Presymptomatic (P) state as an intermediate state before symptomatic Infected (I). Finally, the model also has a probability for the agent to die during the symptomatic (I) state. Agents start as Susceptible except for a number I_0 which start as Infected symptomatic. As Infected agents move, they can infect the cells they pass through and other agents in there. We define three different contagion probabilities for a binomial trial, depending on the agent state: P^P_{cont} for Presymptomatic, P^I_{cont} for Symptomatic and P^A_{cont} for Asymptomatic. If the trial succeeds the infection event triggers, otherwise the agent does not infect at all in this cell. The infection consists of two steps: i) the agent infects other agents with probability $P^{a\to a}_{inf}$, tested individually against each agent in the cell, ii) the agent infects the cell as it passes through with a probability $P^{a\to c}_{inf}$. 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.

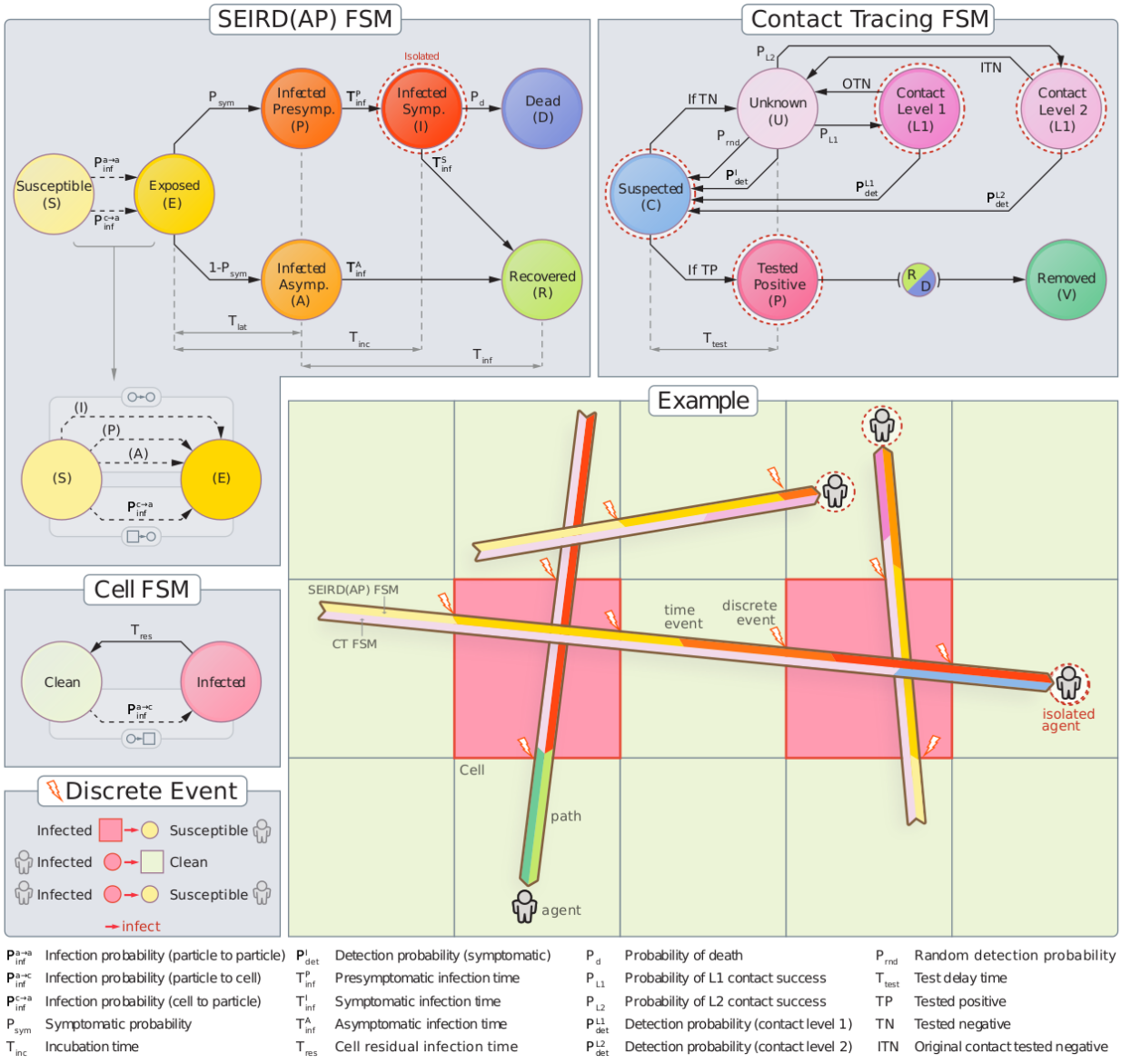

Figure 1: Finite State Machines defining the main dynamics of the model: SEIRD(AP), Contact Tracing and Cells.

