Cell-DEVS Models for the Spread of COVID-19

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Abstract. Improved Susceptible-Infected-Recovered (SIR) models have been used to study the COVID-19 pandemic. Although they can predict epidemiology curves, spatial models cannot be easily built, and cannot model individual interactions. In this research, we show a definition of SIR-based models using the Cell-DEVS formalism (a combination of Cellular Automata and DEVS), showing how to deal with these issues. We validate the equivalence of a simple Cell-DEVS SIR model, and we present a SIIRS model, whose parameters are configured to imitate the spread of SARS-CoV-2 in South Korea. Such models may assist in the decision-making process for defining health policies, such as social distancing, to prevent an uncontrolled expansion of the virus.

Keywords: Cell-DEVS, Cellular Models, Coronavirus, COVID-19, Pandemics.

1 Introduction

Current studies of COVID-19 [1, 2] include theory and methods of infectious disease dynamics. These methods are based on mathematical models that show how the disease spreads. The original Susceptible-Infected-Recovered (SIR) model [3] has been subsequently adapted to study the spread of diseases with a variety of new equations. Some recent extensions represent exposed individuals [4], latency of the disease, and the effect of quarantines [5], as well as the effects of isolation and contact tracing [6].

Several of the advanced models are based on formal mathematical methods, such as network dynamics, ordinary differential equations, finite equation theory, and others. Although these theoretical studies on infectious diseases are useful, they are difficult to apply in practice. Specifically, they have shortcomings for defining contact processes, the behavior of the individuals and the spatial dimension in the model. Cellular automata (CA) allows to develop models that overcome the above-mentioned shortcomings.

Although CA has been successfully applied to develop disease spread models, its discrete-time nature considers time as isomorphic to the natural numbers set $\mathbb{N}$ (i.e., time advances at constant steps). Therefore, all cell states that are supposed to happen between timesteps must be either neglected or delayed matching the simulation

timestep. CA are not trivial to integrate with other models defined in other formalisms, as well as defining advanced timing conditions for each cell. The Cell-DEVS formalism [7] solves these issues by combining CA and the Discrete EVent System Specifications (DEVS) [8] to describe n-dimensional cell spaces as discrete-event models.

Here we illustrate the application of Cell-DEVS to build spatial models of spread of COVID-19. In Section 2, we present related work and introduce Cell-DEVS. Section 3 describes two models for pandemics and illustrates how to build them in Cell-DEVS. Section 4 shows the results of simulations performed under these models.

2 Background

Mathematical models of infectious diseases have been studied since the XVIII century, when Bernoulli proposed a model to analyze the effect of vaccination on the spread of smallpox [9]. In 1927, Kermack and McKendrick published what is considered to be the first modern mathematical model for pandemics [3]. This model classified the population into three different groups: susceptible (S), who can get infected with the disease (I), and then can recover (R). The success of the SIR model led to several improved. For instance, the Susceptible-Exposed-Infected-Recovered (SEIR) models [4] added a new class of infected individuals that cannot transmit the disease: the Exposed (E), which eventually become infected. SIRD models [10] include dying (D) individuals, and SIS models [11] include infected individuals that after overcoming the disease can be susceptible to it again. There are numerous combinations of these methods (e.g., SEIIR, SIRS, SEIRS, SIRDS, or SEIRDS), in which the number of individuals moving from one class to another is described using differential equations.

For the COVID-19 outbreak, numerous mathematical models used SIR-based models of prediction. For example, Danon et al. [1] used a SEIIR model for SARS-CoV-2 in England and Wales, tuning the coefficients of the corresponding differential equations according to estimates from the outbreak in China. Caccavo [2] showed a modified SIRD model that adequately describes the outbreaks of China and Italy by defining time-variant coefficients of the differential equations of the mathematical model.

2.1 SIR-Based Models using Cellular Automata

The mathematical theory and methods of infectious disease dynamics do not include variable susceptibility of the population or the representation of spatial aspects of the spread of the disease. Using CA [12] for SIR-based models can address these issues effectively. For instance, [13] proposed a simple CA model that consider the effect of vaccination. In this model, the cells' population is inhomogeneous, and individuals can travel between neighbors on each time step. Alternatively, [14] presented a geographical CA corresponding to a SIRS model with multiple infection phases. Each phase implies a different probability of spreading the virus, and each infection state presents a variable chance of getting recovered. The cell space has irregular shapes with a varying dimension corresponding to a geographical location, length of the boundaries with the adjacent regions, and road links between two sections.

