

Introduction: Cooperation and the Origin of Life

Cooperation forms the basis of the emergence of natural complexity. Specifically, cooperation allows individual entities to gather together and form more complex organisms, networks, or organizations. We sought to study cooperative behavior on a small-scale with the use of a lattice-based model that implements the *Iterated Prisoner's Dilemma* (IPD), while integrating – in analogy with the biological world – selection, heredity and variability.

The Prisoner's Dilemma

The Prisoner's Dilemma is a formalism that was developed in Game Theory to study cooperative behavior between two individuals. Two players have the possibility to either Cooperate or Defect – without knowing in advance what their opponent's move will be – and are subsequently assigned a score based on the following matrix:

	Cooperate	Defect
Cooperate	R 3	S 0
Defect	T 5	P 1

Table 1. Score matrix of the Prisoner's dilemma. Numbers indicate the PD's score matrix as traditionally defined [1], indicating the reward of the player adopting the strategy in the leftmost column. Mutual cooperation leads to the best possible mean outcome, while defection either leads to the absolute maximum or the absolute minimum reward. Letters refer to the general form of the score matrix for the PD: the game satisfies the PD constraint when $T > R > P > S$.

The Prisoner's Dilemma becomes even more useful in a biochemical/biological context in its *iterated* formulation: in the **Iterated Prisoner's Dilemma**, we model repeated encounters between individual entities and consider the sum total of all interactions.

Strategies

IPD players play according to *strategies* that define their moves according to the information available to them – their previous moves and their opponent's previous moves.

Examples of strategies include: **ALLC** – always cooperate, **ALLD** – always defect, **TFT** – reciprocate the opponent's last move (“Tit-for-Tat”) and RND – play randomly. However, in all simulations conducted, strategy RND was quickly and completely evacuated from the lattice.

The IPD in Biology

Previous work has shown that many interactions in microbiology, biochemistry or ecology can be modeled using the IPD. Examples include:

- a polymerase ribozyme that can be replicated by other polymerases [3]
- catalysts part of an autocatalytic set that catalyse one another's formation [3]
- the RNA phage $\Phi 6$ for which co-infection of the same host creates conflict [4]
- the yeast *Saccharomyces cerevisiae* that can either produce ATP slowly through respiration or speed up the process through additional fermentation [2]

Take away: cooperation is essential in biology, specifically when studying the Origin of Life !

Methods

We implement IPD games on a 128×128 periodic lattice where each site adopts a different strategy, but makes the wrong move with an error rate p . At each iteration of the model,

1. each strategy play against the other strategies in their Moore neighborhood (i.e. the 8 sites surrounding them)
2. strategy is replaced by highest scoring neighbour
3. the error rate is also propagated (**heritability**, *optional*)
4. apply a mutation to the error rate (**replication error**, *optional*)

This gives rise to complex population dynamics and spatial pattern formation:

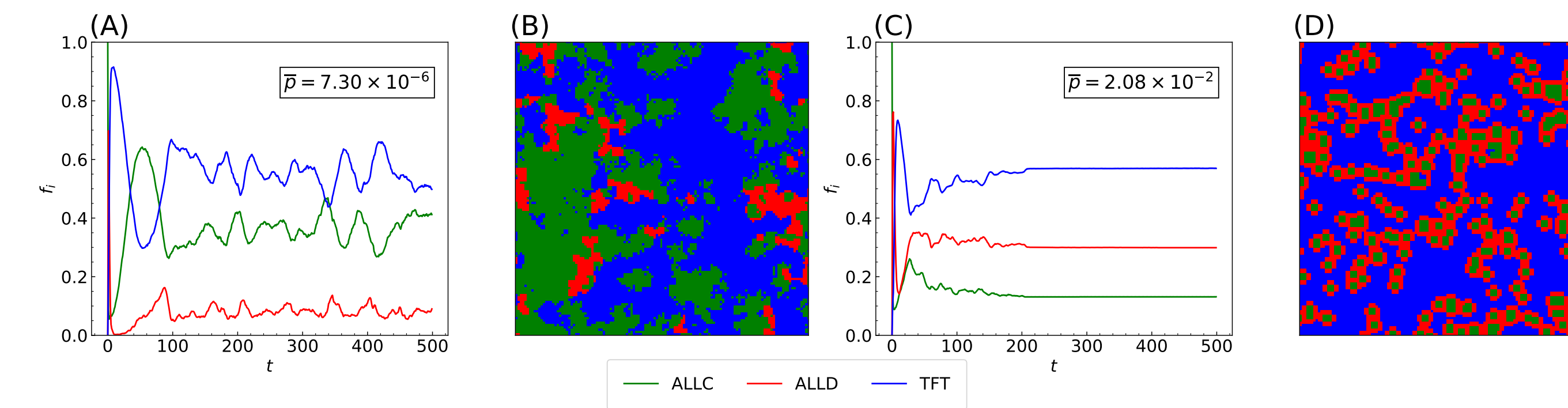


Figure 1. Population evolution and final lattice state for two simulations. Strategies **ALLC**, **ALLD** and **TFT** are initially distributed randomly on the lattice with equal relative frequency.

