Simulating Ecosystems with Wild Demographic Fluctuations

James Blakely, Sophie Bucker, Kanishk Kondaka, Will Sperling, Brayden Yang, Mentor: Keon Abedi

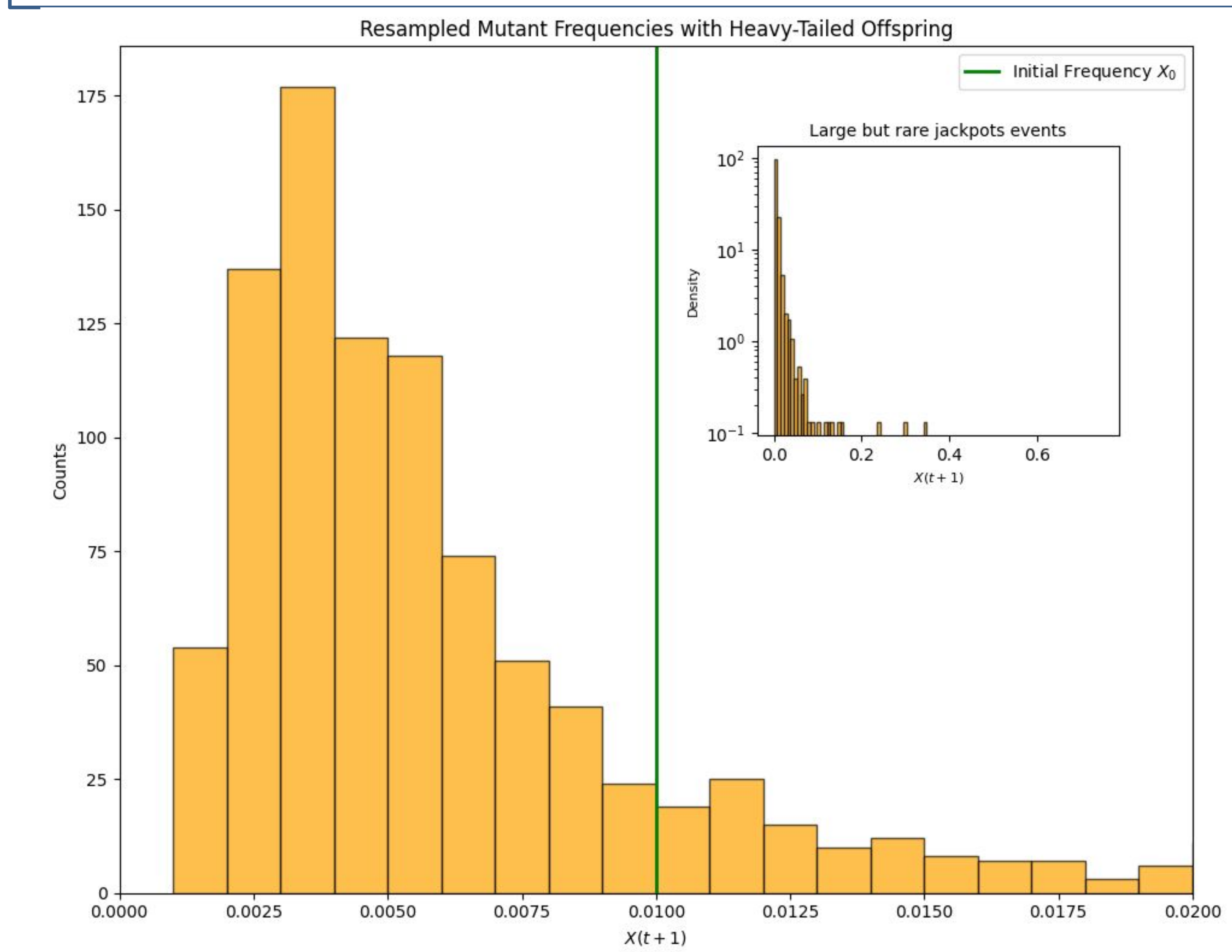
Department of Physics, University of California, Berkeley   Undergraduate Lab at Berkeley, Physics & Astronomy Division

Introduction

- Ecosystems are constantly fluctuating, resulting in organisms with highly variable offspring numbers.
- For e.g., a slow-growing cancer cell at the nutrient-rich edge of a tumor will have far more offspring than a faster-growing one in the middle. This "lucky" occurrence is called a **jackpot event**[1,2].

Do jackpot events alter ecological & epidemiological dynamics?

