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# Agent-Based SARS-CoV-2 Simulation Model

## Model Specification

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### Abstract

To simulate solely the spread of SARS-CoV-2 a variety of methods exists, of which many are probably more suited for prognoses than agent-based models. Yet, in order to evaluate the impact of policies like tracing, individual-based models are required.

We developed an agent based simulation model to reproduce the current outbreak of Covid-19 in Austria that allows for exploratory analysis of tracing in different characteristics. Aim of this work is the presentation of this model and consequent evaluation and comparison of different policies.

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# 1 Model Definition

We will explain our agent-based Covid-19 model based on the ODD (Overview, Design Concepts, Details) protocol by Volker Grimm et.al. [28, 29].

## 1.1 Overview

The modelling of the spread of the disease is based on the interplay of four modules.

1. Population. Altogether the agent-based Covid-19 model is based on the Generic Population Concept (GEPOC, see [18]), a generic stochastic agent-based population model of Austria, that validly depicts the current demographic as well as regional structure of the population on a microscopic level. The flexibility of this population model makes it possible to modify and extend it by almost arbitrary modules for simulation of population-focused research problems.
2. Contacts. In order to develop a basis for infectious contacts, we modified and adapted a contact model previously used for simulation of influenza spread. This model uses a distinction of contacts in different locations (households, schools, workplaces, leisure time) and is based on the POLYMOD study [37], a large survey for tracking social contact behaviour relevant to the spread of infectious diseases.
3. Disease. We implemented a module for the course of the disease that depicts the current pathway of SARS-CoV-2 infected persons starting from infection to recovery and linked it with the prior two modules. **Note, that the current version of the model actually does not depict the illness Covid-19 but solely focuses on the spread of the virus. This strategy was found useful since the feedback from severe disease outcomes such as hospitalisations or deaths was found negligible small for the virus spreading behaviour. Illness specific outcomes are evaluated in post-processing steps, e.g. using specific hospitalisation models.** Thus, in the present model *recovery* is equivalent with the loss of *infectiousness* of the agent.
4. Policies. Moreover, we added a module for implementation of interventions, ranging from contact-reduction policies, hygienic measures, contact tracing to vaccinations. This module is implemented in form of a timeline of events.

### 1.1.1 Purpose

The agent-based Covid-19 model aims to give ideas about the potential impact of certain policies and their combination on the spread of the disease, thus helping decision makers to correctly choose between possible policies by comparing the model outcomes with other important factors such as socioeconomic ones. In order to fulfill this target, it is relevant that the agent-based Covid-19 model validly depicts the current and near future distribution and state of disease progression of infected people and their forecasts.

In the following overview of the model, we will not state any parameter values to focus on the model concept. A full collection of model parameters including values, sources and justifications is found in Section 1.3.3.

### 1.1.2 Entities and State Variables

Each **person-agent** is a model for one inhabitant of the observed country/region. We describe state variables of a person-agent sorted by the corresponding module.

**Population.** Each person-agent contains the population specific state variables *sex*, *date of birth* ( $\cong$  age) and *location*. The latter defines the person-agent’s residence in form of latitude and longitude and uniquely maps to the agent’s municipality, district and federal state.

**Contacts.** Independent on how, where and with whom the person-agent has contacts with, it is assigned an individual scalar *contactivity* parameter that models, how many contacts this agent typically has. This parameter is sampled once at the start of the simulation and remains constant for the whole simulation time. Agents with low *contactivity* have, on average, a smaller number of daily contacts. Moreover each person-agent features a couple of contact network specific properties. These include a *household* and might include a *workplace* or a *schoolclass*. We summarise these as so-called *locations* which stand for network nodes via which the person-agent has contacts with other agents. As well as person-agents, locations have their own coordinate which uniquely maps to political regions. Assignment of person-agents to *locations* is based on distance of the agent’s residence to the position of the *location*. Each day, an agent has a certain number of contacts within each of the *locations*, which essentially leads to spread of the disease. To model contacts apart from these places, every person-agent has an additional amount of leisure time contacts, which are sampled randomly based on a spatially-dependent distribution. Some locations are themselves summarised in so-called *location-collections*: Multiple *schoolclasses* and one *workplace* representing teachers are summarised into one *school*, multiple *households* and one *workplace* representing care home workers are summarised to one *care-home*. If a location is part of a location collection, some contacts are scheduled across different locations within the collection. The contact network is schematically displayed in Figure 1.

For disease spread, contacts between infectious and susceptible agents are important. At each contact the disease is transmitted with a certain probability (see Section 1.1.4).

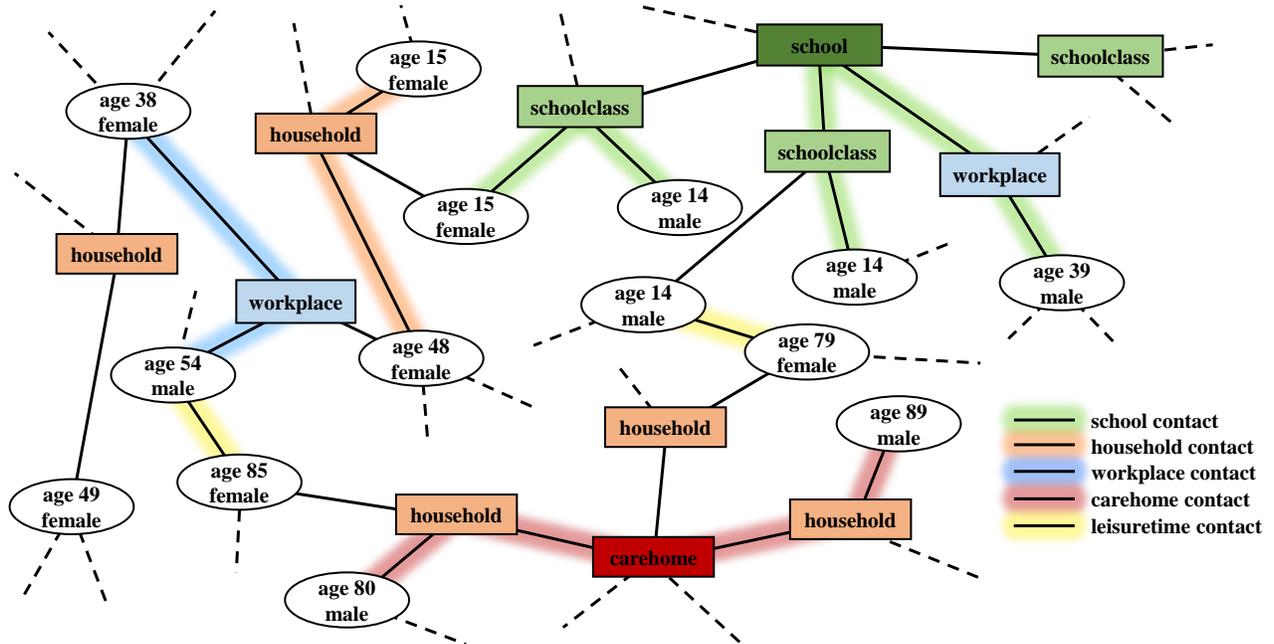


Figure 1: Contact network of agents in the agent-based Covid-19 model. Regular contacts between agents occur via locations (schoolclasses, workplaces and households), location-collections (schools, care-homes), while random leisure time contacts extend the standard contact network.

**Disease.** In order to model the spread of the disease each person-agent has a couple of health states that display the current status of the agent. They stand for certain points within the patient pathway of an infected person and enable or disable, respectively, certain person-agent actions. The disease states relevant for the simulation dynamics are *infected*, *infectious*, *susceptible*, *severity* and *infectiousness*. The prior two are boolean states, that can either be true or false, and multiple of them can be true at a time. The meaning of these attributes is self-explanatory. The state *susceptible* is an array of boolean variables, one for each virus strain to consider in the simulation. To decide the progression of the person-agent’s disease, a state *severity*, which can be *symptomatic* and *asymptomatic* is sampled as soon as the person-agent becomes infected. Disease state *asymptomatic* means that the agent will have no (asymptomatic) or very mild symptoms, so the person is not going to be detected by the standard test. State *symptomatic* means, that the person will issue a test as soon as symptoms set on. As soon as *infectious=true* the person-agent’s contacts become infectious, and the probability of infection is based on a continuous infectiousness curve ( $\beta_{dyn}(t)$ ) that depends on the sampled latency, incubation and recovery time (see Figure 2). Finally, since immunisation and disease progression depends on the virus strain, the current *strain* is also one of the disease states of the person-agent. It is inherited from the infection origin and passed on via secondary infections.

