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Contagion and State Dependent Mutations*

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Abstract

Early results of evolutionary game theory showed that the risk dominant equilibrium is uniquely selected on the long run by the best response dynamics with mutation. Bergin and Lipman (1996) qualified this result by showing that for a given population size the evolutionary process can select any strict Nash equilibrium if the probability of choosing a nonbest reply is state-dependent. This paper shows that the unique selection of the risk dominant equilibrium is robust with respect to state dependent mutation in local interaction games. More precisely, for a given mutation structure there exists a minimum population size beyond which the risk dominant equilibrium is uniquely selected. Our result is driven by contagion and cohesion among players, which exists only in local interaction settings and favors the play of the risk dominant strategy. Our result strengthens the equilibrium selection result of evolutionary game theory.

Keywords: Contagion, state dependent mutations, risk dominance, local interaction games.

JEL classification: C72; D83

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1 Introduction

Kandori, Mailath and Rob (1993) and Young (1993) showed that the risk dominant equilibrium is uniquely selected for global interaction games with mutation. When a group of agents play coordination games among themselves and there is a small mutation probability that each agent may choose a sub-optimal strategy, the risk dominant equilibrium is selected uniquely as the probability of mutation vanishes to zero. While the strength of the result follows from the parsimony of the model, Bergin and Lipman (1996) showed that the result crucially depends on one of the few parameters of the model. In particular they criticized the result in that the equilibrium selection result may be overturned in favor of the payoff dominant equilibrium if state dependent mutation is allowed. If agents are more cautious when playing the payoff dominant action than the risk dominant action and hence they make less mistakes or less experimentations, the equilibrium playing payoff dominant action remains the long-run equilibrium.

This paper reexamines their argument for equilibrium selection in the context of local interaction games. We show that for a given mutation structure there exists a minimum finite population size beyond which the risk dominant equilibrium is uniquely selected. This is because the equilibrium selection turns out to depend on the size of the population in local interaction games. This is also the reason why we obtain a different result from that of Bergin and Lipman (1996). They take the population size as given, and then pick up a mutation structure which selects some long run equilibrium. In contrast, we fix the mutation structure first, then we set the population size sufficiently high so that the risk dominant equilibrium is selected. Since the equilibrium selection is independent of the population size in global interaction games [see the motivating example in Bergin and Lipman (1996)], it does not make a difference in such an environment which parameter is regarded as fixed relative to the other one. In contrast, it matters for local interaction games.

The nature of our result is easiest to understand in the model where each agent plays against the nearest neighbors on a circle of size N as in Ellison (1993). Assume that the mutation rates for each action choices are not uniform, and specifically the payoff dominant action has mutation rate smaller than the risk dominant action by some order. Observe that if there are two adjacent sites playing the risk dominant action, they continue to play the risk dominant action independent of the strategy profile played in the remainder of the circle. Moreover two additional sites next to the two adjacent sites who are already playing the risk dominant strategy switch to the risk dominant action in the next period in the absence of mutation because *contagion* takes place deterministically. Thus, transition from the configuration playing payoff

dominant strategy everywhere to that playing risk dominant strategy everywhere takes just 2 mutations. In contrast, transition from the configuration playing risk dominant strategy everywhere to that playing payoff dominant strategy everywhere require N simultaneous mutations. The *cohesion* between players playing the risk dominant strategy makes it difficult to break that group. The number mutations required to make transition to the state playing risk dominant strategy everywhere is independent of the population size while the transition to the state playing the payoff dominant strategy everywhere depends on the population size. Therefore, if the population is big enough, the latter transition probability can be made much smaller than the former one taking the mutation structure across the two strategies as given. So that the risk dominant equilibrium is uniquely selected in the long run even even if the mutation probability is state dependent.

Recently many attentions were paid to the local interaction games. For instance Anderlini and Ianni (1996), Blume (1993, 1994, 1995), Lee and Valentinyi (2000) and Morris (2000) examined the issue of equilibrium selection in local interaction games with and without mutation. Most notably Ellison (2000) addresses the issue of contagion dynamics and mutation.[1] He develops a powerful technique for the determination of the long-run distribution of a stochastic process when there are many limit states in the system. In contrast we focus narrowly to 2 dimensional local interaction games which has a lot of limit states. In doing so we find that the selection of risk dominant equilibrium is not mainly due to the step-by-step evolution. In particular the saving in the waiting time emphasized in Ellison (2000) does not necessarily favor the propagation of risk dominant strategy since the step-by-step evolution which induces the saving may facilitate the transition from the risk dominant strategy to the payoff strategy as much as the opposite. It is the contagion dynamics which provides huge saving in the number of mutations required for the transition from payoff dominant strategy to the risk dominant strategy.

Since Bergin and Lipman (1996) suggested a careful examination of the implication of the state dependent mutation, more attention has been paid to the issue. For instance van Damme and Weibull (1998) examined the decision making process associated with the cost of mistake. They concluded that the introduction of the state dependent mutation does not overturn the standard equilibrium selection result since the concern for the cost of mistake favors the mutation toward the risk dominant strategy. Similarly, Young (1998) one can relax the uniformity of mutation in a plausible way without changing the stochastically stable outcome. Although refining the decision making process is an important issue, we believe that it is at least as im-

[1] We provide more discussion on the relationship of our result to his approach later

portant to prove the robustness of the equilibrium selection result for a certain environment. In particular, the evolutionary approach deliberately avoids refining the decision making process since a result obtained by refinement would be susceptible to a perturbation in the opposite way. In the absence of a theory which guarantees the validity of a particular refinement, establishing the robustness in a certain environment seems consistent with the general spirit of the research in the area.

The rest of the paper organized as follows. Section 2 describes a 2-dimensional environment of local interaction. Section 3 contains the main result of the paper. This section starts with a renormalization argument, then characterizes the stochastically stable outcome using a series of lemmas and propositions. Section 4 concludes the paper.

2 Local Interaction Game

2.1 A framework of local interaction

There is a population of N^2 players located on a 2 dimensional torus $\Lambda(N) = \mathbb{Z}^2 \cap [0, N)^2$ for $N \geq 1 \pmod N$. A player with address $x \in \Lambda(N)$ interacts with her nearest neighbors. The set of neighbors for the origin is defined by $\mathcal{N} \equiv \{y : \|y\| = 1\}$ where $\|y\| \equiv (|y_1| + |y_2|)$, and the set of neighbors for player x is given by $x + \mathcal{N} \equiv \{y : \|x - y\| = 1\}$, namely the translation of $\mathcal{N}$ by x.

