Symmetry and collective fluctuations in evolutionary games

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We review stochastic evolutionary game theory from a perspective that starts in the very general regression methods of population genetics, and introduces games as a powerful yet flexible framework to unpack the "black box" of development, which generates frequency-dependent fitness maps and which may also structure the transmission process. This approach to evolutionary game theory retains the robustness of regression methods while attempting to progressively refine mechanistic explanations. The tautological nature of normal-form games, and their refinement with the extensive form, are compared as characterizations of mechanism. We develop *symmetry* as the core concept to classify evolutionary game models, and *symmetry breaking* as the source of robust multiscale dynamics. Stochastic population processes generally produce regression models that depend on scale, and the change of parameters at different scales, as well as the emergence of hierarchical units of selection, is due to collective fluctuations. The current paper surveys aspects of symmetry in games, emphasizing qualitative features. A companion paper develops large-deviation methods for multiscale dynamics in evolutionary game theory, and presents quantitative solutions for some of the scale-dependent models in this paper.

I. INTRODUCTION

A review emphasizing the diversity of mechanisms together with robust model selection and interpretation

Evolutionary game theory [1–12] is in principle a rich synthesis of concepts and tools. It can draw on all the methods to treat assortation, replication, transmission, and selection formalized within modern population genetics [13–19], and on the full range of descriptions of structured individual and group interactions from game theory [20–22], including the extensive form [23] and cooperative solution concepts [24].

In a pair of papers we present an overview of evolutionary games that we hope will complement the existing comprehensive reviews of applications and model classes [4, 7, 8, 23], and the more formal treatments from the probability literature emphasizing convergence [6, 25, 26]. Our perspective derives from the problem of robust model selection and analysis, but rather than attempting to address domain-specific validation of models, we will limit our discussion to what can be learned about robustness from the requirement of internal mathematical consistency of theories.[1]

We give a specific form to the problem of robustness by taking seriously the disruptive effects of stochasticity on every level of scientific description with evolutionary games: in natural system dynamics, in estimation, in model analysis, and in prediction. Requiring consistency of a fully stochastic analysis restricts the class of properties that robustly identify models, and leads to nontrivial relations among descriptions of the same system at multiple timescales or scales of aggregation. Although these results follow from a merely technical analysis, we believe they ultimately lead to a clearer understanding of the meanings – and limitations of meaning – of the basic entities and interactions of stochastic evolutionary game theory, as similar analyses have led to reconceptualization of the basic entities and interactions in many-body equilibrium systems [27–30].

We wish also to stress the wide scope and richness of mechanistic explanations available from game models, but to organize this diversity in terms of a small number of unifying principles, to make the world of models both easier to navigate, and less *ad hoc* in practical use.[2] For these reasons we adopt an unconventional approach of deriving games entirely as refinements within the empirically grounded regression methods of Fisher's theorem [32], the Price equation [33–35], and quantitative genetics [36]. The union of rich mechanistic explanation with the conservative approach of statistics is not

[1] The approach of arguing for the plausibility of particular models is standard in existing literature, and in any case requires domain-specific knowledge of each instance to be done properly. We intend the current review to provide a different kind of support for model validation and interpretation, which can be expressed in precise terms from mathematical consideration, but is easy for arguments focused on case-by-case plausibility to overlook.

[2] We particularly wish to avoid turning a summary of mechanistic diversity into a "wilderness of models", as has happened to the concept of bounded rationality [31]. We also wish to avoid reinforcing a separation of paradigms that already exists, in which mechanistically explicit, causally "satisfying" game models are reduced to the status of "toys", expected only to describe what might happen in imagined worlds, while empirically grounded regression methods are often limited to low-order (linear or "additive") models, and accepted as being inherently highly mechanistically ambiguous.

often achieved within evolutionary modeling [15, 37] and game theory,[3] because of difficulties of validating complex models on one hand, and difficulties of searching the space of complex regressions on the other. These may be understood as two aspects of the same problem, and can be argued to define the primary need for theory in biology [40].

The need for a more mathematically-defined, and less case-specific grounding for the basic concepts in evolutionary game theory is perhaps most clearly recognized and addressed in the literature on social evolution [41] and the dynamics of economic innovation [42, 43]. It encompasses the always-essential concepts of the invidual who develops and reproduces, the trait subject to selection, and the role of player, move, strategy, or payoff, in the game structure. It also extends, however, to the concept of the gene, which is often fundamental to the structure of the type space and the choice of transmission model, and hence to the validity of formal models of evolution at all. These difficulties have been less emphasized in biological evolutionary models in recent decades, but we believe that they exist in much the same form, and that they are increasingly being recognized.[4]

Using symmetries to classify and understand the major qualitative distinctions among evolutionary games

Our review will emphasize classification of games according to their robust dynamical features, particularly those that link individual and population-level behavior or that bridge multiple timescales. Our examples are chosen to cover many aspects of normal-form and extensive-form games, and to show how the game description connects to fundamental problems in development, multilevel selection, and multiscale dynamics. We will also make contact with important concepts such as repetition and the Folk Theorems from rational choice game theory [22, 55–57], where an explicit mapping can be made between the evolutionary and strategic interpretations.

A categorization of stochastic game models requires criteria which are robust against errors of parameter estimation, fluctuation effects, and changes of resolution or aggregation scale. For these criteria **symmetries** are the tool of choice [58]. Symmetry has long been used to classify dynamical-system representations of evolutionary games obtained within the approximations of the replicator equation [4, 7]. It is an even more fundamental concept in stochastic evolutionary game theory, where the fact that symmetry is a *scale-invariant* property of systems carries implications for the connection of dynamics across even very large ranges of scale, which can be difficult to compute, or even to prove exist, by direct constructive analysis.

As the dynamical counterpart to symmetry, we review several important classes of fluctuation effects in stochastic evolutionary games. We emphasize effects that lead to qualitative or large quantitative differences between individual interactions and the estimates obtained for them from population averages. Understanding why and how model parameters sometimes must change with the scale of observation or aggregation helps to resolve one source of mechanistic ambiguity in regression-based approaches. With more work, an analysis of collective fluctuations can provide a principled approach, based in symmetry and scaling, to identify the robust features of self-consistent ("closed") model classes [27–29].

Introducing games from a starting point in regression modeling of population processes

We introduce stochastic evolutionary game theory (SEGT) by starting from general features of population processes required in evolutionary models, and extracting games from successive refinements in genotype- and frequency-dependent fitness functions. A repeated theme in our presentation will be that, precisely because of their generality, game models often must be ambiguous with respect to mechanism. This permits evolutionary games to furnish valid descriptions for a wide variety of systems, both organismal and social, but in many cases the connotations that surround "play" are largely irrelevant to either the motivation or meaning of the game.

Therefore, rather than considering diverse, case-specific justifications for games, we focus on a small set of concepts – symmetry groups and their representations, scaling, large-deviation asymptotics, and entropy – which we feel make it possible to characterize the formal contents of the theory in terms that are still conceptual but are free of metaphor. In these functional terms, evolutionary game theory may be entirely derived as a general framework to unpack the "black box" of fitness functions in population processes.[5]

[3] For an example in which structured models *are* systematically extracted by refinement of empirical descriptions, see Ref's. [38, 39].

[4] The existence of individuals and genes was simply *presumed* as a primitive given by the nature of organisms, in the wake of the modern synthesis [44], much as players and motives have been presumed in much of game theory following von Neumann and Morgenstern [20, 45]. These presumptions have grown increasingly unsatisfactory, with the recognition of multilevel selection [17, 46–48], and precise descriptions of modularity in mechanisms of development, regulation, transmission, and selection [49–51] showing the deficiency of a gene concept based only on non-recombining sequences of DNA. Finally, the literature on conventions in economics [52–54], which uses methods identical to ours, has offered meaningful population-level alternatives to the individual as the unit that learns and remembers preferred strategies.

[5] This characterization is not in conflict with the widespread use

This paper considers *qualitative* aspects of model classification, prediction of dynamical properties from symmetries, and connections to a variety of general results in evolutionary dynamics or game theory, including: Hamilton's rule [35, 48], the emergence of coalitional behavior from non-cooperative interactions [22, 24], a symmetry-based analysis of Lewontin's classic model of the chromosome as a unit of selection [37], the origin of genes in relation to extensive-form games [23] and the status of linkage in genic models, and the interpretation of repeated games [55, 60–64]. In a companion paper, we present a suite of methods [65–71] to *quantitatively* analyze effects of collective fluctuations in stochastic evolutionary game models, including extraction of their large-deviation limits [72–74]. Among the many approaches to large-deviation theory (some of which have been applied to SEGT [2, 6, 52–54, 59, 75]), we choose one that makes the connection of dynamics and units of selection across scales conceptually transparent and technically easy. Many solutions worked out in the companion paper provide quantitative examples of results whose qualitative form is presented here.

A. Summary of the development of topics and the main results

The following list summarizes the topics covered, and the main conceptual points to be illustrated by the examples of the remaining sections.

The Price equation: accounting identities, fitness, and closures in population genetics: We introduce evolutionary game theory as a general framework to classify and interpret fitness models within population genetics. We begin with the *Price equation*, an accounting identity for any processes satisfying the assumptions of population genetics, in which *fitness* universally appears as a summary statistic [35].[6] The problem of replacing accounting with prediction, and of defining *closures* for population-process models,[7] leads to the approach of Fisher [32] and Price [33], who replace fitness (the statistic) with models derived from its regression on individual and population states. The generality of fitness models – which population genetics treats as a "black box" – along with the problem of relating these to structure and dynamics either in ontogeny or across generations, *defines* the role for games in evolutionary game theory. The regression approach of Fisher and Price inherently emphasizes robust estimation and model classification. In the ending discussion, this and all of our topics which follow will be brought back together as the foundation for a robust universality classification [28, 29] of evolutionary games.

Information-incorporation and development are complements in evolutionary dynamics: From the formal equivalence between the replicator equation and Bayes's theorem for updating probability distributions [76], we will regard population genetics as comprising the information-incorporating aspects of replication and selection.[8] Incorporated information is what can be transmitted. Its complement contains the aspects of phenotype constructed through non-heritable interactions within generations, which we will term *development* broadly construed. The complementarity of these functions within evolutionary dynamics clarifies the respective roles of population genetics and games within evolutionary game theory.

The emergence of games as a framework to systematically model development: Starting from the Price equation and a need for closures, a general polynomial expansion of frequency-dependent regression models for fitness is equivalent, at order k, to treating development as a k-player normal-form game. This equivalence is a tautology, meaning that it is both assured and highly ambiguous in terms of mechanism. If we know more about the statistics of matching, we may resolve the normal form into contributions from assortation and the individual-level consequences for fitness that are most naturally called payoffs. If we know more about the internal structure of interactions – which may be temporal sequence, signaling or imitation, or even just linkage – then we may refine the normal form to a particular extensive-form game [23]. In this way games emerge as a highly general, if not all-encompassing, framework to model development.

Symmetry and collective fluctuations in evolutionary games: The relation of games obtained through regression at the population level, to interactions at the individual level, will generally change with resolution or aggregation scale in the presence of stochastic perturbations. This makes not only the metaphor of "play", but in many cases any single interpretive metaphor, in-

of evolutionary game theory as a source of equilibrium refinements [1, 5, 20, 59] or a model for reinforcement learning. The emergence of fitness as a summary statistic, and hence of our characterization in terms of fitness, follows from the defining assumptions of population genetics, which all these applications of evolutionary game theory share.

[6] Economists will appreciate the importance of accounting identities as non-trivial constraints, despite their "tautological" nature: that by construction they apply to all well-formed models in the domain for which they are derived.

[7] "Closure" is used as a general term in economics; in population genetics it normally refers to the more specific problem of *moment closure*, which we will show can be handled in a variety of ways.

[8] As population genetics has developed, it has come to integrate with these, at an equally fundamental level, the constraints on replication and selection following from the structure of transmission.

appropriate to refer to the meaning of a model across its whole domain of scales. We therefore classify fitness models in terms of symmetry groups and their representations by population states. Symmetries are a scale-invariant property of systems, and changes in their representations (known as *symmetry breaking* [29, 77, 78]) imply robust predictions for multiscale dynamics. The stochastic effects that are robust within symmetry classes (and that lead to symmetry breaking) are collective fluctuations [74]. We provide examples of major classes of symmetry groups and categories of symmetry breaking, and show how each implies a distinctive form of scale-dependence in fitness or dynamics. These include the emergence of new units of selection or of coalitional behavior from interactions that are non-cooperative at the scale of individual interactions.

The statistical gene in relation to modularity in development: The particulate heredity of genes is one pillar of the Modern Synthesis [44], and is incorporated as an essential assumption within population genetics. The assumption of particulate heredity drives perhaps the widest wedge between evolutionary models of species and attempts to use evolution to describe social, behavioral, or institutional dynamics [42, 43]. We argue here that particulate heredity in any domain – when it exists – results from modularity in development which is either reinforced by selection or recapitulated in transmission mechanisms. The gene, in other words, is a statistical concept that emerges from modularity in development. Using games as our framework to describe the structure of development, we show how coarse-graining which optimizes compression but also preserves dynamical sufficiency of models,[9] leads to genes that reflect game structure. However, the joint criteria of compression and sufficiency also lead generically to multi-level selection, for reasons that have been understood for decades in the theory of variable-length data-compression coding [79–81].

Placing evolutionary models within game theory: A population-genetic emphasis treats games as the source of fitness functions and perhaps modular architecture, which selection uses to stabilize population states for genes and genomes. If we instead treat selection as a means to stabilize the distribution over paths of play that unfold anew within each generation, we arrive at the view of evolutionary stability (or its natural generalization to stochastic stability [2]) as one among many solution concepts within the larger framework of game theory [5], and as a source of equilibrium refinements [20, 52, 59].[10] The time-reflection symmetry between adaptation and prediction is the basis for the *Folk theorem of evolutionary game theory* [7],[11] which notes that fixed point conditions are the same for evolutionary dynamics and the non-cooperative (Nash) equilibrium of rational-choice game theory, and that the criteria for stability can be mapped between the two approaches.

Repetition in evolutionary and rational-choice game theory: re-directing the forces of selection: The *repeated games* are a restricted subset of the extensive forms, in which a single set of matched players repeatedly play a stage game to determine their final payoffs. These games have become a standard framework, in both evolutionary [8, 11, 83] and rational-choice game theory [22, 55–57], to formalize the notion that "cooperation" is a paradox, and then to propose its solution. In repeated games, correlations between moves in different stages can re-direct the force of selection (or equivalently, the non-cooperative equilibrium condition) to favor outcomes very different from those that would be favored by the stage game in isolation. The approach to repeated games in evolutionary and rational-choice game theory has diverged sharply. The *Folk Theorems* of rational-choice game theory [64] emphasize the range of feasible, individually rational outcomes, and use a loosely formalized notion of prior commitment (defined through trigger strategies of varying complexity) to show that any point in this large set can be reached. In evolutionary models with repeated games, the prior agreement is implicit in the population dynamics, and usually a single repeated outcome is selected. We construct the mapping between these two approaches, and show how different forms of coordination during development qualify as signaling, or substitute for forms of public information introduced to expand the non-cooperative solution space in rational-choice theory. In the process we propose a description-length approach to encoding the complexity of strategies [84, 85], which serves to operationalize the notion of "indefinite repetition" often used by trigger strategies in the strategic Folk Theorems.

Evolutionary mechanics and thermodynamics: The classical *replicator equation* [4] often used to study evolutionary games is very restrictive: by omitting explicit fluctuations, it is effectively a single-scale description, and it is based on a particular set of closure assumptions for first moments which can sometimes be invalid, even for infinite-population limits. By deriving games as classes of scale-dependent fitness models for population processes, we make games the mechanical description of an inherently stochastic process. It is now well under-

[9] This is a term coined by Lewontin [37].

[10] Indeed, the alternation of the Evolutionary Stable Strategy to the strategic Nash equilibrium was perhaps more strongly emphasized in the original introduction by Maynard Smith and Price [82], than their application to animal conflict which was the scientific motivation behind their paper.

[11] Despite the appellation, the content of this theorem has only a distant relation to the Folk Theorems for repeated games coined by Aumann and Shapley [55], which we consider next.

stood that the property of *large-deviations scaling* [72–74] defines the emergence of thermodynamic limits in stochastic processes, and powerful methods exist to apply large-deviations theory to quite general classes of models, including evolutionary games. In this paper and the companion, we will provide examples (classified by symmetry representations) of scale-dependence in fitness functions that arises from fluctuations. The difference between individual and aggregate models results from entropy corrections, equivalent to those in any thermodynamic theory. We emphasize entropy corrections created by *neutrality* in the fitness map. Neutrality is a phenomenon of growing interest in the study of evolution and development [86–91], and a simple counting argument suggests that it will be a generic feature of development models that can be described by highly branching extensive-form games.

B. Organization of the remainder

Sec. II introduces the Price equation in its role as an accounting identity and a general framework for regression models of fitness, and discusses problems of closure and robustness. Sec. III introduces genes as a derived concept for the coarse-graining of genotypes, and discusses their relation to extensive-form games, together with an approach to the coarse-graining of games and strategies. Sec. IV reviews symmetry breaking and the major categories of symmetry that distinguish evolutionary game dynamics, and then presents four examples showing robust consequences of symmetry and a game-theoretic approach to neutrality in development. Sec. V notes ways in which symmetry helps clarify conceptual distinctions in open-ended evolution or the interpretation of repeated games, or suggests a unified theory of games incorporating both evolutionary and strategic concepts.

II. THE PRICE EQUATION AND CLOSURES FOR PROCESSES DESCRIBED BY POPULATION GENETICS

The requirement of consistency with particulate heredity places two restrictions on the class of stochastic processes that can be described by population genetics. First, transmitted material must be partitioned among *individuals*, and the space of individual types must be compatible with reproduction that only replicates, rearranges, and mutates a fixed collection of elements. Because these "hereditary particles" may be much more fine-grained than what we will eventually recognize as *genes*,[12], and because many forms of re-arrangement are permitted between parents and offspring,[13] compatibility of the type space will not be very restrictive for our purposes.[14]

The second restriction is that each heritable particle must, at a sufficiently fine resolution, have had a unique ancestor. This requirement leads to an accounting identity for population processes known as the *Price equation* [33, 34], in which offspring can be grouped according to the type of the parent. In such a grouping, *fitness* is always defined as a summary statistic for offspring numbers partitioned by the parent's type [35]. Fitness may be combined with other observable functions of the population state as a basis for descriptive statistics of the population process.

The Price equation goes beyond descriptive statistics if parametric forms are assumed for fitness or transmission parameters as functions of the population state. The most important such assumption (historically) is the assumption of a time-stationary fitness function which, if used as an observable in the Price equation, yields *Fisher's Fundamental Theorem of natural selection* [32] as the contribution from fitness to the change in its own population average.

More generally, the Price equation with parametric models for fitness or transmission statistics provides a framework for hypothesis testing and calibration.[15] However, the projection onto parametric models introduces the need for *closure assumptions*. Parametric models are not generally complete or consistent under time averaging or other forms of aggregation. They therefore do not by themselves define consistent approximation schemes for dynamics.

In this section we first introduce the accounting function of the Price equation to show where the assumption of particulate heredity projects all population-genetic models into a standard form. We then consider parametric models for fitness, showing why they require closure assumptions and introducing the mean-field approxima-

[12] For instance, they may be single nucleotide positions in a genomic model.

[13] These include crossover, various forms of assortation that in chromosome models represent ploidy (although we will ultimately represent these with player roles in games), and even indefinite ratios of the numbers of replicates, as for mitochondria in relation to nuclear genomes.

[14] Recall that Darwin's informal description of reproduction with selection left the map from parent to offspring type almost unspecified. The difference between Darwinian and Lamarckian dynamics is not one that we consider enforced by the formalism of population genetics, because it can change with the scale of description. Epigenetic modification by the environment is Lamarckian if the germ line is the reference for generations, but becomes Darwinian if transmission of epigenetic markers and base sequences within the ontogeny of the germ line are modeled as a population process. What Darwinian evolution excludes is the class of ecological relations in which persistent characters cannot be grouped into collections of individuals, and for these the formalisms of *Niche construction* or *ecosystem engineering* [92] are invoked.

[15] The use of the term here is compatible with its use in economic modeling [93].

tion as one such closure, which is usually taken to be the basis for the replicator or quasispecies equations.

Finally we consider the approximation of arbitrary polynomial expansions for fitness, and show the relation between order-k frequency dependence and use of k-player normal-form games to describe interaction. This tautological equivalence concludes our treatment of the formal aspects of population genetics. In Sec. III we consider 1) the problem of finding compact representations of substructure within the normal form, 2) the way this leads to a concept of genes within population genetics, and 3) the role of more refined game models such as the extensive form in structuring development and leading to the emergence of statistical genes.

A. The accounting identity for one-step processes with given initial state

Transmission models in population genetics are *Markovian*, meaning both that they are memoryless if a sufficiently rich type space is assumed,[16] and that the stochastic process is defined by its time-local behavior in the form of a *generator* acting on the instantaneous population state [94].

Because our interest is the role of games, we will minimize our discussion of the structure of transmission processes. In particular, we will suppose that we may pass freely back and forth between the discrete-time and continuous-time formulation of Markov processes. Our introduction of the Price equation will allow general branching processes (subject to fixed population), but in examples we will assume for simplicity that the discrete-time process can be modeled as a *Moran process* (or birth-death process) [94, 95] on sufficiently short intervals.[17] We fix population size N because this parameter controls fluctuation strengths in models whose other parameters are stationary. Constant fluctuation strength simplifies examples, though our methods (primarily developed in the companion paper) admit changing populations with no formal difference.

In the discrete-time formulation, the one-step process is the generator of the Markov chain. We first describe the accounting identity for one step with known initial state, and then consider the problem of estimating the generator from moments of a distribution over initial states. The moments of the distribution may be more robust summary statistics in small samples, but they are incomplete to identify the one-step process.

1. *Asynchronous branching-and-removal processes with fixed population size*

A population of N individuals is partitioned into D types indexed $i \in 1, \ldots, D$. The population state at any instant is a vector $\mathrm{n} \equiv (\mathrm{n}_1, \ldots, \mathrm{n}_D)$ giving the number of individuals n_i with type i. We leave open the possibility for assortative interactions that determine fitness, but we assume that any such interactions are based on type alone, and that individuals of the same type are interchangeable.

Our types i correspond to "genotypes", in the sense that they govern descent, and are not directly assigned fitness values until a development model has been chosen.[18] Although we retain the term "genotype" to make use of biological intuition for the potential size and complexity of the type space, we will not assume that the space of types $\{i\}$ is built up from more elementary genes. We instead suppose that the type space is primitive and is empirically defined. The identification of genes will follow as we attempt to capture structure in the type space or in development, on which fitness or transmission depend.

Branching and removal (birth and death) occur on discrete time intervals of length Δt. We do not assume that all individuals in the population reproduce in a single time interval (reproduction is asynchronous), and one or more (or zero) offspring may be produced. At each branching event, a set of individuals from the existing population (which may include the parent) are chosen for removal at random to keep the number of individuals N fixed. The type of the offspring may preserve or may differ from that of the parent, but we suppose that the offspring types j have the same values $j \in 1, \ldots, D$ (the type space is complete even if not all types are populated in a generation). This set of branching processes is rich enough to describe imitation dynamics or other

[16] The type space required for the Markov assumption may be too rich to be tractable in many cases. Although in formal models this is not a problem, in practice the Markov assumption is sometimes considered a strong constraint. How much the Markov assumption is sacrificed by coarse-graining the type space, into a description in terms of genes, is a similar question to how much information is lost by approximating fitness with additive or other low-order models.

[17] We wish to emphasize the conceptual distinctions behind model classes. The *inability* to pass freely between discrete and continuous time implies a *synchronization* of generations. Similarly, a continuous-time Markov process aggregated within discrete intervals may approximate a a *Wright-Fisher* process [15, 94], which may be more or less well approximated by a Moran process acting over multiple generations. The *inability* to substitute the Wright-Fisher process by a Moran process implies synchronized reproduction of multiple offspring or multiple deaths.

[18] To the extent that development depends on interaction of multiple genotypes, the closest concept to "phenotype" that appears in this construction will be the vector of payoffs at the outcome of a game. Phenotype is therefore a property of tuples of player-genotypes, which in game theory correspond to *strategy profiles* [22, 64]. The notion of a "genotype-phenotype" map for the individual who is the unit of transmission is not required in this analysis, but could be defined as a summary statistic from projection of game outcomes onto the focal player's payoff, conditioned on the population state.

forms of oblique transmission [23, 41] as well as literal reproduction.

2. Accounting for population number: defining fitness and parametrizing the transmission process

Fisher's contribution to clarifying the concept of fitness came from grouping offspring according to the parent's type [35]. Properties of offspring type whose distribution is conditionally independent of fitness then appear in "environment" terms, which were written in general form by Price. In the accounting of Fisher and Price, numbers in the offspring generation are conventionally denoted by primes.