To make generation of simulation output easier, we sometimes make use of derived parameters such as *undetected* ( $=infected \wedge \neg confirmed$ ) or additional book-keeping variables such as infectious contacts per infected, reinfection or the full infection tree. Stating all these would make this documentation unhandy and difficult to read though.

More on the influence of the state variables and how they change is described in Section 1.1.4.

Table 1: State variables of each person-agent.

Population specific states	
<i>sex</i>	{female,male}
<i>date of birth</i>	date
<i>location</i>	(latitude, longitude)
Contact specific states	
<i>contactivity</i>	$\mathbb{R}^+$
<i>household</i>	<i>household-location</i> (optional) within <i>care-home-location</i>
<i>schoolclass</i>	(optional) <i>schoolclass-location</i> within <i>school-location</i>
<i>workplace</i>	(optional) <i>workplace-location</i>
Disease specific states	
<i>infected</i>	boolean
<i>infectious</i>	boolean
<i>susceptible</i>	boolean for each virus strain
<i>severity</i>	{asymptomatic, symptomatic}
$\beta_{dyn}$	[0, 1]-valued function of $t$
<i>strain</i>	virus strain
Policy specific states	
<i>detected</i>	boolean
<i>quarantined</i>	boolean

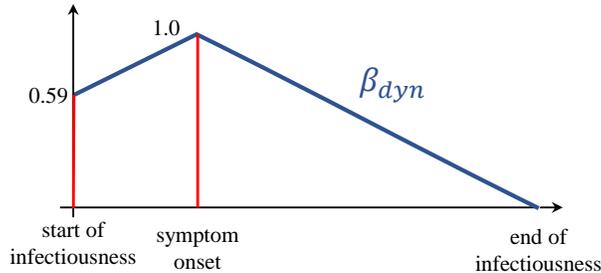


Figure 2: Relative infectiousness  $\beta_{dyn}(t)$  dependent on latency, incubation and sampled end of infectiousness

**Policies.** Policies apply either to person-agent-behaviour directly or indirectly via *locations*. All *locations* except for households are defined *open* or *closed* which marks whether this place is available for having contacts. The directly policy-related states for person-agents relate to the testing regime: An agent with attribute *detected=true* is detected by a SARS-CoV-2 PCR or antigen test. Note, that we don't consider false positive tests in the model. In the current implementation of the model, any *symptomatic* person-agent will become *detected* in the course of its disease. Agents directly marked as *detected* will also be *quarantined*. This state marks isolated agents which limits their contacts. Quarantine is not only issued via positive test but also due to tracing measures (see later).

All person-agent states are summarised in Table 1. For the sake of simplicity of speech we furthermore address mentioned parameters as attributes for the corresponding agents. I.e. an agent with *infectious* set to true will be denoted as “*infectious agent*”.

### 1.1.3 Scales

Unlike other agent-based models it is not possible to validly run the model with a smaller number of agents (e.g. one agent represents 10 or 100 persons in reality) as certain contact-network parameters do not scale this way (average school size, . . .). Consequently, one simulation run always uses agents according to the size and structure of the full population.

### 1.1.4 Process Overview and Scheduling

Like the underlying population model, the agent-based Covid-19 model can be interpreted as a hybrid between a time-discrete and a time-continuous (i.e. event-updated) agent-based model:

The overall simulation updates itself in daily time steps, wherein each step is split into four phases. In the first phase each agent is called once to plan what it aims to do in the course of this time step. In the second phase, each agent is, again, called once to execute all planned actions for this time step in the defined order. In the third step, a couple of global actions, i.e. actions not triggered by any person-agent, are executed. We attribute these actions to the *government agent* as introduced in [18]. These are vaccinations, screening tests and external infections. In the fourth step, a recorder-agent keeps track of all aggregated state variables.

On the microscopic scope, each person-agent is equipped with its own small discrete event simulator. In the mentioned planning phase, each agent schedules certain events for the future which may, but not necessarily must, be scheduled within the current global time step. In the second phase, the agent executes all events that are scheduled for the currently observed time interval, but leaves all events that exceed this scope untouched.

This strategy comes with the following benefits:

- In contrast to solely event-based ABMs, the event queue is distributed among all agents which massively increases the speed for sorting (a solely event-based ABM with millions of complex agents would not be executable in a feasible time).
- In contrast to solely event-based ABMs, usage of daily transition probabilities/rates instead of transition times is possible as well.
- In contrast to solely time-discrete ABMs, agents can operate beyond the scope of time steps and sample continuous time-intervals for their state-transitions.

We shortly describe all actions that are scheduled and executed by one person-agent within one time step sorted by the specified module. We will lay a special focus on the immunisation process. Finally we will explain the actions of the *government agent*.

**Population.** As briefly described in [18], agents trigger birth and death events always via time- and age-dependent probabilities that apply for the observed time step (i.e. the observed day). If one of these events triggers, the agent samples a random time instant within the current time step and schedules the event.

**Contacts.** Also contact specific events are scheduled and executed within the scope of only one time step. We summarize all contact events planned and executed within one timestep in Table 2.

Table 2: Contacts sampled within one time-step. In addition to the depicted conditions, *quarantined* agents don't have any contacts outside their household, *hospitalised* agents don't sample contacts at all. Moreover, *Poi* stands for the poisson distribution, *c* for the individual *contactivity* and *dlc*, *dsc* and *dwc* for *daily leisuretime*, *school* and *work contacts*

condition(s)	contact type	number of contacts per time-step	sampling method
	<i>household</i> contact	size of household - 1	one with every member
	<i>leisuretime</i> contact	$Poi(c \cdot dlc)$	random in whole agent list, based on regional distribution
agent has <i>schoolclass</i> $\wedge$ <i>schoolclass</i> is open $\wedge$ <i>schoolclass</i> is not quarantined	<i>school</i> contact	$Poi(c \cdot dsc)$	fraction randomly in own class, rest randomly in whole school
agent has <i>workplace</i> $\wedge$ <i>workplace</i> is open $\wedge$ <i>workplace</i> is not quarantined $\wedge$ <i>workplace</i> is <b>not</b> part of <i>location collection</i>	<i>workplace</i> contact	$Poi(c \cdot dwc)$	randomly in <i>workplace</i>
agent has <i>workplace</i> $\wedge$ <i>workplace</i> is open $\wedge$ <i>workplace</i> is not quarantined $\wedge$ <i>workplace</i> is part of <i>school</i> or <i>care-home</i>	<i>school/ care-home workplace</i> contact	$Poi(c \cdot dwc)$	randomly in whole <i>school/care-home</i>
<i>household</i> is part of <i>care-home</i> $\wedge$ <i>care-home</i> is open $\wedge$ <i>care-home</i> is not quarantined	<i>care-home</i> contact	$Poi(c \cdot dwc)$	randomly in whole <i>care-home</i>

Contact partners for leisure time are drawn based on an origin-destination matrix on municipality resolution. The latter has been gathered from mobile data (see Tables 4-5).

Anyway, planned contacts are always scheduled for the beginning of the new time-step. Hence, interaction between agents is actually limited to the discrete time steps of the simulation. This guarantees, that the states of both involved agents do not differ between the time of the planning of the event and its execution.

**Disease.** First of all, it is important to mention that the model is not parametrised by a reproduction number  $R_0$  or  $R_{eff}$ , but by a contact-specific probability for a transmission in case of a contact. Nevertheless, the agent-based model provides the opportunity to generate estimates for  $R_0$  and  $R_{eff}$  by its original definition: the average number of secondary infections of an infected agent. Hence, what comes as model input for many traditional SIR models becomes a model output for the agent-based Covid-19 model.

In case of a contact, *infectious* agents spread the virus, in specific the specific virus strain, spreads to *susceptible* agents with a certain *infection probability*. This probability calculates as a product of different factors:

$$P(\text{transmission}) = \min(\beta_{str} \cdot \beta_{dyn} \cdot \beta_{cl} \cdot \beta_{loc} \cdot \beta_{ex}, 1) \quad (1)$$

Where,

- $\beta_{str} = \beta_{str}(st)$  depends on the virus strain *st*. While this value is calibrated for the original SARS-CoV-2, values for virus variants are set according to estimates for excess transmissibility.
- $\beta_{dyn} = \beta_{dyn}(t)$  depicts the current infectiousness of the agent. See above (e.g. Figure 2) for more information.
- $\beta_{loc} = \beta_{loc}(loc)$  depends on the location of the contact. Typically, *household* contacts are weighted more transmissible due to the closeness of the involved persons.