Spoilers: Yes! We find that ecosystem models with skewed-offspring distributions are less stable and exhibit larger (e.g. infection) spikes



Background

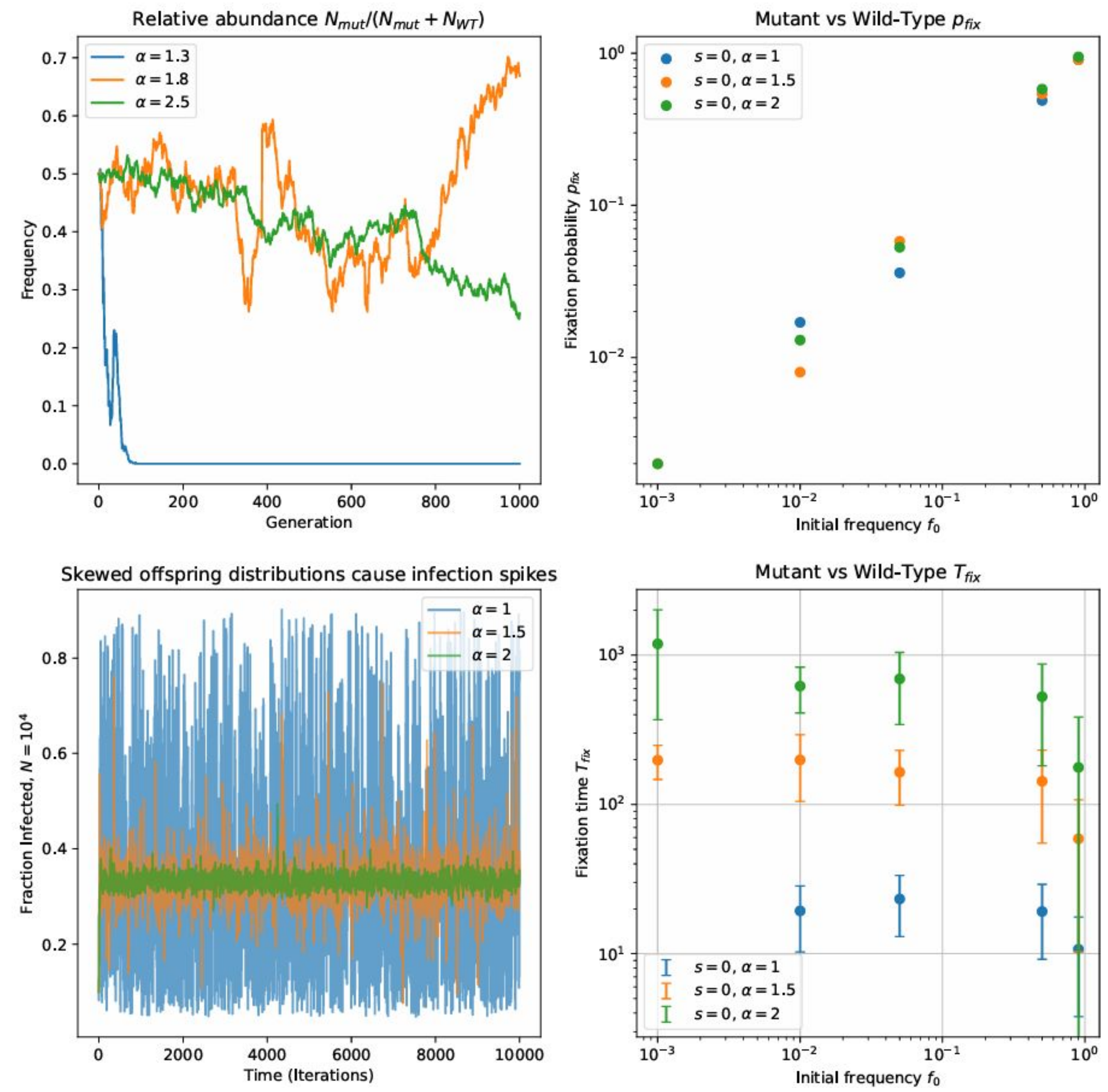
- To model offspring number fluctuations with rare jackpot events, we ran simulations with samples drawn from **heavy-tailed offspring distributions**[1].
- Using the example of microbial ecosystems, we model multiple populations undergoing jackpot events with varying frequency.
- For e.g.: we studied a **susceptible-infected model** for disease dynamics:

$$I = Binomial\left(N, \frac{\beta S_{wild} + (1-\gamma)(1-h)I_{wild}}{S_{wild} + (1-h)I_{wild}}\right), \quad S = N - I.$$

- β = (the infection rate); γ = (the recovery rate)
- S = (susceptible population); $I = N - S$ (infected population)
- h = (reduction in growth rate from infection)

We investigated how different parameters (e.g. α and β) affect how quickly an infection went extinct, the probability of a mutation to fix, and disease dynamics (trajectories) over time.