We let $\mathrm{n}'_{j|i}$ denote the number of offspring of type j from all parents of type i over a single timestep Δt. Fitness is expressed in samples from the one-step branching/removal process as a random variable w_i giving the total number of offspring to parents of type i in that pair of generations,

$$\mathrm{n}_i \mathrm{w}_i \equiv \sum_j \mathrm{n}'_{j|i}. \tag{1}$$

The change in numbers of types between the parent and offspring generations results from both fitness and transmission effects, and is denoted using Δ, as

$$\sum_i \mathrm{n}'_{j|i} \equiv \mathrm{n}_j + \Delta \mathrm{n}_j. \tag{2}$$

If total population number is conserved, we must have $\sum_i (\mathrm{w}_i - 1)\, \mathrm{n}_i = 0$, so the $\{\mathrm{w}_i\}$ cannot all be independent, and we have the constraint

$$N = \sum_i \mathrm{n}_i = \sum_i \sum_j \mathrm{n}'_{j|i}. \tag{3}$$

Selection comprises all processes affecting offspring number independent of their type. All other processes determining the distribution of offspring types are absorbed into the model of transmission. To reflect this division, any distribution of type-j offspring from type-i parents may be separated into a type-preserving term that simply counts offspring, and a remainder whose sum over types j is zero, as

$$\mathrm{n}'_{j|i} = \delta_{ji} \mathrm{n}_i \mathrm{w}_i + \left(\mathrm{n}'_{j|i} - \delta_{ji} \mathrm{n}_i \mathrm{w}_i \right). \tag{4}$$

The average over realizations of the one-step update process, starting from a definite composition n, is denoted $\langle\, \rangle_{\mathrm{n}}$. When applied to the reproduced number $\mathrm{n}'_{j|i}$ in Eq. (4), the conventional notation used is

$$\left\langle \mathrm{n}'_{j|i} \right\rangle_{\mathrm{n}} = \{ \delta_{ji} \left[1 + \Delta t \left(f_i(\mathrm{n}) - \phi(\mathrm{n}) \right) \right] + \Delta t \mu_{ji}(\mathrm{n}) \}\, \mathrm{n}_i. \tag{5}$$

The scaling of terms to linear order in Δt is the basis for a continuous-time limit if the coefficients of Δt are stable in sample averages as $\Delta t \to 0$. By construction from Eq. (4), $\sum_j \mu_{ji}(\mathrm{n}) \equiv 0$, so $\mu(\mathrm{n})$ is a stochastic matrix. We will refer to it as the *mutation matrix*.[19]

For population processes that preserve total number, the D fitness terms f_i cannot be independently determined from the $D-1$ independent components of the $\{\mathrm{w}_i\}$. We therefore write fitness f_i offset from a population-averaged fitness $\phi(\mathrm{n}) \equiv (1/N) \sum_i \mathrm{n}_i f_i(\mathrm{n})$.[20] Although Eq. (5) resembles the classical *replicator equation* [4] in form, it differs in the important respect that it describes actual population numbers n_i. The replicator equation, defined for fractions n_i/N, includes an average fitness term ϕ for any behavior of total population size. Including this term in models of actual population size, as we have noted, implies restrictions on the time-dependence of fluctuations.

In the examples below, with D types, for the sake of simplicity and in order to isolate other roles of symmetry, we consider only the isotropic mutation process

$$\mu_{ij} = 1 - D\delta_{ij}. \tag{6}$$

3. Consequences for arbitrary observable functions of population state

For any functions $g_i(\mathrm{n})$ defining some attribute for each type i in the context of a population composition n, the Price equation provides a decomposition of the intergenerational change in the population average of the $\{g_i\}$, denoted

$$\bar{g}(\mathrm{n}) \equiv \frac{1}{N} \sum_i \mathrm{n}_i g_i(\mathrm{n})\,, \tag{7}$$

into affects attributable to selection and those from environmental change.

Changes in g_i can come from three effects: a change in the distribution of total number of offspring for each type i, the dependence of $g_i(\mathrm{n} + \Delta \mathrm{n})$ on a changed population state, and a redistribution of types due to transmission. Only the first effect is directly due to selection; the remaining two terms are grouped together as environmen-

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[19] When we return to consider models for f and μ in the next subsection, we will admit arbitrary polynomial expansions over $\{n_i\}$ for both terms. Therefore fitness and mutation have some overlap in the terms they can contain. We may identify $\mu_{ji}(\mathrm{n})$ as any collection of terms that form a stochastic matrix and that include a sum over types i different from the focal offspring type j. This contrasts with the fitness terms, which must all contain at least one factor of n_j.

[20] When we derive minimal stochastic process models from their first-moment constraints in the companion paper, the overall magnitude assigned to fitness will appear in the strength of symmetric diffusion, even though it cancels from selection effects.

tal sources of change, written as

$$\Delta g_i(\mathrm{n}) \equiv g_i(\mathrm{n}+\Delta\mathrm{n}) - g_i(\mathrm{n}) + \sum_j g_j(\mathrm{n}+\Delta\mathrm{n}) \left(\frac{\mathrm{n}'_{j|i}}{\mathrm{n}_i \mathrm{w}_i} - \delta_{ji} \right). \tag{8}$$

The difference between the population-average of the $\{g_i\}$ in the parent and offspring generations in a sample may then be decomposed as

$$\begin{aligned}
&\sum_j (\mathrm{n}_j + \Delta\mathrm{n}_j)\, g_j(\mathrm{n}+\Delta\mathrm{n}) - \sum_i \mathrm{n}_i g_i(\mathrm{n}) \\
&= \sum_i \left\{ \sum_j \mathrm{n}'_{j|i} g_j(\mathrm{n}+\Delta\mathrm{n}) - \mathrm{n}_i g_i(\mathrm{n}) \right\} \\
&= \sum_i \mathrm{n}_i g_i(\mathrm{n})\,(\mathrm{w}_i - 1) + \sum_i \mathrm{n}_i \mathrm{w}_i \Delta g_i(\mathrm{n}) \\
&= \sum_i \mathrm{n}_i \left[g_i(\mathrm{n}) - \bar{g}(\mathrm{n}) \right] (\mathrm{w}_i - 1) + \sum_i \mathrm{n}_i \mathrm{w}_i \Delta g_i(\mathrm{n}) \\
&= N \mathrm{Cov}_{\mathrm{n}}(g, \mathrm{w}) + N \mathrm{E}_{\mathrm{n}}(\mathrm{w} \cdot \Delta g(\mathrm{n}))\,.
\end{aligned} \tag{9}$$

In the second right-hand line of Eq. (9), we have used the fact that the averaged fitness $\sum_i \mathrm{n}_i \mathrm{w}_i = N$ to subtract a (free) factor of $\bar{g}$, leaving the covariance in the last line. Eq. (9) is the standard form for the *Price equation*.

The Price equation as a relation among summary statistics is not yet a hypothesis about any form of evolutionary dynamics, but only a description of sample properties of observables g in terms of their covariance with fitness and their residual dependence on transmission. The observable $g_i(\mathrm{n})$ is a function of type i and population state n, but the fitness itself so far is not a function of population state. To go beyond the accounting identity we must propose models for fitness.

B. From summary statistics to models, bringing in the problem of moment closure

To replace summary statistics $\{f_i\}$ and $\{\mu_{ij}\}$ with models, we must make further assumptions about the ensemble in which $\{f_i\}$ and $\{\mu_{ij}\}$ sample the generating process.[21] Frank has emphasized [35] that the Price equation relates three empirical quantities – initial population state, final population state, and process description – and that from any two of these, it may be used to estimate the third.[22] However, summary statistics do not determine the evolution of population states or probability distributions except under additional (often rather strong) assumptions known as *closures*. Therefore most problems of model estimation, phylogentic reconstruction, or prediction, are undefined prior to the choice of closures.

The standard problem in population genetics is the choice of *moment closures* [14, 19], which we consider in this section. Other problems of closure also arise, however, including the selection of robust parametric models of fitness in the presence of dynamical noise or estimation error. We return to the latter in Sec. III.

1. *One-step averages over samples with fixed initial state*

The fitness term (the covariance term) in Eq. (9) is linear in the only variable (w_i) that depends on the one-step update. If it is possible to obtain a sample of updates from a common initial state n, a fitness model may be directly assigned, as

$$\langle \mathrm{w}_i - 1 \rangle_{\mathrm{n}} = \Delta t \left[f_i(\mathrm{n}) - \phi(\mathrm{n}) \right]. \tag{10}$$

The transmission process is not generally identifiable in terms of linear expectations alone, as it may involve covariance of w with the change $\Delta\mathrm{n}$. g itself may also be a non-linear function of n. A linear estimator in the $\left\{ \mathrm{n}'_{j|i} \right\}$ that ignores these covariance terms may be used to approximate $\mu(\mathrm{n})$ by

$$\frac{1}{\langle \mathrm{w}_i \rangle_{\mathrm{n}}} \left\langle \frac{\mathrm{n}'_{j|i}}{\mathrm{n}_i} - \delta_{ji} \mathrm{w}_i \right\rangle_n = \frac{\Delta t \mu_{ji}(\mathrm{n})}{1 + \Delta t \left(f_j(\mathrm{n}) - \phi(\mathrm{n}) \right)}. \tag{11}$$

If we use these sample estimators as models, supposing they are stationary in time, and substitute $f_i(\mathrm{n})$ for the observables $g_i(\mathrm{n})$ in Eq. (9), then the covariance term in the last line expresses *Fisher's Fundamental Theorem* [32].

Note that for the expected number n_j, the linear estimator (11) is exact. We therefore have, as a definition, the relation for first moments under the one-step process

$$\frac{\langle n_j + \Delta n_j \rangle_{\mathrm{n}} - n_j}{\Delta t} = \left[f_j(\mathrm{n}) - \phi(\mathrm{n}) \right] \mathrm{n}_j + \sum_i \mu_{Ji}(\mathrm{n})\, \mathrm{n}_i. \tag{12}$$

In many situations, the formula (12) may be taken from discrete to continuous time, where its form is the basis for the deterministic differential equation known as the replicator equation.

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[21] This ensemble may result from accumulation of generations in a single population, reflecting time-stationarity, but it could instead come from replication of populations whose short-term evolution is tested for reproducibility, even if it is not time-stationary.

[22] Each estimation process reflects one aspect of evolution. Reconstruction of the (typically diachronic) past from synchronic variations and (usually highly random) process models is the object of phylogenetics. Estimation of process models is the attempt to extract evolutionary regularities or laws, and estimation of future states from inferred or assumed laws is the standard problem of prediction.

2. Averages over initial state, and the mean-field approximation for moment closure

Suppose that the robust observables are sample estimators for mean population numbers, where we assume that the true mean values are the first moments of an underlying distribution that we do not know and are trying to infer. We denote the true means by

$$\bar{n}_i \equiv \sum_{\mathrm{n}} \mathrm{n}_i \rho_{\mathrm{n}}, \tag{13}$$

where ρ_{n} is the (generally time-dependent) underlying distribution appropriate to the system context we assume and the set of questions we wish to ask.[23]

$$\begin{aligned}
\bar{n}'_j &\equiv \sum_{\mathrm{n}} \rho'_{\mathrm{n}} \mathrm{n}_j \\
&= \sum_{\mathrm{n}+\Delta\mathrm{n}} \rho'_{\mathrm{n}+\Delta\mathrm{n}} \left(\mathrm{n}_j + \Delta\mathrm{n}_j\right) \\
&= \sum_{\mathrm{n}} \rho_{\mathrm{n}} \sum_i \left\langle \mathrm{n}'_{j|i} \right\rangle_{\mathrm{n}} \\
&= \bar{n}_j + \Delta t \sum_{\mathrm{n}} \rho_{\mathrm{n}} \left\{ \left[f_j(\mathrm{n}) - \phi(\mathrm{n}) \right] \mathrm{n}_j + \sum_i \mu_{ji}(\mathrm{n})\, \mathrm{n}_i \right\}.
\end{aligned} \tag{14}$$

The map (14) from ρ_{n}, $\{f_i\}$, and $\{\mu_{ij}\}$ to $\left\{\bar{n}'_j\right\}$ is exact but non-invertible. In addition to higher-order moments of ρ'_{n} that are not identified by $\left\{\bar{n}'_j\right\}$, the map to the first-moment conditions from interactions of $\{f_i\}$ and $\{\mu_{ij}\}$ with higher-order moments of ρ_{n} is also many-to-one.

A strong *approximate* moment-closure assumption, valid when the distributions ρ_{n} and ρ'_{n} are sharply peaked around a single value, is the *mean-field approximation* (MFA). It replaces Eq. (14) by a relation of similar form, in which $\{f_i\}$ and $\{\mu_{ij}\}$ are simply evaluated at $\bar{\mathrm{n}}$, which is assumed to change slowly as well as having only small fluctuations,

$$\frac{d\bar{n}_j}{dt} = \left[f_j(\bar{n}) - \phi(\bar{n}) \right] \bar{n}_j + \sum_{i=1}^{D} \mu_{ji}(\bar{n})\, \bar{n}_i. \tag{15}$$

The MFA is a useful approximation but not a proper moment closure, in that it will generally be violated even within stochastic models that it was used to define.

[23] That is, ρ_{n} might be a smooth distribution in the context where a stationary population process has acted for a long time, or it might be singular because it is the outcome of a shock, or because it reflects a particular initial sample over types made by the experimenter but outside the model of the evolutionary dynamics.

C. Robustness of moment-closure approximations and the concept of universality classification

Two related but slightly different approaches can be taken to the problem of robustness. They bear slightly different relations to the two dimensions of robustness itself, which are sensitivity of model identification, and prediction in the presence of fluctuations.

The approach of defining and closing recursion relations for moments in population genetics [13, 14] seeks approximate solutions to fixed models. Predictive error comes from errors of moment closure, and identification error can be tested separately with parameter sensitivity analysis.

An alternative approach recognizes (formally) that models themselves are approximate, but that averaging or aggregation can lead to convergence of groups of distinct models into *universality classes*[28]. Each such class is represented by an *effective theory*, which is self-consistent under moment closure up to terms that disappear under aggregation. An approach based on effective theories and universality classification seeks to derive the interchangeable roles of moment-closure and model-identification approximations.

A derivation of universality classes for games is beyond the scope of our review. However, the calculations we present in the companion paper are those used in background-field methods [96] to prove renormalizability, universality classification, and the existence of effective theories.[24]

D. k-player normal forms provide a polynomial expansion in frequency-dependent selection

Suppose we restrict fitness models to be approximated by polynomials in the agent numbers $\{n_i\}$.[25] Then the coefficients of all order-k terms, in the expansion of the vector $[f_i(\mathrm{n})\, \mathrm{n}_i]$, form a rank-$k$ matrix that may be interpreted as the expected-payoff matrix in a k-player normal-form game with random matching.

To order $k \leq 2$ this is the affine expansion

$$f_i = \alpha_i + \sum_j a_{ij} \left(\mathrm{n}_j - \delta_{ij} \right), \tag{16}$$

in which α_i is an inherent fitness of type i, and a_{ij} is the payoff to type i when matched with type j in a two-player normal-form game.[26] By the freedom to define

[24] Examples of renormalization for closely related systems within the Freidlin-Wentzell approach may be found in Ref's. [70, 71].

[25] These are simply the fitness functions that converge to their Taylor's series expansions.

[26] By freedom to vary the magnitude of a_{ij}, we may reference fitness to the case that each agent plays the others, on average, exactly once in a generation.

α_i we adopt the convention of subtracting a term δ_{ij}, so that in the game interpretation, no agent plays against itself.[27] The two-player normal form is the form in which evolutionary game theory was introduced by Maynard Smith and Price [82].[28]

If a finite-order expansion such as Eq. (16) is estimated by regression, for a stochastic process which may additionally have (unmodeled) higher-order frequency dependence, the population-corrected fitness (10) becomes

$$\frac{\mathrm{w}_i - 1}{\Delta t} = \sum_j a_{ij} \left(\mathrm{n}_j - \delta_{ij}\right) - \frac{1}{N} \sum_k \mathrm{n}_k a_{kj} \left(\mathrm{n}_j - \delta_{kj}\right) + \epsilon_i \tag{17}$$

with residual ϵ_i.

The residual will have zero mean if the regression coefficients are chosen to minimize the residual sum of squares (RSS)

$$\sum_i \mathrm{n}_i \epsilon_i^2 = \sum_i \mathrm{n}_i \left(\frac{\mathrm{w}_i - 1}{\Delta t} - \sum_j a_{ij} \left(\mathrm{n}_j - \delta_{ij}\right) + \frac{1}{N} \sum_k \mathrm{n}_k a_{kj} \left(\mathrm{n}_j - \delta_{kj}\right) \right)^2 . \tag{18}$$

We will show in the repeated Prisoner's Dilemma example of Sec. IV E that, although the form of the regression may be valid either as a characterization of individual interaction or by calibration of the population equilibrium that includes fluctuations, the coefficients may differ between the two descriptions. Another reason regression coefficients may not correspond directly to individual-level payoffs is that they incorporate effects of non-random matching of individuals. We will not pursue assortative matching further in this paper, but in repeated game models where strategies can depend on history, behavior can change as a result of "signaling" by early moves in the game. Signaling within the course of play presumably lives on a continuum with abilities to recognize player type that determine whether they are matched at all.

[27] This convention leads to a simple map from the parameters in the fitness function and those in the stochastic-process model, which we develop in the companion paper.

[28] The place of low-order polynomials and therefore low-order games may be more important than their restricted form suggests. The central limit theorem for many kinds of distributions projects infinite-dimensional manifolds onto low-dimensional subspaces under aggregation [28, 97]. In the large-deviations rate functions computed in the companion paper, the parameters in low-order polynomial models of fitness will occupy the place of stable distribution parameters in the central limit theorem. Therefore low-order normal form games may be general in population-level regressions even when they do not describe individual interactions.

III. GENES IN THE PRICE EQUATION AND IN GAME MODELS

We now consider the problem that type spaces which are rich enough to satisfy the Markov condition may be too large to sample, estimate, or analyze. Even with the conventional notion of a gene as "a particular copy of a non-recombining sequence at some locus in some individual" [14], this situation will be common for traits that depend epistatically on a large number of genes. If non-recombining DNA regions cannot be taken to define genes [49] the fallback Markovian genotype space may be as large as the space of sequences, which are too large for natural populations to sample. In the domain of behaviors or institutions, there may be no standard template comparable to DNA to provide a fallback Markovian state space, even if "units of culture" functionally comparable to genes do exist in some approximation.

Noting this range of possibilities, we suppose the "genotypes" $\{i\}$ in our population models are primitive and are empirically defined, and we consider *genes as a basis for the course-graining of genotypes* as the derived quantities. We will introduce genes as *partitions* of genotypes, beginning with the context of normal-form games and general frequency-dependent selection. We then suppose that the normal form can be refined to a particular extensive form game representing the substructure within development. In this case, the abstract problem of optimal partitioning may be replaced with model-based partitions referring to the structure of the game tree.

A. The origin of biological genes in development

To provide intuition for our discussion of the gene as a derived concept, we recall that the particulate heredity of DNA in cell biology is *not* the source of the Mendelian gene of evolution. App. A 1 argues that the error-correcting role of a general-purpose digital substrate such as DNA is optimized precisely when the substrate does not carry the distinctions that define gene boundaries.

The gene as a source of robustness or compression must be sought – even for cells – at the system level of physiology and development. Transmission that violates the modular structure of development will generally produce non-viable phenotypes. To the extent that this exposes the transmission mechanism itself to selection, we may expect that transmission will come to actively preserve developmental modules.

Just as DNA is a reflection but not a source for genes in cell biology, the absence of any close analog to DNA in behavioral or institutional systems need not preclude the existence of a gene concept in these systems. The proper question to ask is why coarse-grained genes have been ubiquitous and relatively simple to identify in cell biology, and whether the mechanisms responsible for this simplicity are active in other domains as well.

1. Arguments from robustness and evolvability, that modularity in system architecture should be common

Modularity in the architecture of complex systems appears common, even though proximate causes such as those in cell physiology and development are system-specific. Arguments that modularity is a condition for hierarchical complexity have been put forward on grounds of robustness [98, 99] and evolvability [100]. They are persuasive, but are not yet sufficiently formal that we see a way to incorporate them into model classification.

2. Genes, data-compression codes, and multilevel selection

The statistical problem of identifying good gene-partitions for genotypes, as we will construe it, has much in common with the problem of finding good data-compression codes. The loci and alleles suggested by simply following the structure of extensive-form game trees will often give good (if not proven-optimal) compressed representations, but they will also require the use of variable-length moment-closures in order to preserve predictive sufficiency. In the language of population genetics, we will be required to keep non-linear *associations* of genes in regressions for fitness – that is, the very genes that account well for variance in fitness with additive models will also usually require the explicit retention of a few contributions from multi-level selection. From experience with variable-length data-compression codes [79, 80], we expect that this property, which arises for partitions motivated by the game tree, will remain even as a property of optimal codes.

B. The space of gene decompositions: disjunction, conjunction, and compression

We will formalize the notion of genes in our models in terms of *discrete partitions* on the space $\{i\}$ of genotypes. We require that the gene description be invertible, so that it changes the representation of genotypes but does not lose information or the Markovian property of completeness.[29] A partition is also known as an *equivalence relation*. The sets in the partition, called *equivalence classes*, correspond to unions of genotypes. In order for a representation by partitions to be invertible, we require multiple equivalence relations, so that each genotype can be uniquely identified by the intersection of the sets from different equivalence relations in which it appears. We will refer to any equivalence relation as a *locus*, and to the equivalence classes in that partition as the *alleles*. A *gene* is a particular allele at a particular locus. We further restrict, without loss of generality, to sets of equivalence relations that are *minimal*, meaning that that no partition consists entirely of sets that may be identified by intersections of sets from the other partitions. We do not, however, require that the decomposition into genes reconstruct the genotype space as a product space, or even that all individuals in the population share the same loci.

A good set of partitions will be one in which many equivalence classes contribute little to the variance that drives population dynamics, and of those that remain, most of the dynamically significant variance is captured in additive models. We now consider the expansion of game parameters in indicator functions for the presence of genes.

1. Disjunction and conjunction systematically approximate type-dependencies with associations of genes

Introduce capital indices $I, J, K, \ldots$ to indicate genes, and indicator functions $\sigma_{Ii}, \ldots$ to map genotypes i into equivalence classes I. Then each indicator function σ_{Ii} is a *disjunction* (logical OR) of indicator functions for the genotypes $\{i \mid i \in I\}$, and each genotype i is a *conjunction* (logical AND) of indicators σ_{Ii} for some collection of genes $\{I \mid i \in I\}$.

Each coefficient in the polynomial expansion of frequency-dependent fitness then has its own expansion in terms of gene-indicator functions. For the coefficients in Eq. (16), this expansion may be written

$$\begin{aligned}
\alpha_i &= \sum_I \sigma_{Ii} \left(\alpha_I^{(0)} + \sum_K \sigma_{Ki} \left(\alpha_{KI}^{(1)} + \sum_M \sigma_{Ki} \alpha_{MKI}^{(2)} + \ldots \right) \right) \\
a_{ij} &= \sum_{IJ} \sigma_{Ii} \sigma_{Jj} \left(a_{IJ}^{(0,0)} + \sum_K \sigma_{Ki} \left(a_{KI,J}^{(1,0)} + \sum_M \sigma_{Mi} a_{MKI,J}^{(2,0)} + \sum_L \sigma_{Lj} a_{KI,LJ}^{(1,1)} + \ldots \right) \right) .
\end{aligned} \tag{19}$$

[29] These two criteria alone permit much more general models than discrete partitions, including continuous *partitions of unity*. We restrict to discrete partitions for simplicity.

The leading coefficients $\left\{\alpha_I^{(0)}\right\}$ in the expansion of α_i give the additive genetic contribution to (inherent) fitness. In population genetics, the higher-order products of indicator functions are known as *associations*, and nonzero coefficients for associations represent epistasis.[30]

Note that the series for payoff a_{ij} contains an expansion in associations for both the focal individual (i) and the opponent (j). We use superscripts $a^{(p,q)}_{I_1,\ldots,I_p,J_1,\ldots,J_q}$ to indicate the orders p and q of "epistatic" interaction in each player.

Just as the polynomial expansion in frequency has uniformly-mixed k-player normal form games as a default interpretation,[31] the joint-gene expansion of payoff coefficients, for suitably chosen partitions, has a default interpretation in terms of interactions on an extensive-form game that refines the normal form, with payoffs that are additive over internal nodes on the game tree. We return to develop this interpretation in the next subsection.

2. Compression, and the competition between obtaining additive variance and minimizing multilevel selection

If partitions of the type space may be chosen so that the number of genes $|\{I\}|$ grows only logarithmically in the number of genotypes $|\{i\}|$, then the truncation of the series (19) to fixed order provides an exponentially compressed representation of the dependence of fitness on genotype. Whether this truncated approximation is good depends on the rate at which the coefficients $\alpha^{(p)}$ and $a^{(p,q)}$ fall off with increasing p or q.

We expect as the general case, even for genes defined purely for the sake of compression, that a typical gene will be pleiotropic, with differing contribution to the additive genetic variance for different traits. That is, a partition I may account for most of the variance of one component of f_i as additive variance, while it contributes epistatically to other components of f_i, perhaps to varying degrees depending on population state. Maximizing additive variance favors the status of I as a gene, while pleiotropy and epistasis make any purely linear model involving I insufficient to explain population dynamics, and require the retention of higher-order associations involving I. This competition is analogous to the problem of designing data-compression codes, which leads (through Fano's theorem [80]) to variable-length codes. We will present an example in Sec. IV G with this property, for which the precise components of variance explained by each higher-order interaction in relation to the main additive term are worked out in App. E 2.

3. The genotype space need not be a product space

The genotype space $\{i\}$ need not be a product space, with each individual having some allele at each of a fixed set of loci. Our population models are free to represent interactions of individuals from multiple "species" or "organelles", a feature easily accommodated in games where player roles are distinguished.

Product spaces are one class within which the number of genes grows logarithmically in the number of genotypes. However, for genes of unequal effect on fitness, a simple product space may not provide the best compressed approximations to fitness. By departing from the assumption of a product space, we may use genes to label higher-order functional modules, for which canalization may have made the wild type insensitive to some forms of mutation or recombination.

C. Extensive-form games, and partitions that reflect structured interactions/development

Genes reflect the existence of structure in the fitness function that, expressed as a normal-form game, makes the representation of the game "compressible" in some way. But if we know the source of structure in the normal form, it may be possible to suggest the compression directly – at least approximately – rather than approaching it as an optimization problem. The way to classify the structure of normal-form payoff matrices in game theory is to refine the game description by providing a particular extensive form [24].

Extensive-form games, and the genetic descriptions they induce, are a large topic,[32] from which we consider three points here: 1) the gene partition induced by extensive-form games, which relates interaction structure to mutation and recombination; 2) the problem of expanding the strategies on repeated games in a systematic

[30] Note that, if we are using players to model haplotypes, and two-player games to model development in a diploid organism, then inherent fitness is a transmitted property of chromosomes (such as meiotic drive), and epistasis refers specifically to *cis*-interactions among genes within a chromosome. *Trans* interactions appear in the payoff matrix. *Dominance* appears as a combination of an inherent fitness from the dominant allele, and a payoff from pairing with any opponent having the dominant allele. A model for dominance with saturation, in a pairing of exactly two chromosomes of types i and j, might be $f_i = a_i + a_i a_j / (a_i + a_j)$, where both $a_i, a_j \in \{a, A\}$. The interaction term then requires a slowly-converging polynomial series to approximate.

[31] This interpretation is only a "default" in the sense that it assumes random matching as a parameter-free model. However, random matching depends on on the representation of types for its meaning. Therefore, like any Bayesian prior, it should be regarded as a particular assumption which is a part of model selection.

