Appendix S1 – ODD Model Description

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We developed a spatially-explicit, stochastic agent-based model (ABM) to simulate airborne and direct droplet-mediated respiratory pathogen transmission in indoor settings. This model was created and executed using the open-source modeling software, NetLogo (Ver. 6. 1. 1 – Willensky 1999). Below, we provide a detailed description of our model in accordance with ODD (Overview, Design concepts, Details) standards outlined by Grimm et al*.* (2020).

1. *Purpose and patterns*

The purpose of this model is to quantify the effect of increasing group density on the probability of respiratory pathogen transmission from infectious individuals to susceptible ones, given varied spatial dimension, vaccination, and risk-reduction behavior (e.g., mask use, social distancing, etc.) levels in indoor settings. The ability of our model to accurately simulate infection events is predicated on its ability to recreate four processes involved in transmission: 1.) Susceptible individuals become infected through inhalation of virions contained within infectious droplets of varying sizes. 2.) Infectious agents expel infectious droplets of varying sizes, and droplets' movement, fallout, and virion-carriage rates vary with droplet size. 3.) Symptomatic infectious agents are likely to infect more susceptible individuals than asymptomatic ones, as coughing expels infectious droplets farther than does breathing or speaking alone (Kwon et al. 2012). 4.) Susceptible individuals' probability of infection can be lessened if individuals employ extra measures to avoid transmission (e.g., wearing face masks).

2. *Entities, state variables, and scales*

There are two mobile agents (i.e., NetLogo agents capable of movement) in our model: *People* and *AirArrows*. *People* in our model represent people congregating in fixed space (e.g., students in a classroom, people watching a movie in a theatre, etc.), while *AirArrows* control the direction of simulated airflow in the space when ventilation-induced airflow is being simulated. Patches (i.e., grid cells in the NetLogo model interface) in our model represent 1×1 m² areas. When a simulation begins, spawned people can be susceptible, infectious and symptomatic (i.e., these agents represent individuals who spread the pathogen via coughing, sneezing, etc.), or infectious and asymptomatic (i.e., these agents represent individuals who spread the infection through breathing or speaking alone). Over the course of any simulation, susceptible agents may become infected with a pathogen following exposure to infectious droplets expelled from symptomatic and/or asymptomatic agents. The global environment dictates the size of the fixed space, total number of agents in the model, number of these agents that are infectious, as well as the dynamics and probabilities of infection events and airborne droplet movement within the model. Global, agent, and patch variables are described in Table S1-1, where we also make the distinction between parameters (i.e., static variables that are unchanging within simulations) and dynamic variables that may vary within simulations. The spatial extent of our model can range

from 1 to ∞ m², and is controlled by the *grid_height* and *grid_length* parameters. Each tick (i.e., one-unit time step) in our model represents a one-minute progression.

3. *Process overview and scheduling*

Model processes are outlined in Figure S1-1 and described in detail herein. Upon initialization, a world with $grid$ height $*$ grid length patches is generated. Following world creation, *n* susceptible people spawn within patches. People spawn one at a time and, if the *social-distance* parameter is > 0 m, they appear at a random location at least *social-distance* from any other person. If there is no available space ≥ *social-distance* from any person, newly spawned people will be placed at a random location as far away from others as possible. If *social-distance* equals 0 m, people spawn in completely random locations. All people are asked to set their heading (i.e., direction they are facing) to a random direction between 0° – 360° if *face-northward* is FALSE, or between 315º – 405º if *face-northward* is TRUE.