There are two pure strategies $\{A, B\}$ for player $x \in \Lambda(N)$. Let $s_t : \Lambda(N) \longrightarrow \{A, B\}$ be a map which represents the strategy chosen by player x at time t. We characterize the dynamics of the population in terms of the set of players playing A, thus

$$S_t = \{x : s_t(x) = A, x \in \Lambda(N)\}. \tag{1}$$

2.2 Coordination game

Consider the 2×2 coordination game given in Table 1. We require that $a > c$, $d > b$ and $(a-c) > (d-b)$ so that both (A, A) and (B, B) are Nash equilibria and (A, A) is the risk dominant one.

All players play the game simultaneously over discrete times. The feature of local interaction is reflected in that the payoff of each player depends on the strategy played by herself and

	A	**B**
A	a, a	b, c
B	c, b	d, d

Table 1: Coordination Game

everyone in the neighborhood. The payoff of player x playing strategy A in period t is given by

$$u_t(x, A) = b + (a - b)\frac{|S_t \cap (x + \mathcal{N})|}{|x + \mathcal{N}|}, \tag{2a}$$

where $|\cdot|$ denotes the cardinality of a set. Similarly, the payoff of player x playing strategy B in period t is given by

$$u_t(x, B) = d + (c - d)\frac{|S_t \cap (x + \mathcal{N})|}{|x + \mathcal{N}|}. \tag{2b}$$

In the absence of mutation, players are assumed to play the myopic best-response: player x in period $t + 1$ chooses

$$s_{t+1}(x) = \arg\max_{\{A,B\}}\{u_t(x, A), u_t(x, B)\}. \tag{3}$$

Thus player x plays A in period $t + 1$ if

$$\frac{|S_t \cap (x + \mathcal{N})|}{|x + \mathcal{N}|} \geq \frac{d - b}{(a - c) + (d - b)} \equiv \theta. \tag{4}$$

Since (A, A) is the risk dominant equilibrium, $\theta < 1/2$.

2.3 State Dependent Mutation

We introduce mutation into the model; the agent may make a mistake or an experiment and thus choose a strategy at random with a small probability. In particular we consider a stochastic process $\{S_t^\varepsilon\}_{t\geq 0}$ which is derived from $\{S_t\}_{t\geq 0}$, allowing for random noise due to mutation. In the configuration of the noise structure, we explicitly recognize the possibility of the state dependent mutation probability.

To ensure state dependence on the one hand, but independence across players and time, we use the following construction. Let

$$p : \Lambda(N) \times [0, 1] \times \{A, B\}^{\Lambda(N)} \times \{A, B\} \longrightarrow [0, 1]$$

be a map which assigns two non-negative numbers, $p(x, \varepsilon, S, A)$ and $p(x, \varepsilon, S, B)$, to player x representing the probability of mutation to A and B, respectively. We require that $p(x, \varepsilon, S, A) + p(x, \varepsilon, S, B) \in [0, 1]$.

Define $\lambda_t(x)$ as the collection of independent random variables uniformly distributed on $[0, 1]$ for all t and $x \in \Lambda(N)$, and define X_t as:

$$X_t(x) = \begin{cases} A & \text{if } \lambda_t(x) \leq p(x, \varepsilon, S^{\varepsilon}_{t-1}, A) \\ B & \text{if } \lambda_t(x) \geq 1 - p(x, \varepsilon, S^{\varepsilon}_{t-1}, B) \\ 0 & \text{otherwise.} \end{cases} \tag{5}$$

$X_t(x)$ is a collection of random variables which are independent across players and time. The specification also ensures that the mutation probability depends on the state of the population, the location of the agent, and so on. In particular, the state of the population includes the state of agent x so that the mutation probability may be different depending on whether the agent plays A or B and one strategy may be chosen with a higher mutation probability than the other one.

We make the following assumption about the mutation structure.

Assumption 1 *The mutation probability satisfies:*

1. $\lim_{\varepsilon \to 0} p(x, \varepsilon, S, \cdot) = 0$ *for all* $x \in \Lambda(N)$ *and* $S \subset \Lambda(N)$*, and*

2. *there exist* $\alpha(A) > 0$ *and* $\alpha(B) > 0$ *such that*

$$\inf_{x \in \Lambda(M), S \subset \Lambda(N)} p(x, \varepsilon, S, A) = \varepsilon^{\alpha(A)} \tag{6a}$$

$$\sup_{x \in \Lambda(M), S \subset \Lambda(N)} p(x, \varepsilon, S, B) = \varepsilon^{\alpha(B)}. \tag{6b}$$

The first assumption is standard. The second one requires that the mutation probabilities are uniformly bounded, i.e. $\varepsilon^{\alpha(A)}$ and $\varepsilon^{\alpha(B)}$ are the lower bound and upper bound on the probability that an agent chooses strategy A and B as a consequence of mutation, respectively. This assumption is not more restrictive than other similar assumptions used in the literature (see Ellison (2000) and Young (1993) for example). Note that $\varepsilon^{\alpha(B)}$ can be of a lower order than $\varepsilon^{\alpha(A)}$, i.e. B can be adopted more frequently than A. Hence this is compatible with the environment for which Bergin-Lipman's result holds for a given population size.

Using the construction, the transition rule for the process $\{S_t^\varepsilon\}_{t\geq 0}$ can be formally written as

$$S_{t+1}^\varepsilon = \Phi(S_t^\varepsilon) \equiv \left\{ x \in \Lambda(N) : X_{t+1}(x) = 0, \frac{|S_t^\varepsilon \cap (x + \mathcal{N})|}{|x + \mathcal{N}|} \geq \theta \right\} \cup \{x : X_{t+1}(x) = A\} \tag{7}$$

Denoting the event where everybody plays A by $\vec{A}$, we are interested in the long-run probability of the event $\vec{A}$:

$$\mu_\varepsilon(\vec{A}) = \lim_{t\to\infty} \Pr(S_t^\varepsilon = \Lambda(N)). \tag{8}$$

In particular our main goal is to characterize $\mu_\varepsilon(\vec{A})$ as $\varepsilon \downarrow 0$.