- $\beta_{cl} = \beta_{cl}(t)$  depicts the current seasonality (climate) and is parametrised with weather data. It is proportional to the concentration rate value suggested in [22].
- $\beta_{ex} = \beta_{ex}(t, region)$  depicts impact of exogenous factors which are not included in the model and depends on time and region. This parameter is typically free for calibration purposes to fit the case numbers to given data. For forecasts it is set to one.

Anyway, an infectious contact triggers the start of the newly-infected agent’s patient-pathway. This pathway describes the different states and stations an agent passes while suffering from the Covid-19 disease and can be interpreted as a sequence of events of which each triggers the next one after a certain sampled duration.

We show this infection strategy in a state chart in Figure 3 and describe how to interpret this figure by explaining the initial steps in the pathway in more detail: As soon as a person-agent becomes infected, its *infected* state is set to *true*, its *susceptible* variable is set to *false* (there are no double-infections in the model), and its *severity* parameter is drawn from a given distribution. Moreover, a *latency period* is sampled according to a distribution as well. The corresponding “Infectious” event is scheduled for the sampled time instant in the future. As soon as this “Infectious” event is executed, the *infectious* parameter is set to *true* and a parallel branch that updates the *infectiousness* is started. After the “Finish Incubation” event, the first branch in the patient’s pathway decides whether the agent continues being detected by the standard test-regime, or continues undetected due to mild or nor symptoms at all. All other elements of the pathway follow analogously. All branches are evaluated with age-class-dependent probabilities (see Section 1.3.3).

After *recovery* (i.e. after the agent is not infectious anymore), the original susceptibility state before the infection is restored. Afterwards, immunity is decided in the *Sample Immunity* event. We explain this process in detail below in paragraph “Immunity Gain and Loss”.

**Policies.** Every policy is modelled as a global event occurring before the planning phase of any of the simulation time steps. Policies are timed-events that are fed into the model as an event-timeline (see Figure 4). The elements of this timeline may include real policies like closure or opening of locations, start of tracing, vaccination rounds (for a full list, see Table 10), but may also contain incidents that change the model behaviour but are not directly related to policies, such as raising hygiene awareness. The most outstanding feature of the model is clearly its ability to model contact tracing policies, since agents are aware of all other agents with which they had contacts. Using simple housekeeping arrays, these can be logged for a certain period of time and used for detection and isolation of contact partners.

Due to the huge flexibility of this strategy, the pool of available policies that can be added and combined in simulation scenarios is huge. In Table 10 the reader finds those which have been included to the canonical main-version of the model and which are used for the most fundamental research problems.

**Immunity Gain and Loss.** The immunisation and immunity-loss process is one of the most important processes in the model. It is crucial to understand that being *immune* ( $\neg$  *susceptible*), depends on the virus strain. That means, that person-agents can be immune against infection with one virus strain yet being susceptible against the other. In the model we distinguish between

- *immunisation cause*, i.e. all things that may lead to immunity such as recovery and vaccination, and
- *immunisation targets*, i.e. all things a person-agent could get immune against, usually infection by a certain virus strain. In some model versions this list is extended by illness-specific outcomes such as severe or critical disease progression.

In the context of this paragraph we denote the implemented immunisation causes by  $X_{1,\dots,n}$  and the targets by  $Y_{1,\dots,m}$ . The *Sample Immunity* event (compare Figure 3) depends on the *immunisation cause*, say  $X_i$ , and evaluates immunity against all  $Y_{1,\dots,m}$ . This is done as follows:

1. For all targets  $Y_j$ , a base probability value  $b_{i,j}$  decides whether immunity is gathered at all. To decide this, a  $U(0,1)$  random number  $u$  is drawn. For all  $j$  with  $u \leq b_{i,j}$ ,  $\text{susceptible}(Y_j)=\text{false}$ . For all  $j$  with  $u > b_{i,j}$ , the susceptibility values is unchanged.
2. Furthermore, a real valued random number  $z$  is drawn using a positive distribution with mean value 1. For all  $j$  selected to become immune, an *Immunity Loss* event is scheduled in  $z \cdot m_{i,j}$  days, where,  $m_{i,j}$  is a scaling factor which can be interpreted as the average number of days until immunity against  $Y_j$  is lost if provided by  $X_i$ .
3. If the prior step now leads to two scheduled *Immunity Loss* events for the same target  $Y_j$ , the earlier one is discarded.

This strategy seems unintuitive and unnecessarily complex, but it helps to model the impact of multiple immunisation events. For example, step 3 allows that a vaccination is able to prolong the immunity previously gained through an infection. Even without availability of specific data any sequence of immunisation events can be evaluated in a plausible way. Moreover it provides all necessary freedom to establish immune-escape variants.

**Government-Agent actions.** In addition to the dynamic processes triggered by the agents alone, three modules are implemented which are triggered by the top-level *government agent*. These are *vaccinations*, *screenings* and *imports*. The following actions take place on daily basis.

Vaccinations. A number of vaccine doses are distributed to capable model agents. Since the current model version, we do not distinguish between different vaccine types anymore but only consider first, second, third, ... shot and regard them by age and region. A person-agent is capable to get a vaccine if (a) it belongs to the correct age class, (b) lives in the correct region, (c) is not currently detected, and (c) fulfils the correct prerequisites (a second vaccine shot can only be issued to a person which already received a first one).

After being vaccinated (*Vaccination* event), a *Sample Immune* event is scheduled after a *vaccine delay* duration. This event renders a person-agent immune against a strain with a given, shot- and strain-dependent probability for a given shot- and strain-dependent time (see Figure 3). The concept is equivalent with the immunisation process after recoveries (see above). Thus the effects of multiple vaccine doses accumulates.

Screening Tests. A certain number of randomly selected non-*confirmed* person-agents are selected and screened for being *infected*. The modelled tests have a certain sensitivity so that *infected* agents are only found with a certain probability. If the test is positive, the found person-agent is labelled as *confirmed* and treated equivalently to the symptomatic agents (compare *Make Test* event in Figure 3).

Imported Cases. A certain number of randomly selected agents with age between 20 and 40 are chosen for having external contacts with the virus, in specific, a certain virus strain. This age class was chosen since it was evaluated to be the most reactive w.r. to the spread of new virus strains. If the selected person-agent is *susceptible* against the strain, the *Infection* event is triggered (compare Figure 3).

This mechanism introduces the virus into the simulation. By varying the distribution for the imports, also new virus strains are introduced in the infection network.



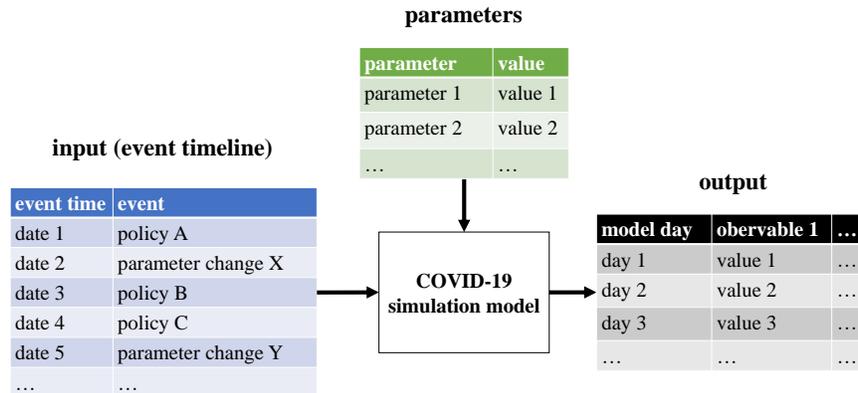


Figure 4: Event-timeline as the input of the simulation model in contrast to standard model parameters.

## 1.2 Design Concept

### 1.2.1 Basic Principles.

Increasing the level of detail from a standard epidemiological model for simulation of disease waves to a model that is capable of dealing with various different policies is a huge step with respect to model complexity. It excludes the use of macroscopic strategies and requires modelling of a contact network and contact behaviour. Consequently a detailed demography, spatial components and stochasticity need to be introduced to the model which come with a huge number of additional parameters and parameter values.

Hence, we were very careful that the agent-based model is designed as simple as possible yet tracking the most important features for evaluation of certain policies. Hereby, many details within the pathway of an infected person and, in particular, lots of details within the personal daily routine are simplified to avoid indeterminable model parameters and unpredictable model dynamics.