The SEIR CA model in [15] has probabilistic state transitions. Each cell represents an individual, and mobility is defined as a reciprocal change in neighboring cells. As each cell describes a single individual, the model was limited to small scenarios, and cannot be extrapolated to large cities with high population density, where the disease could affect the most. Finally, the SEIRDS model in [16] explores how the spread of infectious diseases is affected by population density, gender, and age structure. Infected individuals are not divided into subgroups based on the stage of the disease, and therefore the behavior of the pandemic is more predictable than in the models cited above.

2.2 The Cell-DEVS formalism

The discrete-time nature of CA considers time as isomorphic to the natural numbers set $\mathbb{N}$, and any event between time steps must be either neglected or delayed to match the simulation time base. Furthermore, the synchronism of CA could lead to unnecessary processing. CA are also complex to integrate with other models defined using different systems specifications, and the definition of timing conditions for the cells is difficult.

Cell-DEVS [7] combines CA with Discrete EVent System Specifications (DEVS) [8] to describe n-dimensional cell spaces as discrete-event models. In Cell-DEVS, each cell represents a DEVS atomic model, and the cell space is defined as a DEVS coupled model that interconnects neighboring cells, as seen in Fig. 1 for a 2D Cell-DEVS.

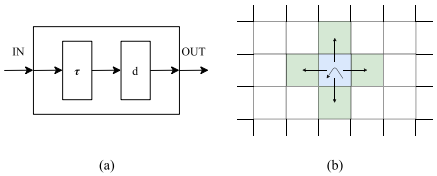


Fig. 1. Cell-DEVS model: (a) schematic of an atomic cell; (b) 2-dimensional Cell-DEVS

As shown in Fig. 1(a), when a cell receives an input, a local computing function τ is activated to compute the next state of the cell. If this is different from the current state, the change is transmitted to neighboring cells after a time delay d specified by a delay function D. Fig. 1(b) shows that outputs from a cell (in the center) are received by the nearby cells using a von Neumann neighborhood. Cells are only active when they receive an external event or when they have a scheduled internal event. Otherwise, cells remain passive. As a result, this discrete-event approach only computes active cells, using a continuous time base (and time advances with events triggered by cells). Cell-DEVS models are equivalent to CA with explicit timing information. Cell-DEVS inherits modularity and hierarchical modeling from DEVS formalism, allowing cells to interact with other models, tools, data sets, and visualization mechanisms, making it easier and efficient to build complex cellular models.

CD++ [17, 18] is a simulator that allows defining models based on the Cell-DEVS and DEVS formal specifications. We define the local transition functions as follows:

```
rule: {PORT_ASSIGN} {NEW_STATE} DELAY {PRECONDITION}
```

When the PRECONDITION is satisfied, the state of the cell changes to the designated NEW_STATE. The PORT_ASSIGN values are transmitted to other components using different ports after waiting the required DELAY. If the PRECONDITION is false, the next rule in the list is evaluated until a rule is satisfied or there are no more rules available.

CD++ visualization engines ease in-depth analyses of the simulation traces for the models under study. The Cell-DEVS Web-viewer [19] allows us to easily visualize simulation results, display cells information and activity with ease.

3 Cell-DEVS Definition of SIR Models

In this section we present SIR Cell-DEVS models implemented in CD++.

3.1 Susceptible-Infected-Recovered (SIR) Model

The SIR model in this section is based on the model in [13] to simulate the spreading of epidemics in a 2D space. At time *t*, cell (i,j) has a number of individuals $N_{i,j}$, and it stores the ratio of individuals on each SIR group as follows: susceptible $S_{i,j}^t$, infected $I_{i,j}^t$, and recovered $R_{i,j}^t$. The model does not consider birth, immigration, or death: the population of each cell remains constant. At every timestep, a portion of cells' susceptible individuals becomes infected according to the following rule:

$$i_{i,j}^t = \min\left(S_{i,j}^{t-1}, S_{i,j}^{t-1} \cdot \sum_{(\alpha,\beta)\in V} c_{i,j}^{(\alpha,\beta)} \cdot m_{i,j}^{(\alpha,\beta)} \cdot \lambda \cdot \frac{N_{i+\alpha,j+\beta}}{N_{i,j}} \cdot I_{i+\alpha,j+\beta}^{t-1}\right) \quad (1)$$