3 Best Response Dynamics with State Dependent Mutations

Our analysis of the long-run distribution of the population consists of two steps. First, we construct a new process which satisfies some sample path inequality with respect to the original process so that it implies a certain distributional inequality.[2] Second, we compute the long-run distribution for the new process and deduce the properties of the limiting distribution of the original process using the distributional inequality. The main benefit of the stepwise approach is the analytical convenience the new process provides.

3.1 Renormalization and Coupling Argument

Suppose that N is even.[3] Let $\Lambda(M)$ be a torus where $M \equiv N/2$, and assign to each $y \in \Lambda(M)$ a set of four players from the original torus $\Lambda(N)$ by

$$H(y) = \left\{ x \in \Lambda(N) : x_i \in \{2y_i, 2y_i + 1\}, i = 1, 2, y \in \Lambda(M) \right\}. \tag{9}$$

We call y and $x \in H(y)$ a team and a team member, respectively. We shall also refer to the population of teams as the renormalized population. Let $z_t : \Lambda(M) \longrightarrow \{A, B\}$ be a map which represents the state of team y at time t, which will be defined shortly. As before, we characterize the dynamics of the population in terms of the set of teams playing A, thus $Z_t^\varepsilon = \{y : z_t(y) = A\}$. We construct an initial configuration Z_0^ε, and a transition rule such that that if the state of a team is A, then all of its team members play A in the original population.[4]

[2] This is called the coupling technique. See Aldous and Fill (1999, Chapter 14).

[3] We discuss later what happens if N is odd.

[4] On the other hand we do not require if a team plays B, all members play B.

First, we define the *initial state* for the renormalized process. At time zero, a team is said to play A if all of its members in the original population play A; otherwise it plays B. Formally, Z_0^ε is defined by

$$Z_0^\varepsilon = \{y : z_0(y) = A\}. \tag{10}$$

where

$$z_0(y) = \begin{cases} A & \text{if} \quad s_0(x) = A \quad \forall x \in H(y) \\ B & \text{otherwise.} \end{cases} \tag{11}$$

Note that

$$\{x \in H(y) : y \in Z_0\} \subset S_0. \tag{12}$$

Thus, the set of agents playing A estimated with the teams in the initial period is a subset of the set of agents playing A in the original population.

First, we construct the transition rule for the renormalized process *without mutation* such that the condition $\{x \in H(y) : y \in Z_t\} \subset S_t$ (if a team plays A, all of its members in the original population play A) satisfied for all $t \geq 0$. Observe that if all members of a team play A in the original population, then the best response for all team members is A. Therefore, if a team plays A, it will never adopt B in the absence of mutation. Moreover, let

$$G_i(y) = \{y - e_i, y + e_i\}, \quad \text{for } i = 1, 2 \qquad \text{where} \quad e_1 = (1, 0) \quad e_2 = (0, 1).$$

That is, $G_i(y)$ represents the two neighbors of team y in coordinate direction i. It is easy to see that if a team playing B encounters two other teams all of whose members are playing A in two different coordinate directions, then all members of the team adopts A in at most 3 periods under the original process (see Figure 1 where the reference to the time indicates the period when the agent adopts A).

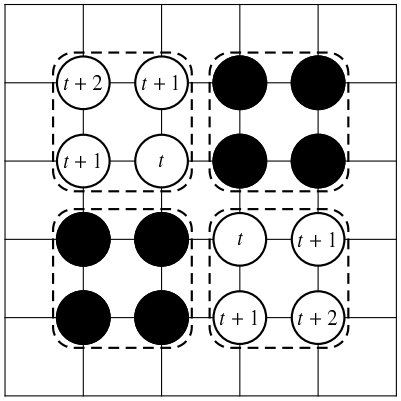
For future reference we summarize the transition rule for the renormalized process in the absence of mutation by the following lemma.

Lemma 1 (Contagion) *In the absence of mutations*

1. *if a team y plays A, it never adopts B,*
2. *if a team y has at least one member playing B, all of its members adopt A if* $G_i(y) \cap Z_t^\varepsilon \neq \emptyset$ *for* $i = 1, 2$.

Second, we construct a random variable representing *mutations* for the renormalized population using the random variable representing mutations for the original population. We do it in

Figure 1: Propagation mechanism for the teams with no mutation



● agents playing A in period t

two steps. Let

$$\xi_t(x) = \begin{cases} A & \text{if } \lambda_t(x) \leq \varepsilon^{\alpha(A)} \\ B & \text{if } \lambda_t(x) \geq 1 - \varepsilon^{\alpha(B)} \\ 0 & \text{otherwise.} \end{cases} \tag{13}$$

Our condition ensures that the event $\{\xi_t(x) = A\}$ implies $\{X_t(x) = A\}$, and $\{X_t(x) = B\}$ implies $\{\xi_t(x) = B\}$. Next, let $Y_t(y)$ be a random variable with values

$$Y_t(y) = \begin{cases} A & \text{if } \xi_{3t}(x) = A \ \forall \ x \in H(y) \\ 0 & \text{if } \xi_n(x) = 0 \ \forall \ x \in H(y) \text{ and } n = 3t-2, 3t-1, 3t \\ B & \text{otherwise} \end{cases} \tag{14}$$

In words $\{Y_t(y) = A\}$ is the event in which all team members mutate to A in period $3t$, $\{Y_t(y) = 0\}$ is the event that no mutation occurred during the period $3t-2, 3t-1, 3t$. Finally, $\{Y_t(y) = B\}$ is the event that at least one team mutated to B during the period $3t-2, 3t-1, 3t$, and all team members do not mutate to A in period $3t$.

There are two important facts about $Y_t(x)$. First, the construction of $Y_t(x)$ and Lemma 1 ensure that the stochastic process for the renormalized population is a Markov process. Secondly, the timing for the renormalized population is different from that of the original population. As we can see on Figure 1, it takes three periods under the original process that all team members adopt A. Therefore the clock for the renormalized population ticks slower, i.e. three periods under the original process correspond to one period under the renormalized one. This is reflected

by the timing convention used in the definition of the random variable $Y_t(x)$.

