

## Nonlinear Dynamics of the Rock-Paper-Scissors Game with Generational Mutations

### **Introduction:**

The well known game of Rock-Paper-Scissors (RPS) is, on the surface level, just another children's playground game or something adults use to decide who pays for parking after a night out. However, biologists, mathematicians and any other professionals who rely on the implementation of game theory have long utilized this simple game setup to better elucidate the dynamics of competition in which an individual has an advantage over one opponent but not the other. This game is of particular interest because, under the right conditions, the system is sent to endless cyclic behavior of varying dominant strategies.

One of the most widely used methodologies for modeling the RPS game relies on the replicator equation which effectively demonstrates the various stable states of the game (ie. coexistence, limit cycles and heteroclinic cycles). In 2015, Toupou and Strogatz showed that, when various mutational regimes are added to this framework, a supercritical hopf bifurcation and subsequent limit cycles can always be achieved by varying both the strength of mutation and cost of losing an interaction (Toupou 2015). For mathematical simplicity, the authors utilized an *additive* mutational scheme to their replicator equation dynamics. This is analogous to adding the potential for mutation among adults in a system.

My model attempts to extend the findings of the previously mentioned paper by implementing identical mutational regimes but by using *multiplicative* mutational schemes in the replicator dynamics. This is biologically analogous to having generational mutations in the offspring of the current population. For these patterns of mutation under a multiplicative lens, we can demonstrate that, once again, under the right conditions a limit cycle will be born about the interior fixed point where we have coexistence of all three strategies in the system. In particular, we once again demonstrate that we can always achieve a stable limit cycle in the limiting case of zero mutation.

Since we are examining mutations on the scale of generations, it is common to look at the analogous biological system of bacteriocin-producing, resistant and sensitive *E. coli*. If we

simplify the system to the three species of toxin producing bacteria, those which are sensitive to the toxin, and those which are resistant to the toxin, then we can see that this biological situation can be effectively modeled by an RPS game. If we use the biologically feasible assumption that toxin sensitive bacteria can invade the resistant population then we have the game setup of: toxin producing > toxin sensitive > toxin resistant > toxin producing. While it is known that a well-mixed population of these types of bacteria without mutations can enter cyclic behavior, it remains unclear whether or not introducing errors in replication (mutation) to the system will allow for similar or different behavior.

In addition to demonstrating that we can once again undergo a hopf bifurcation and produce stable limit cycles with our multiplicative model, we can also sketch a proof of their existence which can be applied to all the mutational patterns presented in the paper.

### Methods:

In order to better understand the modeling approach taken in this paper, it is best to first look at the results of the additive mutational model and compare the findings to that of the multiplicative. The first difference between the models comes from the payoff matrices used. In the Toupou-Strogatz model, the following payoff matrix was utilized:

	Rock	Paper	Scissors
Rock	0	$-(\epsilon + 1)$	1
Paper	1	0	$-(\epsilon + 1)$
Scissors	$-(\epsilon + 1)$	1	0

Table I: Payoff matrix of an RPS game for the Strogatz-Toupou model. A zero-sum game occurs at  $\epsilon = 0$ .

This table was utilized because in the case of competition among adults, where mutation occurs on the same generational level, the payoffs are defined as follows: two players using the same strategy will have no benefit or cost to playing, a player with a dominant strategy will win with no cost (rescaled payoff to 1), and a player with a suboptimal strategy will lose with variable cost based on the parameter  $\epsilon$  (where  $\epsilon = 0$  results in a zero-sum game).

In our model, with generational mutations this payoff matrix must be adapted slightly to reflect the new outcomes:

	Rock	Paper	Scissors
Rock	1/2	$-\epsilon$	1
Paper	1	1/2	$-\epsilon$
Scissors	$-\epsilon$	1	1/2

Table II: Payoff matrix of an RPS game for our new model.

The new payoff matrix reflects the generational changes as a result of each interaction: two players of the same strategy will each increase their populations by the same amount, a player playing a dominant strategy will fully increase their population (rescaled to 1), and when playing a losing strategy there is a variable decrease in population represented by  $\epsilon$  once again (where  $\epsilon = 0$  would represent no change in portion of individuals playing that strategy when losing).

