Proceedings of the 2021 Winter Simulation Conference
S. Kim, B. Feng, K. Smith, S. Masoud, Z. Zheng, C. Szabo and M. Loper, eds.

CELL-DEVS ADVANCED COMPARTMENTAL MODELS OF COVID-19

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ABSTRACT

Susceptible-Infected-Recovered (SIR) models have been used to study the spread of Covid-19. These models have been improved to include other states (exposed, deceased and others) as well as geographical level transmission dynamics. In this research, we present a new model using the Cell-DEVS formalism that addresses asymptomatic cases in a population. We use the model to simulate the spread of Covid-19 through public health units of Ottawa where asymptomatic cases are present and where they are not. The example shows how the model can be used for rapid prototyping at a geographical level. The framework for basic asymptomatic growth has been incorporated and can be adapted to fit the needs of the modeler.

1 INTRODUCTION

Covid-19 has been at the forefront of public health since late 2019. Health officials were left with the task of educating and maintaining the health of their populations. Despite their efforts, in May 2021 many countries are seeing second and third waves of Covid-19 cases leading to further deaths from the disease. Many of these new waves are reaching higher peaks than the that of the first wave. Researchers have started to investigate what could lead to these secondary waves of infections while populations seemed to be following health restrictions. Some of these researchers found asymptomatic carriers could be the cause for the secondary waves of cases we are seeing in mid-2021. An asymptomatic infection is an infection that does not show symptoms; thus, the carrier does not know they have the infection. This leads to the problem of carriers not knowing they have the infection, spreading it to those around them and leading to elevated case counts. These asymptomatic carriers create serious problems for health policy makers as they cannot track them. This leaves them with little options for planning and prevention. One of the few options that health officials have when planning for diseases that are difficult to monitor is simulation and modelling.

Modelling and simulation have been used by public health officials to predict the spread and growth of many diseases. Predictive models give the decision makers an idea of what could be if they decide to quarantine, partially lockdown or apply other measures. This kind of insight allows for public health professionals to make informed decisions. One of the most popular of such models, introduced by Kermack and McKendrick in 1927 [1], classified the population in “compartments”, in particular recognizing those individuals Susceptible to the disease, those who are Infective (i.e. can transmit the disease), and those who Recovered (SIR). These models give public health officials a look into how much a disease might continue to rise depending public health measures put in place. This allows for public health officials to plan public health measures before case counts reach an uncontrollable point. With these models we can incorporate asymptomatic infections to try and predict the impact they will have on symptomatic case counts.

Using rapid prototyping modelling to change and adapt a model on the fly, combined with geographical models can be useful to adapt new research results to allow for modelling at multiple geographical levels.

For example, a user should be able to run an SIR-based model at the province/state level, and then, with minor modifications, run the same model for the neighborhoods in a city. Models should also allow for quick adaptation of the disease(s) users want to model. Compartmental models use several inputs that characterize the disease being modelled, and these characteristics might change depending on the evolution of a disease. One example is the Covid-19 variant B117 that caused significant rises in Covid-19 infections. Rapid prototyping allows for public health officials to efficiently change the characteristics of a disease and re-run simulations showing different case scenarios depending on the given characteristics. These changing characteristics allow for rapid prototyping a virus as it evolves and changes over time.

Geographical models allow for simulations to be run over a given area and can range from continental models to neighborhoods in a city. For instance, in a city, each component in each city neighborhood can use its own defined population and characteristics, allowing for accurate representation of a given geography. The geographical SIR model we present here allows users to input a described group of neighborhoods and run an SIR model through the neighborhoods, allowing for visualization of how a disease might spread through a city, town, or country. In this research we adapted a geographical SIR model to incorporate advanced behavior for COVID-19: the representation of deceased individuals and those that are asymptomatic state. The model was quickly adapted to incorporate the exposed state resulting in a SEIRD model. To do so, we used the Cell-DEVS formalism [2] and the Cadmium Cell-DEVS simulator [3]. The models easily adaptable framework allows for accessible rapid-prototyping and modifications.

Geographical models are useful to understand the spread of the disease and adding new characteristics should be done with simplicity. For instance, asymptomatic infections are a variant of regular infections where none of the general symptoms are presenting. Covid-19 has proven to have a proportion of asymptomatic carriers as high as 80% [4]–[8], and they can spread the disease to at risk individuals unknowingly. Considering these diverse facts, we will discuss the definition and development of a geographical SEAIRD model (Susceptible-Exposed-Asymptomatic-Infective-Recovered-Deceased). The model was developed to allow users to quickly model the impact asymptomatic cases would have on a geographical population. The asymptomatic cases are implemented using a basic infectious/asymptomatic ratio value that can be defined by the user depending on their input. The asymptomatic carriers are defined as slightly more transmissible individuals, which allows us to simulate the effect of not knowing you have the disease would have on those around you. Those who do not know they can spread the disease will expose more people. The model was developed to allow users to experiment with different asymptomatic rates that the population may experience depending on disease characteristics. This has been shown using the public health units of Ontario. The results show how an asymptomatic set of carriers can change the total numbers of cases that a population would experience. The model shows how a greater number of asymptomatic carriers can lead to sharper increases in case counts. The geographical SEAIRD model described gives users a framework to rapidly prototype disease spread in their neighborhood where asymptomatic infections can be considered and incorporated where necessary.