[32] A broader set of examples is developed in the monograph by Cressman [23].

hierarchy of complexity, memory and symmetry, in order to relate repeated games in evolutionary and rational-choice game theory; and 3) the directionality of solutions on game trees from root to leaves, and the asymmetry this creates between transient and recurrent states when strategies are mapped to finite-state automata. We develop examples of each of these points which are used in the evolutionary game models of later sections.

1. The gene partition induced by an extensive-form game

An extensive-form game has three elements: 1) an underlying *game tree*; 2) a collection of *information sets* telling which nodes on the tree are to be regarded as indistinguishable by players;[33] and 3) an assignment of information sets to players, telling who moves at each information set. A *strategy* for a player on an extensive form game is a specification of a move on every information set assigned to the player by that game.

To complete a bit of terminology that will be needed later, when the tree in an extensive-form game has a repeated structure of the same simultaneous or sequential moves by the players, the repeated units are called *plies*. The joint strategies of a collection of agents, who together are sufficient to play a game, is called a *strategy profile*. In an extensive-form game, which further decomposes strategies into sequences of moves, the joint moves of the players at their respective information sets within a ply is called a *move profile*.

If strategies in evolutionary game theory are to serve as genotypes, then any mutation and recombination processes must respect the information-set structure on the extensive-form game. They may alter moves at information sets, singly or jointly, but they cannot introduce distinctions at nodes within an information set,[34] and must under all conditions produce fully-defined strategies.

Any extensive-form game induces a gene decomposition of genotypes, in which the information sets are loci and the moves on each information set are alleles.[35] Mutation is free to change moves in any fashion, while recombination acting on a set of existing strategies may shuffle the moves on different information sets to produce one or more new well-formed strategies. In some cases the topology of the game tree may define a useful notion of adjacency, so that recombination by "crossover" switches all information sets on the sub-tree descended from the crossover point.

The gene decomposition induced by an extensive-form game in this manner does reconstruct the genotype/strategy space as a product space. For games with asymmetric player roles, a distinct product space exists for the subsets of players who can take each role.

2. Separating mechanism from solution concept, and then constraining mechanism

The extensive form for a game separates the *mechanics* by which strategy profiles interact to produce an outcome, from any consequences of that outcome, including payoffs or solution concepts derived from payoffs, which select strategies.[36] The aspects of mechanism that will concern us are the *complexity* of strategic interactions, and the *correlation* among moves that can be created by strategies with memory. Complexity can determine whether symmetry breaking leads to simple or complex ordered population states, and we will measure it by the number of information sets a strategy distinguishes. Long-range correlation can redirect the force of selection, as in the literature on repeated games.

Rational choice game theory, emphasizing inductive solution concepts, has taken a complicated approach to achieving long-range correlation, often using indefinitely repeated games and restricted forms of discounting [56, 64].[37] Evolutionary game theory, with its emphasis on causal mechanisms, has used recursively defined strategies. To bring these two approaches together, we introduce a way to systematically approximate strategies of any complexity on a game tree of indefinite length, with a sequence of finitely defined strategies. We thus replace the loosely formalized notion of "prior agreement" in rational-choice repeated-game theory, with constraints on the complexity or memory of strategies that may reflect costs or other mechanistic limitations.

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[33] Information sets may be used to compensate for deficiencies in the tree representation for handling simultaneous play, or they may reflect limits of complexity or memory imposed on the available strategies.

[34] The purpose of the extensive form is to separate the "reality" defined by the game from the strategic freedom and solution concepts of players. In the reality defined by information sets, distinctions among the nodes within them do not exist as strategic options for players.

[35] This "natural" decomposition is also the one adopted in Ref. [23].

[36] The game also formally separates players from each other, in the sense that a player's strategy is not a function directly of other strategies, but rather of the states of play indexed by the information sets. Information sets are therefore Markovian states in the sequence of play, encapsulating all information about history relevant to future moves, within the frame of a given strategy. The selection of strategies by solution concepts need not be Markovian in this way, and is free to respond to arbitrary non-local properties of strategies, as the concept of subgame perfect Nash equilibrium does in rational-choice game theory. [20].

[37] The general device is the *trigger strategy*, a correlated move sequence based on some notion of "prior agreement". Trigger strategies overcomes Pareto dominance of some move in a stage game through sequences of retaliatory moves, which individually satisfy requirements for rationality, but cumulatively reverse the advantage of the stage move which it is their function to prevent.

3. *Coarse-graining extensive-form games, and complexity and memory of strategies*

Our approach will be to start, not with the strategies, but with the game trees on which they are defined. The information sets on game trees provide a way to make specific complex strategies *impossible*, by eliminating the distinctions among states of play that those strategies require.

We call this procedure **coarse-graining of games**. For any game in extensive form, a *coarse-graining* is another game with the same tree, in which the information sets are unions of information sets in the original game. The original game is then a *refinement* (generally one among several that are possible) of the coarse-grained game.

Here we give a procedure based on coarse-graining to systematically extend the recursive complexity of strategies, and to classify these according to the memory and symmetries enabled by coarse-grained games. We illustrate with a binary, simultaneous-move, total information game. For the sake of giving a form to payoffs that can be used in examples, we will restrict to payoffs that can be written as sums of values from the internal nodes visited in the play of a strategy profile.

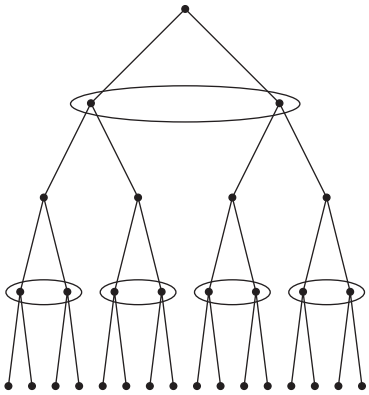


FIG. 1. The two-player game with simultaneous binary moves and complete information. Information sets on alternating rows belong to the first and the second player, and each pair of rows represents one simultaneous move in the game. Two plies of the tree are shown; a tree with indefinitely repeated moves is obtained by extending this and all subsequent trees from their leaf nodes, recursively. Since all paths of play are distinguished by these information conditions, this game tree supports solution concepts such as subgame perfection.

Fig. 1 shows an extensive-form game for two players, each given two simultaneous, pure-strategy moves L and R in each ply. This tree provides the largest possible complexity of strategies and memory consistent with simultaneity and pure moves. We will refer to it as the *total information* game.

For the game in Fig. 1 with $N_{\rm ply}$ plies, the number of information sets assigned to either player is $\left(2^{2N_{\rm ply}}-1\right)/\left(2^2-1\right)$, so the number of possible distinct strategy types is

$$|\{i\}| = 2^{\frac{2^{2N_{\rm ply}}-1}{2^2-1}}. \tag{20}$$

The most general additive normal form giving payoff a_{ij} to an individual of strategy-type i when matched with an individual of type j is then

$$a_{ij} = \frac{1}{N_{\rm ply}} \sum_{k=0}^{N_{\rm ply}-1} \sum_{\alpha^k=L,R} \sum_{\beta^k=L,R} \sigma_{\alpha^k i}\sigma_{\beta^k j} a^{(k)}_{\alpha^k\beta^k}. \tag{21}$$

In Eq. (21) $a^{(k)}_{\alpha\beta}$ is the normal-form payoff in ply k. The α^k, β^k take values L or R at each information set, and we use indicator functions $\sigma_{\alpha^k i}$ that take value 1 for those strategies i with move α^k at position k.[38]

It is important that strategies specify moves at information sets, but payoffs need not be the same at all nodes within a single information set. In this way, the extensive form permits independent control over the complexity and memory of strategic opportunities, and the payoff functions that may be used in *solution concepts* over the available strategies. [24].

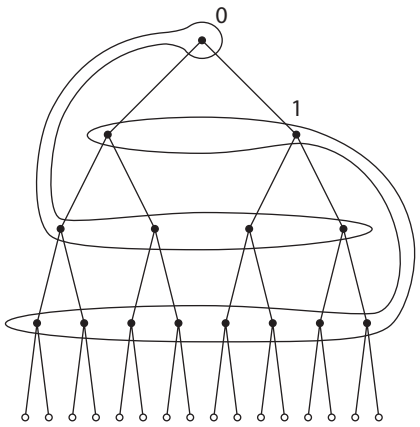


FIG. 2. Maximal coarse-graining of the simultaneous-move game of Fig. 1, to one information set per player. The information sets 0 and 1 belong to the first and second player, and are understood to be extended as plies of the tree are added.

The maximal coarse-graining of the total-information game in Fig. 1 is shown in Fig. 2. Each player has a single information set, and therefore no memory and minimal complexity. The size of the type space is

$$|\{i\}| = 2. \tag{22}$$

[38] The normalization by $N_{\rm ply}$ is arbitrary, but provides a useful way to handle scaling when we consider repeated games in later sections.

This coarse-graining projects the extensive form at any $N_{\rm ply}$ onto the normal form for a single ply, and reduces the payoff a_{ij} of Eq. (21) to a one-ply effective normal form $a_{\alpha\beta}$, averaging the $\left\{a^{(k)}_{\alpha^k\beta^k}\right\}$ with equal weight.

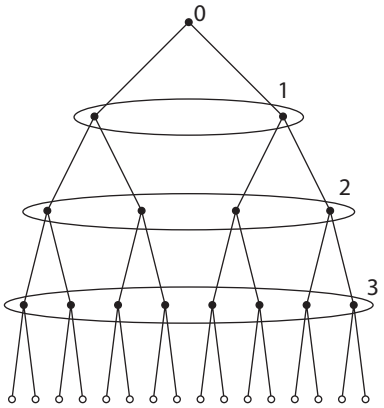


FIG. 3. A coarse-graining of the total information tree 1 that distinguishes plies but not paths of play. Even-numbered information sets belong to the first player, and odd-numbered information sets belong to the second player. In this tree, as plies are added, new information sets are introduced with each ply.

An intermediate coarse-graining of the total-information tree shown in Fig. 3 distinguishes plies and permits strategy complexity to grow with tree size, but precludes memory, because nothing in the history of play is distinguished by the information sets. The number of strategy types is

$$|\{i\}| = 2^{N_{\rm ply}}. \tag{23}$$

The adjacency relation between plies on the tree is the same as that between loci on a linear chromosome, and this tree will be used as the basis for a model that maintains linkage disequilibrium in Sec. IV H.

4. Recursion and repeated games: systematic approximation of strategies

We may generate a variable-length description for strategies of increasing memory by referring each strategy to the simplest coarse-grained tree on which it is defined. We then assign to refinements of the coarse-grained tree, only the new strategies that each refinement first enables.[39] We illustrate this decomposition with the first level of refinement, back from the maximally coarse-grained tree of Fig. 2 toward the total-information tree of Fig. 1.

Strategies L and R, equivalent to a single 2×2 normal form, are the only ones defined on the tree of Fig. 2. The maximally coarse-grained tree permits three distinct refinements having memory-depth of one ply, and equal numbers of L and R moves, shown as Figures 4, 5, and 6. Each tree has three information sets per player and thus

$$|\{i\}| = 2^3 \tag{24}$$

possible strategies. However, the first two strategies on each refined tree, which assign either L or R to all information sets, coincide with those on the tree of Fig. 2, and are not counted again. Therefore each refined tree brings six new strategies.

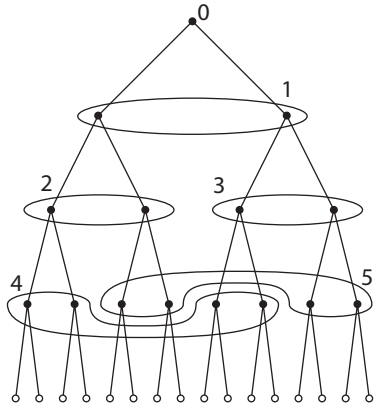


FIG. 4. A tree with memory of the player's own previous move. Here information sets 0 and 1 are opening moves for the first and second player. Information sets 2, 3 are the *recurrent* moves for the first player, and 4, 5 are the recurrent moves for the second player. Information sets representing recurrent moves are to be extended recursively as plies are added to the tree. Strategy REP moves L on information sets 2 or 4 and R on 3 or 5, while strategy ALT moves R on 2 or 4 and L on 3 or 5, thus repeating or alternating the player's previous move.

The refinement tree shown in Fig. 4 describes memory of the player's own previous move. The two new strategies defined on the recurrent moves either repeat the player's previous move (REP), or do the opposite, leading to alternation (ALT).

The refinement shown in Fig. 5 describes memory of the opponent's previous move. The new strategy defined in the recurrent stage of of this tree is tit-for-tat (TFT), which repeats the opponent's last move (and its opposite, anti-tit-for-tat, or ATFT).

[39] In matching a strategy's complexity to its description length, we are following the *minimum description length* approach of Rissanen [84] for representing algorithms. The decomposition of strategies which are defined in a product space over normal forms on each information set, into varying degrees of long-range order, is similar to the decomposition of a direct-product group into its direct-sum components [101]. The product space over normal forms is the most finely resolved genotype space that a coarse-grained extensive-form game admits.

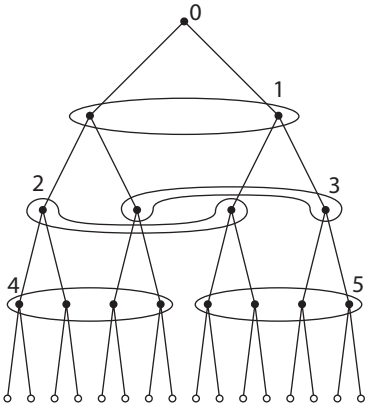


FIG. 5. The coarse-graining of the total-information game of Fig. 1 that defines one-period memory for the opponent's move in the previous round. Strategies 0 and 1 are again opening moves, while 2–5 are recurrent moves. Strategy TFT moves L on sets 2 or 4 and R on sets 3 or 5, and attempts to achieve coordination between players by opening with L on sets 0 or 1.

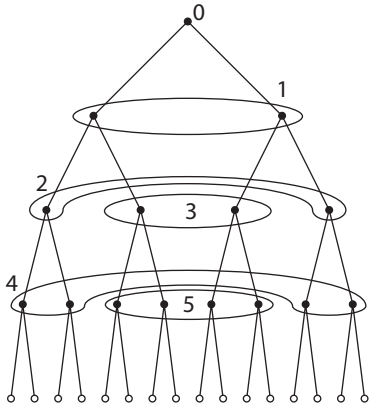


FIG. 6. A tree that distinguishes parity of the players' previous-round moves. Sets 0 and 1 are opening moves, and 2–5 are recurrent. The strategy Win-Stay/Lose-Shift assigns L to recurrent sets 2 or 4, and R to sets 3 or 5. It therefore breaks parity with the recurrent move in every ply, and its opening move is not needed for coordination and does not matter in long repeated games.

Both of the previous refinement-trees have the property that their non-trivial recurrent moves (those that are not all-L or all-R) are *symmetric* with respect to joint exchange of responses L and R together with reflection of the information sets through the vertical. That is, by taking in asymmetric moves from the previous round and generating asymmetric responses, all four of REP, ALT, TFT, and ATFT make symmetrically defined *responses* to their inputs.

The coarse-graining of Fig. 6 describes memory of the *parity* between the player's and opponent's previous move. Its information sets are therefore symmetric under reflection. The two non-trivial recurrent-move strategies on this tree *break* the response symmetry by mapping even or odd information sets to L or R moves. The widely explored strategy of Win-Stay/Lose-Shift [60, 63] (WSLS, also called Pavlov [61]) maps even parity to L and odd parity to R. Its opposite, which we term anti-WSLS or AWSLS, does the reverse.

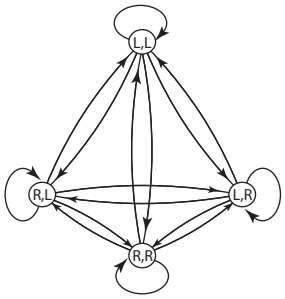
The symmetry or asymmetry in the response of short-memory strategies is fundamental to the way they cope with imperfect moves (called "trembles"). As we show in Sec. IV E 4, this difference of symmetry determines whether the repeated game even uses long-range correlations to redirect the force of selection, or simply modifies the effective normal form over short range.

5. *Finite-state automata and behavior strategies*

As we have shown with the tree of Fig. 3, the importance of memory can be minimized or even removed with the structure of information sets. Models with this property can represent general epistasis in development without an implied directionality.

When memory is used, however, it inherits a direction from the branching of the underlying tree in the extensive form.[40] This directionality need not entail temporal sequence, but it does entail one-way contingency in some form. In order to classify the forms of strategic irreversibility that can result, we map finitely-defined strategies on an indefinitely repeated extensive form game tree to their corresponding finite-state-automata (FSA) [102].

Any finitely defined strategy on a tree of infinite depth must correspond to a coarse-grained tree in which infinitely many nodes are assigned to some information set or sets. Because information sets are Markovian, we may always collapse the tree onto a finite-state graph. For any such graph, a minimal form exists [103]. The states in the FSA may be divided into *transient states*, which are not revisited asymptotically, and *recurrent states*. In the simple examples above, the transient and recurrent states correspond to the opening-move and recurrent-move information sets on the game tree.



[40] This one-way branching during interaction is reminiscent of the one-way branching of population processes described by population genetics.

FIG. 7. Possible moves in the recurrent stage of the repeated game of Fig. 5.

Fig. 7 shows the recurrent FSA for the tree of Fig. 5. The nodes are labeled with the move profiles that arrive at them. This FSA will be the basis for the repeated Prisoners' Dilemma example in Sec. IV E.

Strategies defined by moves on the states of a FSA are known as *behavior strategies* [85]. Depending on the structure of the game tree, behavior strategies may or may not be intrinsically reversible. We may isolate behavior strategies as models of development by using long extensive-form games in which payoffs from opening moves are negligible, though transient states may still coordinate the recurrent moves. An example using behavior strategies in which correlations are removed with trembles is given in Sec. IV E 4, using the recurrent moves from Figures 4, 5, and 6

IV. SYMMETRY AND COLLECTIVE FLUCTUATIONS

We now consider the major robust effects based on symmetry groups and their representations by population states, which distinguish categories of game models. The most important of these is spontaneous symmetry breaking, which may happen to either continuous or discrete symmetry groups, and may lead either to groups of population states whose number does not depend on the size of the state space, or to groups of population states that grow with increasing size. The fact that symmetry remains a property of the system as a whole can be used to prove the existence of properties such as persistent dynamics, in regimes where direct calculation by approximation methods is infeasible.

We first review the essential definitions and summarize the categories of symmetries and symmetry breaking along with their qualitative effects. We then develop a sequence of worked examples demonstrating each class. These models based on symmetry also provide examples of scale-dependent regression coefficients, different coordinating roles for repetition, or the origin of essential multilevel selection, introduced in the preceding sections.

A. Symmetry breaking by short-term dynamical states and restoration by time averaging

We define the symmetries of a game model as the transformations that leave the fitness and transmission coefficients unchanged. These may be subgroups of permutations among agent types (mirror reflection, or cyclic permutation) known as *point groups*, or they may come from displacing the time coordinate by any of a continuous range of values, forming what is known as a *Lie group* [101].

Symmetries written in as properties of the individual-level transition coefficients may be hidden if population states that determine frequency-dependent fitness become asymmetrical. Such asymmetric states may be rest points, limit cycles, or more complex attractors, onto which the population state converges. Because invariances of the underlying model are not changed by "accidental" properties of its solutions, symmetries hidden from the agent-level transition rates by population states must be expressed as *degeneracies* among the eligible population states. For example, if the fitness function has a reflection symmetry under exchange of two types *L*eft and *R*ight, but the population state converges to either a left/right-asymmetric rest point, then there must be two rest points that are identical under the reflection that either rest point alone masks. The same is true for continuous symmetries, which lead when they are broken to a one-dimensional manifold of solutions along a limit cycle or attractor.

We may think of asymmetry of population states as an extension of the asymmetry that any *sample* may have with respect to its underlying distribution, and doing so provides a useful way to formalize changes of representation. If asymmetries in samples of population states decay rapidly – over timescales comparable to the generation time – then the system dynamics is said to be *ergodic*.[41] If asymmetries in sampled population states persist over times much longer than the individual generation time, the population state is said to *spontaneously break* the defining symmetries of the underlying process.[42]

The fact that hidden symmetry implies *strict* degeneracy of population states ensures autonomous dynamics at the population level.[43] Emergent population dynamics will generally be stochastic, and the stochasticity

[41] The technical definition of ergodicity is complicated to express, because it requires that samples from a dynamical trajectory accumulate to a distribution that is statistically equivalent to some reference distribution representing an ensemble. Here we will be concerned only with the ensembles that reflect the defining symmetries, and beyond this we will not require a more precise criterion for ergodicity.

[42] Symmetry breaking is familiar from (second-order) phase transitions [27, 29, 77] in either equilibrium or non-equilibrium stochastic processes. Spontaneous symmetry breaking *sensu stricto* is defined formally by the existence of a large-system limit in which, asymptotically, asymmetric population states persist forever. Because we can always recover the asymptotic property (if it pertains) as a regular limit of finite-time persistence, we consider all forms of hidden symmetry in finite-time as well as infinite-time samples. The more general case includes many important forms of timescale separation, and the scaling approach to the asymptote contains additional information about the symmetry-breaking transition beyond that present in the limiting distribution itself.

[43] This becomes a *non-trivial* property when the timescale separation between population state-changes and the generation time becomes very large. At a small timescale separation, bifurcations between asymmetric states and those between symmetric states may not seem qualitatively different. Both cases have basins

drives convergence to the ergodic distribution – which restores the expression of the underlying symmetries – under sufficiently long time-averaging. In such long-time averages, fluctuations in the population state become an autonomous variable with fitness consequences at the individual level, which then feed back to re-enforce the stability of population states. Spontaneous symmetry breaking is therefore an important source of multiple units of selection, and of dynamics that is coupled over multiple scales.

B. The major categories of symmetries and symmetry breaking

Symmetry breaking, and restoration of an ergodic distribution by a time or ensemble average, are very general phenomena [27, 29, 58, 77]. The particular symmetry groups affected, however, and the ways they are broken by sets of ordered population states, can vary widely. Each type of symmetry and type of symmetry breaking leads to a qualitatively distinctive kind of dynamics at the population scale. Whether a discrete or a continuous underlying symmetry is broken determines whether the characteristic timescale for population dynamics grows exponentially or only polynomially (or not at all) with population size, relative to the generation scale. This difference affects the convergence of asymptotic expansions and thus the computational methods that can be used. More complex forms of symmetry breaking introduce a new kind of macroscopic dynamics known as "creep", and require qualitatively new calculation methods developed in the theory of glasses. [104, 105].

1. *Simple versus complex symmetry breaking: how does the number of ordered system states scale with the size of the state space?*

The distinction between "simple" symmetry breaking (which we will also term *classical*) and "complex" symmetry breaking (which we will also term *glassy*) is made, not based on single models, but based on the behavior of population states in collections of models among which the number of agents, agent states, or some other variable is changed as a scaling variable. In simple symmetry breaking, the number of ordered population states and the symmetry group that relates them do not change as the scale of the underlying system changes. The classical pitchfork and Hopf bifurcations are examples of this kind.

In complex symmetry breaking, the number of ordered population states grows (typically exponentially) with the scale of the underlying system. The model of linkage disequilibrium among heterotic loci on a chromosome [37], in which the number of heterotic loci is the scale parameter, shows an example of complex symmetry breaking. Complex symmetry breaking in equilibrium systems was first worked out as a description of glasses, and is now used to describe a number of other combinatorial optimization problems [104, 106] in which the constraints on a system cannot all be jointly satisfied, a condition known as *frustration*.

2. *Continuous versus discrete broken symmetries, and the consequences for fluctuations*

When a broken symmetry is discrete, as occurs in the example of reflection symmetry for the pitchfork bifurcation, population-level dynamics occurs by hopping between basins of attraction, with hops becoming exponentially rare in a product of the selection strength with population size. Collective fluctuations must overcome kinetic "barriers", and the problem for fluctuation mechanics to solve is to compute the rates and trajectories of population-dynamic escapes from the parameters of the individual-level interaction.

A very different behavior results when the broken symmetry is continuous, as occurs for the supercritical Hopf bifurcation from a fixed point to a limit cycle. In this example the ordered states must form a continuum, along with the group of symmetries that transform one ordered state into another. No "barrier" impedes the action of collective fluctuations, and these therefore accumulate as Brownian motion along the limit-cycle trajectory [107, 108]. The diffusivity for this Brownian motion scales as a polynomial (in our examples, it is constant) in the population size, rather than exponentially as for the case of discrete symmetry breaking.

The Hopf bifurcation shows a special feature of non-equilibrium stochastic processes, including stochastic evolutionary games: the space of states itself is discrete and its symmetry group is the discrete group of cyclic permutations. The Hopf bifurcation, however, does not break this discrete group, but rather breaks the continuous symmetry of *time translation*. The continuity of time translation implies the degeneracy of positions around the limit cycle through a topological argument, for both the mean population state and its fluctuations, a result

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of attraction, and the non-symmetric case may simply have different occupancy times in different attractors. However, if the population dynamics separates exponentially (in some parameter such as selection strength) from the generation scale, in an asymmetric bifurcation the relative state occupancies will separate exponentially as well. In other words, all but the most frequently-occupied state will come *never* to be occupied, and the population state will effectively become non-dynamical. The observation that many properties of stochastic processes are fragile under exponential separation of scales if they are not protected by symmetries is known as the *hierarchy problem*. It is the origin of the statement in Ref. [52], that stochastically stable equilibria will "typically" be unique, suggesting that exceptions involve some form of fine tuning and should occur rarely. The one class of exceptions in which this violation of typicality is not regarded as finely tuned is the case where it is ensured by symmetries.

that would be difficult to derive directly from the fluctuation expansion. The proof that a form of barrier-free dynamics at the population level is protected by an underlying continuous symmetry is the stochastic version of *Goldstone's theorem* [58].[44]

3. Spontaneously broken symmetry contrasted with externally imposed symmetries

Systems that produce degenerate states through symmetry breaking behave differently from systems in which degeneracy is imposed externally by symmetries of the underlying process. We illustrate this below by contrasting a coordination game with broken reflection symmetry, against a deterministic repeated Prisoners' Dilemma game, in which a continuum of population states is rendered degenerate by a defining (and essential!) symmetry between certain strategy pairs. Whereas the hidden symmetry in the coordination game results in discrete two-scale dynamics at any separation of scales, the imposed symmetry of the repeated Prisoners' Dilemma leads to large fluctuations about a unique average population state. The large fluctuations, however, cause the actual mean state to differ from any limit suggested by the naïve mean-field approximation.