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 Exposed. 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). Presymptomatic agents evolve into the symptomatic Infected (I) state after a period $T_{inf}^{P} = T_{inc} - T_{lat}$, where T_{inc} is the incubation time (time since exposure to symptoms onset).

When an agent enters (I) state there is a detection probability P_{det}^{I} reflecting how likely is that the agent contacts the healthcare system, becoming isolated during a period T_{isol}. An isolated agent remains static

and does not infect. When its infection period T^S_{inf} elapses, the agent may evolve either 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 initialization excerpt of the SEIRD(AP) model in Modelica, using the particle-geometry retQSS framework. Position and velocity in the 2D plane are defined by Newtonian laws of motion. Afterwards we define the initial conditions of the algorithm. Arrays are used to store characteristic time periods (time arrays) for agents and cells, defining when *time events* occur, triggering specific state changes in Figure 1. All Cells are initialized in CLEAN state and all agents are initialized in Susceptible state excepting an amount I_0 of initially Exposed agents for whom we set their latency time T_{lat}.

Listing 1: Code excerpt for the initialization of the SEIRD(AP) model.

```
1   model SEIRD(AP)
2   equation  // particle kinetic dynamics
3       for i in 1:N loop  // Derivatives of position and velocity in the x-y plane
4           der(x[i])  = vx[i]; der(y[i])  = vy[i];
5           der(vx[i]) = 0; der(vy[i]) = 0; // No acceleration
6       end for;
7   initial algorithm // system initial conditions
8       geometry_gridSetUp(G,C) // Size of the grid and cells
9       for i in 1:CELLS loop   // Initialize the grid of cells
10        cellInfectionFinishTime[i] = 0
11        cellStatus[i] = CLEAN
12      end for;
13      for i in 1:N loop // Set random initial positions and velocities
14          (x[i], y[i]) = randomXYPoint(G);
15          (vx[i], vy[i]) = randomXYVelocity(V, P_SS, V_SS);
16      end for;
17      for i in 1:N loop // Set time arrays for SEIRD(AP) state change
18          if i <= I_0 then // Initialize as Infected the first I_0 agents
19              latencyStartTime[i] := 0;
20          else
21              latencyStartTime[i] := ∞;
22          end if;
23          infectionStartTime[i] := ∞;
24          symptomsStartTime[i] := ∞;
25          infectionFinishTime[i] := ∞;
26          agentStatus[i] = SUSCEPTIBLE;
27          setAgeGroupProperties(i, P_young, P_adult, P^Y_sym, P^A_sym, P^E_sym, P^Y_d, P^A_d, P^E_d);
28      end for;
```

The main SEIRD(AP) behavior is shown in Listing 2. The **when** clause in Modelica declares event-driven behavior checking for logical condition involving both discrete and continuous variables. If combined with the **time** variable we can define time-driven 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 what comes next.

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: Code excerpt for the SEIRD(AP) epidemiological dynamics.

```
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 // Update properties when an agent enters a
         new cell.
      (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 // 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 Presymp.)
      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 Incorporating Contact Tracing Dynamics

Each pair of agents that share a cell at any given instant become mutual contacts and are included in each other's contact list. The contacts included in these lists are saved 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 of detection, as mentioned before (P^I_{det}). Now, according to the contact tracing intervention strategy, if 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 locate them by calling them or sending messages. Each agent contacted this way 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}), also 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 state in the Contact Tracing FSM. The implementation of Contact Tracing intervention strategy is shown in Listing 3 and Listing 4.

Listing 3: Code excerpt for the initialization of the Contact Tracing model extension. Newly added code highlighted in red. Previous code not shown (highlights 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: Code excerpt for the Contact Tracing model extension. Newly added code highlighted in red. Previous code not shown (highlights 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
    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;
    (same code as before) // Change to Recovered (from Symptomatic or Asymptomatic)
      // Test delay ends, change to Tested Positive or Unknown depending on test result. If test result is positive,
           start moving again.
    when time > testResultTime[i] then
      isolationFinishTime[i] := onTestResult(time, i, TESTED_POSITIVE_ISOLATION_TIME);
      if shouldMove(i) then
        (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;
```