2 BACKGROUND

As discussed in the introduction, we will present advanced compartmental models of COVID-19 using a spatial approach. SIR geographical models have been the forefront of disease growth and spread prediction. With the emergence of the SARS-Cov-2 virus, these models have become more relevant than ever, being used to track and monitor the potential growth of the disease.

SIR models include three compartments, representing different individuals and their state during the spread of the disease: those that are Susceptible (S), Infectious (I) and Recovered (R) [9]. SIR modelling has been extended several times to include new compartments. For instance, SIRD model adds a compartment to model Deceased (D) individuals, including death factors and fatality rates. SEIRD models adds a Exposed (E) state which is used as a transition from susceptible to infected [9]. There are many iterations and additions to the SIR model, these adaptations all follow the standard SIR framework.

Following Ross and Hudson [10]–[12], Kermack and McKendrick [1] defined a compartmental model in 1927. This model classified a given population into three “compartments”: Susceptible, Infectious, and

Recovered (SIR). Their model defined how a population could move from one compartment to another over time. Kermack and McKendrick's work would define the framework and mathematics that many SIR models continue to follow today. The standard SIR model evolved over the years to incorporate more advanced disease spread, the simplest of these evolutions is the SIRD model which incorporates Death [9]. Over time models became significantly more advanced, having several different, complex compartments. Another advancement made in SIR modelling was the addition of geographical information. Sattenspiel and Dietz [13] described a model for the spread of infectious diseases among geographic regions. This work describe how individuals from different regions can become mobile and be in contact with individuals in other regions, resulting in the spread of a disease across regions. The work shows how geographical information can complement the standard SIR model as well as lead to better, more defined results.

In [14] a geographical Cell-DEVS SIR model was described. The model is based on the model described in [15] to simulate the spread of epidemics in a geographical based 2D cell space. The model has described that at time t, a given cell(i, j) has a given population Ni,j. Each cell stores the ratio of individuals in each state as follows:

$$\text{Susceptible(S)} = S_{i,j}^{t} \tag{1.1}$$

$$\text{Infected(I)} = I_{i,j}^{t} \tag{1.2}$$

$$\text{Recovered(R)} = R_{i,j}^{t} \tag{1.3}$$

The SIR model described in [15] and translated to Cell-DEVS in [14] uses a geographical correlation factor defined by the shared boundaries between two cells. The correlation factor is a method the model uses to link two neighborhoods together to allow for interaction between their populations. This method is not necessarily the best method as it does not take other important factors into account. For example, dense population areas and workplace hubs such as downtown Ottawa. With this being said, the method has been shown to work for easily adaptable geographical simulations. Being able to quickly adapt a model to receive new data is crucial when rapid prototyping, this model allows for user to change both the geographical level they are simulating as well as the disease characteristics. The equations for the correlation are as follows:

$$w_{ij} = \frac{z_{ij}}{l_i} \tag{1.4}$$

$$w_{ij} = w_{ji} = \frac{\frac{z_{ij}}{l_i} + \frac{z_{ji}}{l_j}}{2} \tag{1.5}$$

$$c_{ij} = w_{ij} \tag{1.6}$$

Equation (1.4) describes the weighted correlation factor w_{ij}, which uses the two values, the shared boundary length between cells i and j (Z_{ij}) divided by the total boundary length of cell i (l_i). Then, equation (1.5) is used to calculate the weighted correlation factor for cells i and j where they match both directions. This method states that the correlation for i, when moving to j, is the same as j moving to i. Finally, equation (1.6) is used to set the geographical correlation factor between cell i and j, this will be used in Section 3.

The SIR model described in [14] would be used as the foundation for further works. The next iteration of the model incorporated both deaths, and the ability for a cells population to become re-infected after recovering from a disease. In the coming text, the SEIRD model [16] that was used as a framework for our SEAIRD model will be described as it is important to understand the background and changes made when incorporating the asymptomatic stage.