### *Model Definition*

I now present the generalized model which will define all the future mutational regimes based on how the mutational matrix  $Q$  is assigned. We must first suppose that we have a well-mixed population of  $N$  individuals. If we let  $x$ ,  $y$ , and  $z$  represent the proportion of individuals playing rock, paper and scissors respectively then we have the equality  $x + y + z = 1$ , and by using this we can redefine the system to that of two variables rather than three by  $z = 1 - x - y$ .

Using these preliminary statements and assumptions we can define our system of ordinary differential equations as

$$\begin{aligned}\frac{dx}{dt} &= x f_x q_{xx} + y f_y q_{yx} + (1 - x - y) f_z q_{zx} - x \phi \\ \frac{dy}{dt} &= x f_x q_{xy} + y f_y q_{yy} + (1 - x - y) f_z q_{zy} - y \phi\end{aligned}$$

where  $f_i$  is the fitness of strategy  $i$  (the expected payoff against the other strategies of the system),  $q_{ij}$  is the probability that  $i$  mutates into  $j$  (these will be defined based on each mutational schema) and  $\phi = x f_x + y f_y + z f_z$  is the average fitness of the population. For the above reduced model, we can more clearly define the fitness of each strategy as

$$f_x = 1 - \frac{1}{2}x - (\epsilon + 1)y$$

$$f_y = x + \frac{1}{2}y + \varepsilon (1 - x - y)$$

$$f_z = \frac{1}{2} (1 - (1 + 2\varepsilon) x + y).$$

As a result of reducing the dimension of the model, we also have the luxury of mapping trajectories onto the  $x + y + z = 1$  simplex which is achieved by using a simple linear transformation onto the equilateral triangle by

$$\begin{pmatrix} X \\ Y \end{pmatrix} = \begin{pmatrix} 1 & \frac{1}{2} \\ 0 & \frac{\sqrt{3}}{2} \end{pmatrix} \begin{pmatrix} x \\ y \end{pmatrix}$$

## Results:

### *Mutation Case 1: Global Mutation*

The first mutational scheme examined by Toupo is the symmetric case of global mutation where the rate of mutation is equivalent for all transitions (Figure I).

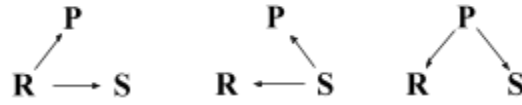


Figure I: Global Mutation Scheme

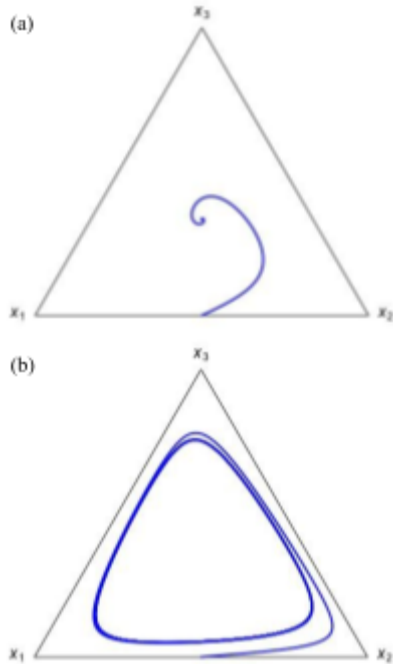


Figure II: (a) Phase portrait corresponding to the stable inner fixed point of the system for large rate of mutation. (b) Phase portrait of limit cycle which is achieved at low rate of mutation and high cost (epsilon).

In the additive mutation case, a system with this form of mutation will exhibit either a stable limit cycle or stable inner fixed point at the symmetric point  $(\frac{1}{3}, \frac{1}{3}, \frac{1}{3})$ , in addition to the fixed points at the vertices of the simplex. These results have been replicated in figure II.