Once initial spawning is completed, *n_infectious* people are randomly selected from the pool of people to transition to the "infectious" health state. Infectious people have a *symp-pr* probability of being classified as "symptomatic" and a 1 - *symp-pr* probability of being "asymptomatic." Then, we carry out vaccination efforts. All people have a *vacc-proportion* probability to be vaccinated. Susceptible people that are vaccinated have a *vacc-immunity probability* (i.e., *vacc-proportion***vacc-immunity**100%) to receive complete immunity from infection, and will no longer be considered "susceptible." Following vaccination efforts, people have a *mod-proportion* probability to wear masks in the simulation, and the affected groups (i.e., susceptible only, infectious only, or both susceptible and infectious individuals) that may transition to wearing masks are designated by the *mod_group* parameter. Masked people are then asked to update their *exposureRisk* and *expectorateRisk* variable values. The default value for each variable is 100%, but will change to the *maskRisk-mod* parameter value.

The final aspect of simulation setup is to establish agents for simulating ventilation airflow if *ventilation* is TRUE. To create a supply and return vent(s), *numSupplyVents* and *numReturnVents* patches on *ventilSupplyWall* and *ventilReturnWall* world borders are designated as supply and return vents, respectively. All non-return-vent patches are asked to spawn a single airArrow. All airArrows are asked to set their heading towards the closest return vent patch. AirArrow headings will be used to direct ventilation airflow. This concludes the simulation setup procedure.

Following setup, the simulation begins in earnest. If *ventilation* is TRUE, the first task to take place each tick is to move droplets towards return vents. We assume very simple ventilationinduced air movement within an enclosed room where air moves only towards the return vent(s), and return vent patches transfer a proportion of droplets present there to supply vent patches while also removing some droplets from the simulation. To achieve this, we ask all patches to count the number of droplets in each size class (Figure S1-2) that will be transferred to the next patch (i.e., $num_{dp} * ventil_movementRate$, where num_{dp} is the number of droplets of a given size class d in patch p). Then we simultaneously ask non-return vent patches to transfer these droplets to patches 1-patch ahead of airArrows, and ask return vent patches to transfer

$$
num_{dp} * ventil_movementRate * (1 - ventil_removalRate)
$$

droplets to supply vents, and to remove

∗ *ventil_movementRate* ∗ *ventil_removalRate*

from the simulation.

Non-ventilation related droplet removal is the second action to occur every tick. This action represents droplet/virion removal from the local environment due to inhalation by individuals, gravitational settling, and general droplet decay. For each droplet size class, we ask all patches to remove

$$
\left\{ \left(\frac{vol_{B}}{1 m * 1 m * expected rate Height} \right) * People_{p} \right) + \left(\frac{Vt_diam_{d}}{expected height} \right) + dropletDecay) \right\} * num_{d_{p}}
$$

droplets, where $\frac{vol_B}{1 \text{ m} * 1 \text{ m} * expected rate height}$ is the proportion of air within the patch inhaled by a single person each minute, $People_n$ is the number of people on patch p, Vt_diam_d is the calculated terminal velocity (i.e., the maximum free-falling speed in m/min, assuming the force of gravity acting on an object is 9.8 m/s²) of droplets in a size class d (Figure S1-3), and num_{d_p} is the number of droplets of a given size class d in patch p. If patches would remove > 100 % of any size class, we ask them to instead set that num_{d_p} to zero. Thus, we ensure that no patch can ever have a negative number of droplets.

Next, we ask patches to diffuse droplets of all size classes to neighbors (i.e., all patches touching them) at rate *diffusionRate*. We once again ask all patches to count the number of droplets in each size class that will be transferred to the neighbors (i.e., ∗ *diffusionRate*). Then, we ask patches to evenly distribute these droplets to neighbors.

Infectious people, $i \in I$, expectorate droplets and exposed susceptible individuals (i.e., noninfectious individuals without vaccine-associated immunity), $s \in S$, may become infected. Each tick, every symptomatic person has a *cough_frequency* probability to cough (i.e., expel droplets relatively far out from themselves), and $(1 - \text{cough_frequency})$ probability to expectorate in accordance with the "non-coughing" schema (i.e., expel droplets relatively close to themselves). Asymptomatic people have no chance to cough, and will expectorate in accordance with the "non-coughing" schema with 100% probability. While droplet spread distance and angle for this schema can be modulated via model input values to reflect numerous activities (e.g., speaking, breathing, etc.), the droplet size distribution is assumed to reflect that of speaking events (Figure S1-2). Thus, parameters referring to aspects of the "non-coughing" schema are coded as "speak" parameters (e.g., *speak_airflow_angle*).