A SEIRD model is described in [16] added the exposed state to the existing SIRD model. The SEIRD model focused on adding the stage in between the susceptible and the infectious stages. When adding the exposed state, the rate at which a cells population would move from susceptible to exposed remained unchanged. But, when the population moved from susceptible to exposed, a new value was considered, the incubation rate, ε. The exposed state is an important addition to the model as it allows for incubation rate simulations to be added. With the added exposed state, the model can simulate realistic calculations showing the time it takes for someone to be exposed to when they show symptoms. The problem with this is not all individuals show symptoms, thus the need for an asymptomatic stage.

In medicine, when a patient tests positive for a disease but shows no symptoms, they are classified as asymptomatic [17]. Asymptomatic carriers can continue shedding the disease to those around them. Generally, asymptomatic carries will continue shedding their disease at a slower rate than those that are symptomatic. The problem that occurs with asymptomatic carriers are they do not know they have the disease; thus, they may not follow the same procedures as someone who knows they are infectious would. For example, someone who has a cough, generally, will try and cover their mouth to protect those around them, if they did not have any noticeable symptoms, they will spread the disease unknowingly [8]. The asymptomatic affect has caused problems in disease tracking and planning for many diseases and epidemiologists, COVID-19 has not been immune to this problem.

The proportion of asymptomatic infections that make up the COVID-19 pandemic has been widely debated. Studies have found the range of asymptomatic COVID-19 infections may been anywhere from 4% - 80% [4]–[8]. The biggest problem limiting these studies is how the authors validate and reliably collect data. Studies which focus on asymptomatic naturally have the difficulty of finding these infections as the carriers are not aware they are infected; thus, they are not tested. With the possibility of having a significant amount of COVID-19 asymptomatic carriers shedding the disease to those around them, it is crucial to understand how much of an impact they are having on overall case counts. The next challenge many studies have is tracking the impact asymptomatic cases have on the overall true case count. It is a difficult task to find asymptomatic cases to begin with; tracking these cases and seeing who they infect is even harder. This leaves modelers with the job of estimating how many asymptomatic carriers are in each population and how many people they are exposing. How can rapid prototyping allow us to quickly test these rates.

Studies have been done on the integration of the asymptomatic state in modelling. One of these studies [18] proposed a SQIARD and SIARD model. The SQIARD model adds a quarantined (Q) state. The model incorporated the asymptomatic state as a transition from the quarantine state. The model splits the population that move from the quarantine state to the asymptomatic or infectious state using a given asymptomatic and infectious rate. The SIARD model in [18] uses a simple transition from the susceptible state to the infectious or asymptomatic state using a given infectious rate. The model provides results that have proven to resemble real world case counts using a few different countries in testing.

Another study [19] took a different approach to modelling asymptomatic infections, proposing an advanced, SIDARTHE model, which also includes a states for individuals that are diagnosed (D), ailing (A), recognized (R), threatened (T), healed (H) and extinct (E). The model takes a new approach to adding asymptomatic by using multiple disease states under one state. When an individual moves from susceptible to infected they can become asymptomatic, infected or undetected. The asymptomatic individuals will then move to diagnosed where they can either develop symptoms and move to the recognized state (infectious) or heal. The authors in [19] consider those who are asymptomatic or undetected to be important as they will not be isolating like someone who is known infectious. The model shows the important of asymptomatic cases and gives a more advanced example of what could come of the model described in section 3.

Finally, a SEAIRD model is presented in [20]. The model shows a similar transition method as those described previously. In this model the authors use an asymptomatic state where, again, an asymptomatic rate α. Where E is the exposed individuals those who move to asymptomatic are defined as αE, the remaining exposed individuals $\alpha(1- E)$ will move to infectious. The model shows how asymptomatic cases can affect the rate and magnitude of new infectious cases in a population. Later, the model described a more advanced model called the SEAIRD-Control model where a quarantine state, and a hospitalization state have been introduced. These extra states can be considered as future additions to our model.

The models discussed above define several methods that can be incorporated into a SEAIRD model. Each model shares a defined asymptomatic rate where a given proportion of the exposed population move to either infectious (symptomatic), or asymptomatic. This method allows for easy, efficient implementation where user can prototype different values without having to change more than the input parameters. To do so, we use a spatial modeling methodology, called Cell-DEVS [2], which allows defining cell spaces based on the Discrete Event Systems Specifications (DEVS) [21]. Cell-DEVS described an n-dimensional cell

space where each cell represents a DEVS atomic model, the cell space containing the n cells is defined as a DEVS coupled model where each cell is connected to its neighboring cells, as in figure 1.