We must first note that for the global mutation case we have the following mutation matrix being plugged in to the general sets of differential equations on page 3:

$$Q = \begin{bmatrix} 1 - \mu & \frac{1}{2}\mu & \frac{1}{2}\mu \\ \frac{1}{2}\mu & 1 - \mu & \frac{1}{2}\mu \\ \frac{1}{2}\mu & \frac{1}{2}\mu & 1 - \mu \end{bmatrix}$$

The multiplicative mutation model yields almost identical results, where it also possesses a stable inner fixed point which is once again the symmetric point  $(\frac{1}{3}, \frac{1}{3}, \frac{1}{3})$ . When the

parameters are varied, this fixed point once again becomes unstable via a supercritical hopf bifurcation and yields a stable limit cycle (phase diagrams in figure III). With respect to *E. Coli*, this means that when all strains are mutating into one another, we can have either a stable coexistence of all strategies or endless cycle of dominant strategies dependent upon the probability of mutation.

### *Mutational Case 2: Single*

#### *Mutation $x \rightarrow y$*

For the case of single mutations, we need only consider two different pathways: flowing from a losing to the winning strategy (ie.  $x \rightarrow y$ ) or flowing from

the winning strategy to the loser (ie.  $y \rightarrow x$ ). Thus we need only show one mutation pattern of each type and have selected the pathways between  $x$  and  $y$ .

The mutation matrix for the first scenario is defined as follows and the values are once again plugged into the dynamic equations on page 3:

$$Q = \begin{bmatrix} q_{xx} & q_{xy} & q_{xz} \\ q_{yx} & q_{yy} & q_{yz} \\ q_{zx} & q_{zy} & q_{zz} \end{bmatrix} = \begin{bmatrix} 1 - \mu & \mu & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

This situation is analogous to the toxin producing bacteria mutating into the toxin resistant strain. Figure IV depicts the phase diagrams for this system of differential equations. Identically to the additive mutation case, we have an unstable fixed point at  $(0, 1, 0)$ , a stable fixed point at  $(0, 0, 1)$ , and either a stable inner fixed at some small perturbation away from the symmetric point, call it  $(\frac{1}{3} + \delta_x, \frac{1}{3} + \delta_y, \frac{1}{3} + \delta_z)$  where the  $\delta$  perturbations tend towards 0 as  $\mu \rightarrow 0$ . In addition, the point  $(1, 0, 0)$  is no longer a fixed point since we have introduced the mutation which forces those playing strategy  $x$  to eventually mutate away into  $y$ .

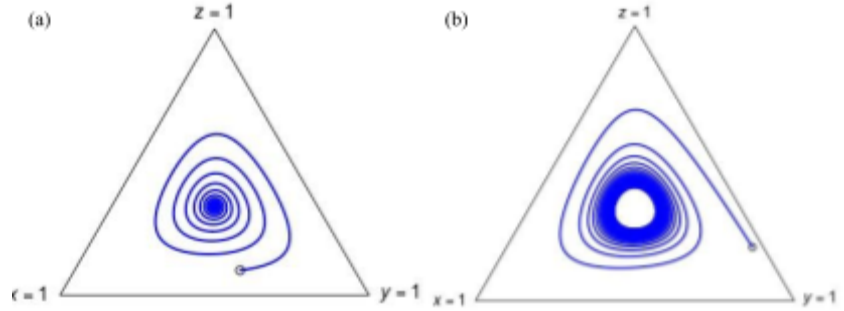


Figure III: (a) Phase diagram for the parameter set which yields a stable inner fixed for the global mutation system in our multiplicative mutation model. (b) Phase diagram for the parameter set which yields a stable limit cycle for the global mutation system in our multiplicative mutation model.

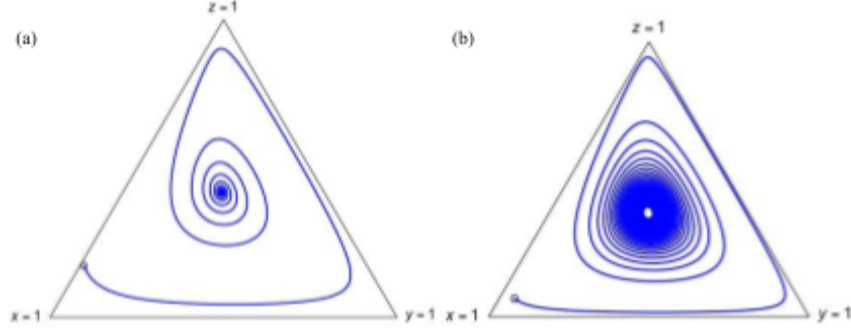


Figure IV: (a) Phase diagram for single mutation from  $x$  to  $y$  with parameter set that produces a stable inner fixed point. (b) Phase diagram for single mutation from  $x$  to  $y$  with parameter set that produces a stable limit cycle after undergoing a supercritical hopf bifurcation.