### 1.2.2 Emergence.

In addition to the classic emergence of nonlinear epidemiological effects, analysis of the effects of interaction between different measures is one of the key objectives of the model. For example, seemingly unconnected policies like *school closure* and *contact reduction for the 65+* might lead to unexpected effects when applied simultaneously. More generally speaking, the model displays that the individual effects of applied policies do not add up linearly.

### 1.2.3 Sensing.

Agents' perception of reality is one of the key problems of modelling Covid-19 as no agent is actually aware of its own disease and, more importantly, infectiousness until symptoms occur. Therefore, agent parameters can be distinguished into two sets: the ones the agent is aware of (e.g. *detected*), and the ones it is not (e.g. *infected*, *infectious*).

Interestingly, besides the individual perception of agents and the perception of an omniscient observer, there is also a third level of perception included into the model: the perception of the general public. While an individual

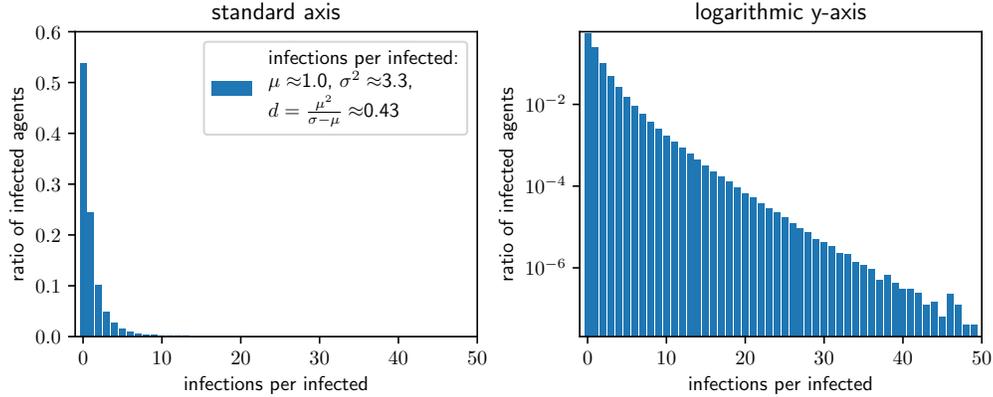


Figure 5: Infections per infected of a fully unconstrained epidemic wave. Note, that such a scenario causes the average number of secondary infections per infected tracked over the whole time-frame (here calculated as  $\mu$ ) to be slightly smaller than 1. The dispersion factor  $d$  can be estimated by the stated formula considering mean and variance of the distribution.

agent knows about its symptoms, the public is not yet aware of this additional infected case, until the person-agent has reacted on the disease, has had itself tested and eventually becomes *confirmed*. Consequently, the levels of perception can be sorted with regards to their amount of knowledge:

omniscient observer > person-agent > general public.

#### 1.2.4 Interaction.

Interaction between agents only occurs in form of contacts at *locations* or *leisure time*. The features provided by the underlying population model make it possible to investigate contacts on a very local level. As described before, *leisure time* contacts are weighted by their regionality, but also *school* and *workplace* contacts depict locality: Using specified latitude and longitude for locations, it is possible to assign person-agents with distance-dependent probabilities (see Section 1.3.1). Consequently, interactions between agents follow a spatially-continuous locally-biased contact network.

#### 1.2.5 Stochasticity.

Basically all model processes, including the initialisation, contain sampling of random numbers. Therefore, Monte Carlo simulation is applied, results of runs are averaged and also their variability is assessed (see Section 2.1).

Yet, besides being time-consuming to flatten, the stochasticity of the model is actually its key strength. It allows to model heterogeneity and skewness of the infection-network which distinguishes the model from classic macroscopic approaches. This specifically refers to the way, how contacts are modelled: Since the person-agent's *contactivity* is initially drawn from a Gamma distribution, the contacts sampled via Poisson distribution result in a so-called Gamma-Poisson mix, which is by definition Negative-Binomial distributed. This strategy allows to directly parametrise the skewness of the contact network to published information on the dispersion factor of Covid-19 clusters (see Figure 5).

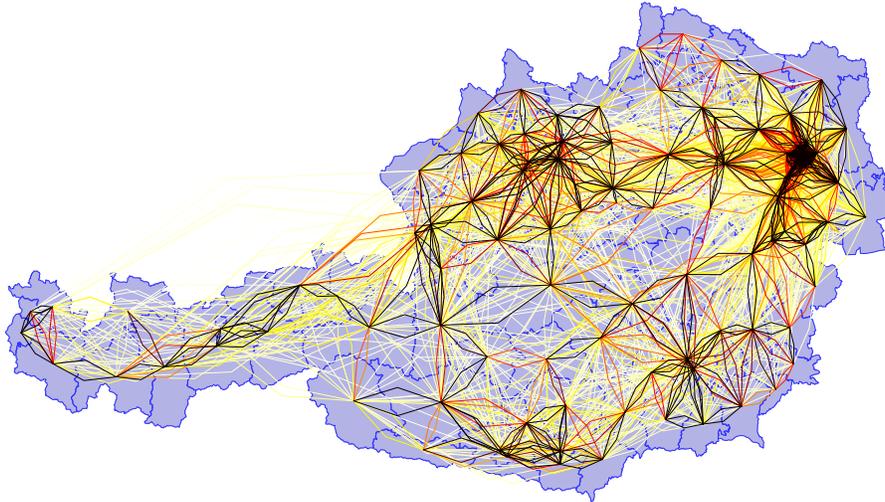


Figure 6: Visualisation of the origin destination matrix used for assignment of persons to locations. Dark red lines indicate many inter-regional contacts, thin white and yellow lines indicate few contacts.

### 1.2.6 Observation.

Inspired by [38], a recorder-agent takes care about tracking and aggregating the current status of the simulation. At the end of each global time step, all person-agents report to the recorder-agent which furthermore keeps track of all necessary aggregated model outputs. This includes for example *confirmed active cases*, *confirmed cumulative cases*, *undetected agents*, *incubating agents*, *recovered agents*, *agents in a certain hospital*, or *average-number of contacts per infectious agent*. If required, numbers can also be tracked with respect to age, sex, regional level and/or contact-location.

## 1.3 Details

Clearly, Section 1.1 could only outline the basic concepts of the model and left a lot of technical and modelling details necessary for a reproducible model definition open. In particular, this refers to the highly non-trivial initialisation process of the model. In this process, two problems occur that require completely different approaches. The first problem considers the generation of the person-agents, locations and hospitals in the first place. The second problem deals with the initialisation of the status quo of the distribution of the disease states of the agents for the specified initial date.

### 1.3.1 Initialisation of Person-agents, Locations and Hospitals.

A lot of problems that deal with the sampling of the initial population have already been solved in the original GEPOC model [18]. In particular this refers to the Delaunay-triangulation-based sampling method for locations. We apply this method to merge information from the national statistics institute and the global human settlement layer [26]. Consequently, besides initialisation of the disease states which is described in the next section, only new methods for location- and hospital-generation had to be implemented.

In general, locations and location collections are initialized in a two step mechanism. First, the location / location collection is created with a certain capacity. Secondly, the location / location collection is filled with agents / locations using a regional distribution. Similar to the sampling algorithm for leisure time contacts,

an origin-destination matrix on district level (see Figure 6) gathered from mobile phone data is used in the following way:

1. Filter the agent list for all agents that are suitable for being assigned to the location.
2. Given a certain location in district  $x$  and municipality  $xx$ , draw a random district  $y$  according to the distribution in the matrix.
3. If  $x \neq y$ , pick a randomly chosen agent from district  $y$ . If this fails, return to 2.
4. If  $x = y$ , a Bernoulli experiment decides to either pick a random agent from  $xx$  or from somewhere else in  $x$ . If this fails, return to 2.

For location collections, we follow more-less the same strategy, with suitable locations instead of agents. We go into more detail about sampling and filling for the specific location types:

Households are initialised given a discrete distribution of their sizes and household members. We distinguish between five groups: children ( $age < 18$ ), male and female adults (18 – 64) and male and female retired (65+). The number of households is created on demand, so that every person-agent can be assigned a household eventually. Household coordinates are drawn according to the same algorithm as creation of person-agents and are filled, as explained above. After a household is successfully filled, all coordinates of all household members are set to the coordinate of the household.