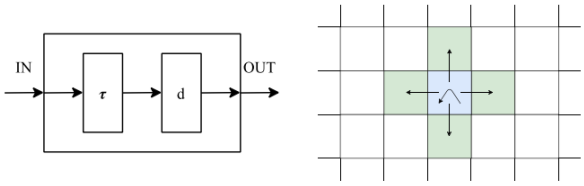

Figure 1. Cell-DEVS model: (a) Atomic cell schematics; (b) 2-dimensional Cell-DEVS neighborhood

We can see both an atomic cell and its schematics as well as a 2-dimensional Cell-DEVS neighborhood. In figure 1(a) we can see that when a cell receives an input, the local computing function τ is activated, this will compute the next state for the cell. If there is a change in the cells state, the change is transmitted to the neighboring cells after a time delay d. We can see how a cell (center) will transmit information to the neighboring cells using a von Neumann neighborhood (although irregular topologies are accepted in Cell-DEVS). The cells in the 2D space default in a passive state, when cells either receive an external event or execute a scheduled internal event they become active. This discrete-event approach only considers and computes active cells using a continuous time base. A delay function is used to define when the cell transmits outputs. Cell-DEVS inherits the modularity and hierarchical modeling ability of the DEVS formalism. This allows for models to better interact with other models, tools, datasets, and visualization tools. This makes Cell-DEVS an easier, and more efficient method to build complex cellular models.

Our SEAIRDS model utilizes the Cadmium tool [3], one of the tools available for use with Cell-DEVS. Cadmium allows users to define model inputs in a clean, efficient JSON format. This is an advantage for our model as it allows for complex geographical inputs that load into the model at run time resulting in a flexible model that allows for efficient rapid prototyping.

3 MODEL SPECIFICATIONS

Our proposed geographical SEAIRD model is based on [14], [16] by adding an asymptomatic state (A) to a state diagram depicted in figure 2. The model is defined as a coupled Cell-DEVS where the cell space represents a geographical region, and each cell (of irregular topology) is a neighborhood in the city/province under study, and it is connected with its neighboring cells (using an irregular topology). Each neighborhood consists of a cell ID, a set of state variables, a model configuration, and neighboring cells correlation factors.

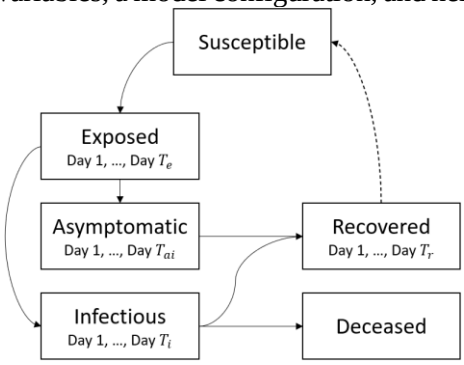

Figure 2. SEAIRD State Diagram

Figure 2 shows that a cell's population starts in a susceptible state and then it can become exposed. From there, the exposed population will move to either asymptomatic or infectious. If asymptomatic, they will eventually become recovered, but if an individual is infectious, they can move to either recovered or death. The dotted line in figure 2 from recovered to susceptible shows how a population can become re-

susceptible after recovery. Each transition from one state to another is based on their defined time behavior. Time in the model is described using a delay function. Exposed, Asymptomatic, Infectious and Recovered states each have a defined set of days that a population can be within the state described by T_e, T_i, T_{ai}, T_r.

Each state has a defined state transition that occurs at each day within the state. The days within each state set of days is described by q = {1, 2, …, T_{state}}. For example, $A^t_{i,a}(q)$, describes the proportion of asymptomatic cases for a populations age group in cell i at asymptomatic state q = {1, 2, …, T_{ai}}, at time t. The asymptomatic state works similarly to the infectious state where an asymptomatic rate is defined to describe the proportion of the population that transitions from exposed to either infectious or asymptomatic.

Our SEAIRD Geographical model is comprised of k unique geographical cells. The six states a population can belong in are S, E, A, I, R, D. The proportion of a populations age group a found in each state can be described with the following notation: $S^t_{i,a}, E^t_{i,a}, A^t_{i,a}, I^t_{i,a}, R^t_{i,a}, D^t_{i,a}$ where i is the cell being described at time *t*. The state transitions are built using Cell-DEVS transition function, which implement the equations defined below. Let us consider *Fa(q)* as the fatality rate of infected stage q for age group *a;* $\lambda a\ (q)$ as their virulence; μa (q) as the mobility rate of age group *a;* ε_a(q) as the incubation rate; and $\gamma_a(q)$ as the recovery rate. Then, c_{ij} is the geographical correlation factor between cells i and j; k_{ij} the correction factor applied to both cells i and j and φ as the asymptomatic proportion of infections. Then:

$$F^{t+1}_{i,a} = F^t_{i,a} + \sum_{q\in\{T_e+1,T_e+2,\ldots,T_e+T_i+T_{ai}\}} \mathrm{F}a(q)\left(I^t_{i,a}(q)\right) \tag{3.1}$$

Equation (3.1) is used to calculate the new deaths at a time *t*. New deaths (*next* day) are the total of current deaths plus the sum of the infectious population that has died the day before. New fatalities are equal to the newly deceased population moving from the infectious state multiplied by the fatality rate. The death transition does not consider asymptomatic infections as they do not lead to fatalities.