### *Mutational Case 2: Single Mutation $y \rightarrow x$*

A more interesting dynamic system arises when the mutation term is added such that a winning strategy will constantly change into its loser counterpart. An example of this would be if resistant bacteria mutate into toxin producing strain. This is done by setting our mutation matrix equal to the following, similar to before:

$$Q = \begin{bmatrix} q_{xx} & q_{xy} & q_{xz} \\ q_{yx} & q_{yy} & q_{yz} \\ q_{zx} & q_{zy} & q_{zz} \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 \\ \mu & 1 - \mu & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

Similar to the other single mutation pathway, this system still has two fixed points at the vertices of  $(0, 0, 1)$  and  $(1, 0, 0)$  since now all of those implementing strategy  $y$  will constantly mutate into  $x$ . This mutation going against the natural gradient of the game allows for the emergence of a new stable state, one of coexistences between strategies  $x$  and  $y$  (as is seen in the Toupo model). This coexistence makes sense because in a system of exclusively players of  $x$  and  $y$ , the  $y$  population will constantly be growing as they beat out the  $x$ . However, these winners are also constantly being mutated into their counterpart. These three states of interest can be seen in the phase diagrams of figure V.

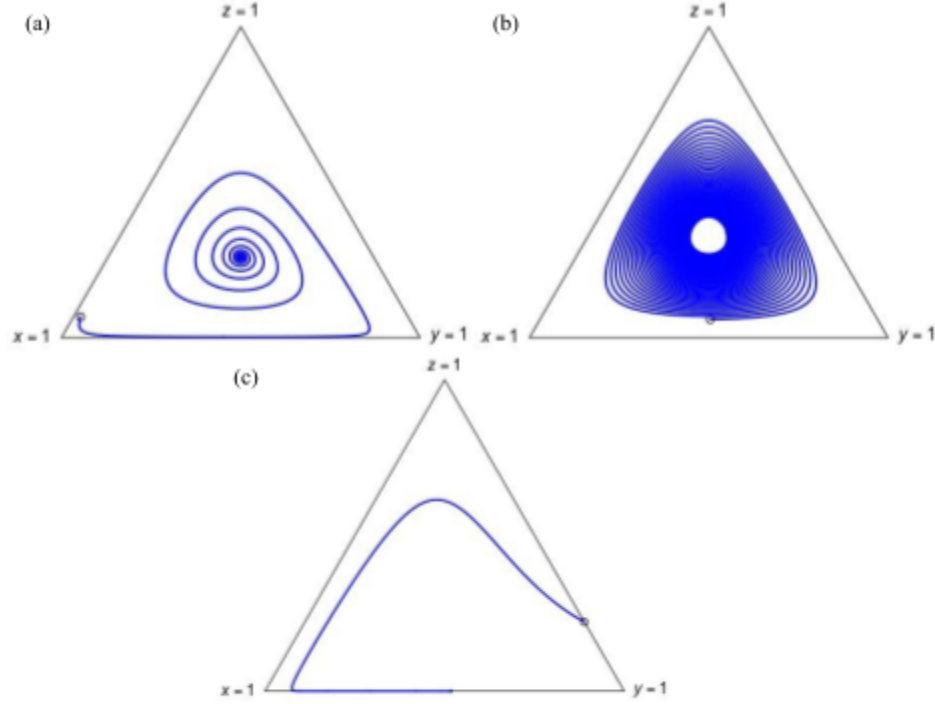


Figure V: (a) Phase diagram for the inner fixed point which is a perturbation of the symmetric interior point. (b) Phase diagram of the stable limit cycle achieved when the inner fixed point undergoes a supercritical hopf bifurcation. (d) The new stable state, coexistence, that arises from the mutation pathway going against the natural flow of the RPS game.