We can use Assumption 1 to provide bounds on the events that a team mutated to A or to B. Setting $\bar{\alpha}(A) = 4\alpha(A)$, we obtain

$$\Pr(Y_t(y) = A) = \varepsilon^{\bar{\alpha}(A)}. \tag{15a}$$

Moreover, a team mutates to B with probability at most $12\varepsilon^{\alpha(B)}$. For small ε there is a $\bar{\alpha}(B)$ such that this probability is at most $\varepsilon^{\bar{\alpha}(B)}$, i.e.

$$\Pr(Y_t(y) = B) \leq \varepsilon^{\bar{\alpha}(B)} \tag{15b}$$

Finally, after the construction of the transition rule without mutations, and defining the random variable representing mutations for the teams, we can construct the *transition rule* for the renormalized process with mutation

$$\begin{aligned} Z^{\varepsilon}_{t+1} = \Psi(Z^{\varepsilon}_t) \equiv & \left\{ y \notin Z^{\varepsilon}_t : Y_{t+1}(y) = 0, G_i(y) \cap Z^{\varepsilon}_t \neq \varnothing, Y_{t+1}(u) = 0, u \in G_i(y)\ i = 1, 2 \right\} \\ & \cup \left\{ y \in Z^{\varepsilon}_t : Y_{t+1}(y) = 0 \right\} \cup \left\{ y \in \Lambda(M) : Y_{t+1}(y) = A \right\}. \end{aligned} \tag{16}$$

Equation (16) can be decomposed to three transition mechanisms, contagion, cohesion, and mutation. *Contagion* is represented by the first bracket. If team y is playing B at time t, and meets two other teams playing A in two different coordinate directions, it chooses A at time $t + 1$. This transition is called contagion. Since we require that neither team y nor any of the two other teams mutate at time $t + 1$, the construction in (14) implies that no members of any of these three teams mutated in the original process at $3t + 1, 3t + 2, 3(t + 1)$. Therefore, all members of team y make a transition from B to A during $3t+1, 3t+2, 3(t+1)$. Consequently, all players in these three teams plays A at time $3(t + 1)$ in the original process. *Cohesion* captured by the second bracket. It indicates that a team playing A continues with the same choice in the absence of mutation. Since $Y_{t+1}(y) = 0$ implies that no member of team y mutates during $3t + 1, 3t + 2, 3(t + 1)$, all members of team y play A at time $3(t + 1)$ in the original process. *Mutation* is represented by the last bracket. Again, the construction of the mutation for the teams ensures that if team y mutates to A at time $t + 1$, so does all of the team members at time $3(t + 1)$ in the original process.

All three mechanisms are inherited from the original population, renormalization makes cohesion explicit and simplifies the way contagion works. The above decomposition also highlights the potential advantage of the renormalized process over the original one. Transition

from B to A can take place both in the presence and the absence of mutation in the original process. In contrast, transition from B to A takes place only as a consequence of mutation in the renormalized process. This simplifies the derivation of the limit distribution of the renormalized process.

It follows from our discussion above that our contraction ensures the desired relationship between the original and renormalized process as stated in the next proposition.

Proposition 1 *The process* $\{Z_t^\varepsilon\}_{t\geq 0}$ *governed by the transition rule* (16) *and starting from the initial condition* (10) *satisfies*

$$\{x \in H(y) : y \in Z_t^\varepsilon\} \subset S_{3t}^\varepsilon. \tag{17}$$

for all t.

Proposition 1 in turn implies an important consequence for our analysis captured by the following statement.

Proposition 2 *If* $\lim_{\varepsilon\to 0} \nu_\varepsilon(\vec{A}) = 1$, *then* $\lim_{\varepsilon\to 0} \mu_\varepsilon(\vec{A}) = 1$ *where* $\nu_\varepsilon(\vec{A})$ *is the long run probability of the event where all teams play A.*

Proof. It suffices to prove that μ_ε dominates ν_ε, i.e. $\nu_\varepsilon(\vec{A}) \leq \mu_\varepsilon(\vec{A})$. This follows from equation (17), i.e. the event that $Z_\infty^\varepsilon = \Lambda(M)$ implies the event that $S_\infty^\varepsilon = \Lambda(N)$. □

3.2 Contagion versus State Dependent Mutation

This subsection presents the main result of the paper. Our main goal is to demonstrate that for any state dependent mutation satisfying the conditions in Assumption 1, contagion dominates mutation: given a mutation structure, the risk dominant equilibrium will be uniquely selected in the long run for a large but finite population.

The proof is built around two important concepts.

Definition 1 (Lock-in) *A state Z is called a lock-in if no team changes strategy in the absence of mutation. The set of these states is denoted by* $\mathcal{L}$.

If strategy A does not spread further through contagion, then the state is a lock-in.[5] The next concept is new.

[5]This state is also called limit sets of the mutationless process in the literature.

Definition 2 (k-restricted lock-in) *Draw an $M \times M$ square representation of the torus. Denote the "left-upper" $k \times k$ square by $R(k)$. Define the restriction of Z to $R(k)$ to be a state where all teams inside $R(k)$ play the strategy according to Z while all teams outside $R(k)$ play B. If the restriction of Z to $R(k)$ is a lock-in, then Z is called a k-restricted lock-in. The set of k-restricted lock-ins is denoted by $\mathcal{L}_k$.*

Put differently, we treat all teams outside $R(k)$ as if they play B. If no team ever adopts A inside $R(k)$ due to contagion, then $Z \in \mathcal{L}_k$. The definition describes the set of states where the configuration outside $R(k)$ does not generate contagion,[6] i.e. there are no contagious effects coming from outside $R(k)$. It is easy to see that a k-restricted lock-in is also an l-restricted lock-in for all $l < k$. Although a restricted lock-in is not necessarily a lock-in, any lock-in Z is a k-restricted lock-in for all $k < M$.

We start by proving a prepartory lemma. Let $\mathcal{L}_k^A$ be the set of k-restricted lock-ins with all teams in $R(k)$ playing A. Moreover, let $\mathcal{Z}_k$ be the set of configurations where at least k teams in $R(k)$ play B.