During expulsion events, droplets spread to patches in front of coughing and speaking infectious people in cones with semi-vertex angles of *cough_airflow_angle* and *speak_airflow_angle*, respectively, and lengths randomly drawn from lognormal distributions. Lognormal distributions were obtained by exponentiating Poisson distributions with known

means and standard deviations, in accordance with methods described by Railsback & Grimm (2011). In our model, lognormal distributions to inform droplet travel distances from coughing and speaking people are generated from known mean and standard deviation pairs: *cough_spread_dist.mean*, *cough_spread_dist.sd*, and *speak_spread_dist.mean*, *speak_spread_dist.sd*, respectively. If infectious people are wearing masks, only the patch they are in is contaminated (i.e., cones of expectoration in these cases have lengths of 0).

The number of droplets that infectious people expel at time t , $dropletNum_{it}$, is determined by sampling from another lognormal distribution with known means of *speak_dropletNum.mean* or *cough_dropletNum.mean*, and standard deviations of *speak_dropletNum.sd* or *cough_dropletNum.sd,* depending on if people are speaking or coughing, then multiplying this samples value by *expectorateRisk_i*. If infectious people are vaccinated (i.e., their *vaccinated?* attribute is set to "TRUE"), the number of droplets expelled is equal to $dropletNum_{it} * vacc$ *virionRiskReduction*). Thus, we allow vaccinated people to be relatively less infectious than their unvaccinated counterparts if users so choose. We assume that people expectorate droplets of 16 size classes, with increasingly large mean diameters. We accept the size class frequency distributions for speaking and coughing events given by Chao et al. (2009) and shown in Figure S1-2, and enforce these distributions in our model. We assume that all droplets are evenly distributed between and within contaminated patches.

After infectious individuals expectorate, we assess if any susceptible individuals will transition to the infected status. The number of virions (i.e., live pathogen capable of causing infection in susceptible individuals) in a patch, $virions_p$, is given by the equation

$$
virions_p = \sum_{d=1}^p (virionsPerML*Vol_d*num_d_p),
$$

where Vol_d is the mean volume (in mL) of droplets in each size class, calculated using the equations presented by Anchordoqui & Chudnovsky (2020). The probability that a susceptible person on patch *p* is infected at any given time is

$$
pr(infection)_{s} = \text{virions}_{p} * \text{virionRisk} * \frac{\text{vol}_{B}}{1 \text{ m} * 1 \text{ m} * \text{expectorateHeight}} * \text{exposureRisk}_{s}
$$

where, *virions* $_p$ is the number of virions in the patch containing the individual.

If *numCohorts* = 1, the simulation ends after *cohort_dur* ticks have elapsed. If *numCohorts* > 1, the simulation will last for numCohorts * cohort_dur ticks. In this case, every cohort_dur ticks, if *rearrange-cohort* is TRUE, all people will move to randomly-selected patches while still adhering to *social-distance* and *personPerPatch-*cap rules. People will set a new heading in accordance with *face-northward*. If *rearrange-cohort* is FALSE, all people are killed, and an equal number of people will spawn while adhering to *social-distance* and *personPerPatch-*cap rules. All people in the new cohort will be susceptible to infection (i.e., infectious people only exist in the first cohort).