### *Mutational Case 3: Double Mutations*

In this situation we have one strategy mutating into the other two at an equal rate of  $\mu$ . There are three different types of this mutation pathway: opposing mutations ( $z \rightarrow y$  and  $x \rightarrow y$ ), mutations in the same direction ( $z \rightarrow x$  and  $x \rightarrow y$ ), and finally bidirectional ( $x \rightarrow y$  and  $y \rightarrow x$ ). Constructing the mutation matrix in the same fashion as before, we can see the various stable states once again as we vary the value of  $\mu$ .

The pathway consisting of opposing mutations yields three stable states, similar to the single mutation against the natural flow presented above: a stable limit cycle about the symmetric center point, a stable inner fixed point, and finally a coexistence between those playing strategies  $y$  and  $z$ . Figure VI shows the phase diagrams transition as we increase the value of  $\mu$ .

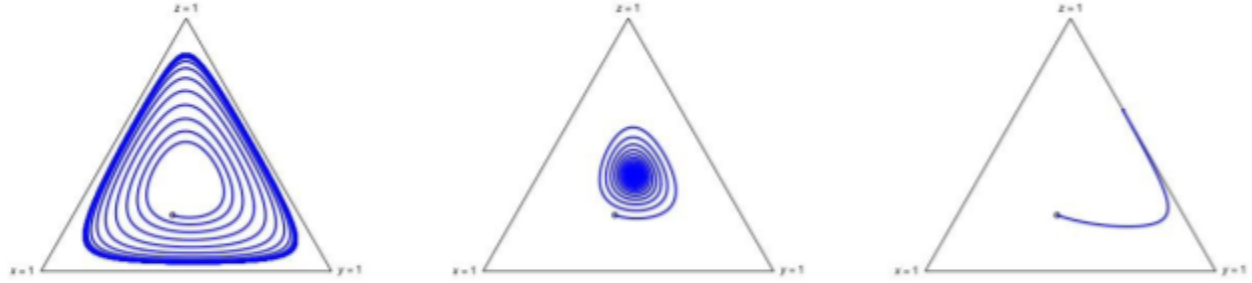


Figure VI: Phase portrait of the opposing mutation pathway. As we increase the value of the mutation parameter, the limit cycle turns into a stable spiral and after a certain point, the only remaining fixed point resides as a coexistence between strategies  $y$  and  $z$ .

The other two pathways consisting of mutations in the same direction and bidirectional mutation yield similar results to all other mutation patterns and also reflect the results of the additive case. Their simplex trajectories are nearly identical so it suffices to just show one (figure VII):

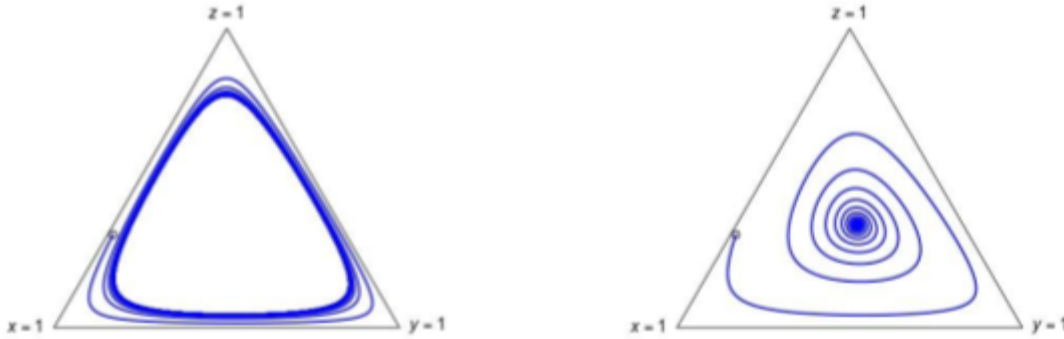


Figure VII: Phase diagrams for the stable limit cycle and subsequent bifurcation to a stable spiral for both the same direction and bidirectional mutation pathways.