Lemma 2 *If $Z \in \mathcal{L} - \vec{A}$, then $Z \in \mathcal{Z}_M$, i.e. it contains at least M teams playing B. If $Z \in \mathcal{L}_k - \mathcal{L}_k^A$, then $Z \in \mathcal{Z}_k$, i.e. it contains at least k teams playing B.*

Proof. Consider the statement for lock-ins. Observe that it is sufficient for the whole population to adopt A in the absence of mutation if at least M teams play A at $t = 0$ and these teams are located on a diagonal. It is easy to see that all teams next to the diagonal have two neighbors in two different coordinate direction playing A. Therefore these teams adopt A by lemma 1. Applying induction, we obtain that all teams adopt A at $t = M - 1$.

Next, suppose that at most $M - 1$ teams play B initially. To see that such a configuration cannot be a lock-in, note that a torus of size $M \times M$ can be viewed as an object consisting of M paralel diagonals of length M. If at most $M - 1$ teams play B at $t = 0$, there must be at least one diagonal on the torus on which all teams play A. Hence our previous argument implies that the the whole population adopts A which proves the first part of our lemma.

Now consider the statement for k-restricted lock-ins. Since a k-restricted lock-in is not a torus but a square which cannot be viewed as an object consisting of paralel diagonals, the previous argument cannot be applied directly. However, a redefinition of the concept "diagonal" for squares solves our problem. View $R(k)$ as consisting of an upper and a lower triangle plus the main diagonal. Pick up a diagonal of length n_1 from the upper triangle and the corresponding

[6] To ensure that the teams at the boundary of the $M \times M$ square have a regular neighborhood structure, we assume that there is an $(M + 1)$st row and column of teams all playing B for all $t \geq 0$.

diagonal of length $n_2 = k - n_1$ from the lower triangle. Let a "diagonal" be defined by these two pieces. The $k-1$ such a "diagonal" plus the main diagonal gives us k "diagonals". Elementary argument shows that if any of the construced diagonals contains k teams playing A, all teams in $R(k)$ eventually adopt A in the absence of mutation. Therefore our previous argument applies proving the second part of the lemma. □

These two results are a very when we need to know the minumum number of mutations required to leave the basin of attraction of $\vec{A}$ or a k-restricted lock-in with all teams in $R(k)$ playing A.

After this preparaton, we shall characterize the limit distribution of the process $\{Z_t^\varepsilon\}$ using a result on first passage times. First, we introduce some notation. Let Z and Z' be two states of $\{Z_t^\varepsilon\}$ and $T_Z = \inf\{t > 0 : Z_t^\varepsilon = Z\}$ be the first passage time to state Z. Let

$$\Pr_Z(Z_t^\varepsilon = Z') \equiv \Pr(Z_t^\varepsilon = Z' | Z_0^\varepsilon = Z).$$

and

$$\Pr_{\mathcal{Z}}(E) = \inf_{Z \in \mathcal{Z}} \Pr_Z(E)$$

for an event E and $\mathcal{Z} \subset \Lambda(M)$. We use the following well-known identity for ergodic Markov-processes [see Durrett (1996, Chapter 5)].

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\vec{A})} = \frac{\Pr_{\vec{A}}\left(T_Z < T_{\vec{A}}\right)}{\Pr_Z\left(T_{\vec{A}} < T_Z\right)} \tag{18}$$

We estimate the numerator and denominator separately. The key is to estimate the latter.

Now we turn to the proof for the main theorem which is constructed via three lemmas. Although they are somewhat technical, we believe that there is a clear intuition behind each of these lemmas. The first lemma establishes the order of the probability of getting from one lock-in to another one which has less teams playing A.

Lemma 3 *Let Z and Z' be two lock-ins with $Z \not\subseteq Z'$. The probability of getting from lock-in Z to lock-in Z' without hitting any other lock-in is given by*

$$\Pr_Z(T_{Z'} < T_{\mathcal{L}-Z'}) = O(\varepsilon^{\tilde{\alpha}(A)|Z \setminus Z'|}). \tag{19}$$

Proof. We estimate the following sum

$$\Pr_Z(T_{Z'} < T_{\mathcal{L}-Z'}) = \sum_{t=1}^{\infty} \Pr_Z(T_{Z'} = t, T_{\mathcal{L}-Z'} > t).$$

First, observe that for any t, the event $\{T_{Z'} = t, T_{\mathcal{L}-Z'} > t\}$ requires that the process leaves Z in the first period since $Z \in \mathcal{L} - Z'$. Next, if the process leaves Z, at least one team adopts A in every period due to contagion until the next lock-in is reached. Since the process may hit $\vec{A}$ at time $t = M^2$, we need at least $\max\{t - M^2, 0\}$ mutations to B not to hit a lock-in by time t. Moreover, since $Z \not\subseteq Z'$ and transition from A to B is not possible without mutation, we need at least $|Z \backslash Z'|$ more mutations to B to reach Z'. Thus, reaching Z' by time t without hitting any other lock-in along a given trajectory has probability at most $\varepsilon^{\bar{\alpha}(B)(|Z|-|Z'|+\max\{t-M^2,0\})}$. We have to multiply this value with the number of possible trajectories. Since each trajectory is t long, choosing a sufficiently large K depending only on M, we can estimate this number by K^t.[7] For ε sufficiently small, the sum in the inequality

$$\Pr_Z(T_{Z'} < T_{\mathcal{L}-Z'}) \leq \varepsilon^{\bar{\alpha}(B)(|Z\backslash Z'|)} \sum_{t=1}^{\infty} K^t \varepsilon^{\bar{\alpha}(B)\max\{t-M^2,0\}}.$$

is convergent. Indeed the sum is $O(1)$ which proves our claim. □

Note that any configuration with less then M teams playing B (other then $\vec{A}$) is not a lock-in by Lemma 2. Therefore this lemma also implies that if the process starts from $\vec{A}$, the probability that it hits any configuration with at least M teams playing B before getting back to $\vec{A}$ is bounded from above by $K\varepsilon^{\bar{\alpha}(A)M}$.

The next lemma formulates a "local" version of this observation. This local version will make it possible to track the evolution of the process more closely and in the end prove our main claim.

Lemma 4 *We have that*

$$\Pr_{\mathcal{L}_k^A}\left(T_{\mathcal{Z}_k} < T_{\mathcal{L}_k^A}\right) \leq K\varepsilon^{\bar{\alpha}(B)k} \tag{20}$$

for some $K > 0$ independent of ε.

Proof. The proof is based on the construction of a shadow process $Z_t^{k,\varepsilon}$, which is intended to keep track of only those events taking place inside of $R(k)$. We prove the claim for the shadow process making use of the previous lemma and then we transform the result to Z_t^{ε} by using coupling argument.