$$E^{t+1}_{i,a}(1) = \sum_{\substack{j\in\{1,2,\ldots,k\}\\ q\in\{T_e+1,T_e+2,\ldots,T_e+T_i\}}} \begin{matrix}\left(c_{ij}k_{ij}\lambda_a(q)\mu_a(q)S^t_{i,a}I^t_j\right)\\ +\left(c_{ij}\lambda_a(q)\mu_a(q)S^t_{i,a}A^t_j\right)\end{matrix} \tag{3.2}$$

Equation (3.2) is used to calculate the newly exposed population. The newly exposed population is a result of the susceptible ones in contact with either the entire (all age groups) infectious population or the asymptomatic population of its neighboring cells *j*. The first part of the equation calculates the proportion of a cell's susceptible population exposed to an infectious individual and the second part the proportion exposed to an asymptomatic individual. Each cell is related to its neighbor by a geographical correlation factor c_{ij} that describes the amount of impact each neighboring cell has on a given cell, including the virulence rates and the mobility rate a given cell's population has with its neighboring cells. Finally, k_{ij} defines a correction factor applied between cells *i* and *j*, applied to the infectious half of the equation to simulate different behavior for infectious population and the asymptomatic population: we consider that asymptomatic individuals to be more carefree, thus they will expose more individuals.

$$E^{t+1}_{i,a}(q \geq 2) = \left(1 - \varepsilon_a(q-1)\right)E^{t+1}_{i,a}(q-1) \tag{3.3}$$

Equation (3.3) describes how the exposed population transitions to the infectious or asymptomatic state. The equation defines the exposed in stage *q* is equal to the exposed of the previous day multiplied by 1- ε_a (q-1). Where ε_a (q-1) defines the incubation rate for an age group *a* for state q – 1. The incubation rate defines the probability of the population moving to infectious or asymptomatic.

$$I^{t+1}_{i,a}(T_e+1) = E^t_{i,a}(T_e) + \sum_{q\in\{1,2,\ldots,T_e-1\}} \left(\varepsilon_a(q)E^t_{i,a}(q)\right)(1-\varphi) \tag{3.4}$$

Equation (3.4) describes the new infectious population that will occupy day 1. The equation considers the exposed population from all stages, and all age groups. As defined above in (3.3), a proportion of the exposed population moves to infectious or asymptomatic depending on the incubation rate ε_a. The rate at

which the exposed population becomes either infectious, or asymptomatic is defined by asymptomatic rate φ. Thus, for the case of new infectious population the rate is defined as (1 – φ).

$$I_{i,a}^{t+1}(q \geq T_e + 2) = I_{i,a}^{t}(q-1) * \left(1 - \gamma_a(q-1) - \mathrm{F}a(q-1)\right) \tag{3.5}$$

Equation (3.5) described the portion of the infected population that move to the next stage. The equation states that the infectious population for stage q equals the population of infectious in the previous stage, q *– 1* minus the population who move to either recovery or death. The portion of the population that move to the recovered or death states is defined by recovery rate γ and fatality rate F respectively.

$$A_{i,a}^{t+1}(T_e + 1) = E_{i,a}^{t}(T_e) + \sum_{q \in \{1,2,\ldots,T_e-1\}} (\varepsilon_a(q) E_{i,a}^{t}(q))\varphi \tag{3.6}$$

$$A_{i,a}^{t+1}(q \geq T_e + 2) = A_{i,a}^{t}(q-1) * \left(1 - \gamma_a(q-1) - \mathrm{F}a(q-1)\right) \tag{3.7}$$

Equations (3.6 and 3.7) define the asymptomatic state behavior following the same rules described in (3.4) and (3.5). Equation (3.6) defines the proportion of the exposed population that moves to the asymptomatic state (here, the asymptomatic population rate remains as φ). Equation (3.7) follows the same rules defined in where the asymptomatic population either moves to the next stage q, recovered or death.

$$\begin{aligned} R_{i,a}^{t+1}(T_e + T_i + 1) = {} & \left(I_{i,a}^{t}(T_e + T_i) + A_{i,a}^{t}(T_e + T_i)\right) + \\ & \sum_{q \in \{T_e+1, T_e+2, \ldots, T_e+T_i-1\}} \gamma_a(q)(I_{i,a}^{t}(q) + A_{i,a}^{t}(q)) \end{aligned} \tag{3.8}$$

Equation (3.8) describes the proportion of infectious or asymptomatic populations that become recovered. The equation defines that the total number of recoveries is equal to the total number of recoveries from the previous day plus the newly recovered population. The current day recoveries are calculated by taking the proportion of infectious and asymptomatic infections that move to the recovered stage using rate γ. Finally, the equation checks for the population that is on the final day of either infectious or asymptomatic, if their population does not move to the death state, they are added to the recovered state.