Externally imposed degeneracies are an important cause of *neutrality* in evolutionary dynamics. Neutrality has been studied in the context of genotype-phenotype maps [86–91], and the use of games to model development introduces a way to study neutrality in the genotype-development-fitness mapping. The distinctive consequence of neutrality in stochastic evolutionary dynamics is that *large* collective fluctuations are important determiners of even the average population state. They can cause population-level regressions of fitness on genotype to differ from the payoffs in the underlying normal-form game, by *entropy* corrections that persist even in infinite-population limits. The companion paper shows how such entropic corrections may be systematically approximated, and the results of those calculations (shown below) agree well with stochastic simulations.

C. Simple discrete symmetry breaking and the emergence of multiscale dynamics

Our first example illustrates simple (classical) spontaneous breaking of a discrete reflection symmetry. The mean-field behavior is a well-known pitchfork bifurcation, to which we add an analysis of collective fluctuations. We study the emergence of the population as a distinct dynamical entity, whose asymmetric states lead to group selection by a standard derivation of Hamilton's rule from the Price equation. We also show how discrete symmetry breaking in evolutionary games has a representation in terms of potential functions similar to the energy potentials of equilibrium thermodynamics.

1. Reflection-symmetry breaking in a coordination game

Consider a population in which agents may have types *L*eft (L), *R*ight (R), and *M*iddle (M). We write the instantaneous population state as a column vector

$$\mathrm{n} \equiv \begin{bmatrix} \mathrm{n_L} \\ \mathrm{n_R} \\ \mathrm{n_M} \end{bmatrix}. \tag{25}$$

We will define the affine model of frequency-dependent fitness by the normal-form payoff matrix in the case of random matching,

$$[a] = \bar{a} \begin{bmatrix} 1 \\ 1 \\ 1 \end{bmatrix} \begin{bmatrix} 1 & 1 & 1 \end{bmatrix} + \begin{bmatrix} a & -a & 0 \\ -a & a & 0 \\ 0 & 0 & 0 \end{bmatrix}. \tag{26}$$

This second-order expansion is completely defined by the reflection symmetry between L and R, equal payoff to M from all types, and the existence of a uniform-population Nash equilibrium under all conditions. The parameters $\bar{a}$ and a are otherwise arbitrary, and the qualitative behavior of the model is associated with broad parameter ranges.

Models of this form converge to their mean-field approximations in the large-population (large-N) limit (15). For weak selection, the model has a single ESS at uniform population compositions. For strong selection, the uniform population remains a rest point but becomes a saddle point, while a pair of left and right rest points emerge as the ESS. The set of these symmetric ESS forms the *Stochastically Stable Set* of Ref's. [2, 52].

We solve for these rest points in App. B 1, in terms of two parameters $\bar{\beta}$, $\bar{\gamma}$. The uniform population state corresponds to $\bar{\beta} = \bar{\gamma} = 0$. The two ESS at $\bar{\beta}, \bar{\gamma} \neq 0$ are related to average population numbers by

$$\frac{n_\mathrm{L} - n_\mathrm{R}}{N} = \frac{2\bar{\beta}}{\sqrt{D-1}} \tag{27}$$

and

$$\frac{n_\mathrm{M}}{N} = \frac{1}{D} + \bar{\gamma}\sqrt{\frac{D-1}{D}}. \tag{28}$$

The left-right asymmetry solves the equation

$$\bar{\beta}^2 \left(D - \frac{Na}{D + Na\,(D-1)\,\bar{\beta}^2} \right) = 1 - \frac{D^2}{Na\,(D-1)}. \tag{29}$$

[44] In equilibrium statistical mechanics, Goldstone's theorem ensures the masslessness of particles brought into existence by symmetry breaking of the vacuum. In the stochastic version, it ensures the existence of noisy clocks.

In terms of this solution, the typical excess of $n_{\mathrm{L}} + n_{\mathrm{R}}$ in *either* ESS is given by

$$\bar{\gamma} = \frac{-1}{\sqrt{D\,(D-1)}} \frac{Na\,(D-1)\,\bar{\beta}^2}{D + Na\,(D-1)\,\bar{\beta}^2}. \tag{30}$$

The excess $\bar{\gamma}$ will serve as a measure of the fitness due to relatedness in correlated group states.

Fig. 8 shows dynamical trajectories solving the mean-field flow equations with payoffs (26) and mutation (6). The payoff strength $a = 0.5$ leads to a critical population size $N = 9$ for instability to bifurcation. The figure shows the strong-selection regime at $N = 16$. In this regime, the deterministic solutions from arbitrary initial conditions flow rapidly toward an arc running through the uniform distribution and the ESS. Diffusion is then slowest along this arc, with departure from the saddle point that is exponential in time with a rate constant linear in N, and approach to the stable points with the same characteristics. Analytic solutions for these values are given in App. B.

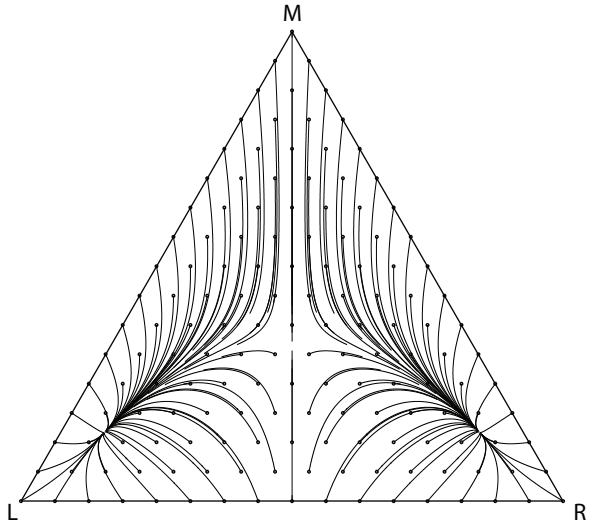

FIG. 8. Discrete symmetry breaking in the normal-form coordination game (26), at $a = 0.5$ and $N = 16$. The simplex of constant N is shown, with the populations of fixed types (L,R,M) indicated at the vertices. Dots represent arbitrary initial values of $\bar{n}$ in the mean-field approximation, and lines are the solutions of the mean-field evolutionary game equation (15) from those initial conditions.

2. *Symmetry breaking in the Price equation and Hamilton's rule*

Hamilton's rule for group selection follows as a general result from the Price equation, whenever the variance within typical group states is smaller than the total variance shown by the population as a whole. The origin of the ensemble over group states does not matter; they may be contemporaneous groups in an island model, or temporally segregated group states of a single population. The transitions in the coordination game (as we will show below) with broken symmetry are typically rapid compared to the time spent in either asymmetric state. Therefore population states at successive times form a natural ensemble of group states, whose frequencies are the same as those sampled independently from an ensemble of model instances. Here we derive the form of Hamilton's rule for the coordination game, following Ref. [35].

We may write the Price equation for the change in population state $\bar{n}$ in a long-time average from times 0 to T, as the sum of terms of the form (14) at single generations,

$$\begin{aligned}
\sum_{\mathrm{n}} \left(\rho_{\mathrm{n},T} - \rho_{\mathrm{n},0}\right) \mathrm{n}_J &= \sum_{t=0}^{T-\Delta t} \sum_{\mathrm{n}} \left(\rho'_{\mathrm{n},t} - \rho_{\mathrm{n},t}\right) \mathrm{n}_J \\
&= \sum_{t=0}^{T-\Delta t} \sum_{\mathrm{n}} \rho_{\mathrm{n},t} \left(\sum_i \left\langle n'_{J|i} \right\rangle_{\mathrm{n}} - \mathrm{n}_J \right) \\
&= \sum_{t=0}^{T-\Delta t} \sum_{\mathrm{n}} \rho_{\mathrm{n},t} \sum_i \mathrm{n}_i \left\{ \delta_{Ji} \langle \mathrm{w}_i - 1 \rangle_{\mathrm{n}} + \sum_j \delta_{Jj} \left\langle \frac{\mathrm{n}'_{j|i}}{\mathrm{n}_i} - \delta_{ji} \mathrm{w}_i \right\rangle_{\mathrm{n}} \right\} \\
&= TN \left\{ \mathrm{Cov}_\rho\big(\delta^J, \mathrm{w}\big) + \mathrm{E}_\rho\big(\mathrm{w} \cdot \Delta\delta^J\big) \right\}.
\end{aligned} \tag{31}$$

Because the population process is Markovian, the update is conditionally independent of t given any state n. The fourth line is a covariance in the time-dependent distribution $\rho_{\vec{n},t}$.

With respect to the same time-dependent distribution,

we may define mean values over the whole interval

$$\bar{n}_J \equiv \frac{\Delta t}{T}\sum_{t=0}^{T-\Delta t}\sum_{\mathrm{n}}\rho_{\mathrm{n},t}\mathrm{n}_J. \tag{32}$$

About the collection of these mean values, the variance of observable δ^J is then

$$V_J \equiv \frac{\Delta t}{T}\sum_{t=0}^{T-\Delta t}\sum_{\mathrm{n}}\rho_{\mathrm{n},t}\frac{1}{N}\sum_i \mathrm{n}_i\Big(\delta_{Ji}-\frac{\bar{n}_J}{N}\Big)^2. \tag{33}$$

If the instantaneous population distributions $\rho_{\mathrm{n},t}$ flip stochastically and symmetrically between rest points, the average $\bar{n}_J$ may be small while the instantaneous averages of the indicator functions δ_{Ji} are large. Thus the relatedness from switching of population states can dominate the transient relatedness from fluctuations about the instantaneous average.

Now we perform a regression for w_i at each time in the same variables as those of Eq. (17), but we require that the set of (fixed) coefficients a_{ij} minimize the squared error in the joint population/time covariance, rather than in the single-time population covariance of Eq. (18).[45] The time-averaged generalization of the regression (18) then becomes

$$\frac{\mathrm{w}_i-1}{\Delta t} = \sum_{kj}\Big(\delta_{ik}-\frac{\mathrm{n}_k}{N}\Big)\left(\alpha_{kj}+a_{kj}\mathrm{n}_j\right)+\mathcal{O}\Big(\frac{1}{N}\Big)+\epsilon_i. \tag{34}$$

We now wish to decompose the covariance term in the long-time Price equation (31). Following Ref. [35] we define a "cost" component for each type, normalized by the variance (33), as

$$\begin{aligned}
-V_JC_J &= \\
&\frac{\bar{n}_J}{N}\frac{\Delta t}{T}\sum_{t=0}^{T-\Delta t}\sum_{\mathrm{n}}\rho_{\mathrm{n},t}\sum_j\Big(\delta_{Jj}-\frac{\mathrm{n}_j}{N}\Big)\left(\alpha_j+\sum_k a_{jk}\mathrm{n}_k\right)\\
&\equiv \frac{\bar{n}_J}{N}\left\langle\sum_j\Big(\delta_{Jj}-\frac{\mathrm{n}_j}{N}\Big)\left(\alpha_j+\sum_k a_{jk}\mathrm{n}_k\right)\right\rangle_\rho\\
&= \frac{\bar{n}_J}{N}\left[\left(\alpha_J+\sum_k a_{Jk}\bar{n}_k\right)\right.\\
&\quad\left.-\frac{1}{N}\sum_j\left(\alpha_j\bar{n}_j+\sum_k a_{jk}\langle\mathrm{n}_j\mathrm{n}_k\rangle_\rho\right)\right].
\end{aligned} \tag{35}$$

The cost measures the fitness contribution from the long-term average population state $\bar{n}$, and in the last line of Eq. (35) we have separated the mean-field approximation from covariance corrections. In the regime of strong selection, the latter will be small.

[45] To reduce clutter in the next several equations, we will collect terms arising from the $-\delta_{ij}$ factor that eliminates self-play into an overall summand at $\mathcal{O}(1/N)$.

In the case where multiple regressions against several predictor types k are used, the "benefit" term in Hamilton's rule is a matrix indexed by "benefactor" k and "beneficiary" J, with components

$$\begin{aligned}
B_{Jk} &\equiv \sum_j\Big[\Big(\delta_{jJ}-\frac{\bar{n}_j}{N}\Big)a_{jk}-a_{kj}\frac{\bar{n}_j}{N}\Big]-\frac{\alpha_k}{N}\\
&= a_{Jk}-\frac{\alpha_k}{N}-\sum_j\left(a_{jk}+a_{kj}\right)\frac{\bar{n}_j}{N}.
\end{aligned} \tag{36}$$

Here "benefit" stands for the difference between the actual payoff coefficients per interaction, and the average in the mean population state $\bar{n}$.

The relatedness between benefactor and beneficiary is also indexed by J and k, and is written as a set of regression coefficients against the variance of the beneficiary, so

$$\begin{aligned}
V_Jr_{Jk} &\equiv \frac{\Delta t}{T}\sum_{t=0}^{T-\Delta t}\sum_{\mathrm{n}}\rho_{\mathrm{n},t}\frac{1}{N}\sum_i\mathrm{n}_i\Big(\delta_{Ji}-\frac{\bar{n}_J}{N}\Big)\left(\mathrm{n}_k-\bar{n}_k\right)\\
&= \frac{\Delta t}{T}\sum_{t=0}^{T-\Delta t}\sum_{\mathrm{n}}\rho_{\mathrm{n},t}\frac{1}{N}\left(\mathrm{n}_J-\bar{n}_J\right)\left(\mathrm{n}_k-\bar{n}_k\right).
\end{aligned} \tag{37}$$

We see from the second line of Eq. (37) that relatedness may be mediated primarily by population state-switching in the realm of strong selection.

Finally we may write the covariance term from Eq. (31) as

$$\begin{aligned}
\mathrm{Cov}_\rho\big(\delta^J,\mathrm{w}\big) &= V_J\left(\sum_k B_{Jk}r_{Jk}-C_J\right)\\
&\quad-\frac{1}{N}\left\langle\left(\mathrm{n}_J-\bar{n}_J\right)\sum_{jk}\left(\mathrm{n}_j-\bar{n}_j\right)\left(\mathrm{n}_k-\bar{n}_k\right)\right\rangle_\rho.
\end{aligned} \tag{38}$$

The least-squares condition for the parameter α_J ensures vanishing of $\mathrm{Cov}_\rho\big(\delta^J,\varepsilon\big)$. In the large-population limit, the third-order variation in the second line of Eq. (38) will be small and asymptotically ignorable compared to terms kept in the first line.

We compute values for C_J, B_{Jk}, and r_{Jk} in App. B 1, and there we also show how the long-term average over population states coarse-grains the type space together with the distribution. The two "types" that are left invariant by reflection, even in the domain of broken symmetry, are the union of L and R types, versus the M types. The fluctuating population state appears within this coarse-graining as a fluctuating phenotype within the $(n_\mathrm{L}+n_\mathrm{R})$-population. Both variants of the fluctuating phenotype share the feature of being capable of coordination which confers benefit when they coordinate on the same phenotype, relative to the M type, who are not capable of coordinating.

3. The large-deviations scaling limit and multiscale dynamics

Systems are said to take on the *large-deviations property* when, under suitable conditions of aggregation, the log-likelihoods for sample fluctuations separate into a scale-independent term that depends only on the structure of the fluctuation, and an overall scale term applicable to all fluctuations [73]. The structure-dependent term is called the *rate function* (or *entropy*) of the large-deviations principle. In this limit, the structure of the state space for aggregate system dynamics has separated from the details of individual behavior, and it becomes possible to regard the aggregate system as a primitive entity in its own right.

Large-deviations scaling is now understood to be the basis for the emergence of thermodynamic descriptions, and the reason that thermodynamics stands as a consistent theory, whether or not it has been derived from an underlying statistical mechanics [109]. The ability to derive rate functions quantitatively (the topic of the companion paper) provides a way to connect models of evolutionary dynamics with multiple scales, in which the same entity appears aggregate at one scale and primitive at another.

4. The role of potentials in representing broken symmetries

The symmetries of potential functions were understood to govern the properties of motion in mechanics with the work of Lagrange and Hamilton [110]. The idea of an *effective potential* whose purpose was entirely to capture the symmetries of states in phase transitions was invented by Landau [77]. Over time it became understood that the energy-scale-dependence of the effective potential came from absorbing fluctuation effects into average parameters [27]. The *free energy landscape* of thermodynamics is just such an effective potential [74]. For this reason, the picture of symmetry breaking in equilibrium systems is almost always conveyed in terms of symmetries of an effective potential.

The use of potentials to visualize symmetry in non-equilibrium stochastic processes is more limited, though they still represent symmetries. A brief summary of the ways in which potentials can be defined for evolutionary games is given in App. A 2. Here we note that, for symmetry-breaking games whose stable trajectories are rest points, it is possible to define a potential function with many of the intuitive properties of the equilibrium potential, including the direct expression of symmetries, and also the control of escape probabilities.

Spontaneous symmetry breaking by pitchfork bifurcation in equilibrium thermodynamics results from effective potentials of the form shown in Fig. 9. The curvature of the wells at their minima determines the strength of mean-regression toward the mean population state and thus the variance of fluctuations. The depth of the wells determines the *rate function*[46] in the large-deviations formula for the rate of escapes from one well to the other.

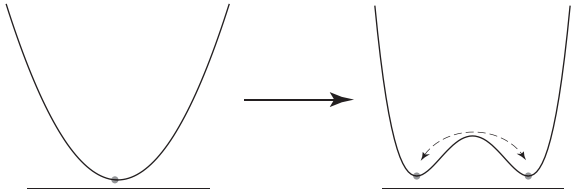

FIG. 9. In equilibrium, the minima of a potential determine the rest points of dynamics, the gradients of the potential in neighborhoods of its minima determine rates of mean regression, and the barrier of the potential between wells determines the leading exponential dependence of escape rates. Phase transitions are described by changes of the potential from the convex form shown on the left to the non-convex form on the right, as an interaction strength is increased. As the wells of an equilibrium potential deepen, the fluctuations in population states are suppressed [6] and the dynamics becomes increasingly well-described by a two-state, continuous-time Markov process, suggested by the dashed line with arrows.

The depths and curvatures of the two wells generally depend on population size N, so the regression rates will generally depend linearly on N and the escape times will depend exponentially on N. Thus, these *quantitative* properties of dynamics will not be independent of the system scale. The underlying reflection symmetry ensures, however, that the minima of the two wells are exactly degenerate at any N. Therefore, unlike the escape time, the relative occupation frequency in these wells does *not* grow exponentially with N, allowing population-scale dynamics at any N.[47]

The quantity we call the "kinematic" potential for the stochastic process of the coordination game is shown in Fig. 10. The white dashed contour is drawn along the saddle of the potential between the stable and saddle rest points. It closely approximates the attracting contour of slowest flow in Fig. 8. We compute escape trajectories in the companion paper, and show that these closely approximate the slow-diffusion contour but travel in the opposite direction.

It can be shown that, in the dynamical system of Freidlin-Wentzell theory that we use to compute es-

[46] See Ref. [73] for the definition of the scale factor and rate function in a large-deviations rate formula. For all of the cases of classical symmetry breaking demonstrated in this review, the scale factor is population size N.

[47] The absence of a protecting symmetry was used in Ref's. [2, 52, 59, 75], along with a selection-strength relaxation protocol analogous to simulated annealing in Ref. [59], to provide a further equilibrium refinement beyond ESS. This equilibrium refinement works because, generically, all but the lowest effective minimum becomes depopulated. The effective minimum, however, depends on non-local properties of the basin of attraction, and therefore requires the inclusion of fluctuation corrections to compute reliably.

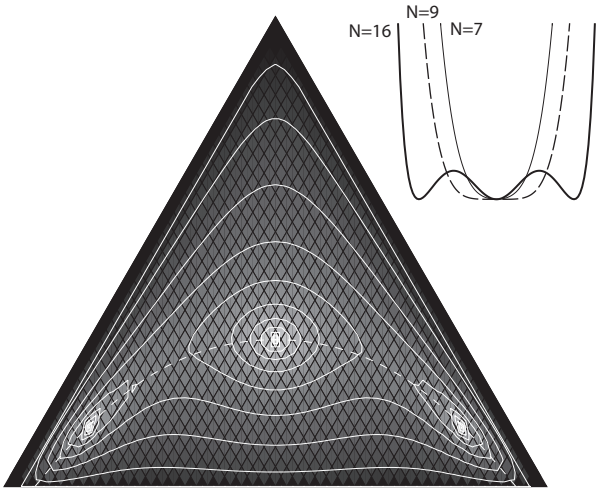

FIG. 10. The "kinematic" potential $V(\nu)$ defined in Eq. (50) of the companion paper, for the coordination game at the parameters shown in Fig. 8. The greyscale and contours are uniform in $\log V(\nu)$. Dashed white line is drawn to follow the saddle of $V(\nu)$, and closely approximates the direction of slowest flow in the diffusive field of Fig. 8. It also approximates the most probable trajectory for escapes. (Inset) Linear plot of the potential along the dashed white contour at three values of N: $N = 7$ stable uniform rest point, $N = 9$ the classical critical value for phase transition, and $N = 16$ the ordered phase of Fig. 8. The symmetry breaking of the potential resembles that shown in Fig. 9 for equilibrium systems, except that in the ordered regime both the saddle point and the stable rest points are zeros.

capes [65], the dynamics of games with broken discrete symmetries converge in the sense of large-deviations theory to lie within one-dimensional *heteroclinic networks* [111, 112]. The orbits of these networks are the deterministic diffusion paths from saddle to stable rest points, and the escape trajectories in the opposite directions. The heteroclinic networks are well approximated for this game by the paths along the saddle of the potential of Fig. 10.

The inset in Fig. 10 shows that the kinematic potential shares the reflection symmetry of its thermodynamic analog, and likewise shows the degeneracy of rest points under change of N through the critical value. It also shows an important respect in which escape trajectories in irreversible stochastic processes differ from those in equilibrium thermodynamics.[48] Equilibrium escapes run between the two minima of the effective potential in Fig. 9, and their motion is fastest at the maximum of the potential which is an unstable equilibrium.[49] Escape trajectories in the stochastic process pass through the saddle, and both enter and exit exponentially slowly compared to their passage through the intermediate configurations. The kinematic potential of Fig. 10 correctly represents these differences, so that the relation of the escape trajectory to the potential is mathematically the same as in equilibrium. The probabilities of escapes have further asymmetries that are not captured in the kinematic potential, which are explained in the companion paper.

5. *Weak and strong selection*

Characteristic timeseries for agent numbers n_L/N and n_R/N, from samples of stochastic simulations of the model (26), are shown in Fig. 11 and Fig. 12. These simulations illustrate the transition from the weak-selection regime $N \gtrsim 9$ to the strong-selection regime $N \gg 9$.

Fig. 11 shows the behavior of n_L/N and n_R/N in a neighborhood of the bifurcation point. In this regime, fluctuations involving large fractions of the population are common, and transitions between the basins of attraction of the two ESS are frequent.

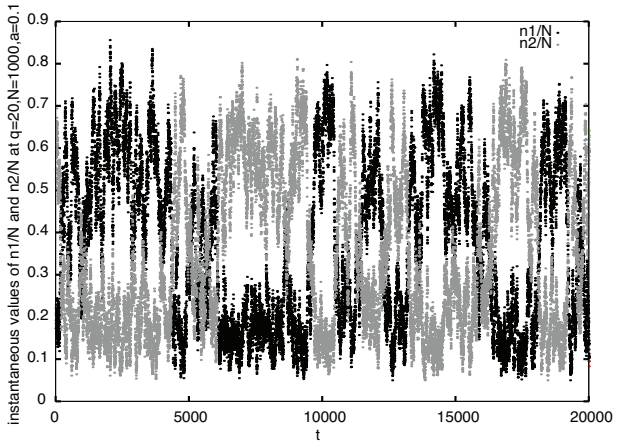

FIG. 11. Characteristic timeseries of two number components n_L and n_R for weakly broken symmetry, in a simulation of the stochastic game model (26). The average occupancies are asymmetric between domains of high-occupancy and low-occupancy, because the numbers n_L and n_R project differently onto the ESS in which they are dominant and in which they are marginal. Fluctuations are also asymmetric between high- and low-occupancy, because the projections onto the saddle path of slowest mean-regression differs.

Fig. 12 shows corresponding timeseries, over the same number of generations as Fig. 11, for a regime of strong

[48] Both of these are known as *instantons* [58], and their mathematical treatment is similar despite different properties of equilibrium and non-equilibrium models.

[49] Escape trajectories are all exponentially improbable paths, compared to fluctuations about the mean population state. Their definition is that, conditional on the occurrence of an escape, they are the least-unlikely trajectories to have mediated that escape..

selection. The typical residence time in domains is now much longer than the time-window shown, so this window was selected to include one such transition. The magnitude of fluctuations has decreased due to strong mean regression, as indicated by the curvatures of the kinematic potential in Fig. 10. More importantly the frequency of large excursions has been suppressed relative to that of smaller ones, leading to rare but sharp transitions between basins of attraction. The distribution over individual types is thus tightly entrained by the mean population state, and this distribution changes as a "coalition".

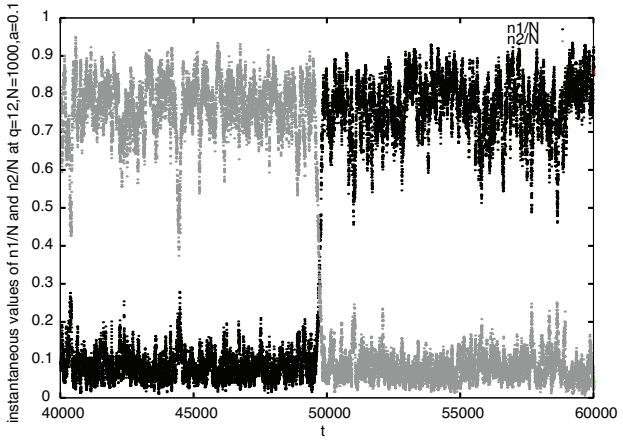

FIG. 12. Characteristic timeseries of number components n_L and n_R for strongly broken symmetry, in comparison to Fig. 11. Population variance about either rest point is now much smaller than the separation between the two ESS, causing the large excursions that lead to escapes to become exponentially rare with N.