Workplaces<sup>1</sup> are initialised with a certain capacity by a *workplace-sampler* based on district-level data about branches of industry. Given the district, the coordinate is sampled re-using the mentioned sampler for person-agent coordinates. Note, that the workplace is hereby also assigned a certain occupation which will be required for sampling of *care-home* and *school workplaces*. Filling of workplaces works analogous to households, yet coordinates of person-agents remain unchanged.

Schools and schoolclasses are initialised based on known distributions w.r.t. average school size and number of pupils in total. We distinguish between schools for children below 14 and older. A *school-sampler* iteratively generates schools and assigns a random number of new created schoolclasses (triangular distribution) with fixed capacity. The process is repeated until the sum of all schoolclass capacities matches the known number of pupils in reality. In a next step, each school is assigned one of the created workplace with branch “teaching” and the school’s coordinate is set to the coordinate of the workplace. In a next step, classes are filled with the same algorithm as workplaces. Clearly, the number of model agents in the group of agents below 18 is larger than the number of pupils. Consequently, we force distribution of all 6 to 14 year old agents, and distribute as many 15 to 18 year old agents as possible. All remaining 15 to 18 year old agents are considered to be working or unemployed.

Care-homes are generated with a fixed size and providing space for a fixed number of inhabitants. Analogous to schools, every care-home is assigned a workplace with the corresponding branch and coordinates are harmonized. Furthermore, suitable households are assigned using the mentioned filling algorithm.

### 1.3.2 Initialisation of the Disease State

The spread of SARS-CoV-2 displays probably better than any other system that the most dangerous enemy is the invisible one. While confirmed infected persons are detected and well known, they hardly contribute to the spread of the disease – they are already isolated properly, and most infections occur even before the onset of symptoms.

Consequently, it is not possible to simply “start” the simulation with a certain number of confirmed cases, acquired for example from official internet sources. Valid values for pre-symptomatic (e.g. persons within

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<sup>1</sup>Workplaces should not be confused with total companies. They rather represent the different teams where the members are in regular contact with each other.

latency and incubation period) and asymptomatic persons need to be acquired as well – yet, this number is hardly measurable in reality.

In order to solve this problem, the model is either started from the start or from a fully serialised image of an other simulation. If started from the beginning, the initial infections are imported via the *government agent* in the course of the daily imported cases. If started from a serialised state, all agents and locations are imported from large, zipped json files. Accordingly, the simulation also provides a function to export its final state to these files.

### 1.3.3 Parametrisation

With respect to parametrisation, we will distinguish between model input and model parameters.

Classical model parameters specify scalar or array-typed model variables that are initialised at the beginning of the simulation and keep their value for the entire simulation time. Examples are the infection probability of the virus strains, or the number of school classes.

In contrast to model parameters, the model input consists of an event-timeline that describes at which point in time a certain incident changes the behaviour of the model. This incident usually refers to the introduction of a policy, e.g. closure of schools or start of tracing, but may also refer to instantaneous changes of model parameters which are related but cannot be directly attributed to policies, e.g. the increase of compliance among the population to increase hygiene.

In the following, we state lists of used parameters and parameter-values (status ) including corresponding sources and/or justifications. They are found in Tables 3 to 9. Table 10 states a list of possible event-timeline elements that can pose as the model’s input.

Table 3: List of population specific parameters (status March 22, 2023)

parameter	description	value	source
<i>birthrates, deathrates, initial population, regional distribution</i>	parameters used by the underlying population model	see source	rates and population tables from Austrian National Statistics Institute [10]. Maps from the Global Human Settlement Project [25] and [4].

### 1.3.4 Calibration

There are various parameters of the model which are calibrated to data and multiple calibration steps have to be done. These are

- $\beta_{str}$  of base virus
- contact reduction in *leisure-time contact number reduction events*
- number and virus strain(s) of imported cases (*change import model event*)
- exogeneous factor  $\beta_{ex}$  (region-dependent values in *change  $\beta_{ex}$  events*)

Goal of the calibration is to recreate a given SARS-CoV-2 transmission dynamics in a certain country. The corresponding reference is the

**7-day moving average of the time dynamics of the number of daily new confirmed cases per age class (person group) and region.**

Key challenge in this process is, that the agent-based model has a large number of agents and consequently a long computation time. Classic meta-heuristic methods such as simulated annealing, genetic algorithm, downhill-simplex algorithm, or particle swarm optimisation, which optimise/calibrate a whole vector of parameters at a time while calling the model thousands of times in a loop, cannot be used in feasible computation time. As a result we simplify the full vector-valued calibration process of the model to multiple sequential scalar calibration processes, in which only one parameter value is optimised. Key to this simplification is to compare the case numbers to the reference of a changed parameter (via a corresponding event) only in a short time-span after the event in which no other un-parametrised event takes place.

We define the following bisection algorithm:

**Calibration Version of Bisection Algorithm.** Let  $y_i, i \in \{0, \dots\}$  define the time-series of the calibration reference and  $x_i(p_1, p_2, \dots, p_n), i \in \{0, \dots\}$  stands for the corresponding model output. Hereby,  $p_1, \dots, p_n$  stand for all scalar parameters of the model which are subject to calibration. We define  $P_i := [p_i^u, p_i^l], i \in \{1, \dots, n\}$  as the upper and lower bound of their parameter spaces.

Furthermore the parameters are sorted with respect to their temporal effect on the outcome: We say, that a parameter  $p_i$  effects the outcome of the simulation after  $t_i$  if

$$\forall p_i, p_i' : x_i(p_1, \dots, p_i, \dots, p_n) = x_i(p_1, \dots, p_i', \dots, p_n), \forall i \in \{0, \dots, t_i\}. \quad (2)$$

We moreover assume that parameters  $p_1, \dots, p_{i-1}$  are already calibrated and their values  $\bar{p}_1, \dots, \bar{p}_{i-1}$  are fixed and investigate the scalar optimisation problem

$$\bar{p}_i = \operatorname{argmin}_{p_i \in P_i} |x_{t_{i+1}}(\bar{p}_1, \dots, \bar{p}_{i-1}, p_i, p_{i+1}, \dots, p_n) - y_{t_{i+1}}| \quad (3)$$

Note, that the values of  $p_{i+1}, \dots, p_n$  are irrelevant for this problem, since they influence the simulation outcome only after  $t_{i+1}$ . Clearly it would be possible to exchange the absolute difference by a more sophisticated norm, like

$$\bar{p}_i = \operatorname{argmin}_{p_i \in P_i} \|(x_{t_{i+1}}(\bar{p}_1, \dots, \bar{p}_{i-1}, p_i, p_{i+1}, \dots, p_n))_{t=t_{i+1}}^{t_{i+1}} - (y_{t_{i+1}})_{t=t_{i+1}}^{t_{i+1}}\|, \quad (4)$$

but experiences showed that a simple comparison of the two numbers at the last possible date produces more stable results.

Anyway, this optimisation problem is furthermore solved using a bisection strategy. To simplify the notation we write:

$$\hat{x}(p_i) = x_{t_{i+1}}(\bar{p}_1, \dots, \bar{p}_{i-1}, p_i, p_{i+1}, \dots, p_n), \quad \hat{y} = y_{t_{i+1}}.$$

Given a predefined number of iterations  $c_{max}$ , the bisection method works as follows:

1. Define the initial interval  $I_1 := p_i^l, I_2 := p_i^u$  and set the iteration counter  $c := 1$ .
2. Evaluate  $S_1 := \operatorname{sign}(\hat{x}(I_1) - \hat{y}), S_2 := \operatorname{sign}(\hat{x}(I_2) - \hat{y})$ .
3. Calculate  $I_3 := 0.5 \cdot (I_1 + I_2)$ .
4. If  $c = c_{max}$ , quit and return  $I_3$ , otherwise continue below.
5. Evaluate  $S_3 = \operatorname{sign}(\hat{x}(I_3) - \hat{y})$ .
6. If  $S_1 \cdot S_3 = 1, S_1 := S_3$ , else  $S_2 := S_3$ . Increment the iteration count  $c$  and continue with step 2.

The found parameter value is precise up to an error of size  $|P| \cdot 2^{-c_{max}}$ . Note, that the first calculation of the signs can be skipped, if the causality between the parameter and the case numbers is known in advance.

There are two event types subject to calibration, which are not parametrised by one scalar variable but by a vector. These are the *leisure-time contact reduction*, which is parametrised w.r. to person groups, and the *change  $\beta_{ex}$  events* which is parametrised w.r. to region. Thus, in many situations a minor adaption of this algorithm is used which compensates this problem using additional assumptions.