The proportion of new infections ($i_{i,j}^t$) depends on the ratio of infected individuals on the neighbors $(i+\alpha, j+\beta)\ \forall \alpha, \beta \in V$, as well as the density ratio between neighbors and the origin cell. It also depends on a connectivity factor $c_{i,j}^{(\alpha,\beta)}$ (the number of means of transportation between two cells), a mobility factor $m_{i,j}^{(\alpha,\beta)}$ (the probability of an individual in a cell $(i+\alpha, j+\beta)$ to move to cell (i,j)), and an infection rate λ. Additionally, a portion of the infected individuals $r_{i,j}^t$, recovers according to the recovery rate γ:

$$r_{i,j}^t = \gamma \cdot I_{i,j}^{t-1} \quad (2)$$

The complete behavior of the model is described as follows:

$$S_{i,j}^t = S_{i,j}^{t-1} - i_{i,j}^t \ ;\ I_{i,j}^t = I_{i,j}^{t-1} + i_{i,j}^t - r_{i,j}^t \ ;\ R_{i,j}^t = R_{i,j}^{t-1} + r_{i,j}^t \quad (3)$$

To ensure that the amount of possible states is finite, the susceptible, infected, and recovered ratios are discretized as follows:

$$DS_{i,j}^t = \frac{[100 \cdot S_{i,j}^t]}{100}, DI_{i,j}^t = \frac{[100 \cdot I_{i,j}^t]}{100}, DR_{i,j}^t = 1 - DS_{i,j}^t - DI_{i,j}^t \quad (4)$$

Code 1 shows the Cell-DEVS implementation of the model in CD++. We first define the size of the cell space, the type of delay (`transport`) and a von Neumann neighborhood. We also define state variables and the ports for each cell.

Code 1. Implementation of the Cell-DEVS SIR model in CD++

```
type: cell              width: 50     height: 50    delay: transport
neighbors: (-1,0) (0,-1) (0,0) (0,1) (1,0)
statevariables: population virulence connection movement i_sus i_infec i_rec
neighborports: initial infec rec pop sus

[sir-rules]
...
rule: {~pop := $population; ~infec:= $i_infec; ~sus:= $i_sus; ~rec:= $i_rec;}
{$i_sus:= round(((0,0)~sus - #(i_effect))*100)/100; $i_infec:= round(((1-#(recov-
ery))*(0,0)~infec+(0,0)~sus-$i_sus)*100)/100; $i_rec:= 1 - $i_sus - $i_infec;}
     1     { (0,0)~initial != -1 }
```

The keyword `statevariables` defines all the variables in the cell: `population` ($N_{i,j}$), `virulence` (λ), `connection` (c), `movement` (m), `i_sus` (DI), `i_infec` (DI) and `i_rec` (DR). The cell's ports used to transmit information to the neighboring cells are defined using the keyword `neighborports` followed by their names: `initial`, `infec`, `rec`, `pop`, `sus`. The transition rule uses the values of the state variables and the inputs received from neighbors. The rule presented represents a part of Equations (4) and (5). If the cell is not in the initial state (`(0,0)~initial!=-1`), we update the proportion of susceptible, infected, and recovered individuals. For example, the proportion of recovered individuals (`$i_rec`) is calculated as in Equation (4). After the delay (1 time unit), the population of the cell and the proportions of S, I, and R are transmitted using the cell ports. Code 2 shows a macro used in the model.

Code 2. Implementation of the macros for the SIR model in Cell-DEVS.

```
#BeginMacro(i_effect)
min((0,0)~sus, (0,0)~sus*$virulence*((0,0)~infec+$connection*$movement/(0,0)~pop*
   ( (1,0)~pop * (1,0)~infec + (-1,0)~pop * (-1,0)~infec +
        (0,1)~pop * (0,1)~infec + (0,-1)~pop * (0,-1)~infec)))
```

This macro is used to calculate the proportion of new infected individuals according to Equation (1) using the Von Newman neighborhood.