Times and probabilities mentioned before are parameters of the model and their values are presented in Table 1 (Appendix A). We assigned some of them as fixed values and we swept others in order to perform some experiments and analyze the results. Time parameters are tuples meaning (T_{min}, T_{max}). These parameters are sampled from a uniform distribution when used.

3 SIMULATION OF A SHARED ROOM-DRIVEN CONTAGION PROCESS

We investigate the emergence of contagion patterns as a disease spreads due to sharing different 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 of SARS-CoV-2 Prather et al. (2020), Chagla et al. (2020).

We analyze 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. We recall that 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 $P_{inf}^{a\to a}$ represent situations when people are less strict in obeying rules about social distancing or wearing face masks. In the case of $P_{inf}^{a\to c}$ and $P_{inf}^{c\to a}$ higher values represent situations of increasingly poorly ventilated rooms. In Figure 2, we show the results of a parameter sweeping experiment of these three probabilities, presented as heatmaps for the reproduction number R in each scenario.

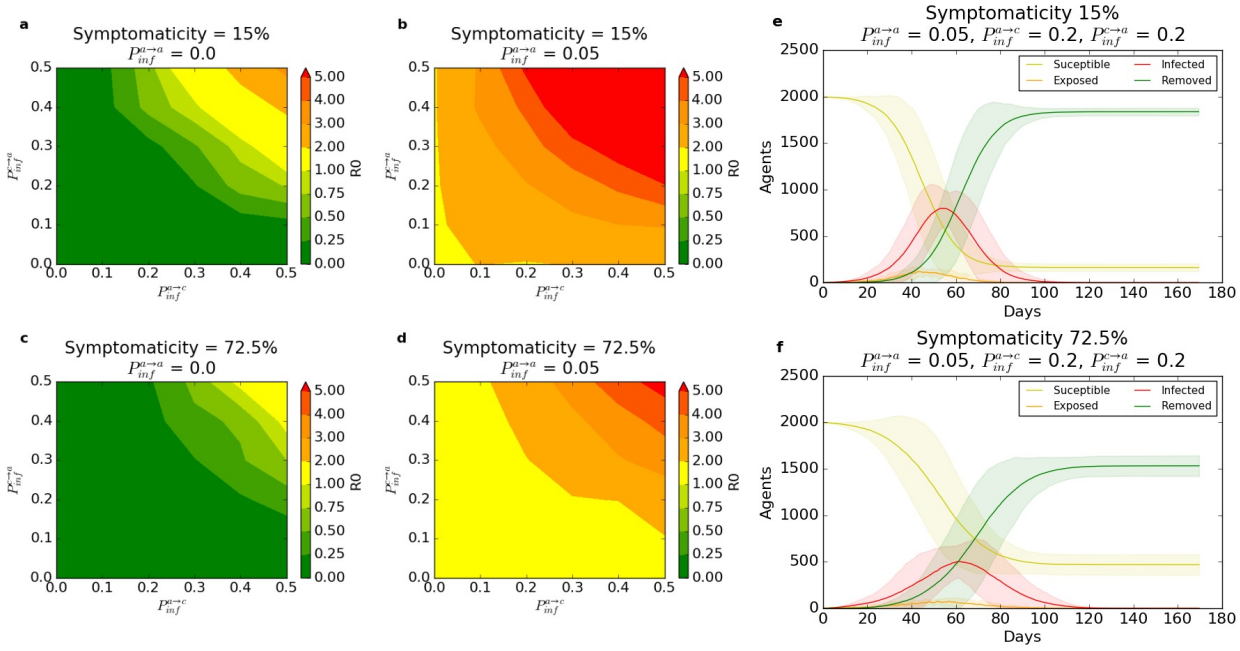

Figure 2: Average Reproduction Number (R) for different scenarios varying the probabilities of infection.