## Discussion:

In the results we have demonstrated that in this multiplicative (or generational) mutation model, we can always achieve stable limit cycles via Hopf bifurcations. We can also develop a proof for the existence of limit cycles. The following is a proof for the case of global mutations (mutational case 1 above). I was unable to create a proof for the general  $Q$  matrix, which would therefore be a proof under all mutational pathways, but it can be seen that if you simply change the values of the parameters in the probability matrix and carry out the same proof methodology you can find the correct conditions under which a hopf bifurcation will occur in each of the above systems.



## Proof of Hopf Bifurcation for Global Mutation Pathway:

Let  $\mathbf{g}(\mathbf{x}) : \mathbb{R}^N \rightarrow \mathbb{R}^N$  be  $\mathbf{g}(\mathbf{x}) = [g_1(\mathbf{x}), \dots, g_N(\mathbf{x})]$  where  $\mathbf{g}(\mathbf{x})$  defines the transformation of the vector  $\mathbf{x}$  into the system of ordinary differential equations defined by

$$\dot{x}_i = \sum_{j=1}^N x_j f_j(\mathbf{x}) q_{ji} - x_i \phi := g_i(\mathbf{x}) \quad (1)$$

where each  $f_i(\mathbf{x})$  and the  $\phi$  term are identical to how they were defined at the start of the paper. We can also define our simplex as

$$\Delta_n = \{\mathbf{x} \in \mathbb{R}^{n+1} | x_i \geq 0, \mathbf{x}^T \mathbf{1} = 1\} \quad (2)$$

Now we need to define our equation for mapping the  $N$ -dimensional system to the reduced  $(N-1)$  dimension by the following (note that  $\hat{\mathbf{x}}$  is the  $\mathbf{x}$  vector reduced to  $N-1$  dimensions by eliminating the last element):

$$\dot{x}_i = w_i(\hat{\mathbf{x}}), i \in \{1, 2, \dots, N-1\}, \quad (3)$$

$$w_i(\hat{\mathbf{x}}) := g_i(x_1, x_2, \dots, x_{N-1}, 1 - \sum_{j=1}^{N-1} x_j) \quad (4)$$

Some final notation to be aware of (this  $Q$  matrix is specific to the global mutation pathway):

$$B = \begin{bmatrix} 1/2 & -\epsilon & 1 \\ 1 & 1/2 & -\epsilon \\ -\epsilon & 1 & 1/2 \end{bmatrix}, Q = \begin{bmatrix} 1-\mu & \frac{1}{2}\mu & \frac{1}{2}\mu \\ \frac{1}{2}\mu & 1-\mu & \frac{1}{2}\mu \\ \frac{1}{2}\mu & \frac{1}{2}\mu & 1-\mu \end{bmatrix}$$

Now that everything is defined properly, we can create a proof for when limit cycles *cannot* exist in our global mutation scheme. This proof implements Bendixson's Criterion (Strogatz 2015) by finding the divergence of the reduced system of equations and finding the conditions under which this value is semi-definite.

$$\nabla \cdot \mathbf{w}(\hat{\mathbf{x}}) = \sum_{i=1}^{N-1} \frac{\partial w_i}{\partial x_i} = \sum_{i=1}^N \frac{\partial g_i}{\partial x_i} - \sum_{i=1}^N \frac{\partial g_i}{\partial x_N} \quad (5)$$

$$\begin{aligned} \sum_{i=1}^N \frac{\partial g_i}{\partial x_i} - \sum_{i=1}^N \frac{\partial g_i}{\partial x_N} &= \sum_i \frac{\partial}{\partial x_i} [x_i (f_i q_{ii} - \phi) + \sum_{j \neq i} x_j f_j q_{ji}] - \sum_i \frac{\partial}{\partial x_N} [\sum_{i=1}^N \sum_{j=1}^N x_j f_j q_{ji} - x_i \phi] \\ &= \sum_i [f_i q_{ii} + x_i q_{ii} \frac{\partial f_i}{\partial x_i} - \phi - x_i \frac{\partial \phi}{\partial x_i} + \sum_{j \neq i} x_j q_{ji} f_{ji}] - \frac{\partial}{\partial x_N} [\phi (1 - \sum_{i=1}^N x_i)] \end{aligned}$$