4. *Design concepts*

Infection in our model is driven by inhalation of virions contained in droplets of varying sizes. Fomite-driven transmission, by design, is outside the scope of our model. Regarding aerosol transmission, for simplicity, we assume that droplets fall from *expectorateHeight* m at terminal velocity and our droplet-size distribution represents post-evaporation sizes. These assumptions are reasonable given the rapid speed at which droplets evaporate and reach terminal velocity (Noakes *et al.* 2006; Xie *et al.* 2007; Anchordoqui & Chudnovsky 2020), and allow us to discount local humidity, temperature, and micro-scale airflow effects on the spatial distribution of droplets within the model. Droplet size class terminal velocity is calculated using the equations presented by Anchordoqui & Chudnovsky (2020), and droplet sizes incapable of settling on the ground from *expectorateHeight* m within one tick (i.e., one minute) are allowed to move between patches via ventilation- and diffusion-induced airflow. As the number of virions within a patch is dependent on the number of droplets in each size class, spatial infection-risk heterogeneity is therefore a function of global airflow parameters and the placement of infectious people throughout the simulated world. For simplicity, we assume that mechanism of dropletmediated pathogen transmission is the same (i.e., inhalation) for droplets of all size classes. We do realize, however, that in reality larger droplets are relatively less-likely to be inhaled and instead mediate transmission through contact with unprotected mucus membranes (Milton 2020).

We assume that the volume of air in each patch at any given time is *expectorateHeight* m³ (i.e., 1 m $*$ 1 m $*$ *expectorateHeight*) and that droplets are evenly distributed within patches. Thus, the per-capita number of virions that people inhale each tick is equal to

> $virions_p *$ vol_B 1 m * 1 m * expectorateHeight .

Wearing a mask to reduce successful pathogen transmission in our model modulates the number of droplets expelled by infectious people and the proportion of virions inhaled by susceptible individuals. Previous research has quantified the extent to which using personal protective equipment may reduce risk of infection with a respiratory pathogen (Jefferson et al. 2008), and recent work has shown that masks reduce the number of aerosols expelled by wearers (Asadi *et al.* 2020). Therefore, we chose to use masks to modify the individual-level probability that susceptible individuals will become infected given exposure to infectious droplets in their patch (i.e., *exposureRisk*), and the number of droplets individuals will expectorate on any given tick (i.e., *expectorateRisk*). In our model, mask use scales both of these variables equally. We acknowledge that making these scaling factors equivalent may be unrealistic however, and intend to make this a focus of future model improvement if and when more detailed information on mask-induced effects on pathogen transmission become available.

For simplicity, we assume that vaccinations do not confer partial immunity. Susceptible people that get vaccinated either transition to the "immune" state or remain completely susceptible, with no additional protective benefits. However, we do allow users to specify reductions in infectiousness associated with vaccination efforts if they so choose.

As previously noted, in our model we characterize infectious people as asymptomatic or symptomatic. "Symptomatic" here refers to agents representing individuals that present any respiratory-disease symptoms (e.g., coughing, sneezing, etc.). We parameterize droplet behavior for symptomatic and asymptomatic collectives separately because we expect them to drive infections through different means. For example, asymptomatic individuals will likely spread infectious droplets by simply breathing near or talking to susceptible people. Symptomatic individuals, on the other hand, may also frequently spread droplets through coughing, sneezing, or similar events. Airflow angle and velocity associated with these means of infection are substantially different (Kwon *et al.* 2012) and as such, necessitate separate parameters if both symptomatic and asymptomatic agents can exist in simulations simultaneously.

Agents in our model have extremely limited movement (i.e., unless *rearrange-cohort* is TRUE and *num-cohorts* > 1, people will be completely unmoving), but people are spawned relatively far away from one another if the *social distance* parameter is > 0 . As such, our model is best used for estimating transmission risk associated with scenarios where individuals are generally unmoving (e.g., students in a classroom). Scenarios like students watching a presentation at the front the room or patrons attending a show in a theatre can be further emulated if users so choose by setting face-northward to TRUE. Users may also simulate wellmixed population interactions by setting *rearrange-cohort* to TRUE and *num-cohorts* > 1. Activity-specific movements may modulate infection risk (e.g., doctors must get close to patients in order to physically examine them), but are outside the scope of our model.