To extend the strategy presented before, we need to make additional simplification steps to change the problem type from a multidimensional to a scalar. In this case, we assume that in the investigated outcomes (cases per federalstate / per person group) depend from the parametrised values in the event independent from each other, at least in the investigated time horizon. The adaption is given as follows:

**Calibration Version of Bisection Algorithm - Simultaneous Strategy.** In this version, the reference  $\hat{y}$ , the outcome  $\hat{x}$  and the parameter  $p_i$  are vectors with equal lengths  $d$ . All operations in the iteration are executed pointwise:

1. Define the initial interval  $\vec{I}_1 := \vec{p}_i^l, \vec{I}_2 := \vec{p}_i^u$  and set the iteration counter  $c := 1$ .
2. Evaluate  $\vec{S}_1 := (\text{sign}(\hat{x}(I_1)_k - \hat{y}_k))_{k=1}^d, \vec{S}_2 := (\text{sign}(\hat{x}(I_2) - \hat{y}))_{k=1}^d$ .
3. Calculate  $\vec{I}_3 := 0.5 \cdot (\vec{I}_1 + \vec{I}_2)$ .
4. If  $c = c_{max}$ , quit and return  $\vec{I}_3$ , otherwise continue below.
5. Evaluate  $\vec{S}_3 = (\text{sign}(\hat{x}(I_3) - \hat{y}))_{k=1}^d$ .
6. For all  $k = 1, \dots, d$ : if  $(\vec{S}_1)_k \cdot (\vec{S}_3)_k = 1$ ,  $(\vec{S}_1)_k := (\vec{S}_3)_k$ , else  $(\vec{S}_2)_k := (\vec{S}_3)_k$ . Increment the iteration count  $c$  and continue with step 2.

The most important feature of both algorithms is the fact, that one only needs to run a comparably short time-snipped of the simulation. Say, parameter  $p_i$  is calibrated, the simulation state (i.e. the whole agent population and their contact network) can be serialised to a file at time  $t_i$ . For the calibration, the population can be deserialised and the simulation only needs to be run from  $t_i$  to  $t_{i+1}$  in the loop of the bisection strategy. Note, that full serialisation and deserialisation of the state of a model with the given size and complexity is a huge technological challenge which we will not address in this model documentation, though.

We furthermore explain how we apply the two algorithms in order to generate a model fully calibrated and fine tuned to the case data and on the example of the dynamics of the Austrian SARS-CoV-2 pandemic and involved data. That means, some of these steps might not be valid for other countries where the progression of the pandemic was crucially different. Our reference data are the Austrian data for new confirmed cases from the Epidemiologisches Meldesystem collected by AGES [2].

### Step 1. Parameter $\beta_{str}$ of base virus.

Clearly, there is no valid data available for direct parametrisation of the  $\beta_{str}$  parameter of the original SARS-CoV-2 virus which is the most fundamental of the factors that decide about a transmission in case of a direct contact. First of all, this parameter is hardly measurable in reality and moreover strongly depends on the definition of “contact”. Consequently, this parameter needs to be fitted in the course of a calibration loop.

The calibration experiment is set up as follows:

1. The simulation model is setup with an empty timeline and to simulate from Feb 1<sup>st</sup> to June 1<sup>st</sup> 2020.
2. An event changing  $\beta_{loc}$  is added to the timeline for April 10<sup>th</sup>.
3. Taking  $R_0 = 2.5$  as reference for the original virus, an event which reduces the overall transmissions by  $1/2.5 = 40\%$  should stop the increase and eventually lead to constant case numbers (the strategy is

comparable to [17]). Thus, the *betaloc* event is parametrised to decrease the infection-probability in all locations accordingly.

4. A bisection algorithm (see above) varies the  $\beta_{str}$  parameter of the original virus. The target outcome is the ratio between the average number of new infections in the last week of April compared to the last week of May. The closer the ratio matches 1, the more accurately the modelled disease has  $R_0 = 2.5$ .

Note: In this small time period and due to the small case incidence, the impact of the increasing number of immune individuals is negligible for the transmission dynamics.

### **Step 2. Policy events and their parameters for the first wave.**

In the next step, the policy events for the first wave and the first lockdown in Austria are parametrised. See Step 4 for strategic details.

### **Step 3. Imported cases.**

In particular over the summer months, the internal reproduction rate, i.e. the transmissions that happened within the country, in Austria was way below 1. Hence, the case incidence is mainly driven by externally imported cases.

In our calibration strategy, we assume that the number of daily imported new cases is directly proportional to the reported number of overnight stays in Austrian hotels by foreign tourists. The proportionality constant is fitted so that the steady-state level of new confirmed cases in July and August 2020 match with the case data (we minimise the area between the two curves after 7-day-moving average flattening). The model with calibrated first wave (Step 2) is used as basis. A bisection strategy is used.

### **Step 4. Policy events and their parameters.**

As soon as the base transmissibility of the original virus and imported cases are calibrated, the event timeline has to be set up. In this process ...

- ... all policies the selected country has established are translated to their corresponding events (closure of locations, quarantine laws, tracing strategies, hygiene measures,...),
- ... all holiday-related closure of schools or workplaces are translated to events,
- ... variant split events are generated to change the split of the variants of the imported cases.

Altogether, the established event timeline has several free parameters which need to be fitted. In particular this refers to the overall reduction of leisuretime contacts (per person grup) for which neither expert estimates nor measured values are available, and the import time of new variants. We use the bisection strategies introduced earlier.

**Step 5. Exogenous factors  $\beta_{ex}$ .** Even if all policies in reality are properly parametrised, still differences between model results and real case numbers will be given. Reasons for these differences can be manifold and range from changing adherence, short-time weather effects, single mass gatherings, changed test system, or simply randomness. To fix these, also events changing the  $\beta_{ex}$  parameter (i.e. *change  $\beta_{ex}$  events*) are added to the timeline in biweekly intervals. Their region-dependent values are calibrated using the simultaneous variant of the bisection strategy introduced earlier.

## 2 Model Implementation

Simulation of agent-based models like the agent-based Covid-19 model is a huge challenge with respect to computational performance. As the model cannot be scaled down, almost 9 Million interacting agents need to be included into the model in order to simulate the spread of the disease in Austria.

These high demands exclude most of the available libraries and software for agent-based modelling including AnyLogic [27], NetLogo [41], MESA [35], JADE [14] or Repast Symphony [39]. Most of these simulators cannot be used as their generic features for creating live visual output generates too much overheads.

Consequently, we decided to use our own agent-based simulation environment ABT (Agent-Based template, see [3]), developed in 2019 by dwh GmbH in cooperation with TU Wien. The environment is implemented in JAVA and specifically designed for supporting reproducible simulation of large-scale agent-based systems.

The next section contains more technical details about the implementation.

### 2.1 Technical Implementation Details

The implementation of the agent-based Covid-19 model uses JAVA 11 and applies the *UniformRandomProvider* random number generator (RNG) by Apache Commons [1]. This RNG implements a 64 bit version of the Mersenne Twister [36] and exceeds the standard RNG of JAVA, a simple Linear Congruential Generator, in both performance and quality.

The simulation itself is always executed in a Monte Carlo setting and several runs with different RNG seeds are averaged. Due to the huge number of agents, a Law-of-Large-Numbers-effect can be observed (similar to [15] Chapter 5.2), and the standard deviation of the model output is always comparably small. Consequently, Monte Carlo replication numbers of 10 to 20 are usually enough to estimate the mean sufficiently well (we apply the algorithms from [19, 31]).

## 3 Features and Limitations

Due to the highly flexible policy timeline, the model is capable of testing and combining lots of different policies in different characteristics at different times. Hence, it can easily depict almost any specified policy announced in reality, if estimates for the policy parameters are available.

The latter statement particularly refers to combination of policies: although the model correctly depicts the epidemiological impact of the combination of policies, the social impact needs to be parametrised manually or calibrated. For instance, the causal relation between closed schools and intensified parent-children contacts needs to be parametrised and is not given by the model dynamics.

Unfortunately, as the model cannot be scaled down, a huge number of agents lead to long computation times, and the necessity of Monte Carlo simulation for flattening of stochastic results increases the time required to get simulation output even further. Consequently, the simulation's capabilities of dealing with multi-variate calibration problems are limited. Consequently, the model is well capable but unhandy to generate (short-time) prognoses.