3.2 Susceptible-Infected-Recovered-Susceptible Model

The model in this section, based on the model in [14], defines a Cell-DEVS representation of a SIRS model. As in the previous case, each cell (i,j) has a fixed, heterogeneous population $N_{i,j}$ divided into three groups: S, I, R. Once infected, individuals remain ill from 1 to T_I days, after which they are immune for T_R days, after which they become susceptible again. The infected group can be divided into subsets depending on the percentage of individuals that have been infected during p consecutive days. Recovered individuals are classified based on how many days they have been immune:

$$I_{i,j}^t = \left\{ I_{i,j}^t(p) \middle| p \in \{1, \ldots, T_I\} \right\}, R_{i,j}^t = \left\{ R_{i,j}^t(r) \middle| r \in \{1, \ldots, T_R\} \right\}, \quad (5)$$

The ratio of individuals that become infected at time t ($i_{i,j}^t$) is described in Equation (6). The proportion of new infections depends on the ratio of infected individuals in the neighboring cells and the population density ratio between neighboring cells and the

origin. This model also considers a connectivity factor $c_{i,j}^{(\alpha,\beta)}$, a mobility factor $m_{i,j}^{(\alpha,\beta)}$, and an infection rate $\lambda(p)$. In this model, the infection rate varies with the stage of the illness (low in the first days of the infection, and high in the last days.

$$i_{i,j}^{t} = \min\left(S_{i,j}^{t-1}, S_{i,j}^{t-1} \cdot \sum_{\substack{(\alpha,\beta)\in V \\ p\in\{1,\dots,T_I\}}} c_{i,j}^{(\alpha,\beta)} \cdot m_{i,j}^{(\alpha,\beta)} \cdot \lambda(p) \cdot \frac{N_{i+\alpha,j+\beta}}{N_{i,j}} \cdot I_{i+\alpha,j+\beta}^{t-1}(p)\right) \tag{6}$$

We also need to consider that the set of individuals in the last day of immunity, $R_{i,j}^{t-1}(r)$, become susceptible again. Hence:

$$S_{i,j}^{t} = S_{i,j}^{t-1} - i_{i,j}^{t} + R_{i,j}^{t-1}(r) \tag{7}$$

In each infected state $I_{i,j}^{t}(p)$, the recovery rate function ($\gamma(p)$) represents the probability for infected individuals to overpass the disease after being infected during p consecutive days. As the maximum allowed days of the disease is T_I, we set $\gamma(T_I) = 1$. Thus, the proportion of people infected for p consecutive days is equal to the proportion of people that have been ill during $p-1$ days in a row and did not recover:

$$I_{i,j}^{t}(p) = \begin{cases} i_{i,j}^{t} & , \text{ if } p = 1 \\ \left(1-\gamma(p-1)\right) \cdot I_{i,j}^{t-1}(p-1) & , \text{ if } 1 < p \le T_I \end{cases} \tag{8}$$

The first recovered state ($R_{i,j}^{t}(1)$) is the sum of the last infected state and the recovered individuals of the other infected states. The following recovered states simply takes the value of the previous recovered states.

$$R_{i,j}^{t}(r) = \begin{cases} \sum_{p\in\{1,\dots,T_I\}} \gamma(p) \cdot I_{i,j}^{t-1}(p) & , \text{if } r = 1 \\ R_{i,j}^{t-1}(r-1) & , \text{if } 1 < r \le T_R \end{cases} \tag{9}$$

The SIRS model was defined using CD++ as a Cell-DEVS model similar to the SIR model explained in Section 3.1. This new version includes different state variables and ports, and new local transition rules to calculate the new state of the cells. In Code 3, we include a part of the rules used to represents Equations (7-9).