Fig. 13 shows the effect of the transition between weak and strong selection, on the time-averaged description of the population state. A moving window was used to time-average $(\mathrm{n}_L - \mathrm{n}_R)/N$, corresponding to the T-generation average of Eq. (31). We denote this average $\langle \mathrm{n}_L - \mathrm{n}_R \rangle_T / N$. The ensemble-average of this quantity over window positions is then plotted in absolute value versus $1/N$. In the realm of weak selection, any window long enough to suppress fluctuations within a basin of attraction has high probability to include a transition, and the ensemble mean of $|\langle \mathrm{n}_L - \mathrm{n}_R \rangle_T| / N$ falls rapidly to zero. Thus even short time averages fail to reflect the analytic solution for the rest points given in App. B, shown in the solid line. The point of departure of the time-averaged population statistic from the analytic result depends sensitively on window length in this weak-selection regime.

In the regime of strong selection, population states are persistent, permitting long windows to average over Poisson fluctuations about the population mean while rarely including transitions. In this regime, a time window shorter than the typical persistence time readily identifies group states as dynamical variables in the two-state

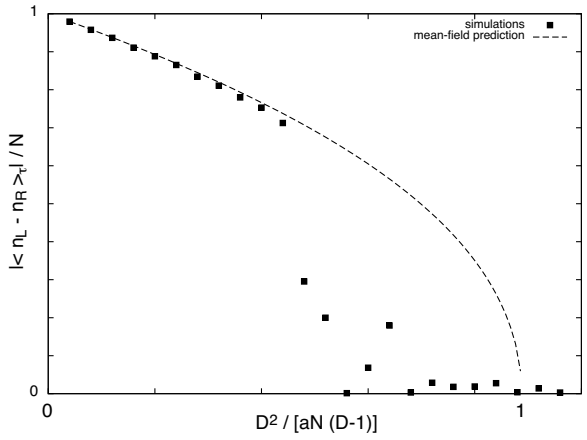

FIG. 13. Short-term breaking and long-term restoration of symmetry. The mean-field approximation for asymmetry plotted against the time-averaged population composition in samples. Average population asymmetry $\left|\langle \mathrm{n}_L - \mathrm{n}_R \rangle_\tau\right|$, over windows of length $\tau/\Delta t$ generations, is plotted versus $D^2/[aN(D-1)]$, which takes value 1 at the mean-field critical point. When state changes have rate $\gtrsim 1/\tau$, the time-average $\left|\langle \mathrm{n}_L - \mathrm{n}_R \rangle_\tau\right|$ (symbols) approximates the symmetric, ergodic distribution, departing from the analytic formula (dashed line) for mean asymmetry given by Eq. (29) – even though the typical population state is asymmetric as shown in Fig. 11. When state changes have rate $\ll 1/\tau$, $\left|\langle \mathrm{n}_L - \mathrm{n}_R \rangle_\tau\right|$ captures the time-dependence of a two-state stochastic process (symbols follow the line). The sample average begins to deviate from the analytic form when the typical residence time is comparable to the window timescale. For weak symmetry breaking as in Fig. 11, changes in τ of order unity result in visible shifts of the transition point on the diagram. For strong symmetry breaking as in Fig. 12, changes of τ by factors $\gg 1$ are required to shift the transition point, corresponding to the exponential dependence of the residence times on N.

Markov process. The persistence time, which grows exponentially with population size in the strong-selection regime, sets the rate of convergence to the ergodic distribution over L/R population states. The group-level description is not sensitively dependent on window length, because a large separation of timescales has opened between Poisson fluctuations whose frequencies are polynomial in $1/N$, and escapes whose frequencies are exponential in $-N$.

6. *Generic group selection versus literal kin selection*

Group selection is defined by the need to include the relatedness r_{jk} in the covariance term (38) of the Price equation, in order to generate the correct population dynamics. Whether or not relatedness depends on descent of mutants from a common ancestor – hence on literal kinship – depends on the particular scaling behavior of first-passage times, as a function of the number of types, the strength of selection, and group size.

Increasing the number of types increases the importance of kinship, by standard Muller's-ratchet arguments for the infinite-alleles model.[50] An important property of independent-mutation models of this kind is that first-passage time scales exponentially in both selection strength and population size. The number of concurrent new mutants does not depend on selection strength, and depends only linearly on population size. As long as these scaling properties of the model are roughly preserved, we expect that, among states with sufficient mutants to tip the population, the likelihood that almost all mutants are kin will increase exponentially with both selection strength and population size.

7. *The transition from relatedness to the emergence of coalitional behavior from non-cooperative interactions*

The frequency with which subsets of a population deviate from the mean population behavior (in these examples) becomes exponentially rare in the regime of strong selection, which is the essence of the large-deviations principle [26, 73]. When the evolutionary game equation has a unique solution, the suppression of deviations describes convergence to an adaptive optimum. When broken symmetry requires multiple population states and autonomous dynamics, we may view the population as a *coalition*, and the convergence of individual behaviors to the population mean as a mechanism for the emergence of coalitional behavior from non-cooperative behavior.

The ability of coalitions to entrain the strategy choices of individuals is the basis of *cooperative solution concepts* in strategic game theory [22, 24]. Like other aspects of game structure, the mechanisms that define coalitions, such as side-payments to share game payoffs, are assumed to be exogenous and given. Symmetry breaking provides one mechanism to endogenize the formation of coalitional behavior, and may provide an instance in which evolutionary game theory provides a mechanistic underpinning to strategically useful concepts. We provide an explicit map from the shared benefit of coordinated group states to a form of side-payment in App. B 1 c

In two respects, however, the particular mechanism of symmetry breaking may miss concepts of importance in cooperative game theory. Symmetry breaking will not generally lead to rest points for all possible combinations of coalitional action, so that concepts such as the core of a cooperative game may not be fully represented.[51] Likewise, since coalition formation is given in cooperative game theory, it has no characteristic timescale or inertia, and switching of the population is effectively free between any coalitional states. The long timescales for stability of population states that accompanies suppression of deviations may fail to capture this flexibility. An important problem in systems ranging from neuronal memory [113] to social dynamics [38] is how slow-timescale variables may emerge which confer order on short-term individual behavior, but which may also rapidly switch in response to particular sets of local events.

D. Simple continuous symmetry breaking and a new role for time in non-equilibrium processes

The next example illustrates simple (classical) breaking of a continuous symmetry. We extend the well-known mean-field behavior of Hopf bifurcation from a fixed point to a limit cycle to include the role of collective fluctuations. The type space in this model has no continuous symmetries, yet the Hopf bifurcation results in a continuous set of solutions because the broken symmetry is continuous time translation. The stochastic Goldstone theorem for continuous broken symmetry ensures that as long as the winding number (a topological feature) of the limit cycle is not broken, the ordered state will represent the continuous symmetry of time in both the mean field and the fluctuations, rather than the discrete symmetry of the type space.

1. *Continuous degeneracy of the order parameter even in discrete type spaces*

The simplest game with a supercritical Hopf bifurcation is the totally-symmetric Rock-Paper-Scissors (RPS) game [4].[52]

Types are indexed (R,P,S), and the population state is denoted

$$\mathrm{n} \equiv \begin{bmatrix} \mathrm{n_R} \\ \mathrm{n_P} \\ \mathrm{n_S} \end{bmatrix} . \tag{39}$$

The normal-form payoff matrix assuming random matching is [8]

$$[a] = \bar{a} \begin{bmatrix} 1 \\ 1 \\ 1 \end{bmatrix} \begin{bmatrix} 1 & 1 & 1 \end{bmatrix} + \begin{bmatrix} & -a & b \\ b & & -a \\ -a & b & \end{bmatrix} . \tag{40}$$

[50] Such example games should be understood to represent minimal representations *after* good gene compressions have been found. Two factors that increase the number of relevant types are the number of alleles for a given relevant locus, and the degree of pleiotropy of the trait, which may require the indices i in the model to represent high-order associations of genes, resulting in high type numbers even if the number of alleles of each gene is small.

[51] The core, like the feasible set in repeated game theory, emphasizes maximum strategic flexibility. If restrictive mechanisms are desired which enable the formation of only a subset of coalitions, symmetry breaking may be applicable.

[52] Similar conclusions apply, however, to a wide variety of stochastic processes with limit cycles, and many of these have been developed in reaction-diffusion theory [107, 108].

This form is uniquely specified, up to the magnitudes of a and b, by antisymmetry of payoffs and invariance under cyclic permutation of the agent types.

The RPS model, like the coordination game, converges to its mean-field limit in all respects besides the accumulation of noise along the limit cycle in the broken phase. The bifurcation in MFA is derived by introducing a radius variable

$$r^2 \equiv \frac{\bar{n}_{\mathrm{R}}^2 + \bar{n}_{\mathrm{P}}^2 + \bar{n}_{\mathrm{S}}^2}{N^2} - \frac{1}{D}. \tag{41}$$

Evaluating Eq. (15) for $\log r^2$ gives

$$\frac{d \log r^2}{dt} = (a-b)\, N \left[\frac{1}{D} - \frac{2D}{(a-b)\, N} + \mathcal{O}(r) - r^2 \right]. \tag{42}$$

The term denoted $\mathcal{O}(r)$ is oscillatory in the angular coordinate on the simplex, and does not accumulate over time, as revealed by the simple Floquet analysis in App. C.

From Eq. (42), $a < b + 2D^2/N$ gives a unique, stable, static equilibrium at $r = 0$ (the uniformly mixed population). When $a > b + 2D^2/N$, the uniform population becomes unstable, and all solutions to Eq. (15) converge to a limit cycle, as shown in Fig. 14. Near the bifurcation, the cycle is approximately circular, with a mean value given by

$$\bar{r}^2 \approx \frac{1}{D} - \frac{2D}{(a-b)\, N}. \tag{43}$$

Mutation is responsible for the stability of the limit cycle along an interior trajectory, which otherwise would accumulate to the simplex boundary.

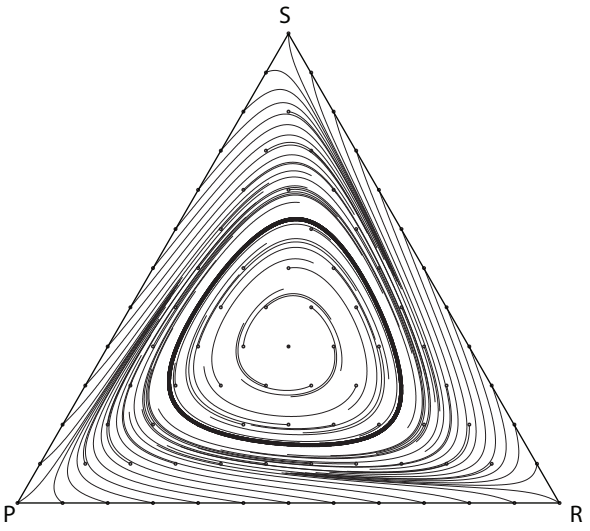

FIG. 14. The standard limit cycle for the rock-paper-scissors game (40) with parameters $a = 0.7$, $b = 0.2$, and $N = 48$. Dots represent initial mean states $\bar{n}$, and light lines are solutions to Eq. (15) from those initial states. Heavy line is the limit cycle, to which all solutions converge.

2. *Symmetries governing the Hopf bifurcation act on a space of histories*

Fig. 14 shows dynamical trajectories solving the mean-field flow equations with payoffs (40) and mutation (6). The projection onto the type space makes it difficult to see, from symmetry alone, why the ordered population state should follow a limit cycle or why fluctuations should not cause the cycle to get "stuck" in one of the corners, so that the ordered states would respect the point-group symmetry of the type space.

Fig. 15 shows the relevant symmetries of the problem by embedding population states in a 3-dimensional space of both types and time. As developed at length in Ref. [74] and in the companion paper, the elementary entities to which non-equilibrium stochastic processes assign probabilities are not single-time configurations, but entire *histories* extended over time. In Fig. 15, the history representing the limit cycle is a spiral path that winds around the symmetric fixed point, and which may be periodically identified in time at integer multiples of the limit-cycle period. The symmetry that transforms different ordered histories is translation in *time* (vertical displacement in the graph). Because the ordered histories couple types and time, this translation maps to a rotation on the projected limit cycle as well.[53] The *winding number* of any ordered history about the vertical axis through the unstable rest point is a topological feature of the ordered history, which must change by "breaking" the path in order for the continuous circle of trajectories to be replaced with a discrete set transformed by the point-group symmetry of the type space.

3. *Stochastic Goldstone's theorem and noisy clocks*

The quantitative derivation of the stochastic Goldstone theorem requires the dynamical-system representation from the Freidlin-Wentzell theory [65] for stochastic processes, which we derive in Sec. III B 3 of the companion paper. The theorem states that the cumulant expansion in the distribution for fluctuations has zero-eigenvalue modes corresponding to the direction of symmetry around the limit cycle. We derive only low-order approximations to this cumulant expansion, to produce quantitative estimates for fluctuations transverse to, and

[53] To appreciate that it is the time translation which is fundamental, note that the time translation acts *uniformly* on the spiraling history. If we wish to project this into an equivalent map on the limit cycle in the base space, the map is not a uniform advance along the line element of the limit cycle, but one that "stretches" or "compresses" different segments to reflect the different rates of advance along the cycle.

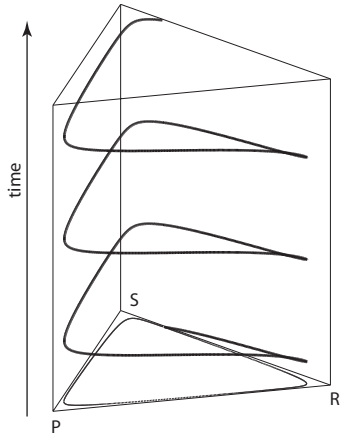

FIG. 15. The order parameter in a space of histories, corresponding to a limit cycle, is an extended-time path. The projection of the limit cycle onto the space of types is shown in the base plane of the graph. For a limit cycle near the boundary of the RPS simplex, the cycle slows at the vertices, as shown by vertical inclination of the trajectory. Yet time translation generates the set of all cycles, unless a topological phase transition changes joint symmetry of time translation and winding around the cycle.

Brownian motion along, the cycle, which we compare to simulations in the next section. It is well-understood that low-order approximations to cumulant expansions are not by themselves reliable, especially for exact cancellations such as zero eigenvalues. The hidden symmetry of time-translation implies, however, that these zero eigenvalues exist at *all* order of approximation, and even when the approximate expansion in $1/N$ fails to converge, as long as the topological winding number of the average ordered history persists.

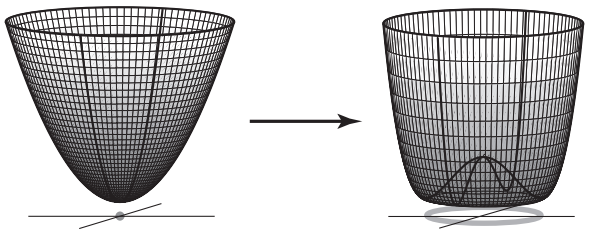

FIG. 16. Spontaneous symmetry breaking in a two-dimensional equilibrium system. The exact degeneracy of a continuous, one-dimensional loop of solutions requires a rotational symmetry in the underlying potential. Cross-sections of the potential are otherwise equivalent to the one-dimensional case of Fig. 9. The exact degeneracy around the loop in the equilibrium potential ensures that arbitrarily small energies can reach any minimum, independent of the steepness of the mean regression in the radial direction. These most-accessible deformations are known as the *Goldstone modes* in the ordered state.

The analogy of the limit cycle in RPS to a continuous circle of degenerate ordered states that leads to Goldstone's theorem for equilibrium thermodynamics is limited. Ordinarily time-translation symmetry is not broken by the solutions of equilibrium thermodynamics.[54] Therefore, in order for a continuous symmetry to emerge among spatial ordered states, the potential would need to have the form shown in Fig. 16. The type space of RPS has no such rotational symmetry, and the kinematic potential corresponding to Fig. 10 for reflection symmetry shows a single zero, at the unstable rest point for the uniform population. It also does not have constant value along the spatial projection of the limit cycle.

4. Quantitative results for fluctuation strength in unbroken and broken RPS

If we expand the instantaneous radial coordinate in the RPS simplex about its mean value as $r \equiv \bar{r} + r'$, with $\bar{r}$ satisfying Eq. (43), we may use the results of the companion paper to estimate the fluctuation magnitude for r' in either the broken or unbroken phase.

In the unbroken phase where $\bar{r} \equiv 0$, the estimate is

$$\left\langle (r')^2 \right\rangle = \frac{2D}{N} \frac{\bar{a} - \frac{a-b}{3} + \frac{1}{N}}{\left| a - b - \frac{2D^2}{N} \right|}. \tag{44}$$

In the broken phase, fluctuations must be divided into two components. For weakly broken symmetry, where the limit cycle is nearly circular and r' describes a radial fluctuation, we may write $\bar{r}\theta'$ for fluctuations in the direction tangential to the limit cycle.

The quantitative consequence of Goldstone's theorem is that radial fluctuations regress, producing a stationary distribution independent of initial conditions at late time. In contrast, tangential fluctuations accumulate linearly in time, and are therefore described by a diffusion constant and must also be referenced to an initial distribution. The quantitative relation between the width of the radial distribution and the diffusion constant for the Brownian motion is given by

[54] Ordinarily equilibrium thermodynamics is *defined* through physical context, as a theory of time-independent states. However, the mathematics of equilibrium may readily be extended by analytic

$$\left\langle \begin{bmatrix} r' \\ \bar{r}\theta' \end{bmatrix} \begin{bmatrix} r' & \bar{r}\theta' \end{bmatrix} \right\rangle_t \to \begin{bmatrix} 0 & \\ & 1 \end{bmatrix} \left\langle \begin{bmatrix} r' \\ \bar{r}\theta' \end{bmatrix} \begin{bmatrix} r' & \bar{r}\theta' \end{bmatrix} \right\rangle_{t'} \begin{bmatrix} 0 & \\ & 1 \end{bmatrix} + \left(\bar{a} - \frac{a-b}{3} + \frac{1}{N} \right) \begin{bmatrix} \frac{D}{2N} \frac{1}{\left| a-b-\frac{2D^2}{N} \right|} & \\ & t-t' \end{bmatrix} \quad (45)$$

Fig. 17 compares time averages of simulation results for the squared radius $\langle r^2 \rangle$ to these expressions. In the unbroken phase, fluctuations dominate the average, and agree closely with the Gaussian-order estimate of Eq. (44). In the broken phase, $\langle r^2 \rangle$ is dominated by the limit cycle itself, which we estimate from numerical simulations of the solutions to the mean-field dynamics (15).

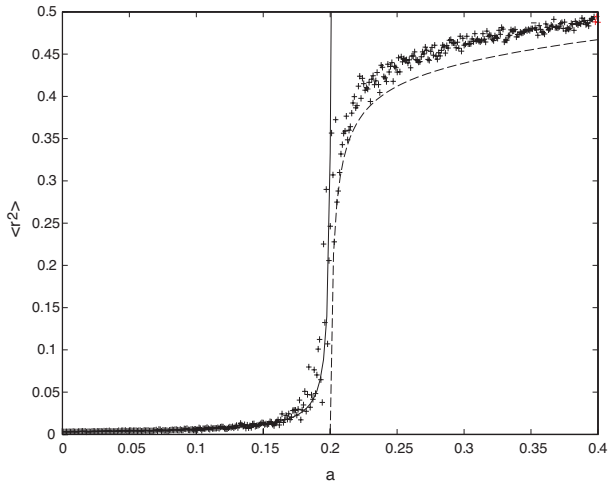

FIG. 17. Comparison of fluctuation results from analytic estimates and simulations. Time-averaged squared radii $\langle r^2 \rangle$ (crosses), sampled at all a values, are well-defined without regard to the circular approximation to the Frenet frame. Eq. (44) for radial variance in the unbroken phase (solid) agrees closely. In the broken phase, where most limit cycles are close to the periphery and the Gaussian fluctuation approximation (45) is poor, time averages $\langle r^2 \rangle$ over an integer number of limit cycles are computed numerically (dashed line). These underestimate stochastic $\langle r^2 \rangle$ by omission of variance terms. Here $b = 0.2$ and $N = 10000$.

Certain features of this model lead to a rapid transition away from the critical point and into the strongly non-gaussian regime of fluctuations near the boundary of the simplex, making quantitative comparison to the radial and tangential estimates of Eq. (45) difficult. If we insert Eq. (43) for the mean $\bar{r}^2$ on the limit cycle, into Eq. (45) for $\left\langle (r')^2 \right\rangle$, we obtain the result that for large N,

$$\left\langle (r')^2 \right\rangle \approx \frac{\bar{a}}{(2D\bar{r})^2} \quad (46)$$

near the critical point, asymptotically independently of N. For the parameters in Fig. 16 and $\bar{a} \sim 1$ (needed so that $\bar{a} \gtrsim a$), $\bar{r}$ only becomes large enough for $\sqrt{\left\langle (r')^2 \right\rangle} \ll 1$ near the simplex boundary. At interior positions of the limit cycle, large fluctuations fill most of the simplex. This feature, together with the coordinate singularity at $r = 0$, makes numerical assignment of fluctuations to the Frenet frame[55] ambiguous. The Gaussian approximation for fluctuations is therefore corrected by boundary terms, until the limit cycle enters the strongly non-linear regime near the boundary. A thorough treatment of the oscillatory diffusion constants of very similar models in this non-linear regime is given in Ref. [107, 108], so we do not duplicate that analysis here.

E. Collective fluctuations that impact model estimation and model interpretation

We next consider a model in which no symmetries are spontaneously broken, but a symmetry is imposed as a defining feature of the model, which makes both the play and the fitness of two types of agents indistinguishable when only they occur in a population. Exogenous symmetries of this kind are less widely modeled with evolutionary game theory than the bifurcations of the last two sections, because they result in mean-field approximations that show *no fitness difference* between agent types in certain population contexts. Therefore the MFA itself does not define a solution. The problems created by such *neutrality* in the fitness map are widely understood, but to our knowledge, a systematic solution of these problems based on symmetries and collective fluctuations has not been carried out.

We choose a canonical form of the repeated Prisoners' Dilemma (RPD) game [8] as our example because it touches many of the key concepts that arise from the use of games to model evolution and development. Repetition redirects the force of selection to favor high penetrance of a cooperative move C, which is strictly dominated in the stage game, but neutrality in the normal

continuation to the range of thermodynamically reversible dynamics, and systems in this domain may break time-translation symmetry [114, 115]. The paired state of superconductors may also be said to break time-translation, in the collective phase of the pair wave function, though this is not an observable under most conditions [116].

[55] The Frenet frame is the coordinate system instantaneously tangent of the limit cycle, explained in App. C.

form of the repeated game is essential to this result.[56]

At the same time as neutrality is essential, it is *fragile*. The formally undefined solution of a strictly neutral evolutionary game can be regulated with mutation, which introduces naïve "defector" types as a kind of *police* to stabilize a mixed, cooperative population. However, in the large-population limit, the police do no work, and the mean-field equilibrium based on their function is not, in fact, the equilibrium about which the population settles. We compute the correct equilibrium, which is stabilized not by the vanishingly small mean fraction of police, but by large fluctuations among all types in the population. This role of fluctuations leads to a normal-form expansion for fitness with the properties of RPD either at the individual or the population-average level, but with different coefficients at the two levels.

The other fragilities stemming from a solution that requires neutrality come from non-deterministic moves (called "trembles") in the repeated game, and from crossover leading toward linkage equilibrium if we model the game moves as genes. We explore the consequences of trembles, and develop a small combinatorial example to suggest that neutrality of this form will become typical in models with large extensive-form games. Finally, we consider the dynamical maintenance of linkage disequilibrium in RPD, and show why fourth-order associations in the regression model for fitness are essential to explain linkage and hence the maintenance of neutrality.

1. *Externally-imposed symmetries: neutrality and the fragility of the mean-field replicator equation*

The normal-form Prisoners' Dilemma is the simplest non-cooperative game in which a Pareto-superior strategy profile (C, C) is strictly dominated by the Pareto-inferior profile (D, D) [117]. Therefore it has become the most widely-used stage game against which to study what can be accomplished with repetition. The finitely repeated Prisoners' Dilemma (RPD) in evolutionary game theory uses a combination of recursively-defined strategies in an extensive-form game, with a replicator dynamic, to achieve high penetrance of (C, C) as the move profile that determines population-averaged fitness. It may be understood as showing how repetition together with evolutionary stability can select Pareto superiority over stage-game dominance, while remaining within the space of non-cooperative solution concepts.

Minimal RPD is played by two memory-zero strategies labeled ALLC and ALLD, identical to C and D in the stage game, and a single memory-one strategy. The zero-memory strategies are defined on the maximally coarse-grained tree of Fig. 2, and the memory-one strategy uses only opponent's move in the previous round, and so is one of the six additional strategies on the coarse-grained game tree of Fig. 5. The memory-one strategy, known as Tit-for-Tat (TFT) [62], consists of an opening move C, followed by a recurrent-stage move that copies the opponent's move in the preceding round. The subset of moves from the finite-state machine of Fig. 7 – with an initial state added – that are visited by *deterministic* RPD are shown in Fig. 18.[57]

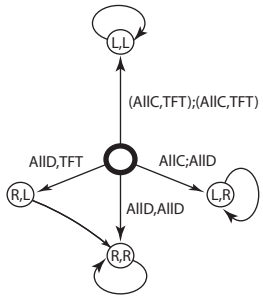

FIG. 18. Subset of transitions from the finite-state automaton (FSA) of Fig. 7 produced by the deterministic repeated Prisoner's Dilemma. Each possible *pair* of information sets Fig. 7 corresponds to a state of the FSA. An initial state (heavy circle) represents the state of play before either player has moved. The moves out of this state correspond to the moves from information sets 0 and 1 in Fig. 7. After any stage has been played, since only memory-one is possible, pairs of information sets are uniquely labeled by the pairs of moves that lead to them, in the same manner as sufficient statistics are associated with symbolic histories in [103, 118]. (Player roles are symmetric, and only ordered matches are shown to simplify the diagram while emphasizing strategy asymmetries.)

The population state in RPD is denoted by a vector

$$\mathrm{n} \equiv \begin{bmatrix} \mathrm{n_C} \\ \mathrm{n_D} \\ \mathrm{n_T} \end{bmatrix}. \tag{47}$$

The normal-form payoff, following Rapoport's notation [117], is given by

$$[a] = \bar{a} \begin{bmatrix} 1 \\ 1 \\ 1 \end{bmatrix} \begin{bmatrix} 1 & 1 & 1 \end{bmatrix} + \begin{bmatrix} R & S & R \\ T & P & P + \epsilon_{TP} \\ R & P + \epsilon_{SP} & R \end{bmatrix}. \tag{48}$$

The payoffs R, S, T, and P, for moves C and D are those of the stage game. The corrections to infinitely-repeated play when TFT plays D, $\epsilon_{TP} \equiv (T - P)/N_{\mathrm{ply}}$ and $\epsilon_{SP} \equiv (S - P)/N_{\mathrm{ply}}$, account for first-round effects.