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Table 4: List of contact specific parameters (1/2, status March 22, 2023). Note that all parameter values are specified for the standard model without policies. The  $\Gamma$ -distribution is given as  $\Gamma(k, \theta)$ .

parameter	description	value	source
<i>contactivity</i> (hence-forth <i>ct</i> )	individual parameter to scale the average number of contacts per day, to ensure the skewness of the contact-network	$X \sim \Gamma(0.6, 1/0.6)$	calibrated to match a network dispersion factor of 0.43 as published in [5]
<i>leisure time contacts per day</i>	number of leisure time transmission-relevant contacts of an agent per day	$X \sim Poi(ct \cdot 6.11)$	based on the results of the POLYMOD study [37]
<i>workplace contacts per day</i>	number of transmission-relevant contacts at work (if assigned) of an agent per day. Same values used for care-home contacts.	$X \sim Poi(ct \cdot 5.28)$	based on the results of the POLYMOD study [37]
<i>school contacts per day</i>	number of transmission-relevant contacts at school (if assigned) of an agent per day	$X \sim Poi(ct \cdot 4.64)$	based on the results of the POLYMOD study [37]
<i>contact in other class probability</i>	probability of a pupil to draw a contact partner from the whole school and not only its own class	10%	Estimate
<i>household sizes and structure</i>	distribution of household sizes and structure	see source	distribution and structure from freely accessible tables for household statistics from the Austrian National Statistics Institute [10]
<i>school class sizes</i>	Capacity of school classes	20 for schools with pupils below 14, 23 otherwise	gathered from a publication of the Austrian National Statistics Institute [11]
<i>school sizes</i>	The actual number of schools and pupils were gathered to calculate the average number of classes per school. Based on this average, the class-capacities of schools in the simulation are sampled with triangular distribution.	$X \sim Tri(\mu/3, \mu, 5\mu/3)$ , with $\mu = \frac{pupils}{schools}$	counts gathered from a publication of the Austrian National Statistics Institute [11]. Bounds for triangular distributed were estimated.
<i>workplace sizes</i>	discrete distribution of workplace sizes	see source	gathered from a survey [12] by the Austrian National Statistics Institute
<i>workplace branches</i>	Industrial branch parameter of the <i>workplace</i> . We distinguish 21 branches according to the top level code of the ÖNACE 2008 norm. Two additional branches, care-home workers (Q.86) and teachers (P.85.2-4), were added for obvious reasons	see source	Austrian National Statistics Institute (see [7] for federalstate data, data on district level behind pay-wall)
<i>care-home units</i>	The actual number of care-homes w staff and residents were gathered to calculate the number of care-home units given a maximum capacity of 2023 residents	see source	counts gathered from freely accessible tables from the Austrian National Statistics Institute [8]

Table 5: List of contact specific parameters (2/2, status March 22, 2023). Note that all parameter values are specified for the standard model without policies.

parameter	description	value	source
<i>regional sampling of leisure time contacts</i>	leisure time contact partners are sampled based on origin-destination matrices on municipality level	Average fraction of all stays of persons from municipality X within municipality Y for all municipalities X and Y of Austria	gathered from mobile phone data evaluated for January 2020
<i>regional distribution of schools, workplaces and care-homes</i>	schools, workplaces and care-homes are created based on known information about workers, teachers and care-home employees per district. The actual coordinate in the district is sampled using the sampling algorithm of the underlying population model		see Table 3 and <i>workplace branches</i> in Table 4
<i>regional assignment of schools, workplaces and care-homes</i>	inhabitants of schools, workplaces and care-homes are assigned based on origin-destination matrices on district level	Average fraction of all stays of persons from district X within district Y for all district X and Y of Austria	gathered from mobile phone data evaluated for January 2020

Table 6: List of virus/disease specific parameters (probabilities, status March 22, 2023).

parameter	description	value	source
$\beta_{str}$	Strain dependent factor to determine the probability that a contact between a susceptible and an infected agent leads to a transmission.	base 0.055 Alpha 0.077 Delta 0.1386 BA.1 0.16632 BA.2 0.24948 >BA.2 0.24948	Value for base variant is calibrated (see Section 1.3.4). Values for other variants are taken from literature ([23], [6]) and fine tuned with additional models [16].
$\beta_{loc}$	Strain dependent factor to determine the probability that a contact between a susceptible and an infected agent leads to a transmission. Is changed by policy events.	Base setting: Same for all regions, 1 in all locations except households, 5 in households	The value for households was adjusted to fit the measured household attack rate.
$\beta_{cl}$	Climate dependent factor to determine the probability that a contact between a susceptible and an infected agent leads to a transmission.	see source	Concentration rate value calculated according to [22] for Austrian Weather data (ZAMG, <a href="https://www.zamg.ac.at/cms/de/aktuell">https://www.zamg.ac.at/cms/de/aktuell</a> ).
$\beta_{dyn}$	Models the virus load in an infectious agent as a severity-dependent function of time, that scales the infection probability in case of a contact	see Figure 2	merged information about the shedding duration from [21] and qualitative information about the shape of the curve from [30]
$\beta_{ex}$	Exogenous factor to determine the probability that a contact between a susceptible and an infected agent leads to a transmission. Changed by events for calibration purposes.	default 1	free parameter for calibration
<i>detection probability</i>	probability of an infected person to get detected by a test. Changed in model timeline.	initial values (spring 2020): [ 3, 9, 22, 20, 24, 28, 20, 21, 33, 58 ]% for 10 year age-classes	Regularly updated with the methods in [40] using screening- and sero-prevalence studies and, later, by comparison with wastewater analysis data.

Table 7: Immunisation and immunity-waning probabilities and distributions (status March 22, 2023).  
 probability that event leads to immunity. Note that effectiveness is stated for the “ $i$ -th  
 vaccination dose” and not “after  $i$  vaccinations”.

cause / target	others	Alpha	Delta	Omicron BA.1	Omicron BA.2	Omicron BA.5
others	1.00 <sup>(1)</sup>	1.00 <sup>(1)</sup>	1.00 <sup>(2)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>
Alpha	1.00 <sup>(1)</sup>	1.00 <sup>(1)</sup>	1.00 <sup>(2)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>
Delta	1.00 <sup>(1)</sup>	1.00 <sup>(1)</sup>	1.00 <sup>(2)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>
Omicron BA.1	1.00 <sup>(1)</sup>	1.00 <sup>(1)</sup>	1.00 <sup>(2)</sup>	1.00 <sup>(4)</sup>	1.00 <sup>(5)</sup>	1.00 <sup>(3)</sup>
Omicron BA.2	1.00 <sup>(1)</sup>	1.00 <sup>(1)</sup>	1.00 <sup>(2)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(4)</sup>	1.00 <sup>(3)</sup>
Omicron BA.5	1.00 <sup>(1)</sup>	1.00 <sup>(1)</sup>	1.00 <sup>(2)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(3)</sup>	1.00 <sup>(4)</sup>
1st dose	0.60 <sup>(1)</sup>	0.60 <sup>(1)</sup>	0.60 <sup>(2)</sup>	0.36 <sup>(6)</sup>	0.25 <sup>(7)</sup>	0.25 <sup>(7)</sup>
2nd dose	0.73 <sup>(1)</sup>	0.73 <sup>(1)</sup>	0.73 <sup>(2)</sup>	0.35 <sup>(6)</sup>	0.22 <sup>(7)</sup>	0.22 <sup>(7)</sup>
3rd dose	0.89 <sup>(1)</sup>	0.89 <sup>(1)</sup>	0.89 <sup>(8)</sup>	0.72 <sup>(6)</sup>	0.47 <sup>(7)</sup>	0.47 <sup>(7)</sup>
4th dose	0.89 <sup>(9)</sup>	0.89 <sup>(9)</sup>	0.89 <sup>(9)</sup>	0.72 <sup>(9)</sup>	0.47 <sup>(7)</sup>	0.47 <sup>(7)</sup>

distribution of immunity duration in days (values stand for the scale parameter of a Weibull  
 distribution with shape 1.5)