$$R_{i,a}^{t+1}(q) = R_{i,a}^{t}(q-1) \tag{3.9}$$
$$q \epsilon \{T_e + T_i + 2, T_e + T_i + 3, \ldots, T_e + T_i - 1\}$$

$$R_{i,a}^{t+1}(T_e + T_i + T_r) = R_{i,a}^{t}(T_e + T_i + T_r) + R_{i,a}^{t}(T_e + T_i + T_r - 1) \tag{3.10}$$

Equations (3.9 and 3.10) are used only if re-susceptibility is not enabled. Once the recovered population reaches the final day of recovery, they remain there for the rest of the simulation time.

$$R_{i,a}^{t+1}(q) = R_{i,a}^{t}(q-1) \tag{3.11}$$
$$q \epsilon \{T_e + T_i + 2, T_e + T_i + 3, \ldots, T_e + T_i + T_r\}$$

Equation (3.11) is an equation only used when re-susceptibility is enabled, i.e., patients who are now recovered will go through each day of recovery, when they reach the final day of recovery the population will move back into the susceptible population pool where they can be re-exposed.

$$\begin{aligned} S_{i,a}^{t+1} = {} & 1 - \textstyle\sum_{q=1}^{T_e} E_{i,a}^{t+1}(q) - \sum_{q=T_e+1}^{T_e+T_i} I_{i,a}^{t+1}(q) \\ & - \textstyle\sum_{q=T_e+1}^{T_e+Ti} A_{i,a}^{t+1}(q) - \sum_{q=T_e+T_i+1}^{T_e+Ti+Tr} R_{i,a}^{t+1}(q) - F_{i,a}^{t+1} \end{aligned} \tag{3.12}$$

Equation (3.12) is a "special equation" needed for the integrity of the model. Since we know that any given population starts in the susceptible state (excluding the starting cell) then the population that is not in any other state should remain susceptible.

We implemented the equations above, and when all the geographical cells are defined, they are placed into a top level coupled cell model called *geographical_coupled*, with configuration seen in Figure 3. At runtime, the *geographical_coupled* model is initialized using cells data provided from in a JSON input file

(using the methods described in the top model class *cadmium::celldevs:cells_coupled<T,C,S,V>*; Figure 3 shows how this coupled cell model is defined).

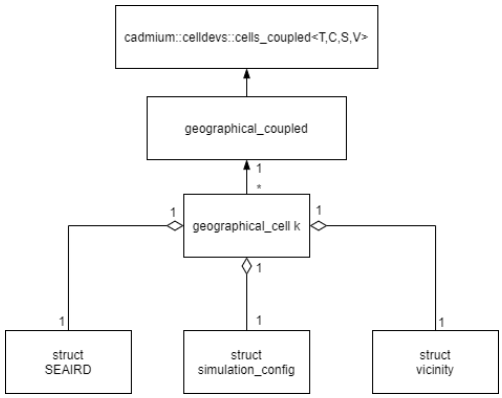

Figure 3. SEAIRD Coupled Cell Diagram

Figures 4 and 5 show code snippets that define the SEAIRD structure and the simulation configuration. Figure 4 describes the state variables discussed above (which relate to those described in figure 2).

```
struct seaird {
        std::vector<double> age_group_proportions;
        std::vector<double> susceptible;
        std::vector<std::vector<double>> exposed;
        std::vector<std::vector<double>> infected;
        std::vector<std::vector<double>> asymptomatic;
        std::vector<std::vector<double>> recovered;
        std::vector<double> fatalities;
        std::unordered_map<std::string,hysteresis_factor> hysteresis_factors;
        double population;
        std::vector<double> disobedient; ...
};
```

Figure 4. SEAIRD configuration code

Each cell contains the relevant information defined in the SEAIRD configuration file. At run time, each cell has a unique population which is divided into described age groups. Each cells population will then be divided into one of the six states. Generally, if modelling the beginning of a pandemic, a single cell will hold the initial case(s) and the remaining cells will be 100% susceptible. At *t=0* the proportion of a cell's population in each state is defined in by the values provided in the SEAIRD structure. Defining the SEAIRD structure at run time with user defined inputs allows users to choose the point of time they want to start a model (if they are interested in the middle of a pandemic they can tailor the input values to hold the number of population are in each state at that time).

```
struct simulation_config {
        int prec_divider;
        using phase_rates = std::vector<std::vector<double>>;

        phase_rates virulence_rates;
        phase_rates incubation_rates;
        phase_rates recovery_rates;
        phase_rates mobility_rates;
        phase_rates fatality_rates;
        double asymptomatic_rates;
        bool SIIRS_model = true;
};
```

Figure 5. Simulation configuration

In figure 5 the simulation configuration is declared; these values are used to change the ways a population transferred from one state to another. These structures define the *geographical_cell* atomic model presented in Figure 3. A *geographical_cell* atomic model is defined for each geographical cell in the model, these cells make up a *geographical_coupled* model where each cell is connected by a correlation factor. Finally, this geographical_coupled model is defined by methods found in class *cadmium::celldevs::cells_coupled<T,C,S,V>*.