We calculate the average R for a full simulation as follows: first, we calculate the number of infections produced by an agent during its infectious period, for each agent. Afterwards, we take the average of these values for all agents over the full simulation time. Each point in the heatmap is calculated as the average of 10 repetitions of the stochastic simulation with the same model parameters.

Asymptomatic rate are yet under discussion, many studies tried to estimate it, but the variability of information available is still very high. Studies on contact tracing report an average asymptomatic rate of 20% Syangtan et al. (2020), but there is other evidences showing more than 50% Byambasuren et al. (2020) and also rare values 80% were presented Hu et al. (2020). For this reason, we decided to compare 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 come up because of the different probabilities 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, we set them as: $P_{Young} = 0.25$ and $P_{Adult} = 0.5$. As agents not showing any symptom do not self-isolate, it is expected that a high proportion of asymptomatic agents would make the infection to spread faster. In the bottom row we tested a high proportion of symptomatic agents (72.5%) where it can be seen that the infection consistently spreads at lower rates (smaller R values for the same parameters, as compared to the top row). In general, as 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 driven by the 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 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 matching any known virus (not shown in the figure).

Finally, in order to find suitable values to assign for these parameters, we looked for configurations having an average R between 2.5 and 3.0. As this criteria could be met by several combination of values we decided to select them being the minimal values required to reach this criteria. This will be of great help to analyze scenarios as lower bounds and each probability could increase separately. From now on we will adopt $P_{inf}^{a\to a} = 0.05$, $P_{inf}^{c\to a} = 0.25$, $P_{inf}^{a\to c} = 0.25$.

4 SIMULATION OF A CONTACT TRACING STRATEGIES AND DIFFERENT AGE GROUPS

We move forward and analyze contact tracing and isolation dynamics, a type of non-pharmaceutical intervention, on top of the SEIRD(AP) dynamics studied in the previous section. We analyze two different aspects of this intervention: 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 sweeping experiments over these parameters to estimate the amount of resources needed for this intervention strategy, such as the number of calls per day required to effectively decrease the infection spread intensity, 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 3 scenarios with different proportions of Young, Adult and Elderly agents, and studied how this structure impacts in the efficiency of the contact tracing mechanism. 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, there is not much difference between Adult and Elderly populations while the main difference is detected when we compare them against the Young population. The FES is consistently higher for a younger population structure because they have lower symptomatic probability. The larger the proportion of asymptomatic agents, the fewer the number of isolations, impacting on higher values of FES.

Finally, according to the scenarios analyzed, we can conclude that L2 contacts (contacts of contacts) should not be contacted because investing resources on this mechanism does not seam to pay off, leading to consistently higher values of FES (implying more hospitalized and deceased agents). Even when trying to capture only one L2 contact for each L1 contact (panels d,e,f) the process ends with a higher proportion of infections as compared to the strategy where no L2 contacts are considered (panels a,b,c).

5 DISCUSSION

Lockdowns as the only type of cotrol strategy is known to be a very traumatic non-pharmaceutical intervention from the economic point of view (Ahir (2020), Bai et al. (2020)). Thus, we still need to develop and optimize complementary strategies effective enough in terms of decreasing the spread of the virus. Even if agents take care of the infection, for example, by using masks or maintaining social distancing, the effect of aerosols in poorly ventilated rooms can still be very dramatic. This supports the hypothesis that a very high proportion of the contagion could be via aerosols in confined spaces.

Contact tracing is considered a promising intervention strategy in post lockdown scenarios. It has been shown that its efficiency decreases noticeably with increasing delays (Hellewell et al. (2020), Kretzschmar et al. (2020)). In our results this effect can only be observed for scenarios with FES in the order of 40% and below, i.e. in those where the epidemic has a lighter final impact. Yet, for FES of 50% or higher, the contact tracing delay seems play a less relevant role. This could be related to the effect of the cell-to-particle contagions, as the viral load of an agent that infected a cell (via aerosols) can last beyond