cause / target	others	Alpha	Delta	Omicron BA.1	Omicron BA.2	Omicron BA.5
others	819 <sup>(1)</sup>	819 <sup>(1)</sup>	819 <sup>(2)</sup>	111 <sup>(3)</sup>	111 <sup>(3)</sup>	111 <sup>(3)</sup>
Alpha	819 <sup>(1)</sup>	819 <sup>(1)</sup>	819 <sup>(2)</sup>	111 <sup>(3)</sup>	111 <sup>(3)</sup>	111 <sup>(3)</sup>
Delta	819 <sup>(1)</sup>	819 <sup>(1)</sup>	819 <sup>(2)</sup>	111 <sup>(3)</sup>	111 <sup>(3)</sup>	111 <sup>(3)</sup>
Omicron BA.1	819 <sup>(1)</sup>	819 <sup>(1)</sup>	819 <sup>(2)</sup>	400 <sup>(4)</sup>	111 <sup>(5)</sup>	111 <sup>(3)</sup>
Omicron BA.2	819 <sup>(1)</sup>	819 <sup>(1)</sup>	819 <sup>(2)</sup>	400 <sup>(10)</sup>	400 <sup>(4)</sup>	111 <sup>(3)</sup>
Omicron BA.5	819 <sup>(1)</sup>	819 <sup>(1)</sup>	819 <sup>(2)</sup>	400 <sup>(10)</sup>	400 <sup>(10)</sup>	400 <sup>(4)</sup>
1st dose	292 <sup>(1)</sup>	292 <sup>(1)</sup>	292 <sup>(2)</sup>	57 <sup>(6)</sup>	57 <sup>(7)</sup>	57 <sup>(7)</sup>
2nd dose	224 <sup>(1)</sup>	224 <sup>(1)</sup>	224 <sup>(2)</sup>	186 <sup>(6)</sup>	186 <sup>(7)</sup>	186 <sup>(7)</sup>
3rd dose	220 <sup>(1)</sup>	220 <sup>(1)</sup>	220 <sup>(8)</sup>	149 <sup>(6)</sup>	149 <sup>(7)</sup>	149 <sup>(7)</sup>
4th dose	220 <sup>(9)</sup>	220 <sup>(9)</sup>	220 <sup>(9)</sup>	149 <sup>(9)</sup>	149 <sup>(7)</sup>	149 <sup>(7)</sup>

sources

- (1) Assumed equal effectiveness as against Delta  
 (2) Effectiveness calculated from all EMS-registered cases/reinfections between Oct 1st to  
 Nov 1st,2021  
 (3) Assuming equal cross immunity between all Omicron subtypes (original data for BA.1  
 to BA.2)  
 (4) Calibrated using an external immunity wanig model (see <sup>2</sup>) respectively.  
 (5) Effectiveness of recovery calculated from registered cases/reinfections between Feb 10  
 and Feb 20,2022  
 (6) Tseng, Hung Fu, Bradley K Ackerson, Yi Luo, Lina S Sy, Carla Talarico, Yun Tian,  
 Katia Bruxvoort, et al.2022. "Effectiveness of MRNA-1273 against SARS-CoV-2 Omicron  
 and Delta Variants."MedRxiv  
 (7) Vaccines estimated 30 percent less effective against BA.2 and BA.4/5 compared to BA.1  
 (8) SARS-CoV-2 variants of concern and variants under investigation in England, Technical  
 briefing 31, UK Health Security Agency, 202112-10  
 (9) Estimated to be equally effective as 3rd dose  
 (10) Assumed higher protection against earlier variants.

Table 8: List of virus/disease specific parameters (durations (in days), status ).

parameter	description	value	source
<i>reaction duration</i>	time between symptom on-set and testing of the agent which furthermore leads to its confirmation and home isolation. Depends on date (correlates with test availability).	2020/02-2020/05: Weib(1.33, 5.67) 2020/06-2021/08: Weib(1.30, 2.90) 2021/09-2021/10: Weib(1.43, 2.59) from 2021/11: Weib(1.53, 1.99)	processed from officially reported data (Epidemiologisches Meldesystem [24])
<i>incubation time</i>	time between infection and symptom on-set. Depends on the virus variant.	base Weib(2.06, 6.10) Alpha Weib(2.06, 5.98) Delta Weib(2.06, 5.27) BA.1 Weib(2.06, 4.09) BA.2 Weib(2.06, 4.09) BA.5 Weib(2.06, 4.09)	Base Weibull distribution fitted to [33]. Scale of other variants adjusted to [42].
<i>pre-symptomatic time</i>	time between start of infectiousness period and symptom onset. Equivalent with the difference between incubation and latency time.	Weib(3.77, 2.13)	Weibull distribution fitted to data from [13].
<i>recovery time unconfirmed</i>	time between end of latency duration and recovery for unconfirmed persons (usually asymptomatic). Depends on the virus variant.	base Weib(1.51, 8.04) Alpha Weib(1.51, 8.04) Delta Weib(1.51, 8.04) BA.1 Weib(1.51, 4.82) BA.2 Weib(1.51, 4.82) BA.5 Weib(1.51, 4.82)	based on the fitted distribution for [34] in the systematic review [20]. Reduced to 60% for Omicron variants.
<i>recovery time confirmed</i>	time between symptom onset and recovery for confirmed persons (mostly mild symptomatic).	base Weib(1.51, 14.86) Alpha Weib(1.51, 14.86) Delta Weib(1.51, 14.86) BA.1 Weib(1.51, 8.92) BA.2 Weib(1.51, 8.92) BA.5 Weib(1.51, 8.92)	based on the mean value (13.4) of the collected papers in [20]. Assumed same shape as undetected duration. Estimated to be reduced to 60% for Omicron variants based on various studies.

Table 9: List of parameter specific for vaccinations, imports and screenings. Since parameters change rapidly in time we cannot state their values.

parameter	description	source
<i>vaccinations per day</i>	Number, type and target group of daily vaccinated person-agents	directly taken from our interface to the Austrian vaccination data (E-Impfpass, <a href="https://www.elga.gv.at/e-impfpass/e-impfpass/">https://www.elga.gv.at/e-impfpass/e-impfpass/</a> )
<i>screenings per day</i>	Number and age-class of daily screened person-agents.	Estimated from Austrian reports on issued tests per federalstate. The highest value is around 100 000 tests in Vienna in 2021 per day. Updated in timeline (see Table 10).
<i>imports per day</i>	Number of daily imported cases per federalstate.	Assumed a linear relation between number of tourist overnight stays ([9]) and imported cases. The proportionality factor was calibrated (see Section 1.3.4). Automatically updated every month, but can be changed manually with an event as well (see Table 10)
<i>imported variants</i>	Split of imported virus strains.	Calibrated according to [32]. Until Sep. 2022 we consider the variants Alpha, Delta, Omicron BA.1, BA.2 and BA.5. Parameter updated in timeline (see Table 10).

Table 10: List of event-timeline elements that can pose for the (main-version of the) model’s input including their effect and, if available, options for the event parametrisation.

event	description	parameters
<i>leisure-time contact number reduction event</i>	Based on an age-class (child, adult, retired) and/or region (municipality, districts, federalstates) dependent probability, an agent may “reject” a leisure-time contact with a different agent. As the rejection happens symmetrically, the probabilities multiply.	affected region; age-class-dependent fraction by which daily leisure-time contacts are reduced
<i>hygiene event</i>	Depicts changes in the hygiene awareness or hygiene policies by changing $\beta_{loc}$ . E.g. distancing or face mask wearing.	value per <i>location</i>
<i>change symptomatic test system event</i>	Changes the detection rate and/or the duration between symptom onset and test.	percentage for detection for 10-year age classes; new scale parameter for the Weibull distribution for the reaction time (see also Table 8)
<i>change screening test system event</i>	Changes the number and target groups for the daily screening tests.	Number of screened persons per day and per federalstate for each target group.
<i>change import model event</i>	Changes the number of daily imported cases and/or their virus strain distribution.	Number of imported cases per federalstate; List of strains to draw from
<i>change vaccination model event</i>	Changes the file-source for the imported vaccination doses. Can be valuable for vaccination scenarios.	path to new file.
<i>location closing/opening event</i>	Fraction of locations of a certain type are closed/opened in this policy.	affected location type; fraction of locations of this type that remain open / are opened
<i>start/end tracing event</i>	Starts/ends with location or contact tracing measures. For location tracing all members of a newly <i>confirmed</i> agent’s location are put under preventive isolation for a certain period of time. For contact tracing, recorded contacts of a newly <i>confirmed</i> agent are put under preventive isolation.	affected location type; length of preventive quarantine length; contact tracing compliance
<i>vaccination round event</i>	Distributes a number of given vaccine doses of a certain type to capable model agents in <b>addition</b> to the daily standard vaccinations	number of doses; type of vaccine (e.g. first, second,... dose); target groups; time delay
<i>change event</i> $\beta_{ex}$	Changes $\beta_{ex}$ . Usually only used by calibration routines.	New value of $\beta_{ex}$ per federalstate.