4 CASE STUDY: ONTARIO PUBLIC HEALTH

The SEAIRDS results generated below are generated using source data from the 34 Ontario public health units where the population is generated using census data. A configuration file is built using a geo package to determine shared boundaries (correlation factor) between each public health unit. The graphs shown below are created using the graphing tools in [14] as well as R [22] and plotly [23]. The coming results will show and compare the affect the asymptomatic state has when added to the simulation. The simulations shown below have been run with the parameters in Table 1:

Table 1: Test case configuration

Parameter	**Value**
Population	Varies per cell based on census data
Age Groups	[0.216, 0.279, 0.268, 0.193, 0.044]
Disobedience	[0.29, 0.25, 0.23, 0.21, 0.24]
Asymptomatic Rate	Varies per simulation (See figure definition)
Virulence Rate	0.1 across all states and age groups
Incubation Rates	14 day profile
Mobility Rates (At run time)	1.0 across all states and age groups
Recovery Rates	0.07 across all states and age groups
Fatality Rates	0.005 across all states and age groups
Infection correction factors (lockdown)	0.001: [0.60, 0.0008], 0.005: [0.50, 0.003], 0.01: [0.40, 0.005], 0.03: [0.30, 0.015], 0.08: [0.20, 0.0005], 0.15: [0.1, 0.08], 0.20: [0.01, 0.12]

In Table 1 the parameters for the model are described. We start with the population of each geographical area. We then use a vector of values to represent the proportion of the total population in each age group. Age groups are an abstract set of values defined by the modeler; in our case, individuals 0-12, 13-19; 20-44; 45-65, and over 65 years old. Disobedience follows the same format as age groups where the values in the vector represent the proportion of each age group that is disobedient to lockdowns. The asymptomatic rate is the proportion of the exposed population that become asymptomatic (the rest become infectious). The virulence rate represents the rate at which the disease spreads, the value represents the amount of age groups population that is in contact with the infected population per day of the infection. Incubation rate is the proportion of the exposed population that become infectious or asymptomatic, defined using a 14 day profile where each day a proportion of the exposed population will move to the next state [16]. Mobility rates define the freedom of the population to move (1.0 mobility rating means the population can move freely). Mobility rates are defined for each age group for each day of the infection. Recovery rates define the proportion of an age groups infected population that will recover each day of the infection. Fatality rates defines the proportion of an age group infected population that will move to the deceased state on each day. Infection correction factors describe the proportion of the population that needs to be infected before a lockdown is put in place. The values can be described as follows: ‘Proportion of population infected to start lockdown’: [‘mobility modifier’, ‘Proportion of population infected to life lockdown’] where mobility modifier reduces the mobility of a cell by the given value.

Figure 6 shows how the results of this test case looks with 0% asymptomatic cases. The results show the steady rise of exposed individuals, then 1-14 days after their exposer they become infected.

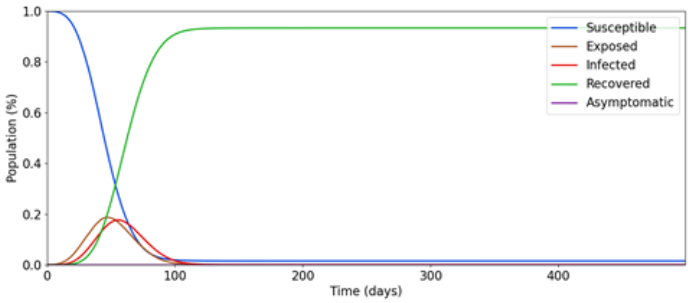

Figure 6. SEAIRD Model - 0% Asymptomatic

Next, we studied the effect of an 80% asymptomatic rate on the same parameters. The graph in figure 7 shows a lower rate of infectious carriers and a higher rate of asymptomatic carriers. With this higher rate of asymptomatic carriers out total exposed population hits a higher peak than it had without asymptomatic cases. This higher exposed count is due to the different asymptomatic and infectious carriers have on the susceptible population. Since the asymptomatic carriers travel more than the infectious carriers more of the population is exposed to them, causing higher overall infections.