[56] The neutrality is between a a naïve C strategy and a *defensive* strategy that can protect both naïve C and itself.

[57] We consider the effect of "trembling-hand" moves in the next subsection.

The equality of the four R entries in Eq. 48 (any combination of ALLC and TFT) is the important feature that distinguishes deterministic RPD from the Prisoners' Dilemma stage game. This feature makes a *neutral space* of all population states with $n_D = 0$. The connection of these population states by mutation and replication makes the space into a *neutral network* [86–91]. Neutrality is an externally imposed symmetry of the model. Together with the ordering of payoffs R, S, T, and P for the stage game, these define the qualitative features of minimal RPD.

Fig. 19 shows solutions to the mean-field dynamics (15) with payoffs (48). For the parameters used, the corrections ϵ_{TP} and ϵ_{SP} are ignorable compared to mutation, and the ratio of selection strength to mutation rate is set by N. Neutrality between ALLC and TFT results in a contour of slowest diffusion (the concentrating contour for flow lines) toward the rest point of the classical equations. Comparing the two panels with $N = 100$ versus $N = 300$ in Fig. 19 shows that the speed of approach along this line becomes slower (flowlines bend more sharply into it) and the offset of the line from the pure C-T axis smaller, both $\sim 1/N$. The asymptotically zero MFA flow at $1/N \to 0$ is responsible for large fluctuations between ALLC and TFT (comprising finite fractions of the population) in the stochastic model.

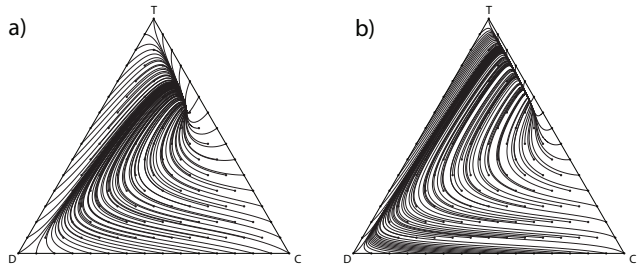

FIG. 19. Repeated play of the standard Prisoners' dilemma between types ALLC, ALLD, and TFT, labeled C, D, and T. Neutrality leads to one vanishing eigenvalue of the deterministic replicator, and a line of absorbing states between ALLC and TFT. Finite mutation rate produces an interior solution, in which a small number of ALLD types distinguish ALLC from TFT. Parameters are $R = 0.6$, $S = 0.1$, $T = 0.8$, $P = 0.3$, and $N_{\rm ply} = 150$ in both panels. Number of players – $N = 100$ (a) versus and $N = 300$ (b) – determines the convergence of the slow-flow line and rest point toward the C-T axis.

2. *Collective fluctuations relate model parameters across scales*

The speed of approach toward a rest point in the deterministic evolutionary game equation determines the rate of mean regression for fluctuations in the stochastic process. For a fixed rate of random type change, the half-width of fluctuations is inverse to the rate of mean regression. The convergence scales as $1/N$, while the population size scales as N along the direction of slowest approach in Fig. 19. Therefore, these two scaling effects cancel and in the large-population limit, fluctuations of a finite *fraction* of agents characterize the stationary distribution at large N.

The self-consistent average of the flow equations for mean population state, in its own fluctuation distribution, are derived in Sec. III C of the companion paper. Fig. 20 shows the consequences of fluctuation corrections to the mean large-N rest points.

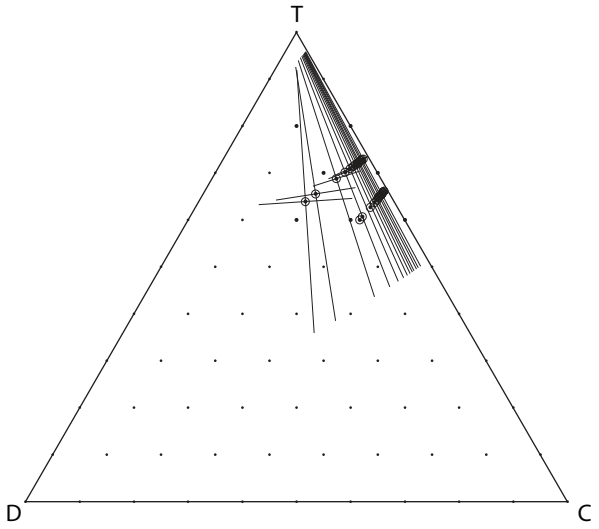

FIG. 20. Self-consistent solutions for rest points of the mean behavior of the (infinite-population) stochastic replicator with neutral directions (points with crosses) show systematic deviation from the rest points of the deterministic replicator (points without crosses).

The circles without axes drawn through them in the figure are the rest points corresponding to Fig. 19, for a sequence of increasing N.[58] The mean population compositions, when self-consistently averaged over fluctuations, are shown as the circles with crosses. The crosses identify both the major and minor axes of the noise covariance as it would appear in a Langevin equation for this system (heavy small axes), and the resulting fluctuations in population state from the combination of noise accumulation and relaxation (light large axes). Both sequences have regular limits at large N, but if the observed limits were fit by regression to the naïve deterministic model, either payoff or mutation parameters would be forced to differ by $\mathcal{O}(1)$ from the values experienced by the individuals.

This example illustrates an important general way to understand and resolve singular limits in evolutionary

[58] N values used were, from left to right in the figure: 90, 100, 150, 200, 250, 300, 350, 400, 450, 500, 600, 700, 900.

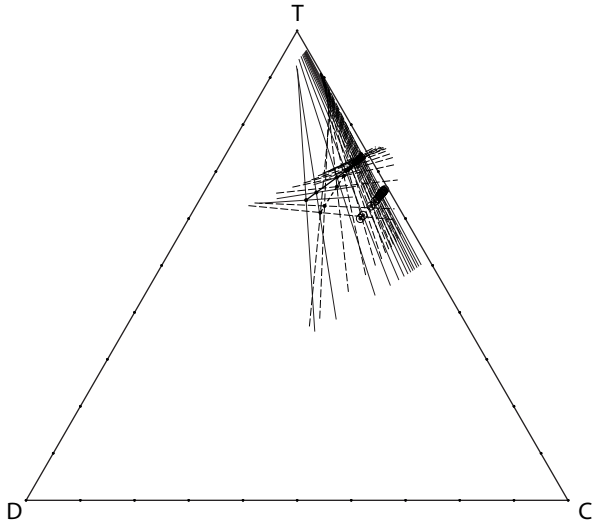

FIG. 21. Simulation results (dashed) compared to the leading-order fluctuation corrections (solid) and to the naïve mean-field limit (points with rings), for the same sequence of N values as in Fig. 20.

game theory. In the deterministic theory, the strength of policing scales as the strength of mutation, $\sim 1/N$. The policed mean-field rest point has a regular limit as $1/N \to 0$. Yet the formal theory at "$1/N \equiv 0$" has no well-defined equilibrium. This is a removable singularity, but it reflects the non-removable feature that in a finite range of small $1/N$, the policing is doing nearly no work on the population.

The interpretation of the removable singularity is that the policing function is *fragile* at large N. In the stochastic theory, fragile policing never reaches the singular limit, because within the interior of the simplex, the stabilizing function shifts from the mean-field parameters to the distribution over fluctuations.

3. *The map from population states of RPD to the Folk Theorems of rational-choice game theory*

For games in the normal form, the association between evolutionary and rational-choice game theory known as the *Folk Theorem of Evolutionary Game Theory* [4, 82] associates rest points of the replicator equation with Nash equilibria, and stable ESS with strict Nash equilibria. Under this association, homogeneous populations map to pure strategies in the strategic interpretation, and heterogeneous, randomly matched populations map to mixed strategies.

The mapping from the evolutionary to the rational-choice approach to repeated games is less apparent, because both refer to the normal form of the stage game, but they use it in different ways. A simple evolutionary repeated game such as our RPD example introduces one new type of agent (TFT) distinguished by memory, and a larger normal-form representation (48) of the repeated game, determined by the way that memory implements a kind of player-identification and long-range correlation.

The Folk Theorems of rational-choice game theory [55–57, 64, 119] define a *feasible set* of move profiles, and derive trigger strategies that are compatible with individual rationality but can prevent deviation from any pre-arranged move profile in the set. However, the set of non-cooperative, mixed strategies of the stage game is too restrictive to define a feasible set that captures long-range correlation in the repeated game, so the rational choice Folk Theorems introduce additional models of "public signals" that extend the notion of non-cooperative equilibrium to include specific forms of coordination.

In App. F we construct the map from population processes to the two key requirements of the rational-choice approach. The first shows how neutrality involving ALLC, TFT, and ALLD makes repeated move profiles in evolutionary RPD *strictly individually rational* by the strategic definition. The second shows how the signaling coordination by TFT – which falls outside the non-cooperative solution concept – is mapped through the population state to the notion of a public signal in the non-cooperative game. When these correspondences are established, it becomes clear how the evolutionary population dynamics of these three strategies form the equivalent of a "prior agreement" to implement a particular coordinated move profile within the feasible set.

The function of the population state that maps to the public signal is $\Phi \equiv \mathrm{n}_T/\left(\mathrm{n}_C + \mathrm{n}_T\right) \in [0,1]$, which has the interpretation of a reliability. The feasible set of non-cooperative move profiles in the stage game is shown in Fig. 26 of App. F, and maps to the entire space of population states in Fig. 19; move profiles outside this set cannot be achieved by random matching of players in the evolutionary game. In particular, each value of Φ – exogenously given in non-cooperative repeated game theory – corresponds to a ray from the vertex D to some point on the C-T line in Fig. 19. The value $\Phi = 0$ is the boundary of the feasible set formed by independent mixed strategies, which maps to the D-C line. The opposite boundary $\Phi = 1$ comprises the fully-coordinated mixed strategies, and maps to the D-T line.[59]

4. *Trembles in finitely-repeated Prisoners' Dilemma and other sources of fragility in overcoming stage-game dominance*

Neutrality between a policing strategy, and the unconditionally cooperative strategy ALLC, is essential for the

[59] We show in App. F how the same population state may be differently decomposed, into a population of "blind" memory-zero players, and the "sighted" type TFT. In the latter decomposition, sighted players achieve the signal-mediated coordination of the evolutionary supergame, using moves that cannot be described with non-cooperative solution concepts.

normal-form payoffs to direct the force of selection to overcome the dominance of ALLC by ALLD in the stage game. In addition to the fragility of policing at large N, the property of neutrality itself is fragile in many limits. A symmetry analysis of the game trees and strategies that are used to work around various dimensions of fragility can be very helpful in understanding when different games even address the same coordination problem, and when they do not.

The most widely studied fragility of RPD replaces the deterministic repeated game by one with "trembling-hand" moves [20], which have a small independent probability in each round to be opposite the move in the deterministic strategy. Trembles, in very long repeated games, unravel the ability of TFT to coordinate and hence to achieve neutrality with ALLC and assure high penetrance of the C move, as shown in Fig. 22. In the limit of infinite repetition, the coordination of deterministic RPD is a (non-removable!) singular limit with respect to any probability of trembles. This fragility, like the fragility of policing, can be regulated by choosing the number of repetitions in relation to the rate at which trembles cause coordination in TFT to decay to the equivalent of random play. However, sufficiently long repeated games are required to reduce first-move effects in the payoffs (48) below the randomizing strength of mutation, so the fragility of trembles is meaningful and is tied to the fragility of policing at large N.

An attempt to circumvent the fragility of coordination that is displayed by TFT with trembles, which has come up intermittently over the years, is to replace TFT with a parity strategy known as "Pavlov" (or Win-stay/lose-shift, WSLS) [60, 61, 63]. The parity strategy is defined on the game tree of Fig. 6, and moves left (C) for even parity and right (D) for odd parity.

It is important to understand that replacing TFT by WSLS does much more than address trembles. The "other's-move" game tree of Fig. 5 is symmetric under interchange of left and right moves, so there is *no* value-asymmetry in the move-space of a population of strategies containing ALLC, ALLD, and TFT. All value asymmetry is written explicitly in the payoffs of the stage game. Thus the problem solved in deterministic RPD is to redirect the force of selection using only properties made explicit within the stage game. WSLS takes the left/right symmetric information sets of the parity tree of Fig. 6 and imposes an *asymmetric* response, converting even parity to C and odd parity to D. This asymmetry, being a feature of the *type* of the player, is constant not only over stages within the repeated game, but over the matches that a player experiences within a generation. It is robust against trembles because it fundamentally does not rely on correlations within the repeated game at all. Rather, it introduces an asymmetry into the type space,[60] which happens to be matched to the Pareto-dominance of Prisoners' Dilemma. The asymmetries of the problem, however, are no longer completely represented by the payoffs of the stage game, so the problem addressed is fundamentally different from that of redirecting the force of selection through correlations within development.

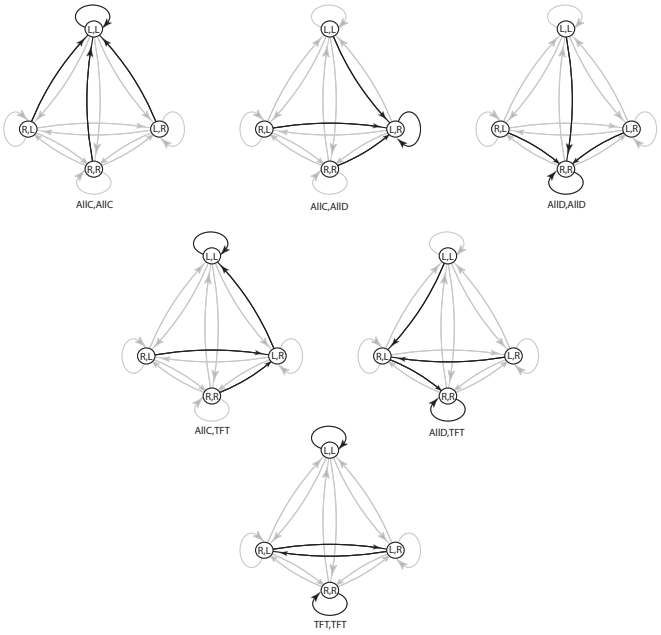

FIG. 22. Decomposition of the possible recurrent moves (heavy lines) from Fig. 7 according to the matched strategies. (Only ordered matches are shown, as in Fig. 18, to aid viewing.) All strategy pairs formed from ALLC, ALLD, and TFT – except TFT,TFT – are robust against both recombination and trembles (trembles result in moves in light grey), returning players within no more than two moves to the absorbing states. TFT,TFT, however, will sustain three recurrent patterns, one of which involves a two-state alternation. If the opening move is mixed as in Sec. IV G, each sub-graph is stable. If instead non-canonical starting states result from unbiased trembles, the three sub-graphs (L,L, L,R/R,L, R,R) are visited in ratios 1:2:1. Mixing of sub-graphs in these ratios leads to the same mixture of payoffs as pairing of equal-weight mixed strategies (purely random play) for both players. In long-repeated supergames, convergence to this distribution over the repeated move profiles removes the neutrality of TFT with ALLC, requiring replacement of TFT by WSLS [60] to evolve cooperation. The rate of trembles in relation to the number of repetitions may be used to tune the transition between the deterministic and random limits.

Pavlov strategies have been described as providing robust evolutionary solutions to the "paradox of cooperation". However, these strategies are themselves fragile against innovation. They were known already to Rapoport [117], who characterized them with the

[60] In the language of the previous century, this asymmetry is "instinctive" to WSLS, in contrast to something constructed within the course of development.

name "The Simpleton" [62]. In the tournaments of Axelrod where TFT achieved a high percentile of wins, Pavlov strategies typically fared in the lowest quarter percentile [120]. It seems appropriate to say that, much as TFT relies on sufficient reliability to create the essential feature of neutrality with ALLC, Pavlov relies on sufficiently limiting the inventory of behaviors admitted within the arena of selection.

F. Neutrality will be a typical feature of extensive-form games

In deterministic RPD, the neutrality between ALLC and TFT is a form of *payoff neutrality*, externally imposed but not a feature required by the structure of the game itself. A simple counting argument suggests that, in extensive-form games with more plies, a different *structural* neutrality may become very typical. Structural neutrality is distinguished from mere payoff neutrality, in that structural neutrality is enforced by the combinatorics of the extensive-form game, no matter what payoffs are assigned at the leaves.

The counting argument goes as follows; we will use the total information simultaneous-move tree of Fig. 1 as an example. Eq. (20) gives the total number of strategies for one player on a tree of $N_{\rm ply}$ plies. The total number of strategy profiles (pairs of strategies for both players) is then

$$|\{i\}|^2 = 4^{\frac{2^{2N_{\rm ply}}-1}{2^2-1}}. \tag{49}$$

However, the number of leaves on the game tree is only $4^{N_{\rm ply}}$. Therefore in a large game tree many strategy pairs must lead to the same leaf and, no matter how payoffs are assigned, to the same payoff. A change to either strategy in a profile, on a node that is not played, must be neutral to the payoff of both players. If the mutation process defines a notion of adjacency between strategies, then neutral sets are given the topology of neutral networks.

To demonstrate structural neutrality, we continue with the paradigm of trembling-hand play of a repeated game with two-player, normal-form stage game, but we admit a more complete and balanced collection of memory-one strategies. Rapoport's notation will continue to denote payoffs in the stage game, but in this example we are not concerned with their numerical values or the particular equilibria of the stage game.

We consider the three memory-one trees in Figures 4, 5, and 6. These trees jointly admit 18 new strategies with equal numbers of L and R moves, in addition to the two strategies from Fig. 2. From these four trees, we select $2^3 = 8$ strategies (two binary complements from each tree) that contain TFT and its opposite (from Fig. 5), WSLS and its opposite (from Fig. 6), and strategies REP and ALT (from Fig. 4) that repeat or alternate the player's own previous move.

The recurrent finite-state diagrams for all pairs of these strategies are shown in Table II of App. D. Ignoring first-move effects, the payoffs from a long-time trembling hand recurrent move for all pairings of these strategies against one another are shown in Table I.

	ALLC	TFT	WSLW	REP	ALT	AWSLS	ATFT	ALLD
ALLC	R	R	$\frac{R+T}{2}$	$\frac{R+T}{2}$	$\frac{R+T}{2}$	$\frac{R+T}{2}$	T	T
TFT	R	X	X	$\frac{R+P}{2}$	$\frac{T+S}{2}$	X	X	P
WSLW	$\frac{R+S}{2}$	X	R	X	X	S	X	$\frac{P+T}{2}$
REP	$\frac{R+S}{2}$	$\frac{R+P}{2}$	X	X	X	X	$\frac{T+S}{2}$	$\frac{P+T}{2}$
ALT	$\frac{R+S}{2}$	$\frac{T+S}{2}$	X	X	X	X	$\frac{R+P}{2}$	$\frac{P+T}{2}$
AWSLS	$\frac{R+S}{2}$	X	T	X	X	P	X	$\frac{P+T}{2}$
ATFT	S	X	X	$\frac{T+S}{2}$	$\frac{R+P}{2}$	X	X	T
ALLD	S	P	$\frac{P+S}{2}$	$\frac{P+S}{2}$	$\frac{P+S}{2}$	$\frac{P+S}{2}$	S	P

TABLE I. Payoffs for the player whose strategies are listed across the top of the table, when paired with the player listed on the left. Strategies have memory zero or one, and even numbers of L and R moves. X stands for the equally mixed payoff $(R+T+P+S)/4$, which would be achieved also by random play. Note that, like the payoffs (48) of RPD, the table goes into its transpose under $T \leftrightarrow S$.

The eight strategies may be assigned a three-digit binary "genotype", given by

$$\begin{aligned} 000 &\leftrightarrow \text{ALLC} \\ 001 &\leftrightarrow \text{TFT} \\ 010 &\leftrightarrow \text{WSLS} \\ 011 &\leftrightarrow \text{REP} \\ 100 &\leftrightarrow \text{ALT} \\ 101 &\leftrightarrow \text{AWSLS} \\ 110 &\leftrightarrow \text{ATFT} \\ 111 &\leftrightarrow \text{ALLD}, \end{aligned} \tag{50}$$

and illustrated in Fig. 23. We may introduce a "Hamming" mutation process that allows single bit-flips, moving player types one step over edges in the cube. In this mutation metric, each strategy is antipodal to its binary complement, and the two zero-memory strategies ALLC and ALLD are each connected to their three refinements in the memory-one trees.

The neutral sets of an evolutionary game with these strategy profiles are readily seen from Table I. The mutation process requires a six-dimensional hypercube to illustrate, but the neutral networks formed by the Hamming mutation process can be illustrated with ties in the original matrix, as shown in Fig. 24.

Even for this small strategy set, the neutral space consists of two large, interwoven cycles of 12 pairs, one chain of 3 pairs, ten sets with 2 pairs, and 13 singletons. The adjacency structure of these neutral networks to one another under the Hamming mutation can be seen from the graph of the hypercube in Fig. 25. In the figure, horizontal moves represent strategy changes by single bit flips on the hypercube of Fig. 23 for the first player; vertical moves represent strategy changes by single bit flips for the second player. The smallest squares represent flips

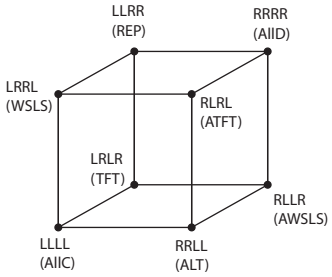

FIG. 23. An assignment of the eight strategies of Table I to the vertices of a cube. Mutation moves player types between vertices connected by an edge. The Left or Right move at each node in the information sets (2-3) is indicated above the strategy name at each vertex.

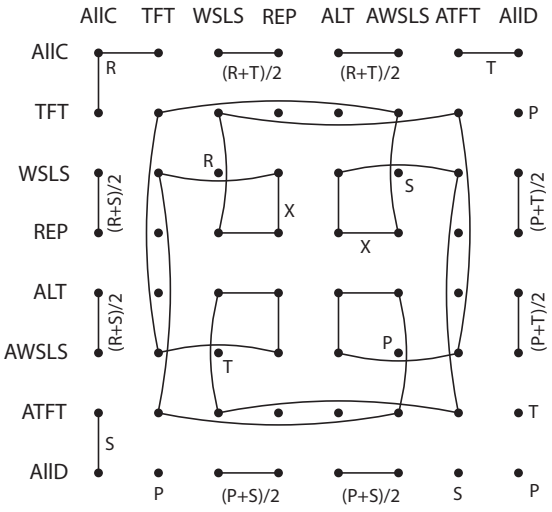

FIG. 24. The neutral networks of Fig. 23 drawn on the payoff Table I. Neutral networks are labeled with the values of the nodes to which they correspond, and as in the table X stands for $(R+T+P+S)/4$. There are two neutral networks for each of $(R+T)/2$, $(R+S)/2$, $(P+T)/2$, $(P+S)/2$, and X. Eight internal nodes, which have no nontrivial networks, remain unlabeled for clarity.

in the direction from ALLC to TFT. The squares formed by the next level of nesting represent flips in the direction from ALLC to WSLS. The outermost level of nesting represents flips in the direction from ALLC to ALT.

The spaces in which large fluctuations can exist will depend on both the cardinality and the topology (degree of convexity) of these neutral sets. We anticipate that, for extensive-form games with many plies, even if most fluctuations in a single direction are not large, the combinations that accumulate from structural neutrality will lead to important fluctuation effects in many cases.

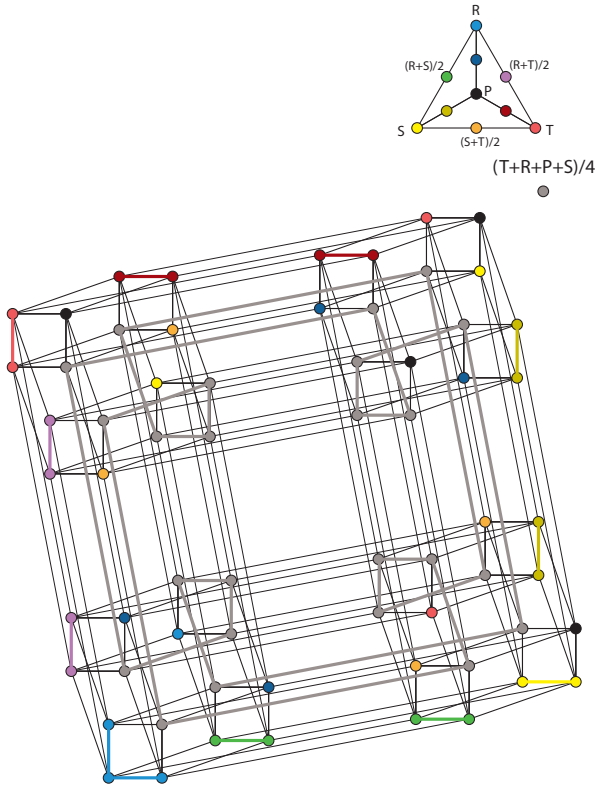

FIG. 25. Hypercube of strategy pairs from Table I, showing the adjacency induced from the mutation metric of Fig. 23.[a]

[a] In the color version of the figure payoff values are indicated by the color scheme. (The three intermediates $(R+P)/2$, $(T+P)/2$, $(S+P)/2$ are not labeled for clarity.) Neutral networks in the hypercube are drawn with heavy links in the same colors as the nodes they connect.

In the b/w version of the figure, neutral nets are indicated with heavy links, and labeled with their corresponding payoffs. The two networks with payoffs $(T+R+P+S)/4$ are shown solid and dashed, to make them easier to follow visually.

G. Multilevel selection and explanatory sufficiency in the repeated-game setting

Many approaches to overcoming paradoxes of stage-game dominance require the maintenance of *linkage* between moves that enable coordination and moves that confer fitness. Examples include costly signaling or the recognition of kinship or identity.[61] The requirement to actively maintain linkage is another source of fragility in such coordinated solutions.