Combining terms and noting that  $\phi = \mathbf{x}^T B \mathbf{x}$

$$\begin{aligned}
& (1 - \mu)\mathbf{1}^T B \mathbf{x} + \frac{1}{2}(1 - \mu) - N\phi - \mathbf{x}^T(B + B^T)\mathbf{x} + \sum_i \sum_{j \neq i} x_j q_{ji} b_{ji} \\
& (1 - \mu)\mathbf{1}^T B \mathbf{x} + \frac{1}{2}(1 - \mu) - \mathbf{x}^T(NB + B^T)\mathbf{x} + \frac{1}{2}\mu(1 - \epsilon)
\end{aligned} \tag{6}$$

Now we setup the following inequality to determine the conditions under which the divergence  $(\nabla \cdot \mathbf{w}(\hat{\mathbf{x}}))$  is semi-definite, and thus by the Bendixson Criterion limit cycles cannot exist.

$$\max_{x \in \Delta_{N-1}} (1 - \mu)\mathbf{1}^T B \mathbf{x} + \frac{1}{2}(1 - \mu) + \frac{1}{2}\mu(1 - \epsilon) \leq \min_{x \in \Delta_{N-1}} \mathbf{x}^T[NB + B^T]\mathbf{x} \tag{7}$$

After simplifying and identifying the maximum / minimum values of both sides of the inequality we have the following condition for which limit cycles cannot exist:

$$\mu_C > \frac{2\epsilon}{3(3 - \epsilon)} \tag{8}$$

We can now prove that for  $\mu < \mu_c$  we will have a stable limit cycle about the fixed point  $(x^*, y^*) = (\frac{1}{3}, \frac{1}{3})$ . This proof implements the Hopf Bifurcation theorem (Guckenheimer 1983). The Jacobian of the reduced dimension vector field  $\mathbf{w}(\hat{\mathbf{x}})$  at this fixed point has eigenvalues

$$\lambda = \frac{1}{12}(2\epsilon - 9\mu + 3\epsilon\mu \pm i\sqrt{3(1 + \epsilon)^2(3\mu - 1)^2}) \tag{9}$$

and from this we can see that the  $\Re(\lambda) = 0$  at  $\mu = \mu_C$  and thus the eigenvalues at this point are *purely* imaginary. Using the Hopf Bifurcation Theorem, we now need to show that

$$\frac{d}{d\mu}(\Re(\lambda))|_{\mu=\mu_C} \neq 0$$

which when plugging in our value for  $\Re(\lambda)$ , we get

$$\epsilon \neq 3$$

Which is always true since we have limited  $0 \leq \epsilon \leq 1$ . Therefore, a Hopf bifurcation occurs at  $\mu = \mu_C$  via the Hopf Bifurcation Theorem.

Our results show that under any mutation pattern, we achieve stable limit cycles or cyclic behavior among competing strategies in the RPS game. It is reasonable to assume that a three strategy population of *E. coli* as described at the start of this paper would follow similar dynamics. The introduction of probability of error in replication is a very real aspect of bacterial life and as such it is interesting to see the theoretical effects it would have on a well-mixed population, in particular the fact that even a very small probability of error in mutation (as is expected in life) will induce this cyclic dominance of different bacterial strains.

A limitation of my work comes from my lack of access to more scientific computing power. As a result, I was not able to generate bifurcation diagrams for the systems modeled as Toupo did in the focal paper and the systems without the symmetric inner fixed point at  $(\frac{1}{3}, \frac{1}{3}, \frac{1}{3})$ , but rather a slight perturbation of this, were much too complex for my scripts to effectively analyze in the same way that the symmetric global mutation pathway was done above. If I were to extend this paper further, it would be wise to identify the bifurcation curves for the Hopf bifurcation of all the systems and subsequently look for similarities and differences among the different mutation schemes. In addition, a proof using just the general  $Q$  matrix, rather than an instance of it with already assigned values of each  $q_{ij}$ , would be a better and hopefully more elegant proof of the ever present limit cycles of the RPS game with mutation.

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