Results

- Assigning different values of the error rate to the agents influences the success of each strategy. For example, at very low error rates, **ALLC** and **TFT** have much more success than **ALLD**, whereas at high error rates it's the opposite (Fig. 2A). Note the log. horizontal scale!
- Including the *heredity* of error rates changes the dynamics of the system (Fig. 2B). Defectors no longer dominate at high error rates!
- Finally, when error rates are additionally subject to *mutations*, the overall picture changes totally: cooperation takes over the lattice for a wide range of initial error rate distributions (Fig. 2C).

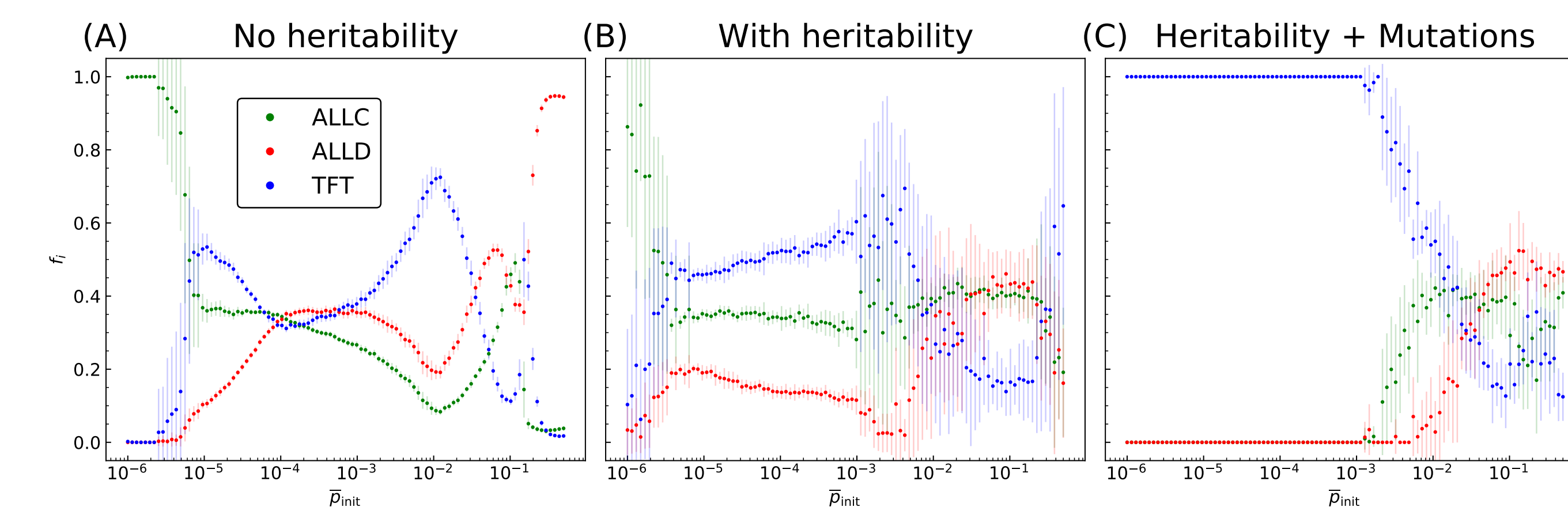


Figure 2. Relative population frequency with respect to the total population of agents following either a strategy of unconditional cooperation (**ALLC**), unconditional defection (**ALLD**) or mutual reciprocity (**TFT**), as a function of the mean error rate of the initial distribution \bar{p} . (A) Error rates are immutable. (B) Error rates become heritable. (C) Error rates become subject to mutations.

Results (*continued*)

Mutation Favors Cooperative Behavior

Analyzing statistical ensembles of simulations while varying the mutation rate μ and the extent of mutations σ_p reveals that there is a *transition* towards cooperative behavior that depends on both parameters. As we increase either parameter past some thresholds, **TFT** invades the lattice – i.e. cooperative behavior emerges!

There is however an upper limit above which cooperation starts to decline – at very high mutation probability, we see a steep decline in **TFT** populations (far right of Fig. 3A-B). This is analogous with the breakdown of heredity in Eigen's error threshold.