First we construct the shadow process as follows. At the initial state, teams inside $R(k)$ in $Z_0^{k,\varepsilon}$ play the same strategy they play in Z_0^{ε}. However, teams outside $R(k)$ in $Z_0^{k,\varepsilon}$ play B. This is to ensure that we focus only on the "local problem", i.e. what happens inside $R(k)$. We define

[7] Different K's appearing in different formulas may be different.

the transition rule as follows. Teams inside $R(k)$ in $Z_t^{k,\varepsilon}$ mutate to a given strategy if they do so inside $R(k)$ in Z_t^{ε}. Contagion within $R(k)$ in $Z_t^{k,\varepsilon}$ takes place according to the standard rule for teams. Finally, teams outside $R(k)$ in $Z_t^{k,\varepsilon}$ play B forever.

It is easy to see that $Z_t^{k,\varepsilon} \subset Z_t^{\varepsilon}$ for all $t \geq 0$. It is true for $t = 0$ by construction. Since no team ever plays A outside $R(k)$ in $Z_t^{k,\varepsilon}$, we only have to show that our claim is true inside $R(k)$. First, mutation generates teams playing A under $Z_t^{k,\varepsilon}$ whenever it does $R(k)$ under Z_t^{ε}. Secondly, since contagion is induced only by A and teams outside $R(k)$ play B forever, teams outside $R(k)$ have no contagious effects on teams inside $R(k)$ in $Z_t^{k,\varepsilon}$. Since this is not true for Z_t^{ε}, we have $Z_t^{k,\varepsilon} \subset Z_t^{\varepsilon}$.

Consider now the shadow process $Z_t^{k,\varepsilon}$. Lemma 2 and 3 imply for the shadow process that the probability of getting to $\mathcal{Z}_k$ before returning to $\mathcal{L}_k^A$ is bounded from above by $K\varepsilon^{\bar{\alpha}(B)k}$.[8] Condition $Z_t^{k,\varepsilon} \subset Z_t^{\varepsilon}$ implies that if Z_t^{ε} hits $\mathcal{Z}_k$ so does $Z_t^{k,\varepsilon}$, and conversely if $Z_t^{k,\varepsilon}$ returns to $\mathcal{L}_k^A$ so does Z_t^{ε}. Thus if the required inequality holds for $Z_t^{k,\varepsilon}$ it also holds for Z_t^{ε}. The lemma is proved. □

The next lemma builds on the previous two lemmas to provide the key to the proof of our main claim.

Lemma 5 *Let*

$$m \equiv \left\lceil \frac{\bar{\alpha}(A)}{\bar{\alpha}(B)} \right\rceil. \tag{21}$$

where $\lceil . \rceil$ *is the smallest integer greater than the term inside. For all* $M > m$

$$\frac{1}{\Pr_Z\left(T_{\vec{A}} < T_Z\right)} = O(\varepsilon^{\bar{\alpha}(A)m}) \tag{22}$$

for any $Z \in \mathcal{L} - \vec{A}$.

Proof. We construct an event which is a subset of the event $\{T_{\vec{A}} < T_Z\}$, and estimate its probability from below. The key step of the proof is to show that once all teams in $R(k)$ play A with $k \geq m$, there is a positive probability independent of ε, that all teams in $R(k+1)$ will play A before returning to Z. Thus m plays the role of a critical mass; once a sufficiently large set of teams (measured by m) is conquered by strategy A, it is easy to proceed. So the difficulty lies in reaching this critical mass of m, and hence the bound $\varepsilon^{\bar{\alpha}(A)m}$.

Since $Z \in \mathcal{L} - \vec{A}$, it follows from lemma 2 that it contains at least M teams playing B. Draw a $M \times M$ square representation of the torus such that the uppermost and leftmost team plays B in

[8] Note that from the point of view of the shadow process the set $\mathcal{L}_k^A$ could be considered to be a singleton since teams outside $R(k)$ do not count anyway.

Z. Denote the "left upper" $k \times k$ square in this $M \times M$ torus by $R(k)$, and the diagonal elements by $D(k)$. Observe that this construction ensures that $Z \in \mathcal{L}_k - \mathcal{L}_k^A$ for all $k = m, \ldots, M$ which implies by lemma 2 that $Z \in \mathcal{Z}_k$ for all $k = m, \ldots, M$.

To estimate $\Pr_Z\left(T_{\vec{A}} < T_Z\right)$ from below, consider the following inequality:

$$\Pr_Z\left(T_{\vec{A}} < T_Z\right) \geq \Pr_Z\left(T_{\mathcal{L}_m^A} < T_Z\right) \prod_{k=m}^{M-1} \Pr_{\mathcal{L}_k^A}\left(T_{\mathcal{L}_{k+1}^A} < T_{\mathcal{Z}_k}\right). \tag{23}$$

To see why this inequality holds, first note that $\mathcal{L}_M^A = \vec{A}$. Next observe that the first term is the probability that the process visits an m-restricted lock-in where all teams in $R(m)$ play A before getting back to Z. The second term is the product of probabilities. Each term in this product measures the probability that the process passes from a k-restricted lock-in to a $k+1$-restricted lock-in without hitting $\mathcal{Z}_k$. Along such a path the process does not hit Z because $Z \in \mathcal{Z}_k$ for all $k = m, \ldots, M$. Since this chain of events is sufficient but not necessary for $\left\{T_{\vec{A}} < T_Z\right\}$ to occur, its probability is a lower bound on $\Pr_Z\left(T_{\vec{A}} < T_Z\right)$. Thus the inequality indeed holds.

We now turn to estimate each term on the right hand side of the above inequality separately. We shall complete this estimation in two steps.

Step 1. Suppose that at least the teams in $D(m)$ mutate to A in the initial period and no mutations to B take place during the next m periods. Lemma 1 implies that all teams in $R(m)$ play A after at most m periods, and we have not returned to Z. Thus

$$\Pr_Z\left(T_{\mathcal{L}_m^A} < T_Z\right) \geq K_1 \varepsilon^{\bar{\alpha}(A)m} \tag{24}$$

for some $K_1 > 0$ and ε small.