Code 3. Implementation of the rules for the SIR model in Cell-DEVS.

```
rule: {~pop:=$population; ~sus_0:=$i_sus_0; ~inf_1:=$i_inf_1; ~inf_2:= $i_inf_2;
        ... ~rec_24:= $i_rec_24; ~rec_25:=$i_rec_25; ~rec_26:= $i_rec_26; ...}
          { $i_rec_28:= $i_rec_27; $i_rec_27:= $i_rec_26;
                $i_rec_23:= $i_inf_22 + #(local_recovered);
                  $i_inf_22:= round((1 - $recovered_rate) * $i_inf_21*100)/100;
        ...
                  $i_inf_2:= round((1 - $recovered_rate) * $i_inf_1*100)/100;
                  $i_inf_1:= #(internal_infected) + #(external_infected);
                  $i_sus_0:= 1-$i_inf_1-...-$i_inf_22-$i_rec_23 -...- $i_rec_28;}
        1 { (0,0)~initial != -1}
```

If the cell is not in the initial state, we calculate the proportion of individuals on each state of the disease. In this case, there are susceptible individuals (`$i_sus_0`), infected individuals on different phases of the disease (e.g., `$inf_1` represents the proportion

of individuals in the first day of infection) and recovered individuals with different immunity time left (e.g., `$i_rec_28` represents the proportion of individuals in the last day of the immunity). The proportion of infected individuals in the second day of the disease (`$inf_2`) is calculated as the proportion of infected individuals the on the first day of disease minus the ratio of individuals who recovered `(1-$recovered_rate) *$i_inf_1` as in Equation (8). Then, the value is discretized to two significant digits. The rules to calculate the rest of the state variables are defined similarly. After a delay, (i.e., 1 time unit), the population of the cell and the proportion of R, I and S individuals are transmitted using the ports of the cell.

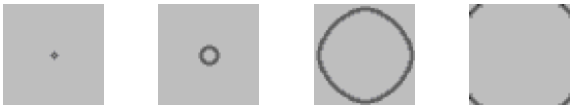
4 Case Studies

In this section, we present results of simulations using the two Cell-DEVS models described above. All models showed in this section represent a 50x50 space with a range 1 von Neumann neighborhood. The time units used correspond to one day.

4.1 SIR Model Simulation Results

The basic SIR allows exploring critical factors such as the infection and recovery rates. In this section we show that the results in [13] can be reproduced using our Cell-DEVS version of the model, using the same parameters than in the original model. The population of every cell $N_{i,j}$, is 100 individuals. Initially, only the cell in the middle contains infected individuals. The proportion of infected individuals of this cell, $I^0_{25,25}$ is 0.3. The remaining people in the cell are susceptible to infection ($S^0_{25,25} = 0.7$ and $R^0_{25,25} = 0$). The infection rate λ is 0.6, the recovery rate γ is 0.4, and the connectivity factor $c_{i,j}^{(\alpha,\beta)}$ is 1. The mobility factor is 1 for the cell itself, and 0.5 for the rest of the neighbors.

Fig. 2 shows the percentage of infected individuals per cell. Cells in light grey correspond to areas with no infected individuals, while dark grey and black cells correspond to areas with a significant ratio of the population infected.



(a) (b) (c) (d)

Fig. 2. Infected rate reported by simulations at day (a) 5, (b) 11, (c) 42, and (d) 60.

At day 5, shown in Fig. 2(a), only a few cells of the center of the cell space reported infection cases. As time advances, nearby cells get infected, whereas previous cells with cases start to get immunity, increasing the percentage of recovered individuals. The peak number of cases occurs the 42nd day, displayed in Fig. 2(c).

Fig. 3 shows the evolution of the percentage of population that is susceptible (red dashed line), infected (blue line), and recovered (green dash-dot line). At time 0, almost all the individuals are susceptible (except a 0.012% of the population that is ill from the

beginning, all in cell (25,25)). As time advances, more people become infected, significantly reducing the percentage of susceptible individuals, and increasing the number of infected ones. The increment of infected individuals is less pronounced, as every day a 40% of sick people recover. At the end, 100% of the population has recovered. These results are the same than those presented in [13].

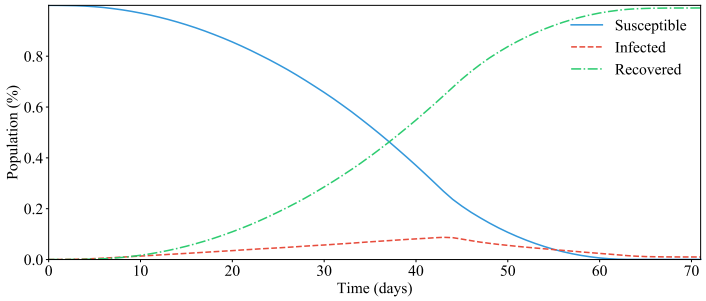


Fig. 3. Evolution of the susceptible, infected, and recovered individuals.