This is a simple model with little adaptive agent behavior, 6 collectives for people agents (i.e., "susceptible," "immune", "infectious: asymptomatic & vaccinated", and "infectious: symptomatic & vaccinated", "infectious: asymptomatic & unvaccinated", and "infectious: symptomatic & unvaccinated), and only one action that can be considered to be a direct interaction between agents. That is, when a simulation begins or cohorts are rearranged/replaced, newly-spawned people learn where previously-spawned ones exist and attempt to ensure that sufficient space exists between themselves and others in accordance with the *social_distance* parameter value. Their objective is to maintain effective social distances to minimize infection risk. Accordingly, model outputs (e.g., the number of susceptible people infected, time to first infection, and average inter-agent distance) are influenced by emergent patterns triggered by this behavior. No other examples of adaptive behavior, sensing, prediction, or learning, as defined by Grimm et al*.* (2020), exist.

Stochasticity is introduced to the model in six ways during simulation initialization, then is further incorporated in four actions that take place during each subsequent time step. At initialization stochasticity is introduced when: 1.) people decide their initial placement, 2.) subsets of people are randomly designated as infectious, 3.) *vacc-proportion* ∗ 100% of people become vaccinated, 4.) *vacc-immunity* ∗ 100% of vaccinated susceptible people transition to "immune," 5.) *mod-proportion* ∗ 100% of people's *exposureRisk* and *expectorateRisk* values are changed from 1 to *maskRisk-mod*, and 6.) if *ventilation* is TRUE but *equallySpaceVents* is FALSE, return and supply vent locations will be randomly decided (though these locations will still be confined to appropriate walls of the world). During each time step, stochasticity plays a role in: 7.) determining if infectious agents expel droplets, 8.) drawing droplet travel distances from lognormal distributions, 9.) drawing the number of droplets produced in expectoration events from lognormal distributions, and 10.) assessing whether exposed susceptible agents transition to "infected" status. We incorporated stochasticity into these processes to introduce plausible variation into simulations.

The key outputs of this model are: 1.) the number of successful infections (i.e., susceptible agents' health statuses changed from "healthy" to "infected") each tick, and 2.) the time of the first successful infection in the simulation. In addition to the primary outputs, our model also keeps track of the average distance (in m) between individuals, and all "infected" people record the number of droplets of each size class contained within their patch at the time of infection. This allows us to not only assess parameter effects on transmission rates, but also estimate the proportion of people infected by aerosols.

5. *Initialization*

All global parameters aside from those controlling transmission mechanics (e.g., airflow angles, mean and standard deviation travel distances, number of virions in droplets, etc.) or airflow rates influence model initialization (i.e., how many agents and patches are created, where they spawn, and what their initial state-variable values are). Model actions associated with initialization are outlined in Section 3. Herein we discuss the rationale in allowing the aforementioned parameters to vary between simulations.

The primary purpose of this model is to assess the effect of population density on transmission risk. Population density in our model, expressed in terms of people/ $m²$, is given by the equation

\boldsymbol{n} _ℎℎ∗_ℎ .

We allow the size of our modeled world to vary, in addition to *n*, as there may be an interaction between world size and *social_distance* levels that may ultimately cause the observed number of infections to vary. Afterall, the maximum distance that agents can spread out from one another is limited by the space available to them.

We tried to make the model flexible enough to test multiple hypotheses about implementing risk-reducing strategies (e.g., social distancing, mask use, etc.). This is the primary impetus adding the *social_distance*, *maskRisk-mod*, and *mod-proportion* parameters. Similarly, the *vol_B* parameter exists so that we can assess how infection risk changes in response to different group activities people may be participating in that are associated with different breathing rates (e.g., choir practice vs. attending a lecture), the *virionsPerML* and *virionRisk* parameters exist to ensure that our model can be used to simulate transmission of different pathogens for which these values are known or can be estimated.

6. *Input data*

No model processes are driven by external data. No external data are imported into the model.

7. *Sub-models*

All sub-models are comprehensively described in sections 3-5 and outlined in Figure S1-1.