In this section we expand minimal RPD into a gene-based population process with genes described from the

[61] Since many of the moves described occur at a very high level of development (or are institutional in origin), we refer heavily to the "statistical" nature of the gene, and to "crossover" in models of linkage as a coarse-grained reflection of any process that disrupts the integration of behaviors while preserving the identity of the individual behaviors in some adequate approximation.

coarse-grained extensive-form tree of Fig. 5. We regard the information sets on the tree as loci, and permit crossover between the first move and the recurrent move of the game tree. The moves C or D are allowed as alleles on every information set. The possible opening moves will therefore be C and D, and four recurrent-move alleles will be possible. Three are C, D, and the recurrent move from TFT, which we will denote T. (The fourth is the complement to recurrent T, shown as ATFT in the previous subsection. We will suppress this possibility from the analysis, in order to simplify the presentation and because it never plays a significant role in late-time stable populations.)

The resulting process demonstrates the following general properties involving genes based on the extensive form: 1) the asymmetry in fitness contribution between different loci, here opening-move and recurrent-move on the coarse-grained tree; 2) the non-equivalence of different recurring moves for providing contextual importance to the opening move; and 3) the way an additive model for which opening and recurrent moves are very good explanatory loci can still require multilevel selection to achieve any explanatory sufficiency over the main behavior of the population process, in this case ensuring high penetrance of the C move.

In the spirit of refining regression models to identify mechanism statistically, we will construct best-estimators for model fitness in a sequence of increasing polynomial order, starting from additive models, and terminating when a stable, dynamically sufficient model has been identified. As higher-order models are used, parts of the regression coefficients, which depended on population state in lower-order regressions, are successively transferred to higher-order interactions where they become constants. Nothing less than a a fourth-order interaction term in the genetic model for fitness leads to regression coefficients that can successfully account for emergence of (C, C) as the most-frequent move profile, without explicit reference to the population state as an external variable.[62]

When crossover and mutation at all loci are introduced into evolutionary RPD, the genotype space expands to include eight types (of which we consider six). Using ($'$) to denote a genotype with the same recurrent move as the types in canonical RPD, and opposite opening move, we denote the composition vector

$$\mathrm{n} \equiv \begin{bmatrix} \mathrm{n_C} \\ \mathrm{n_{C'}} \\ \mathrm{n_D} \\ \mathrm{n_{D'}} \\ \mathrm{n_T} \\ \mathrm{n_{T'}} \end{bmatrix}, \tag{51}$$

The repeated-play payoffs to agents in this larger population a'_{ij} equal the values of a_{ij} for types with the same move in the recurrent stage to $\mathcal{O}(1/N_{\mathrm{ply}})$, for all pairings except T and T'. For these latter four pairings, the payoffs become

$$[a']_{TT'} = \bar{a} \begin{bmatrix} 1 \\ 1 \end{bmatrix} \begin{bmatrix} 1 & 1 \end{bmatrix} + \begin{bmatrix} R & \frac{1}{2}(S+T) \\ \frac{1}{2}(S+T) & P \end{bmatrix}. \tag{52}$$

The values in Eq. (52) follow from the states visited in the bottom diagram in Fig. 22.

App. E provides an analysis of the dynamics of this population. First, we show that the type T' is suppressed by selection relative to T, so that the population comes to be dominated by canonical TFT, and whatever mixture of C and D types is created by crossover and mutation. (The latter effects are silent for C and D types, so they serve only as an enhanced reservoir of mutants that may back-cross with T, reducing the degree of linkage to produce TFT.) We then develop a sequence of regression models, and show that the fourth-order interaction term is both necessary and sufficient to explain the presence of T as a competitive allele, which in turn is the requirement for (C, C) move profiles to occur with high frequency.

H. Complex discrete symmetry breaking through selection on both genes and covariance

Our last example emphasizes the use of extensive-form games in a traditional model of development, and the emergence of complex symmetry breaking from a fitness function with a large permutation symmetry group.

The example originates in a model of linked heterosis due to Lewontin [37]. It illustrates well the use of games to model heredity in diploid organisms, where chromosomes (or regions within chromosomes not disrupted by crossover within a given generation) are the hereditary "individuals", and the diploid "organism" that develops from the match of a pair of chromosomes is represented by play of the game. The linked-heterosis model was presented as one mechanism to explain the high frequency of observed heterozygosity in natural populations, without recourse to the assumptions of neutral theory, but with plausible (meaning, small) levels of heterosis at each locus. The main result of the model is that selection can act cooperatively at many loci, amplifying the effects of heterosis, by effectively selecting for linkage between nearby loci. The model isolates selection on linkage by removing all other sources of fitness differentiation among alleles through an imposed symmetry.[63]

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[62] For comparison, the linked-heterosis model developed next will require only second-order interactions.

[63] Random matching, together with recombination that is not subsequently filtered by selection, lead eventually to the condition of Hardy-Weinberg/Linkage-Equilibrium [15] (HWLE, also known as the *Wright manifold*). This thoroughly-studied limit in pop-

This example illustrates a new role for collective fluctuations, as transient sample excesses of linkage in the population become the only source of fitness differences among genotypes, and through that mechanism become self-amplifying. It also illustrates that plies in an extensive form game, under appropriate information conditions, can be equivalent to loci on a chromosome with respect to epistasis and crossover, without connotation of any temporal sequence in development. Finally, it illustrates the parallel role of repetition within and between generations, by showing the homology between linkage in a repeated game, and the expression for relatedness in Hamilton's rule derived in Sec. IV C 2.

1. *An anti-coordination game that isolates the dynamics of linkage from a neutral manifold of allele frequencies*

The extensive-form game that captures both heterosis and linkage is defined on the tree of Fig. 3, with crossover between successive plies.[64] The gene indices are $I \in \left\{L^0, R^0, \ldots, L^{N_{\rm ply}-1}, R^{N_{\rm ply}-1}\right\}$, where the superscript indexes the locus. Indicator functions σ_{Ii} take value one if strategy i has move I at the appropriate locus. Since a fully-specified strategy must have $\sigma_{L^k i} + \sigma_{R^k i} = 1$ at every k, we may represent strategies compactly in terms of a "spin" variable

$$\sigma_i^k \equiv \sigma_{L^k i} - \sigma_{R^k i}, \tag{53}$$

which takes value ± 1 according to the move at locus k.

Plies serve as loci, and a model that treats all loci identically corresponds to a repeated game. The normal-form stage game for heterosis at each locus is an anticoordination game, which we may write by replacing a by $-a$ in the upper-left 2×2 sub-matrix for moves L and R in Eq. (26). Setting $a = 1$ without loss of generality, the payoff when two genotypes i and j are matched is symmetric in i and j, given by

$$a_{ij} = -\frac{1}{2N_{\rm ply}} \sum_{k=0}^{N_{\rm ply}-1} \sigma_i^k \sigma_j^k. \tag{54}$$

These two equations define the genotype space and the payoffs per-match for a pair of individual chromosomes.

2. *Fluctuations are responsible for population covariance and linkage disequilibrium*

In systems where different genotypes have different fitness at mean-field approximation,[65] an "ecological" regression model, in which many matches per generation determine mean fitness, captures the main cumulative effects on population dynamics.[66] In this model of linked heterosis, not only does a single play of the game literally represent the life of a single diploid organism, but the large sample fluctuations that come from pair matching will be essential to driving linkage disequilibrium. Therefore we begin by contrasting the mean fitness of alleles, which approaches zero for balanced populations, with the fluctuations in fitness due to sampling of partners within generations.

The population-averaged frequency of the indicator function $\sigma^k = \sigma_{L^k} - \sigma_{R^k}$ at each locus k, in a population state n, is given by

$$\left\langle \sigma^k \right\rangle_{\rm n} \equiv \sum_j \frac{{\rm n}_j}{N} \sigma_j^k. \tag{55}$$

The fitness of any complete genotype i in population n, as it would appear in expectation under random pairing against all members of the population, is then

$$f_i = -\frac{1}{2N_{\rm ply}} \sum_{k=0}^{N_{\rm ply}-1} \sigma_i^k \left\langle \sigma^k \right\rangle_{\rm n}. \tag{56}$$

The corresponding population-averaged fitness is

$$\phi = \sum_i \frac{{\rm n}_i}{N} f_i = -\frac{1}{2N_{\rm ply}} \sum_{k=0}^{N_{\rm ply}-1} \left\langle \sigma^k \right\rangle_{\rm n}^2. \tag{57}$$

These are reference values, which provide estimates of the cumulative selection strength on alleles over many generations. In any population with balanced alleles (all $\left\langle \sigma^k \right\rangle \approx 0$), both f_i and ϕ will be near zero. Therefore it can only be fluctuation about these values from *particular* pairings that leads to non-random association at the population level.

The average correlation of alleles at two loci k and k' in the population is given by

$$C_{kk'} \equiv \sum_i \frac{{\rm n}_i}{N} \sigma_i^k \sigma_i^{k'}. \tag{58}$$

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ulation genetics leads to simplifications for the identification and analysis of extensive-form games, considered in Ref. [23]. We will assume random matching and thus Hardy-Weinberg equilibrium, but in the limit of strong selection, the absorbing states of the Lewontin model will lie maximally far from linkage equilibrium.

Because the strength of selection determines whether heterogeneity or drift to fixation will be the persistent state of the population, we regard the contribution of linkage to the strength of selection as a kind of Hill-Robertson effect. Like other Hill-Robertson effects [19], the cause of population structure will not be found in the direct selection coefficients, but will depend on fluctuations in fitness.

[64] Here we do not explicitly model crossover. We consider only the effects of selection, particularly in relation to fluctuations, and for other properties of solutions we draw on Ref. [37].

[65] All previous models have been of this sort, except for the neutrality between ALLC and TFT in the model of the repeated Prisoners' Dilemma.

[66] Indeed, the generation structure in such models is of secondary importance, and can be coarse-grained if appropriate relatedness terms are included in the covariance matrix, as shown in Sec. IV C 2.

Subtracting the expected values of these alleles in the population state n leaves the covariance between loci,

$$\mathrm{Cov}\Big(\sigma^k, \sigma^{k'}\Big) = C_{kk'} - \big\langle \sigma^k \big\rangle_{\mathrm{n}} \Big\langle \sigma^{k'} \Big\rangle_{\mathrm{n}} . \tag{59}$$

The covariance at a single locus k is the variance of σ^k, which evaluates to $\mathrm{Cov}\big(\sigma^k, \sigma^k\big) = \mathrm{Var}\big(\sigma^k\big) = 1 - \big\langle \sigma^k \big\rangle_{\mathrm{n}}^2$.

Referring back to the form of the Price equation (12), and letting σ^k for an allele be the indicator function whose frequency in the population we wish to follow, shows that allele frequency is controlled by the covariance (59) in state n, as

$$\begin{aligned} \frac{d}{dt}\big\langle \sigma^k \big\rangle_{\mathrm{n}} &= -\frac{1}{2N_{\mathrm{ply}}} \sum_{k'=0}^{N_{\mathrm{ply}}-1} \mathrm{Cov}\Big(\sigma^k, \sigma^{k'}\Big) \Big\langle \sigma^{k'} \Big\rangle_{\mathrm{n}} \\ &= -\frac{1}{2N_{\mathrm{ply}}} \Big(1 - \big\langle \sigma^k \big\rangle_{\mathrm{n}}^2\Big) \big\langle \sigma^k \big\rangle_{\mathrm{n}} - \frac{1}{2N_{\mathrm{ply}}} \sum_{k' \neq k} \mathrm{Cov}\Big(\sigma^k, \sigma^{k'}\Big) \Big\langle \sigma^{k'} \Big\rangle_{\mathrm{n}} \end{aligned} \tag{60}$$

Comparing Eq. (60) to the expression (38) for Hamilton's rule, we see that linkage disequilibrium plays the same role with respect to moves in the extensive-form game, that relatedness plays among populations that fluctuate on the generation scale.

If covariance in the population remains near zero, the rate of mean regression for any focal allele will fall as $1/2N_{\mathrm{ply}}$ with increasing N_{ply}, because of the normalization assumed for fitness in Eq. (54). However, if covariance is large over a range of adjacent plies whose number grows with N_{ply}, the mean regression for $\big\langle \sigma^k \big\rangle$ may remain stronger than $1/2N_{\mathrm{ply}}$, ensuring the preservation of heterozygosity against drift in the population even with very weak heterosis per locus. This mechanism for the maintenance of heterozygosity was the motivation behind this model in Ref. [37].

A similar construction may be performed for whole genotypes. We now suppose that a single pairing of zygotes determines the fitness of both partners within each generation. The mean fitness of type i relative to the population average remains the same as the expectation over multiple pairings, given by

$$f_i - \phi = -\frac{1}{2N_{\mathrm{ply}}} \sum_{k=0}^{N_{\mathrm{ply}}-1} \big(\sigma_i^k - \big\langle \sigma^k \big\rangle_{\mathrm{n}}\big) \big\langle \sigma^k \big\rangle_{\mathrm{n}} . \tag{61}$$

The variance in the expected number of offspring left by any individual of type i, due to random pairing of players, is much larger for single pairings than for samples of the population average. The sample fluctuation now depends on details of the probabilities of reproduction, which we absorb into a characteristic time increment Δt. The time increment per typical reproduction scales the overall variance in reproductive success, which is then given by

$$(\Delta t)^2 \sum_j \frac{\mathrm{n}_j}{N} (a_{ij} - f_i)^2 = \frac{(\Delta t)^2}{4N_{\mathrm{ply}}^2} \sum_{k,k'} \sigma_i^k \sigma_i^{k'} \mathrm{Cov}\Big(\sigma^k, \sigma^{k'}\Big) . \tag{62}$$

The expected number of offspring from *all* parents of type i in the population is then

$$\langle \mathrm{n}_i' \rangle = \mathrm{n}_i \left(1 + \Delta t \left(f_i - \phi\right)\right) . \tag{63}$$

By the central limit theorem for independently drawn partners for each of n_i individuals, the corresponding variance in offspring due to the distribution over pairings is then

$$\begin{aligned} \Big\langle \big(\mathrm{n}_i' - \langle \mathrm{n}_i' \rangle\big)^2 \Big\rangle &= (\Delta t)^2 \mathrm{n}_i \sum_j \frac{\mathrm{n}_j}{N} (a_{ij} - f_i)^2 \\ &= \frac{(\Delta t)^2 \mathrm{n}_i}{4N_{\mathrm{ply}}^2} \sum_{k,k'} \sigma_i^k \sigma_i^{k'} \mathrm{Cov}\Big(\sigma^k, \sigma^{k'}\Big) . \end{aligned} \tag{64}$$

These expressions for reproductive variance due to sampling may be used to estimate dynamics for the covariance itself. In App. G we make these estimates using a stochastic differential equation (Langevin equation) in which the variance of the fluctuation field is given by Eq. (64).

The Appendix shows that that fluctuations in genotype numbers n_i transiently polarize the population to favor of growth of genotypes that are complementary (heterotic at many loci) to the transiently increased type i, in proportion to the reproductive variance given in Eq. (62). Positive covariance between fluctuations in the number n_i, and fluctuations in the complements that enhance the fitness of i through heterosis, lead to an *effective fitness*. The magnitude of this effect, which comes from the second-order term in the stochastic differential equation, is given by Eq. (G14) in the appendix.

The effective fitness is largest for genotypes that overlap with the covariance in the population, and their own reproduction then reinforces this covariance, leading to growth in the linkage of alleles in the population. The variance in total reproductive success, averaged over the

population, is proportional to

$$\sum_{i,j} \frac{\mathrm{n}_i}{N}\frac{\mathrm{n}_j}{N}(a_{ij}-\phi)^2 = \frac{1}{4N_{\mathrm{ply}}^2}\sum_{k,k'}\left(C^2_{k,k'} - \left\langle \sigma^k \right\rangle_{\mathrm{n}}\left\langle \sigma^{k'} \right\rangle_{\mathrm{n}}\right). \tag{65}$$

The population-level variance (65) is maximized when $C_{k,k'} \to 1, \forall k,k'$, and $\left\langle \sigma^k \right\rangle_{\mathrm{n}} \to 0, \forall k$. This condition is met on any of $2^{\left(N_{\mathrm{ply}}-1\right)}$ independent solutions, each having exactly 2 population types that oppose each other at each locus. Which type carries which allele at a locus is otherwise arbitrary, reflecting the independence and L-R exchange symmetry of payoffs at each move. These are absorbing states of the replicator dynamic, in the absence of mutation or crossover.

3. The group of representations by ordered population states

Ref. [37] (Ch. 6) shows that, if crossover is introduced with equal probability between any two adjacent plies k,k', the resulting stable distributions have correlation coefficients falling off as $C^2_{k,k'} \approx e^{-2|k-k'|/N_{\mathrm{corr}}}$. The correlation length N_{corr} is proportional to the ratio between the rate of crossover and the magnitude of the payoff per allele, which is $1/N_{\mathrm{ply}}$ in Eq. (54). Therefore a fixed probability of crossover between any two adjacent loci leads to a correlation length scaling as a fixed fraction of the whole-genome length, which defines the continuum-limit description of the chromosome as a unit of selection [37].[67]

The number of possible genotypes occupied with significant frequency in any such correlated equilibrium is $\sim 2^{\left(N_{\mathrm{ply}}-N_{\mathrm{corr}}\right)}$. Therefore the number of independent and mutually exclusive population-state equilibria is $\sim 2^{N_{\mathrm{corr}}}$. Any population equilibrium will be characterized by a pair of mirror images of a "consensus" sequence which produce the greatest heterosis when the two complements are matched.

In the presence of both mutation and sampling fluctuations from matching, population states polarized around any given consensus pair are not asymptotically stable, because symmetry among all loci guarantees an exact degeneracy of all $2^{\left(N_{\mathrm{ply}}-1\right)}$ mirror-image genotypes as possible consensus pairs. Therefore polarized population states will be subject to drift on a timescale which is slower than the generation timescale and increasing with N_{corr}.[68]

[67] Readers familiar with the notion of "dressed" particles in renormalized condensed-matter physics [29, 121] may recognize these correlated regions as "dressed" genes, and the overall construction as the "effective theory" of the chromosome.

[68] Drift of this form in systems with large number of mutually exclusive equilibria, and with very slow time constants, is sometimes given the name *creep*.

This model possesses *no* additive, heritable, component of zygote fitness from any allele, even if the population has attained one of its optima. Instead, a regression of fitness in a particular population background yields interaction terms with coefficients $\propto \mathrm{Cov}\left(\sigma^k, \sigma^{k'}\right)$ between any two loci k and k' within the same genome. Interaction leads to a fitness function f_i^{eff} in Eq. (G14) in which *epistasis* has replaced the additive interaction terms of Eq. (54). Therefore regression models in the Price equation, which attempt to characterize fitness of haplotypes, can at most reflect the symmetry broken by the population state. Their magnitude will be predictable from first principles, but the particular haplotypes favored will not.

V. DISCUSSION AND CONCLUSIONS

In this review we have attempted to show how symmetry provides a bridge between the robust regression methods of Fisher and Price, and the imputation of mechanisms and causation to development and evolution. Our main use of symmetry has been to assign distinctive forms of of multiscale dynamics to classes of game models, and to connect descriptions at multiple scales through the aggregate properties of collective fluctuations. A secondary use of symmetry has been to classify extensive-form games and the strategies defined on them.

We have tried to suggest the richness of paradigms beyond two-player normal-form games, but to avoid turning them into a wilderness of models, by emphasizing robust effects and universality, and by linking these to very general concepts such as neutrality, the origin of the gene concept, or multilevel selection. Beyond these general themes, we mention three specific points that we believe are clarified from the perspective of symmetry.

Complex symmetry breaking and open-ended evolution

Simple symmetry breaking is widely recognized in evolutionary games, but complex or glassy symmetry breaking less so. We have emphasized it here, because glasses are the only class of statistical systems whose stable ordered states are well understood from first principles, for which a single symmetry-breaking mechanism can produce an "open-ended" collection of essentially degenerate ordered states. The property of "open-endedness" of viable forms is often regarded as a hallmark of Darwinian evolution [122], and the inability of classical symmetry breaking to produce this feature has stood as a barrier between the theory of stability through large-deviations and phase transition, and the paradigm of Darwinian evolution. Glasses and their non-equilibrium counterparts combine the large-deviations theory of stable states with open-endedness of the number and symmetry among states.

The glasses still remain in many ways an unsatisfying

paradigm for Darwinian evolution. The well-understood cases require a high degree of statistical homogeneity in the underlying system and therefore in the hidden symmetry group, unlike the remarkable heterogeneity within and among organisms and ecosystems. Also, a general feature of glass phases is that the ordered states are discrete, and transitions between them are "saltations". Evolution in the biosphere appears to combine gradual and punctuated transformations, and extant models of complex symmetry breaking do not produce this combination in a natural way.

The root of "as if" arguments in universality

Both economics, justifying evolutionary solution concepts, and biology, justifying the game representation, commonly employ "as if" arguments. Economic agents rationalize as if they know the evolutionary optimum, because those who didn't have been eliminated from the economy [123]. Animals interact as if under the rules of a game, when those rules reflect either constrained or highly generic dimensions of conflict [82].

From a stochastic-process perspective, an even more elementary justification may be convergence of many paradigms to the same low-order descriptions as a consequence of averaging over fluctuations. In constructing complex, hierarchical dynamics, nature builds using what is robust [98, 99], because it better supports induction and is more easily maintained. In some cases it may be that the emergence of both game structures and predictive models encoding rational behavior follow a scaffold that originates in regression.

Toward a more unified theory of games

Our three views of repeated games show that the overcoming of dominant stage-game strategies is not a single "paradox", but rather a collection of differently framed problems that are not always comparable. The "prior agreement" or "public signals" of the rational-choice Folk Theorems serve as abstractions for law, conventions, or other institutions whose justification comes from outside the game being formalized. While not required to provide coercive force (this is the reason to restrict to strictly individually rational punishment strategies), these institutions are required to provide significant coordination services.

The mechanistic approach more commonly pursued in evolutionary repeated-game theory can provide both constraining force and coordination, from limitations of memory or complexity. Yet even here, the different solutions to trembling-hand moves address two distinct problems. Win-Stay/Lose-Shift frames cooperation as a paradox of *crypticity*; the dominance defined by the stage game is not the only local force, if a strategy (WSLS) exists that binds asymmetric local action directly to the generation scale. The tit-for-tat solution, in contrast, depends on maintaining long-range *correlation* within an encounter. The symmetry of the recurrent move in TFT makes this strategy more flexible against diverse opponents [62], but more fragile against trembles [63].

A more unified theory of games might integrate the empirical, mechanism-from-regression approach presented here, with the recognition that both prediction and reinforcement are co-present in the solution concepts to many games.[69] Limited prediction or coordination may be enabled by mechanisms which have been evolved or learned through reinforcement, and whose costs derive from their memory or complexity.

Acknowledgments

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Appendix A: Historical notes

1. The limited role of DNA in creating the gene concept for cell biology

a. A reminder: why DNA is not the source of genes

The chemical properties of the DNA molecule are separated from the information it carries (in sequences), to a degree that is unique among the systems in biochemistry. This enables DNA to serve as a near-universal carrier of information, and an excellent platform for error correction.[70] To a good approximation, every ester linkage in the molecule is equivalent. The Mendelian gene, when it coincides with a non-recombining sequence of DNA, is defined by the *inequivalence* between positions where recombination is nearly free and those where it is effectively forbidden. Many such regions correspond to protein-folding domains, while others may come from suppressed

[69] This may be seen even in the interplay between backward induction and qualitative position evaluation in hard combinatorial games [124, 125].

[70] General-purpose error correction is achieved by identifying and repairing error states, without the repair mechanism's needing to know what message the system should carry.

crossover within operons. Recombination may be forbidden by lethality of most crossovers, or it may disappear if almost all loci sweep to fixation within a gene in a population. The particulate heredity of DNA may serve these functions by providing a reliable substrate for error correction, but is optimized to be the *last* source in the cell for distinctions between recombining and non-recombining bonds.

b. *How the gene concept got lost with the understanding of epistasis as a source of blending inheritance*

The statistical origin of the gene concept might have retained more of the salience it deserves in biological thought, had it not been submerged under solutions to quantitatively greater problems. The problem of accounting for blending inheritance [126], and the covariance of characters in development [127], in a Mendelian model, required the ability to treat epistasis and pleiotropy abstractly without necessarily having models of development. The demonstration that this could be done at least in principle resolved the major conflicts that had driven the modern synthesis [44]. Showing that blending inheritance *can* be achieved from particulate heredity is equivalent to demonstrating that digital systems can simulate analog systems; the successes of the modern synthesis did not (of course) demonstrate that the underlying systems *were* digital at the coarse level of genes required for practical modeling. The optimism that DNA would provide a reification of genes further pushed critical thought about the justification for genes into the background. The modern return of inquiry into the origins of particulate heredity has been forced by studies of post-transcriptional modification and regulation, which can show modularity at many levels [50, 51].

2. Uses of potentials in non-equilibrium stochastic processes

The term "potential" can be applied in at least four ways to stochastic evolutionary games. Only one of these preserves the feature of the potential energy in mechanics, that its local minima are fixed points of the dynamical system, so that these furnish a representation of the symmetries of population states. We will briefly summarize here the meanings of the four uses, and their relations to each other.

The most common usage refers to *potential games* [4], for which the vectors of population change (15) may be written as the gradient of a scalar "potential" function of the state $\bar{n}$. Non-equilibrium stochastic processes do not generally admit such potential descriptions, so the potential games are a restricted class. The results presented here do not depend on any of the simplifications provided by potential games, so we do not assume these restrictions.

Two generally-applicable notions of potential emerge from Freidlin-Wentzell theory [65], which we develop in the companion paper as a method to extract leading asymptotics of probability distributions. They correspond formally to the mechanical and thermodynamic potentials in equilibrium systems, and they will always reflect the symmetries of population states. However, they exist in a dynamical space where "momentum" reflects constraints from inference about the past, and does not correspond to the momentum of mechanics that appears in equilibrium potentials.[71] A quantity known as the *quasipotential* [128–132] arises as the rate function in a large-deviation principle [72, 73] for fluctuation probabilities at the macroscale. It is so-named because it is a non-equilibrium generalization of the thermodynamic potentials such as the entropy or free energies. A slight variant on the quasipotential is constructed by Young [52], which counts events of improbable movement rather than accumulating their log-likelihoods.

The quasipotential is constructed as an action functional for the dynamical system defined by Freidlin-Wentzell theory. Within that functional, a third kind of potential fills the role of a Hamiltonian. This potential equals the Liouville operator (constructed in the companion paper) that evolves the moment-generating functions for probability densities over population states. The Freidlin-Wentzell Hamiltonian is like an energy function in that it is conserved in systems without explicit time-dependence, and provides the symplectic structure on the space of stationary solutions. However, due to important differences that arise from irreversibility, this dynamical-system Hamiltonian generally does not carry the interpretation of the mechanical potential responsible for trapping and domain escapes[72] in thermodynamics.