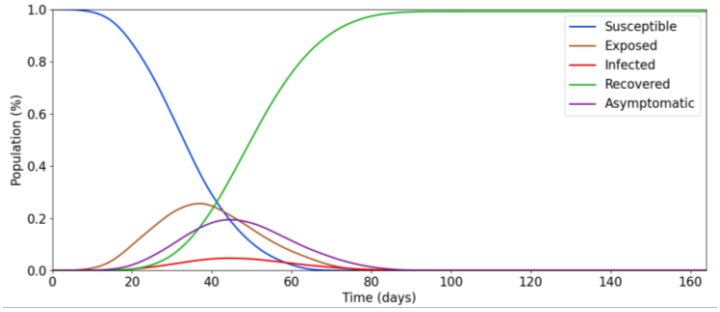

Figure 7. SEAIRD Model - 80% asymptomatic

Figure 8 shows a single cell and how its population transitions through the states with a 0% asymptomatic rate. It should be noted the cell is still exposed to its neighboring cells. When examining the graph at day 16 we can see a cumulative exposed population of 35.7%, cumulative infectious population of 18.9%, cumulative fatalities of 0.3%, and since we have no asymptomatic infections, an asymptomatic infected population of 0%.

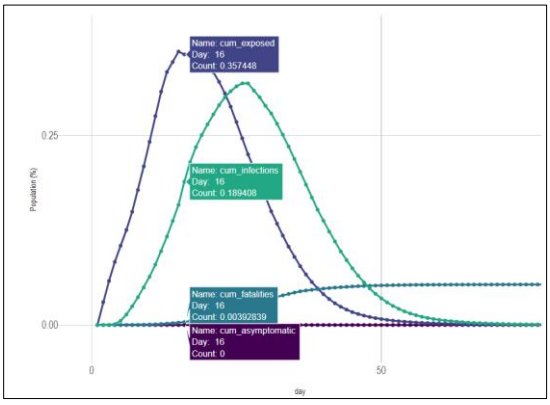

Figure 8. Single Cell SEAIRD - 0% Asymptomatic

If we then compare this to a graph showing the curves with an asymptomatic rate of 80% (Figure 9), we can see that at day 16 we now have a cumulative exposed population of 55.7%, cumulative infectious population of 4.9%, cumulative fatalities of 0.08% and an asymptomatic population of 20.2%. We can see that exposed has grown to 55%, from 35% this large growth can be accredited to the asymptomatic carriers spreading the disease to the surrounding neighbors at a higher rate than the infectious population. We can also see that we have fewer deaths in this simulation, this is linked to the fewer infectious cases.

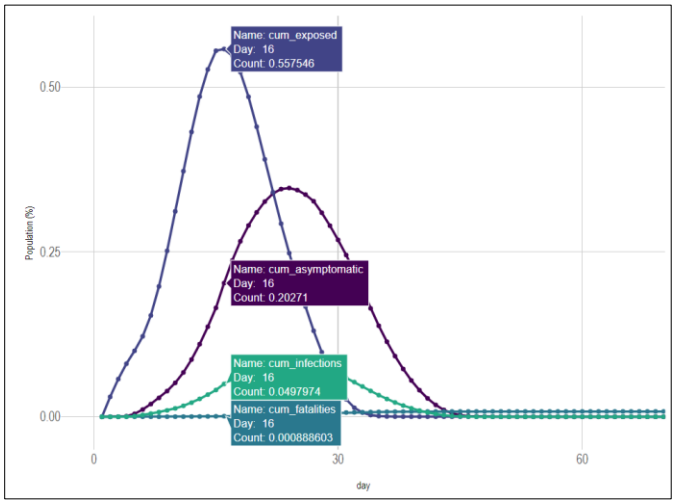

Figure 9. Single Cell SEAIRD - 80% Asymptomatic

To summarize the results shown above in Figures 5 and 6. We can now simulate the affect a "invisible" population of disease carriers may have on a pandemic. If studies show that 80% of the total COVID-19 cases are asymptomatic and the case counts show 5% of a population are infected, we can expect that 20% more of the population is also infected but not showing symptoms and not aware they are infected. These asymptomatic individuals will be spreading the disease to the healthy population and they may have no idea, causing rises in the number of exposed individuals, resulting in more infectious and deadly cases.

5 CONCLUSIONS

We presented the definition of a model that allows users to create quick simulations to help simulate how much of an impact asymptomatic cases would have on disease case counts. Another important note about the model, although it has been built around the COVID-19 pandemic, it can be used for any other disease. This can be done in a quick, efficient manner. If users have the relevant information for a disease along with the asymptomatic rate at which a disease transmits, they can simply change the parameters and re-run the model. The model also gives the users the ability to efficiently adapt the geographical level that the model is being run on. Future adaptations plan on incorporating asymptomatic disease transmission rates (modified virulence rates), asymptomatic rates by age group and finally, a more accurate susceptible to exposed transition.

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