Step 2. We prove now that the probability of getting from $\mathcal{L}_k^A$ to $\mathcal{L}_{k+1}^A$ without hitting $\mathcal{Z}_k$ is bounded from below by a constant independently of ε. To find this bound we recycle our construction of the shadow process from the previous lemma to construct an event which is a subset of $\left\{T_{\mathcal{L}_{k+1}^A} < T_{\bar{\mathcal{L}}_k^A}\right\}$.

Let $\tilde{T}_{\mathcal{Z}}^i$ be the ith hitting times of the shadow process $Z_t^{k,\varepsilon}$ on some subset $\mathcal{Z}$ of the $M \times M$

representation of the torus. Consider the following inequality:

$$\Pr_{\mathcal{L}_k^A}\left(T_{\mathcal{L}_{k+1}^A} < T_{\mathcal{Z}_k}\right) \geq \sum_{i=1}^{\infty} \Pr_{\mathcal{L}_k^A}\left(\tilde{T}^i_{\mathcal{L}_k^A} < \tilde{T}_{\mathcal{Z}_k}\right)$$
$$\times \Pr\left(\begin{array}{c}\text{The team in } D(k+1)/D(k) \text{ does not mutate to } A \text{ in } Z_t^\varepsilon \text{ for the first} \\ (i-1) \text{ times when } Z_t^{k,\varepsilon} \text{ hits } \mathcal{L}_k^A, \text{ but it does at the } i \text{ times.}\end{array}\right)$$
$$\times \Pr\Big(\text{No mutation to } B \text{ occurs in } \Lambda(M) \text{ for } k+1 \text{ periods}\Big).$$

The right hand side captures the following event. The shadow process starting from $\mathcal{L}_k^A$ returns to $\mathcal{L}_k^A$ several times without hitting $\mathcal{Z}_k$. One of these times the next team along the diagonal mutates to A. Finally, no mutation takes place for the next $k+1$ periods after that. We can write the probability of this joint event in a product form because of the Markov property and because the shadow process is independent of what mutations take place outside $R(k)$ in Z_t^ε.

If that this joint event happens, then all teams in $R(k)$ play A in Z_t^ε and Z_t^ε did not hit $\mathcal{Z}_k$ on the way because $Z_t^{k,\varepsilon} \subset Z_t^\varepsilon$ by construction. Since the next team along the diagonal mutated to A, and no mutations happened afterwards for $k+1$ periods, contagion drove all teams in $R(k+1)$ to play A in the end. Therefore the probability of this joint event is indeed a lower bound for the right-hand side.

We estimate each terms on the right hand side separately. The strong Markov property of the stopping times $\tilde{T}^i_{\mathcal{L}_k^A}$, $i = 1, 2, \ldots$ and Lemma 4 imply for the *first* event on the right hand side that

$$\Pr_{\mathcal{L}_k^A}\left(\tilde{T}^i_{\mathcal{L}_k^A} < \tilde{T}_{\mathcal{Z}_k}\right) \geq \left(1 - K\varepsilon^{\bar{\alpha}(B)k}\right)^i \geq \left(1 - K\varepsilon^{\bar{\alpha}(A)}\right)^i,$$

where we used the fact that $\bar{\alpha}(A) \leq \bar{\alpha}(B)k$ for all $k \geq m$ by construction. The the *second* term equals to $\left(1 - \varepsilon^{\bar{\alpha}(A)}\right)^{i-1} \varepsilon^{\bar{\alpha}(A)}$. Finally, the probability of the *third* term, i.e. no team mutates to B for $k+1$ periods is bounded from below by $\left(1 - \varepsilon^{\bar{\alpha}(B)}\right)^{M^2(k+1)}$.

Putting these three pieces together yields

$$\begin{aligned}\Pr_{\mathcal{L}_k^A}\left(T_{\mathcal{L}_{k+1}^A} < T_{\mathcal{Z}_k}\right) &\geq \sum_{i=1}^{\infty} \left(1 - K\varepsilon^{\bar{\alpha}(A)}\right)^i \left(1 - \varepsilon^{\bar{\alpha}(A)}\right)^{i-1} \varepsilon^{\bar{\alpha}(A)} \left(1 - \varepsilon^{\bar{\alpha}(B)}\right)^{M^2(k+1)} \\ &= \varepsilon^{\bar{\alpha}(A)}(1 - \varepsilon^{\bar{\alpha}(B)})^{M^2(k+1)} \frac{1 - K\varepsilon^{\bar{\alpha}(A)}}{K\varepsilon^{\bar{\alpha}(A)} + \varepsilon^{\bar{\alpha}(A)} - K\varepsilon^{2\bar{\alpha}(A)}} \\ &\geq \varepsilon^{\bar{\alpha}(A)}(1 - \varepsilon^{\bar{\alpha}(B)})^{M^2(k+1)} \frac{1 - K\varepsilon^{\bar{\alpha}(A)}}{(1+K)\varepsilon^{\bar{\alpha}(A)}} \geq K_2. \end{aligned} \tag{25}$$

for some $K_2 > 0$ independent of ε. The second step is complete.

Putting the results from equation (24) and (25) together proves the lemma. □

The heart of the previous lemma is step 2 where we assess the outcome of a "race": the next diagonal team to mutates to A before too many teams at wrong locations inside $R(k)$ mutate to B. The probability of the event that the next diagonal team mutates to A at each occasion when all teams play A inside $R(k)$ is $\varepsilon^{\tilde{\alpha}(A)}$. The probability of the event that the process hits a k-restricted lock-in with positive number of teams playing B in $R(k)$ is at most $K\varepsilon^{\tilde{\alpha}(B)k}$. The fact that the ratio of these two probabilities is bounded from below by a constant for $k \geq m$ implies that the set of teams playing A has a strong growth property for small ε. This is due to two factors. One is cohesion: once a large group of teams play A, it is very hard to destroy them. Secondly, contagion ensures that only one additional team is required for the large group playing A to grow beyond its boundaries. Intuitively, m acts as a "critical mass" in the argument; once the teams in $R(m)$ play A, the critical mass has been reached and it is easy to proceed.

The next theorem states our main result.

Theorem 1 *For $M \geq m$*

$$\lim_{\varepsilon \to 0} \nu_\varepsilon(\vec{A}) = 1,$$

where m is defined in (21)*.*

Proof. We prove our claim in two steps. The first step focuses on $Z \in \mathcal{L} - \vec{A}$ whereas the second step considers $Z \notin \mathcal{L}$.