4.2 SIRS Model Simulation Results

The SIRS model provides a finer grain parametrization, allowing the representation of more complex epidemics. It establishes a fixed number of days for the infected and recovered phases, and it allows to define different infection and recovery rates for each infected state. This makes possible to reflect government measures and changes in the population behavior. We show an example of these kind of dynamic scenarios. We use this model to mimic an approximate behavior of the spread of the SARS-CoV-2 in South Korea using real data for defining infection and recovery rates [20].

We configure a 50 x 50 grid, with a population of $N_{i,j} = 100$ individuals per cell. We set the connectivity factor in 1 and the mobility factor to 0.6 for the neighboring cells. Again, we use the cell in the middle to trigger the epidemic ($I_{25,25} = 0.3$, $S_{25,25} = 0.7$, $R_{25,25} = 0$). We set the infection phase length T_I to 22 days. The individuals experience the first symptoms in the 4^{th} day and isolate themselves in the 8^{th} day. Until this event, we establish a fixed infection rate λ of 0.15. The rest of the period they are considered isolated, and their λ is reduced to 0.01. The recovery rate γ is set to 0.07 for all the infected states. For this disease, we consider an immunity period of six days since there is not validated data for COVID-19. Fig. 4 show the evolution of this spread.

(a) (b) (c) (d)

Fig. 4. Infected rate reported by simulations at day (a) 10, (b) 50, (c) 90, and (d) 140.

Here, light grey cells correspond to areas with no infected individuals. For the rest of them, the darker the background color is, the more non-susceptible (i.e., infected and recovered) individuals are present in the cell. As time goes by, the recovered individuals finish their immunity and becomes susceptible again.

Fig. 5 shows how these non-susceptible ratios evolve. The infected population grows at a constant rate until day 93. After this moment, all the individuals have been exposed to the disease, so the infected ratio starts to decrease. As the proportion of infections decreases, the susceptible population increases again, reaching the maximum level of susceptible population at the end of the simulation. The recovered proportion remains low due to the short recovery period length of the scenario under study.

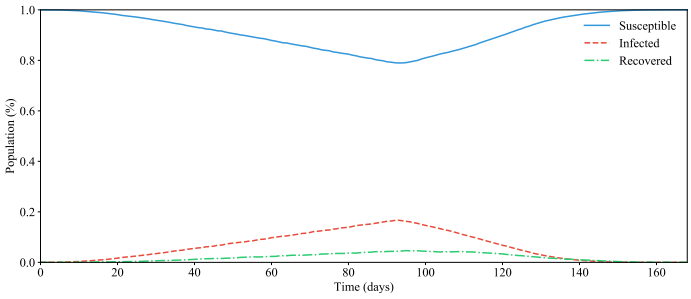


Fig. 5. Evolution of the susceptible, infected, and recovered individuals.

5 Conclusions

We presented a Cell-DEVS definition to simulate and study the spread of disease, focusing on the COVID-19 pandemic. We adapted the model in [13] to validate our approach, which was easily adapted to follow the specifications presented in [14], and we configured the simulation parameters using data from the spread of SARS-CoV-2 in South Korea [20].

In Cell-DEVS models, cells are only active when one or more cells in the neighborhood set notify a state change or any other external event is scheduled. Otherwise, cells stay passive, without requiring any extra computation. Compared with CA, where all cells are active in every simulation step, our approach saves computation time.

Another advantage of Cell-DEVS is its event-triggered time base. Time advances in a continuous timeline when events happen. Thus, the time advance is not fixed into a simulation step. This feature allows defining models where different cells' states have different time spams. We proved that we could define Cell-DEVS models equivalent to any CA with more accurate timing information with no additional effort.

As future work, we will define a SIRDS model that considers the death rate of the pandemic. Furthermore, model parameters such as connectivity or mobility factors will depend also on the different infection phases. With this model, we will be able to define a more precise model that considers more scenarios with complex government policies, such as limiting people mobility depending on the presence of symptoms, or the enforcement of using masks.

The implementations of the models are available at https://github.com/Simulation-Everywhere-Models/COVID-Cell-DEVS-ACRI2020, and a number of simulation scenarios, including the ones presented here, can be found at https://bit.ly/3aiDM4j.

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