8. *References*

- 1. Anchordoqui LA, Chudnovsky EM. A physicist view of COVID-19 airborne infection through convective airflow in indoor spaces. SciMed J. 2020;2:68-72. doi: 10.28991/SciMedJ-2020-02-SI-5.
- 2. Asadi S, Cappa CD, Barreda S, Wexler AS, Bouvier NM, Ristenpart WD. Efficacy of masks and face coverings in controlling outward aerosol particle emission from expiratory activities. Sci Rep. 2020;10:15665. doi: 10.1038/s41598-020-72798-7.
- 3. Chao CYH, Wan MP, Morawska L, Johnson GR, Ritovski ZD, Hargreaves M, et al. Characterization of expiration air jets and droplet size distributions immediately at the mouth opening. J. Aerosol Sci. 2009;40(2):122-133. doi: 10.1016/j.jaerosci.2008.10.003.
- 4. Grimm V, Railsback SF, Vincenot CE, Berger U, Gallagher C, DeAngelis DL, et al. The ODD protocol for describing agent-based and other simulation models: a second update to improve clarity, replication, and structural realism. J Artif Soc Soc Simul. 2020;23(2):7. doi: 10.18564/jasss.4259.
- 5. Jefferson T, Foxlee R, Del Mar C, Dooley L, Ferroni E, Hewak B, et al. Physical interventions to interrupt or reduce the spread of respiratory viruses: systematic review. BMJ. 2008;2008(336):77. doi: 10.1136/bmj.39393.510347.BE.
- 6. Kwon S-B, Park J, Jang J, Cho Y, Park D-S, Kim C, et al. Study on the initial velocity distribution of exhaled air from coughing and speaking. Chemosphere. 2012;87(11):1260- 1264. doi: 10.1016/j.chemosphere.2012.01.032.
- 7. Milton DK. A Rosetta Stone for understanding infectious drops and aerosols. *Journal of the* Ped Infect Dis Soc. 2020;9(4):413-415. https://doi.org/10.1093/jpids/piaa079.
- 8. Noakes CJ, Beggs CB, Sleigh PA, Kerr KG. Modelling the transmission of airborne infections in enclosed spaces. Epidem Inf*.* 2006;134(5):1082-1091. doi: 10.1017/s0950268806005875.
- 9. Railsback SF, Grimm V. Agent-Based and Individual-Based Modeling: a Practical Introduction, 1st edition. Princeton University Press: Princeton, New Jersey, U.S.A. 2011. pp. 195-208.
- 10. Wilensky U. NetLogo. Center for Connected Learning and Computer-Based Modeling, Northwestern University, Evanston, IL. http://ccl.northwestern.edu/netlogo/. 1999 [cited 2021 Jan 21].
- 11. Xie X, Li Y, Chwang ATY, Ho PL, Seto WH. How far droplets can move in indoor environments – revisiting the Wells evaporation–falling curve. Indoor Air. 2007;17(3):211- 225. doi: 10.1111/j.1600-0668.2007.00469.x.

9*. Tables*

10. *Figures*

Figure S1-1. Simplified model overview. Bulleted sub-models are listed in the order that they take place within the model. Simulation bullet points repeat each model tick.

Figure S1-2. Distribution of droplet sizes during expectoration events. Distributions of size classes during coughing and speaking events are based on findings of Chao *et al.* (2009), and represent mean observed droplet-size measurements they recorded 60 mm away from individuals' mouths immediately following these activities.

Figure S1-3. Droplet dynamics for supply-vent and non-supply-vent patches. a.) When modeling ventilation, droplet input to and removal from patches are functions of fixed rates (solid arrows) and probabilistic expectoration from symptomatic and asymptomatic people within range (dashed arrows). b.) Equations for fixed effects on within-patch droplet dynamics. Supply patches receive input from Return-vent patches in addition to diffusion from nearby neighbors. Non-supply-vent patches do not receive input from Return vents. Droplet output is a function of ventilation airflow parameters, diffusion to neighbors, a fixed decay rate, and inhalation by people within the patch.