Step 1. Consider the states $Z \in \mathcal{L} - \vec{A}$. We have that

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\vec{A})} = \frac{P_A(T_Z < T_A)}{P_Z(T_A < T_Z)}.$$

The previous lemma implies

$$\frac{1}{P_Z(T_A < T_Z)} = O\left(\varepsilon^{-\tilde{\alpha}(A)m}\right)$$

for $M \geq m$.

Since Z is a lock-in and $Z \neq \vec{A}$, Z has at least M teams playing B. It follows from Lemma 3 that

$$P_A(T_Z < T_A) = O\left(\varepsilon^{\tilde{\alpha}(B)M}\right).$$

Indeed, starting from $\vec{A}$, the first lock-in on the way requires at least M mutations to B. Putting these together yields

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\vec{A})} = O\left(\varepsilon^{\tilde{\alpha}(B)M - \tilde{\alpha}(A)m}\right).$$

In particular, for $M \geq m$,

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\vec{A})} \to 0$$

as $\varepsilon \to 0$.

Step 2. Consider the states $Z \notin \mathcal{L}$. Let $\hat{Z}$ be the state to which Z converges in the absence of mutations. Then

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\hat{Z})} = \frac{P_{\hat{Z}}(T_Z < T_{\hat{Z}})}{P_Z(T_{\hat{Z}} < T_Z)}.$$

The denominator is of order 1, because if no mutation occurs for the next M^2 periods (which happens with probability approaching 1) then we hit $\hat{Z}$ without hitting Z for sure. The numerator goes to zero as $\varepsilon \to 0$ because of our Assumption 1. Indeed, a mutation is required in the first step, otherwise we would stay at $\hat{Z}$. The aforementioned condition guarantees that all mutation probabilities tend to zero.

Therefore

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\hat{Z})} \to 0$$

as $\varepsilon \to 0$. This holds true for $\hat{Z} = \vec{A}$ as well as other lock-ins. All in all we have that

$$\frac{\nu_\varepsilon(Z)}{\nu_\varepsilon(\vec{A})} \to 0$$

for any Z such that $Z \neq \vec{A}$. The proof is complete. □

Putting together this result and Proposition 2 yields that for $N \geq 2M$ even, the only long-run stochastically stable set of the original $\{S_t^\varepsilon\}_{t\geq 0}$ process is $\vec{A}$.

It remains to consider the situation when N is odd. Let $N = 2M + 1$. Consider a a $2M \times 2M$ rectangle on the torus. Note that throughout the proofs so far we have not used the fact that the environment is a torus, and not an $M \times M$ square. Thus the theorem is applicable for the $2M \times 2M$ square on the torus. Note that if all teams in the $2M \times 2M$ square are playing A, then contagion in the absence of mutation implies that the $(2M + 1)$st column and row will also play A in a finite time period. Since mutation probabilities go to zero, we can conclude that $\vec{A}$ is the only long-run stochastically stable set of the game for $N = 2M + 1$ too. Thus the following corollary holds.

Corollary 1 *For $N \geq 2m$ we have*

$$\lim_{\varepsilon \to 0} \mu_\varepsilon(\vec{A}) = 1$$

where m is defined in (21)*, and μ_ε is the unique invariant distribution of S_t^ε.*

4 Conclusion

In contrast to the crucial role of the state dependent mutation in the equilibrium selection for global interaction games, the detail of mutation structure does not matter much for the local interaction games. The result strengthens the equilibrium selection result of the evolutionary game theory. Moreover it justifies the approach which deliberately avoids a detailed formulation of the decision making process. Finally considering the fact that the situation captured by the approach is mainly social phenomena involving many loosely related agents, the local interaction framework could be a better description than the global interaction framework.

Our result can be regarded as a generalization of Ellison's (2000) step-by-step evolution approach to identify the long run stochastically stable set in a local interaction setting. His approach relies on the concepts of radius and modified coradius to characterize the evolution toward the limit set. The radius measures how easy to leave the basin of attraction of the limit set. The modified coradius measures how difficult to get into the basin of attraction of the limit set. In particular, computing the modified coradius involves to count the number of mutations required for the process to leave the basin of attractions of intermediate limit sets (lock-ins in our terminology). The modified coradius captures the idea that what matters on the long run is how the process gets from one lock-in to the next before reaching the the long run stochastically stable set of a model. However, it is not easy to extend the radius-modified coradius argument to the case of state dependent mutations. The complications comes from the fact that if mutations are state dependent, then it matters through which type of mutations the process leaves the basin of attraction of an intermediate limit set. It is much more complicated to estimate the coradius of an intermediate limit set unless the limit set has a regular shape, for example, it is a rectangle. Therefore, we decided to construct a direct proof of our claim.

Our proof builds on the idea that we can characterize the evolution by looking at how the process passes through configurations which are *locally* regular, i.e. they contain a connected island, a square of players playing A. These are our restricted lock-ins. We show that we can abstract away how the configuration looks outside such a square, and that we can focus on how the process evolves step-by-step from one square of players playing A to the next larger square until the whole population plays A.

The argument for our main result can be extended to a more general setting with a higher dimension, a larger interaction range and many strategies. Using results from Blume (1995), one can construct stable teams for more *general interaction ranges*. Since contagion also works in a similar fashion as in our case, the argument of our proof applicable. Furthermore, *higher*

dimensional environment also allows the construction of teams, and the arguments of Schonmann (1992) imply that the contagion mechanism works in the same way, so our proof can be applied. Finally, if we consider *many strategies*, but there is a unique strategy A such that it is best response to any configuration where at least half of the opponents play A, then stable teams can be constructed, and our proof can be applied.

We can also draw some conclusion about the relationship between mutation and contagion in a more *general local interaction environment* than the torus; see Morris (2000) and Young (1998, Chapter 6). The existence of cohesive groups and the contagion mechanism is essential for our argument to work. If both cohesion and contagion favors strategy A, the intuition behind our result implies that the number of mutation required to eliminate a large group playing A will depend on the size of the group. In contrast, to propagate strategy A through contagion requires few strategies independent of the population size. This asymmetry would ensure that results on state-dependent mutation similar to ours can be obtained in a more general spatial environment.

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