Finally, repeating the same simulations while removing the effects of spatiality (i.e. randomizing the Moore neighborhoods) eliminates all the transitions observed with spatiality included. In other words, spatiality is a critical element of the transition towards cooperative behavior!

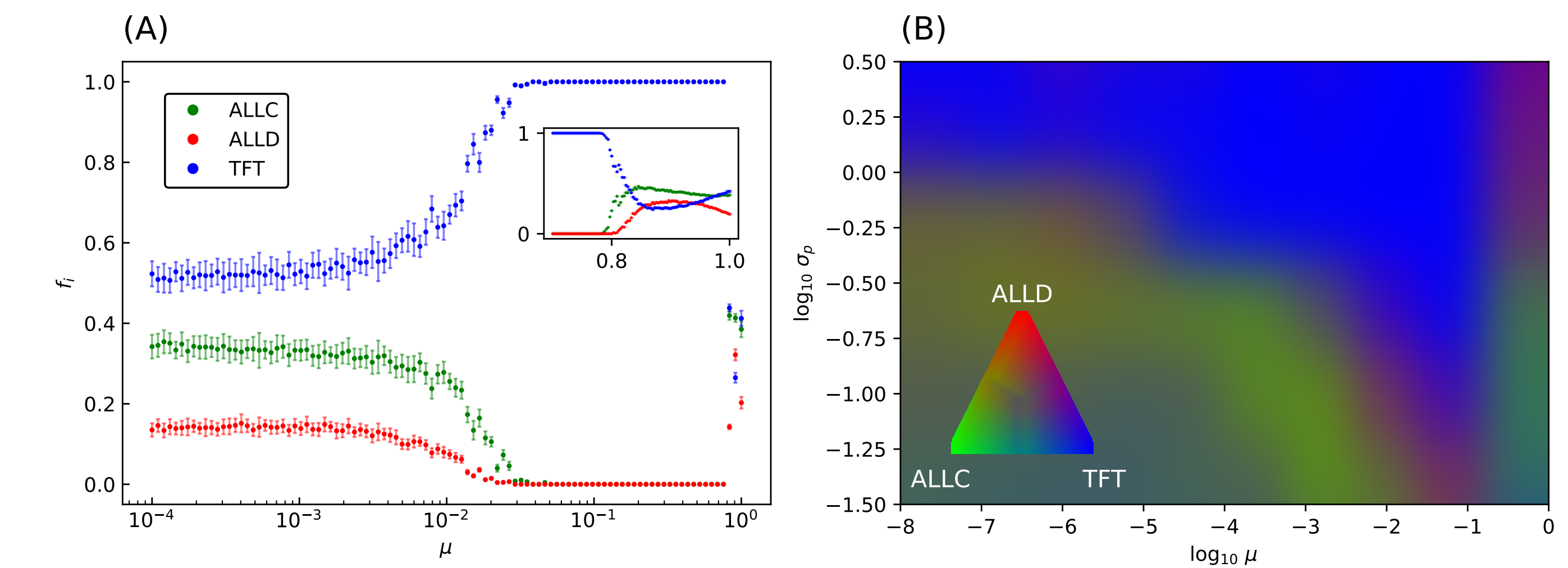


Figure 3. (A) Relative population frequency for each strategy as a function of the mutation rate μ . Increasing the mutation probability μ drives a continuous transition of the final population fractions towards invasion by **TFT**. (B) Relative population frequency of mutual cooperative behavior (**TFT**) as a function of error rates mutation rate μ and the standard deviation of mutations σ_p . The transition towards a domination by **TFT** depends on both the mutation rate μ and the scale σ_p of mutations. Mutation rates exceeding a critical threshold ($\mu > 0.8$) further prevent generalized cooperation from emerging for a wide range of error rates.

Conclusion

- Variability (i.e. mutations) favors the emergence of cooperative behavior!
- **For biologists:** the triad of evolutionary biology (selection–heredity–variation) seems both necessary *and* sufficient for (biochemical) cooperation to emerge
- **For astrobiologists:** the Habitable Zone might be wider than we think (e.g. near M-dwarfs, thermodynamical perturbations, geologically violent environment, active host stars with high UV flux, etc.)

References

- [1] Robert M. Axelrod. *The Evolution of Cooperation*. Basic Books, New York, rev. ed edition, 1984.
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- [3] Paul G. Higgs and Niles Lehman. The RNA World: Molecular cooperation at the origins of life. *Nature Reviews Genetics*, 16(1):7–17, January 2015.
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