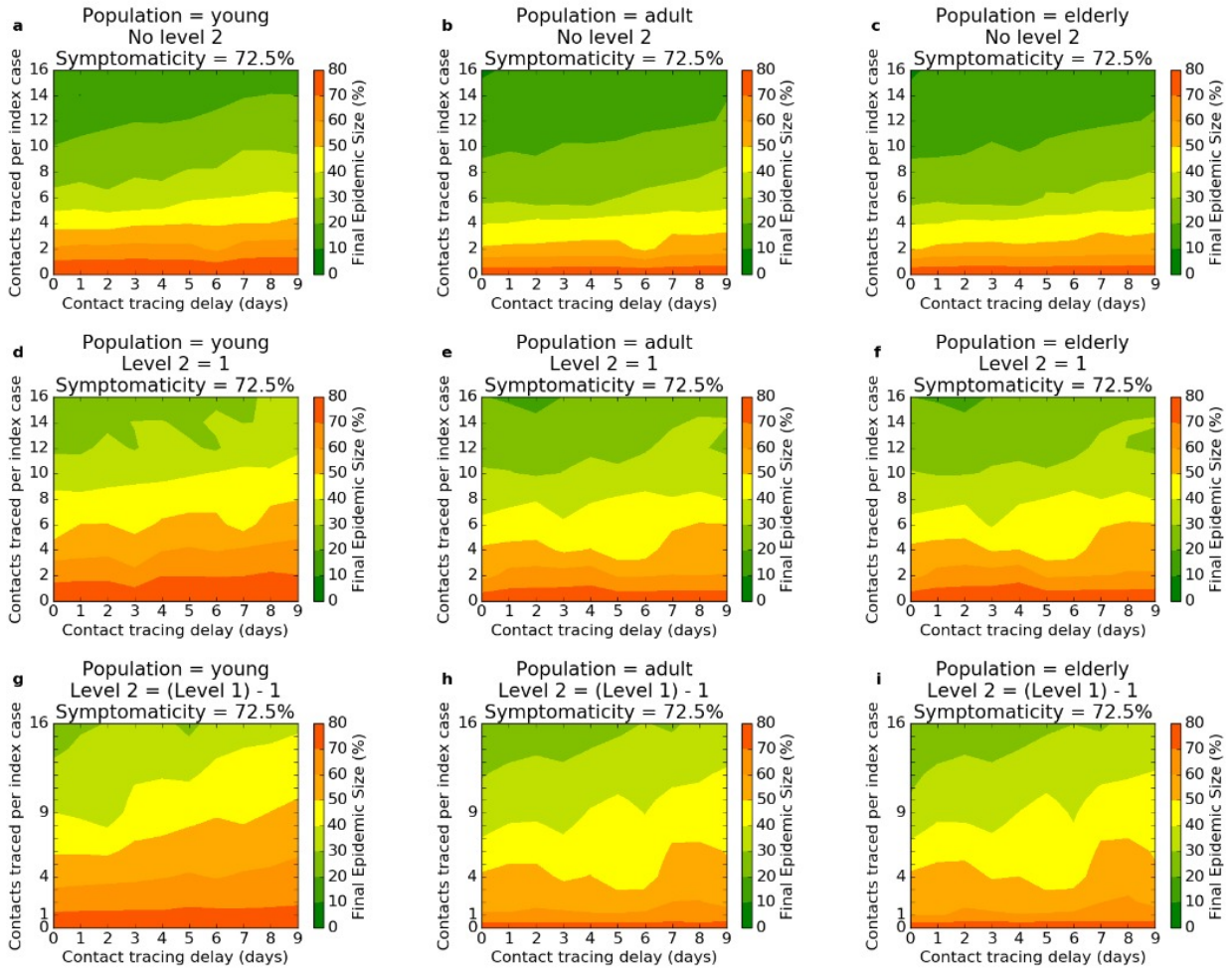

Figure 3: Final Epidemic Size (FES) for varied contact tracing strategies and different age groups.

the moment where the agent is detected and isolated. Such interactions between the contact tracing and the cell-mediated dynamics deserve further investigation. On the other hand, we obtained that the number of contacts traced per index case is a key parameter to optimize the strategy.

Finally, consistently with other studies (He et al. (2020), Moghadas et al. (2020)) we verified that asymptomatic and presymptomatic transmission is one of the main factors supporting the emergence of the spread.