Analysis



(Left to right)
Fig 1: Relative abundance of "mutant" strain in environments with different values of alpha
Fig 2: Fixation probability (Pfix) versus initial frequency F0
Fig 3: Species % of population over generations where colors represent different values of alpha
Fig 4: Fixation times (as as a function of initial frequency) are shorter in environments with jackpot events

Methodology

Time-step procedure

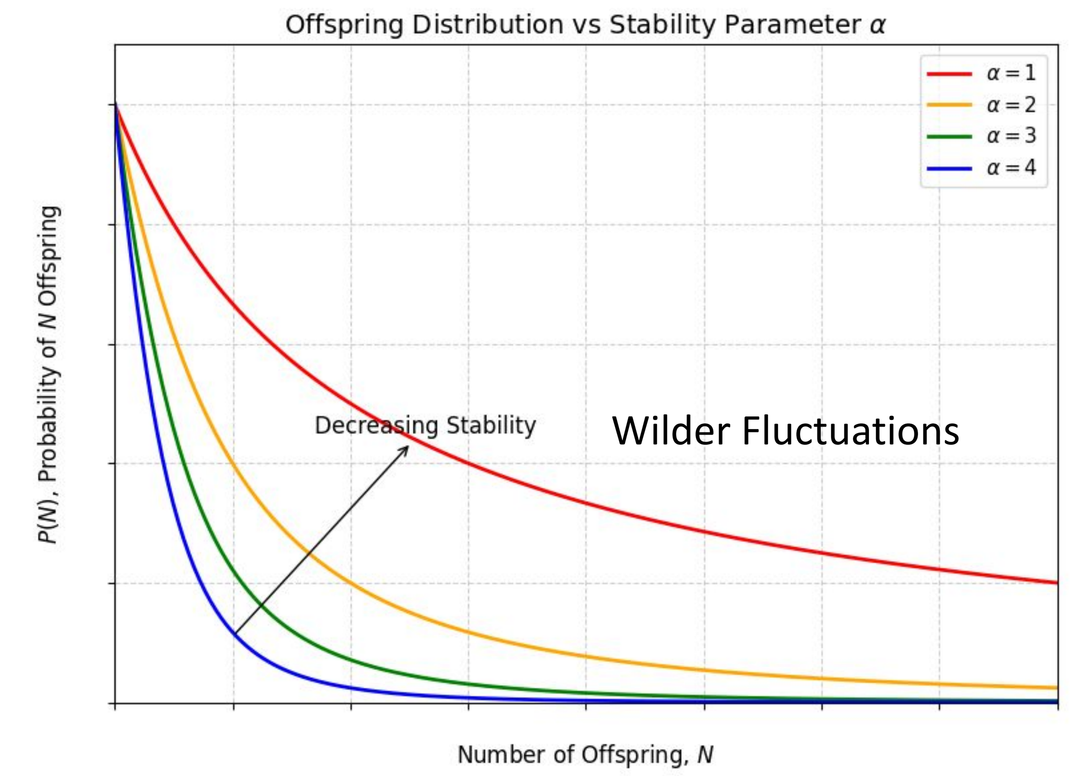
- Mutant microbes produces M offspring drawn from a heavy-tailed distribution, i.e. a probability distribution $P(n) \propto 1/n^{1+\alpha}$. Wild-types make W.
- Let the mutant have growth rate $1+s$ and the wild-type have growth rate 1.
- We binomially sample from the offspring, choosing mutant with a probability of $p = (1+s)\ M / M + W$ and wild-type with probability $1 - p$.

Types of simulations we ran

- Fixation probabilities based on varying initial population frequencies
- Dominant/recessive allele frequencies and fixation times
- Extinction rates for the infection and for minority populations
- Relative fitnesses for species with different α values
- Population dynamics fixations when modifying α, β, and γ
- Trajectories of wild-type and mutant populations over time

Conclusion

- **Wild-fluctuations makes the system less stable!**
  - Larger values of α increase chances of jackpot events in populations, causing populations to fix more frequently more rapidly.



- What we learned
  - Implementing simulations, understanding how species interact and evolve, and how stochasticity impacts these biological systems.
  - This framework can be applied to many different scenarios: from microbial interactions to macroscopic predator-prey relationships

Open Questions

- When do ecosystems behave predictably (i.e., according to natural selection) vs. stochastically (genetic drift)?
- Can we infer the strength of fluctuations acting in microbial populations by looking at genetic data (e.g. DNA sequences)?
- Do wild fluctuations help or hinder pathogen evolution?

REFERENCES

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