This manuscript was compiled on October 16, 2023

Please provide an abstract of no more than 250 words in a single paragraph. Abstracts should explain to the general reader the major contributions of the article. References in the abstract must be cited in full within the abstract itself and cited in the text.

Keyword 1 | Keyword 2 | Keyword 3 | ...

A fter the SARS pandemic in 2005, researchers noted that disease transmission was not homogeneous: some small number of infected individuals – "super spreaders" – were responsible for a disproportionately large number of total infections (?). Subsequent research noted a similar pattern in other diseases: between 15% to 20% of individuals cased 75% to 85% of infections in diseases as diverse as measles, smallpox, monkeypox, HIV and others (?). The COVID-19 pandemic follows a similar pattern: transmission is dominated by superspreading events (SSEs) in which a superspreading individual infects many people over a short time (?) (?).

To address this missing piece, we introduce the risk structured "hot-spot" susceptible-infected-recovered (hsSIR) model, which gives each individual in a population a probability of spending time in a single "hot-spot" location where disease spreads more readily. These probabilities are fixed for each individual over the course of the simulation. The distribution of these risk probabilities introduces a risk structure to the population and allows us to investigate how different distributions of risk taking affect the dynamics and outcomes of a disease outbreak. We find that, compared to the standard SIR model, a small number of initial infections is less likely to lead to a large outbreak – in agreement with SSE theory [@plos2020]. But when large outbreaks do occur they tend to infect the highest risk individuals early and are more explosive; peaking faster and often higher than the base model. We also find the decline in case numbers is faster than would otherwise be expected, as the pool of high risk individuals is exhausted early; which leads in many cases to a smaller number of total infections over the entire course of the outbreak. We corroborate and provide theoretical bases for these findings through the introduction of a branching process model and an integrodifferential model.

Agent Based hsSIR Model

We develop an agent-based model in which N individuals in a fixed population are susceptible (S), infected (I) or recovered (R) (Fig 1). Each day, each infected agent may transmit an infection to any susceptible agent with fixed probability β_C (homogeneous community spread – Fig 1Aiii). At the same time, each agent independently visits the "hot spot" with probability ρ_i ; then disease spreads from each infected individual in the hot spot to each susceptible individual there with probability β_R (hot spot spread – Fig 1Aiii). ρ_i is fixed for each individual but varies between individuals, we consider different distributions of risk across the population and different levels of β_R and β_C (Fig 1B).

Individuals recover from infection after a fixed number of days D. We start each simulation by choosing one individual at random to be infected, and continue until all agents are either susceptible or recovered.

[Introduce calculated quantities, and cite the table here.]

Results

Large Outbreaks Less Likely. If one person in a small population or community becomes infected, they have a chance of recovering before spreading the disease to anyone else; or spreading to only a small number who themselves recover before the initial infection becomes an outbreak. On the other hand, as soon as more than a very small number of secondary infections happen, a larger outbreak is all but guaranteed. This probability of a large outbreak as a result of one initial

Significance Statement

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Author affiliations: ^aQuantitative Ecology and Resource Management Program, University of Washington, Seattle, WA 98195, USA (UW); ^bSchool of Aquatic and Fishery Sciences, UW; ^cVaccine and Infectious Disease Division, Fred Hutchinson Cancer Research Center, Seattle, 98195 WA, USA; ^dDepartment of Applied Mathematics. UW

Please provide details of author contributions here.
Please declare any competing interests here.

¹A.O.(Author One) contributed equally to this work with A.T. (Author Two) (remove if not applicable).

²To whom correspondence should be addressed. E-mail: author.twoemail.com