6 CONCLUSIONS

In this work we extended the retQSS particle-geometry modeling and simulation framework to tackle varied types of dynamics relevant to the transmission of the Sars-CoV-2 virus. We were able produce compact, easy to read specifications of hybrid dynamics that combine a continuous subsystem (the kinetic movement of particles) with very frequent discrete-events (cell boundary crossings) and a complex set of time events attached to Finite State Machines that influence each other.

By adopting a Modelica-based specification, the intricacies of solving numerically all possible interactions between the continuous, discrete-event and time event-based dynamics are hidden to the modeler. This facilitates interdisciplinary work on complex models. For example in this work we combine varied

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 though a cell-mediated mesoscopic process.

The experimental results were consistent with other works in the literature, indicating the analysis performed provides sound indications about the effect of contact tracing techniques on the progression of an epidemic that relies heavily on airborne infections.

Building on these results we plan to continue this work by performing more comprehensive parameter sweeping experiments to assess sensitivity, incorporate more accurate probability distributions for characteristic time periods of the infection, and include other non-pharmaceutical interventions such as intermittent lockdowns.

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Appendices

A Model parameters

Table 1: Parameters of the model.

Ambient parameters	Symbol	Values	Units	Type
Total # of agents	N	2000	Agents	Fixed
Initial # of infected	I_0	5	Agents	Fixed
Grid size (GxG)	G	10	Cells	Fixed
Cell size (CxC)	C	4	Distance	Fixed
Default velocity	V	1	Distance/Time	Fixed
Population parameters				
Super spreader probability	P_{SS}	0.05	Probability	Fixed
Super spreader velocity	V_{SS}	4	Distance/Time	Fixed
Young probability	P_{young}	[0.25,0.5]	Probability	Sweep
Adult probability	P_{adult}	[0.25,0.5]	Probability	Sweep
SEIRD(AP) parameters				
Infection probability (agent to agent)	$P_{inf}^{a\to a}$	[0,0.05]	Probability	Sweep
Infection probability (agent to cell)	$P_{inf}^{a\to c}$	[0-0.5]	Probability	Sweep
Infection probability (cell to agent)	$P_{inf}^{c\to a}$	[0-0.5]	Probability	Sweep
Cell residual time	T_{res}	U(0.5,1)	Days	Fixed
Symptomatic probability (young)	P_{sym}^{Y}	[0.0,0.5]	Probability	Sweep
Symptomatic probability (adult)	P_{sym}^{A}	[0.2,0.8]	Probability	Sweep
Symptomatic probability (elderly)	P_{sym}^{E}	[0.2,0.8]	Probability	Sweep
Contagion probability (presymptomatic)	P_{cont}^{P}	0.5	Probability	Fixed
Contagion probability (symptomatic)	P_{cont}^{I}	1.0	Probability	Fixed
Contagion probability (asymptomatic)	P_{cont}^{A}	0.5	Probability	Fixed
Latency time	T_{lat}	U(1,3)	Days	Fixed
Symptomatic detection probability	P_{det}^{I}	0.9	Probability	Fixed
Isolation time	T_{isol}	20	Days	Fixed
Infection time (presymptomatic)	T_{inf}^{P}	U(4,7)	Days	Fixed
Infection time (symptomatic)	T_{inf}^{I}	U(8,11)	Days	Fixed
Infection time (asymptomatic)	T_{inf}^{A}	U(12,18)	Days	Fixed
Death probability (young)	P_{d}^{Y}	0.0	Probability	Fixed
Death probability (adult)	P_{d}^{A}	0.005	Probability	Fixed
Death probability (elderly)	P_{d}^{E}	0.03	Probability	Fixed
Contact tracing parameters				
Random detection probability	P_{rnd}	0.01	Probability	Fixed
Symptomatic detection probability (L1/L2)	$P_{det}^{L1}/P_{det}^{L2}$	0.8/0.9	Probability	Fixed
Number of contacts traced (L1/L2)	C_{L1}/C_{L2}	[0-16]	Individuals	Sweep
Contact success prob. (L1/L2)	P_{L1}/P_{L2}	0.95/0.95	Probability	Fixed
Contact delay time (L1/L2)	T_{L1}/T_{L2}	[0-9]	Days	Sweep
Test delay time	T_{test}	U(0.5,2)	Days	Fixed
Contact remaining probability base	P_{rem}	0.95	Probability	Fixed
Contact remaining probability decay	D_{rem}	0.95	Factor	Fixed