The last case is our "kinematic" use of the term potential. For games with discrete symmetry that may be hidden by strong selection, the Freidlin-Wentzell dynamical system represents the population state as a particle moving in an energy potential with an additional velocity-dependent force [74]. The fixed-points of the energy potential (having zero velocity) are also the rest points of the dynamical system and so are rest points of the evolutionary dynamics. For systems with point-group symmetry, the local minima of the kinematic potential furnish a representation of the broken symmetries. We construct these potentials in the companion paper, and they provide our graphical representations of broken symmetry in the main text.

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[71] For a systematic description of the meaning of position and momentum coordinates in Freidlin-Wentzell theory with respect to inference, see Ref. [74].

[72] For a discussion of equilibrium potentials and escapes, see Ref. [58], Ch. 7.

Appendix B: Relatedness and effective fitness for models with bifurcations

1. Emergence of a "genic" distinction between coordinating and non-coordinating types in the case of pitchfork bifurcation

To give an example of a component of effective fitness that emerges in long-term regressions as a result of transient relatedness, we consider the coordination game of Sec. IV C. For this model, the time-segregating counterpart to Hamilton's rule, developed in Sec. IV C 2, is as similar as possible to Hamilton's rule in a spatial model with two patches. The dynamics of ordered populations in the coordination game may be described by a two-state Markov process, with symmetric and time-independent probabilities of "hopping" between them, over any time intervals longer than a typical escape time. Therefore the time average of Eq. (31), for the population composition in which an individual finds itself, decomposes into a set of uncorrelated states on the generational scale, which could as well be spatial islands as sequential snapshots in time.

a. Simplex coordinates and rest points of the replicator dynamic with mutation

We first provide an explicit basis in which to represent the population fractions of the three types *L*eft, *R*ight, and *M*iddle. We denote by $(\hat{e}_0, \hat{e}_1, \hat{e}_2)$ the three basis vectors

$$\hat{e}_0 \equiv \frac{1}{\sqrt{D}}\begin{bmatrix}1\\1\\1\end{bmatrix};\ \hat{e}_1 \equiv \frac{1}{\sqrt{D-1}}\begin{bmatrix}1\\-1\\0\end{bmatrix};\ \hat{e}_2 \equiv \frac{1}{\sqrt{D\,(D-1)}}\begin{bmatrix}-1\\-1\\2\end{bmatrix}, \tag{B1}$$

the last two of which form an orthonormal basis in the simplex $\sum_i \mathrm{n}_i/N \equiv 1$. (As in the main text, we keep D explicit to follow normalization factors.)

A general population state with the coordinates of Eq. (25) may be expanded in two simplex coordinates β and γ as

$$\frac{n}{N} = \frac{1}{\sqrt{D}}\hat{e}_0 \pm \beta\hat{e}_1 + \gamma\hat{e}_2. \tag{B2}$$

The basis vectors are chosen so that

$$\begin{aligned}\frac{n_{\mathrm{L/R}}}{N} &= \frac{1}{D} \pm \frac{\beta}{\sqrt{D-1}} - \frac{\gamma}{\sqrt{D\,(D-1)}}\\ \frac{n_{\mathrm{M}}}{N} &= \frac{1}{D} + \gamma\sqrt{\frac{D-1}{D}}.\end{aligned} \tag{B3}$$

Vector $\hat{e}_2$ is the vertical axis in the figures of the main text, distinguishing M from L and R, while $\hat{e}_1$ is the horizontal axis, distinguishing L and R from each other.

The non-constant part of the payoff matrix in Eq. (26) is written in the simplex basis vectors as

$$[a] = a\,(D-1)\,\hat{e}_1\hat{e}_1^T. \tag{B4}$$

The isotropic mutation matrix used in all models in the text is written in the simplex basis as

$$[\mu] = -D\left(\hat{e}_1\hat{e}_1^T + \hat{e}_2\hat{e}_2^T\right). \tag{B5}$$

The Nash equilibria of this game in the ordered-population regime are then $\bar{\beta} = \bar{\gamma} = 0$ (saddle point), and the two ESS solutions $\pm\left|\bar{\beta}\right|, \bar{\gamma}$ given in the main text Equations (29) and (30). In a time-average long enough that the population state has flipped many times between the left and right ordered forms, the time-averaged population number for each type $J \in \{\mathrm{L}, \mathrm{R}, \mathrm{M}\}$ becomes

$$\frac{\bar{n}_J}{N} = \left(\frac{1}{\sqrt{D}}\hat{e}_0 + \bar{\gamma}\hat{e}_2.\right)_J. \tag{B6}$$

A regression on this long-time average would resolve only the distinction between type M and the union of types L and R, corresponding to indicator variables σ_{M} and $\sigma_{\mathrm{L+R}}$ which partitioned the population into two sets. We could characterize this "genic" description by presence/absence at a single locus of an allele for "ability to coordinate", irrespective of which move (L or R) the players coordinate *on*. In this course description, only $\bar{\gamma}$ is directly estimated from the average population state, so we regard the typical population configuration as a random variable converging in probability on one of the two values $\pm\left|\beta(\bar{\gamma})\right|$, where the solutions to the equation

$$\beta^2(\bar{\gamma}) = \frac{-\bar{\gamma}}{\sqrt{D\,(D-1)}} + \frac{1}{D} - \frac{D^2}{Na\,(D-1)} \tag{B7}$$

will be proportional to the typical relatedness among individuals in the long-time average.

In the decomposition of Sec. IV C 2, the expression (35) for "cost" – the regression of fitness on individual type J in the context of the *long-time average* population composition – evaluates to

$$\begin{aligned}V_J C_J &= a\,(D-1)\,\beta^2(\bar{\gamma})\,\bar{n}_J\\ &= Na\,(D-1)\,\beta^2(\bar{\gamma})\left(\frac{1}{\sqrt{D}}\hat{e}_0 + \bar{\gamma}\hat{e}_2.\right)_J.\end{aligned} \tag{B8}$$

The expression (36) for the benefit conferred by any type J on any type k – the regression of k's fitness given encounters with type J in the *instantaneous* population – evaluates to

$$B_{Jk} = a\,(D-1)\,(\hat{e}_1)_J(\hat{e}_1)_k. \tag{B9}$$

Finally, the relatedness (37) between types J and k determining their frequency of encounter – the regression of the frequency of type J in the environment on the indicator variable for type k – evaluates to

$$V_J r_{Jk} = N\beta^2(\bar{\gamma})\,(\hat{e}_1)_J(\hat{e}_1)_k. \tag{B10}$$

Whereas the cost (B8) contains a term decreasing linearly in $\bar{\gamma}$, the product of benefit and relatedness projects into the same subspace spanned by $\hat{e}_0$ and $\hat{e}_2$ with constant coefficients, as

$$\begin{aligned}\sum_k B_{Jk}V_J r_{Jk} &= Na\,(D-1)\,\beta^2(\bar{\gamma})\left[(\hat{e}_1)_J\right]^2 \\ &= Na\,(D-1)\,\beta^2(\bar{\gamma})\left(\frac{1}{\sqrt{D}}\hat{e}_0 + \frac{1}{\sqrt{D\,(D-1)}}\hat{e}_2.\right)_J.\end{aligned} \tag{B11}$$

b. Absorbing relatedness from time segregation into effective fitness in a time-averaged model for two populations

We may now see how nonzero average relatedness leads to time-evolution in the "genic" description in terms of $\pm$ "alleles for coordination", and how we may absorb relatedness terms into an effective fitness parameter in the regression on slow timescales. We suppose that the weak average selective advantage of types $\mathrm{L}+\mathrm{R}$ leads to a slow shift out of type M that can be described in a coarse-grained theory over long times, while the strong selection between L and R in most population configurations leads to internal fluctuations of the $\mathrm{L}+\mathrm{R}$ population that we absorb into the coefficient of relatedness. The Price equation (31) becomes an equation of motion for $\bar{\gamma}$ written as

$$\begin{aligned}&\mathrm{Cov}_\rho\left(\vec{\delta}^J, w\right) + \mathrm{E}_\rho\left(w\Delta\vec{\delta}^J\right) = \frac{d\bar{\gamma}}{dt}(\hat{e}_2)_J \\ &= V_J\left(\sum_k B_{Jk}r_{Jk} - C_J\right) + \frac{1}{N}\sum_k \mu_{Jk}\bar{n}_k \\ &= -\left[Na\,(D-1)\,\beta^2(\bar{\gamma})\left(\frac{1}{\sqrt{D\,(D-1)}} + \bar{\gamma}\right) + D\bar{\gamma}\right](\hat{e}_2)_J.\end{aligned} \tag{B12}$$

(As required, terms proportional to $\hat{e}_0$ in the direct (B8) and population-mediated (B11) fitness have canceled, leaving motion only within the simplex.)

Recognizing that

$$\begin{aligned}\bar{\gamma} &= \sqrt{\frac{D}{D-1}}\left(\frac{n_\mathrm{M}}{N} - \frac{1}{D}\right) \\ &= -\sqrt{\frac{D}{D-1}}\left(\frac{n_\mathrm{L}+n_\mathrm{R}}{N} - \frac{D-1}{D}\right),\end{aligned} \tag{B13}$$

Eq. (B12) is, as required, an evolutionary game equation for a game between two types, M and L+R. The quantity expressed explicitly in Eq. (B12) is $\bar{n}_\mathrm{M}$ (in parentheses in the third line), which we may interpret as a combination of an intrinsic fitness $f_\mathrm{M} \equiv 0$ minus a population-averaged fitness $\phi \equiv (\bar{n}_L + \bar{n}_\mathrm{R})\,f_{\mathrm{L+R}}/N$. The effective fitness for the joint population $\mathrm{L}+\mathrm{R}$ is then given by

$$\begin{aligned}f_{\mathrm{L+R}} &= \frac{N\phi}{\bar{n}_\mathrm{L}+\bar{n}_\mathrm{R}} \\ &= a\,(D-1)\,\frac{N^2\beta^2(\bar{\gamma})}{\bar{n}_\mathrm{L}+\bar{n}_\mathrm{R}} \\ &= a\,(D-1)\,\frac{\left\langle\left(n_\mathrm{L}-n_\mathrm{R}\right)^2\right\rangle_\rho}{4\,(\bar{n}_\mathrm{L}+\bar{n}_\mathrm{R})}.\end{aligned} \tag{B14}$$

c. Scaling of within- and between-population variance, and the emergence of coalitional action

For a Poisson process, $\left\langle\left(n_\mathrm{L}-n_\mathrm{R}\right)^2\right\rangle_\rho / (\bar{n}_\mathrm{L}+\bar{n}_\mathrm{R})$ in Eq. (B14) would typically scale as N^0. This scaling is characteristic both of the symmetric state when selection is too weak to lead to bifurcation, and of the population fluctuations about either of the asymmetric states *individually* in the ordered regime. In order to produce fixed $\bar{\gamma}$ in a population with large N, we must scale $a \sim 1/N$ so that fitness remains in a fixed proportion to mutation. With this scaling, the contribution to excess fitness for the $\mathrm{L}+\mathrm{R}$ population from Poisson fluctuations is therefore $\mathcal{O}(1/N)$, comparable to terms we have ignored.

In contrast, in the ordered regime produced when $Na\,(D-1) > D^2$ – so that related sub-populations at $(n_\mathrm{L}-n_\mathrm{R})/N \sim \pm 2\beta(\bar{\gamma})/\sqrt{D-1}$ become typical – the variance $\left\langle\left(n_\mathrm{L}-n_\mathrm{R}\right)^2\right\rangle_\rho / (\bar{n}_\mathrm{L}+\bar{n}_\mathrm{R})$ scales as N^1, and $\mathrm{L}+\mathrm{R}$ types have an asymptotically finite excess fitness in the infinite-population limit. In the limit $Na\,(D-1)/D^2 \gg 1$, where the within-group variance remains $\mathcal{O}(1)$ as N grows large, it becomes sensible to regard *the group* as the dynamical actor, whose strategy is the $\pm$ sign for β in Eq. (B3). As the benefits to relatedness are apportioned equally to all group members, they constitute a sort of side-payment, leading to an interpretation of the group as acting in coalitional form relative to agents of type M, who are effectively independent.

This transition from large fluctuations and Hamilton-type relatedness near the bifurcation point, to an effective description of the group as a coalition, provides an example of the emergence of both coalition-constrained moves and coalition switching, suggested as a mechanism to support cooperative solution concepts in Sec. IV C 7. A feature of the pitchfork-bifurcation model that makes the notion of "emergent" coalitions natural is the fact that $\bar{\beta}$ scales as $\sqrt{\bar{\gamma}}$ near the bifurcation point. That is,

the mean-regression leading to exponential suppression of deviations from the instantaneous L- or R-dominated population state, may be made arbitrarily stronger than the fitness difference $\propto \bar{\gamma}$ that distinguishes the actors *within* the L + R coalition from the M types that make up a distinct (single-member) coalition.

Appendix C: Frenet coordinates on the limit cycle in the Rock-Paper-Scissors game

A *Frenet coordinate system* on a limit cycle is one whose principle axes are instantaneously tangent and normal to the cycle [107, 108]. These principle axes provide a convenient separation between mean-regressing noise normal to the cycle and Brownian motion tangent to it. For weakly broken RPS, the limit cycle is nearly circular and the Frenet coordinates approximate polar coordinates. A *Floquet* analysis integrates properties around cycles, to arrive at the long-time repeated dynamical states or convergence toward them.

In this appendix we compute the leading dependence of the radial coordinate on angle around the cycle, and verify that the transformation from polar to Frenet coordinates approaches the identity.[73] By showing that the deviations from circularity are harmonic at leading order, we justify the omission of oscillatory terms in the mean-field radius estimates in the main text.

Define polar coordinates (r, θ) on the unit simplex, so that $\theta = 0$ corresponds to the axis $\mathrm{n_R} = \mathrm{n_P}$. The reference scale for radius, $\bar{r}$, will be the value suggested by Eq. (43) of the text,

$$\bar{r}^2 \equiv \frac{1}{D} - \frac{2D}{N\,(a-b)}. \tag{C1}$$

The mean rate of advance of the phase θ is

$$\omega = \frac{a+b}{2\sqrt{3}} \tag{C2}$$

A normalized radius coordinate will be denoted

$$\rho \equiv \frac{r}{\bar{r}} \tag{C3}$$

Three further combinations of the game parameters appear in the exact stationary point equations:

$$\begin{aligned} A_1 &\equiv \bar{r}^2 \frac{a-b}{2\omega} \\ 2A_2^2 &\equiv \bar{r}^2 + A_1^2 \\ \tan\alpha &\equiv \frac{1}{3}\frac{a-b}{2\omega} \end{aligned} \tag{C4}$$

The small expansion parameter will be $\bar{r}$, in terms of which $A_2 \sim \mathcal{O}(\bar{r})$, $A_1 \sim \mathcal{O}\big(\bar{r}^2\big)$.

Then the stationary point condition from the mean-field equation of motion (15), in (ρ, θ) coordinates, becomes

$$\begin{aligned} \frac{d\rho}{\omega\, d\tau} &= \rho\left[A_2\rho\sin\left(3\theta+\alpha\right) + A_1\left(1-\rho^2\right)\right] \\ \frac{\rho\, d\theta}{\omega\, d\tau} &= -\rho\left[1-\eta_2\rho\cos\left(3\theta+\alpha\right)\right]. \end{aligned} \tag{C5}$$

The limit-cycle trajectories are not perfect circles, as shown in Fig. 14, and the angle of inclination to a pure radial vector due to the variation of ρ is

$$\begin{aligned} \frac{\dot{\rho}}{\rho\dot{\theta}} &= -\frac{A_2\rho\sin\left(3\theta+\alpha\right) + A_1\left(1-\rho^2\right)}{1-\eta_2\rho\cos\left(3\theta+\alpha\right)} \\ &\equiv \tan\xi \\ &\equiv \frac{d\log\rho}{d\theta}. \end{aligned} \tag{C6}$$

These polar coordinates lead to a simple small-parameter expansion if we solve Eq. (C6) for the limit-cycle trajectory, to give

$$\bar{\rho}\big(\bar{\theta}\big) = 1 + \frac{A_2}{3}\cos\left(3\bar{\theta}+\alpha\right) + \mathcal{O}\big(\bar{r}^2\big). \tag{C7}$$

At leading order $r = \bar{r}$ around the cycle, and the deviation at $\mathcal{O}\big(\bar{r}^2\big)$ is oscillatory with period three. This term cancels around one cycle, giving the approximate radial convergence equation (42) in the main text.

[73] A more explicit computation of the orthogonal transformation to Frenet coordinates is provided in the companion paper, where fluctuation variances are computed.

Appendix D: Recurrent finite-state automata for eight strategies in trembling-hand repeated Prisoners' Dilemma

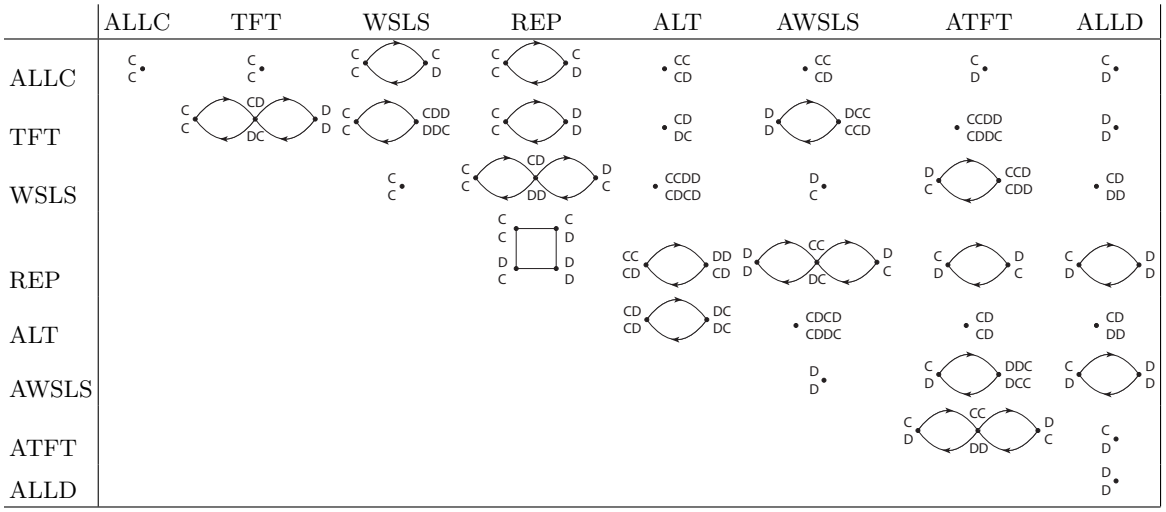

TABLE II. Recurrent finite-state automata visited by the eight strategies in trembling-hand Prisoners' Dilemma. Each node indicates a recurrent state, and the sequence of row- and column-player moves in that state is indicated. Arcs between the nodes are created by trembles with rare frequency per move. Transients and opening-move effects are not shown. In cases where nodes have cycles of different lengths, the probabilities on arcs are equal per term in the cycle. The practical consequence is that all cycles containing all four move profiles lead to equal visits when averaged over the late-time distribution over the recurrent states. Entries in the lower triangle (omitted for clarity) are the mirror images of those in the upper triangle.

Appendix E: Interaction terms between first-move and recurrent move in Prisoners' Dilemma

In this appendix we compute the mean-field dynamics of first-move and recurrent move alleles for the repeated Prisoners' Dilemma, supposing that these may vary independently by mutation or crossover. To simplify the analysis, we will not compute or present the full mean-field dynamics in six-dimensional type space, but rather will consider the direction of departure from an initial state with arbitrary allele frequencies in linkage equilibrium. Our interest will be the fitness differences between canonical and mutant TFT that result in a a dynamically maintained linkage of opening C with the recurrent TFT move. We will then consider the extent to which this is approximated by additive models with different levels of interaction terms.

1. Dependence of fitness on opening and recurrent moves

The canonical RPD strategies ALLC, ALLD, and TFT are defined by particular pairings of first moves with recurrent moves. Let primes $'$ indicate strategies with the same recurrent move but the opposite pairing. We will use superscript ^{0} to indicate quantities associated with opening moves, and $^\infty$ to indicate quantities associated with recurrent moves.[74]

[74] Changing first moves in ALLC and ALLD has no effect in the limit of large games. Substituting TFT$'$ for TFT, however, has the effect of producing a "dual" game. Duality here means that the roles of C and D are exchanged, and the sign of the payoffs is then reversed. Thus, as C is a repelling point in the game with TFT, D is the corresponding attracting point in the game with TFT$'$, which is thus eliminated apart from mutation and crossover effects.

We denote the opening moves by C and D, and recurrent moves by C, D, and T. As in the examples of the figures, we will suppose that the payoffs satisfy $R+P-S-T=0$. We will not write expressions for mutation and crossover explicitly, but we suppose that such terms are kept nonzero as regulators, and specifically that they are large enough that we may ignore terms of order $1/N_{\text{rounds}}$. Approximation $\approx$ instead of equality $=$ will indicate corrections of order $1/N_{\text{rounds}}$.

The population numbers for each of the recurrent moves are then the sums

$$\begin{aligned} \mathrm{n}_C^\infty &\equiv \mathrm{n}_C + \mathrm{n}_{C'} \\ \mathrm{n}_D^\infty &\equiv \mathrm{n}_D + \mathrm{n}_{D'} \\ \mathrm{n}_T^\infty &\equiv \mathrm{n}_T + \mathrm{n}_{T'}. \end{aligned} \tag{E1}$$

The corresponding population numbers for each of the first moves are given by

$$\begin{aligned} \mathrm{n}_C^0 &\equiv \mathrm{n}_C + \mathrm{n}_{D'} + \mathrm{n}_T \\ \mathrm{n}_D^0 &\equiv \mathrm{n}_{C'} + \mathrm{n}_D + \mathrm{n}_{T'}. \end{aligned} \tag{E2}$$

The fitness coefficients associated with the recurrent-move alleles in Eq. (E1) are readily obtained by taking the appropriate sums of the replicator equation on fully-specified types, and are given by

$$\begin{bmatrix} f_C^\infty \\ f_D^\infty \\ f_T^\infty \end{bmatrix} \approx \begin{bmatrix} R & S & R \\ T & P & P \\ R & P & X \end{bmatrix} \begin{bmatrix} \mathrm{n}_C^\infty \\ \mathrm{n}_D^\infty \\ \mathrm{n}_T^\infty \end{bmatrix}. \tag{E3}$$

The regression coefficients R, S, T, and P, are constants as in the main text, but X depends on population composition. With the assumption $R+P-S-T=0$, it is given by

$$X\mathrm{n}_T^\infty \approx R\mathrm{n}_T + P\mathrm{n}_{T'}. \tag{E4}$$

The fitnesses of types incorporating recurrent C and D are approximately independent of the opening move,

$$\begin{bmatrix} f_C - f_C^\infty \\ f_{C'} - f_C^\infty \end{bmatrix} \approx \begin{bmatrix} f_D - f_D^\infty \\ f_{D'} - f_D^\infty \end{bmatrix} \approx \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \tag{E5}$$

while those incorporating recurrent T depend on the first move as

$$\begin{bmatrix} f_T - f_T^\infty \\ f_{T'} - f_T^\infty \end{bmatrix} \approx \frac{R-P}{2} \begin{bmatrix} \mathrm{n}_{T'} \\ -\mathrm{n}_T \end{bmatrix}. \tag{E6}$$

Because $R-P>0$, mutant/recombinant TFT$'$ will be suppressed by selection relative to standard TFT, justifying our omission of the former in the examples of the main text.

If we denote by f_C^0 and f_D^0 the first-move fitnesses, and note that the mean fitness for alleles is the same as that for the population under any decomposition, which we have denoted ϕ, then the fitness differences for opening-move alleles are

$$\begin{aligned} \begin{bmatrix} f_C^0 - \phi \\ f_D^0 - \phi \end{bmatrix} &\approx \frac{1}{N} \begin{bmatrix} \mathrm{n}_D^0 \\ -\mathrm{n}_C^0 \end{bmatrix} \left[\left(\frac{\mathrm{n}_T}{\mathrm{n}_C^0} - \frac{\mathrm{n}_{T'}}{\mathrm{n}_D^0} \right) \begin{bmatrix} R & P & \frac{R+P}{2} \end{bmatrix} \begin{bmatrix} \mathrm{n}_C^\infty \\ \mathrm{n}_D^\infty \\ \mathrm{n}_T^\infty \end{bmatrix} + \frac{R-P}{2} \left(\frac{(\mathrm{n}_T)^2}{\mathrm{n}_C^0} + \frac{(\mathrm{n}_{T'})^2}{\mathrm{n}_D^0} \right) \right] \\ &\to \left(\frac{\mathrm{n}_T^\infty}{N} \right)^2 \begin{bmatrix} \mathrm{n}_D^0 \\ -\mathrm{n}_C^0 \end{bmatrix} \frac{R-P}{2} \end{aligned} \tag{E7}$$

The first line is the general form, and the second line indicates the form if the population is found in a linkage equilibrium where $n_T/\mathrm{n}_C^0 = \mathrm{n}_{T'}/\mathrm{n}_D^0 = \mathrm{n}_T^\infty/N$.

The first term on the first line of Eq. (E7) is a constant background part of the fitness of recurrent T, which simply carries along whichever first move is linked to it in excess, while the second term is a linkage-dependent, but always positive, preference for TFT over TFT$'$. If $\mathrm{n}_{T'} \to 0$, the correlation with n_C^0 is total, and the fitness in the first line is governed by the regression coefficients $\begin{bmatrix} R & P & R \end{bmatrix}$, with positive sign. If $\mathrm{n}_T \to 0$, the correlation with n_D^0 is total, and the fitness is governed by regression coefficients $\begin{bmatrix} R & P & P \end{bmatrix}$, with negative sign. Unless the population is sufficiently far from a linkage equilibrium in favor of $\mathrm{n}_{T'}$, we will have $f_C^0 \geq \phi^0/N \geq f_D^0$, and n_D^0 will be suppressed in proportion to its correlation with recurrent T.[75]

In the linkage-equilibrium case, we have

$$\begin{bmatrix} f_T - f_T^\infty \\ f_{T'} - f_T^\infty \end{bmatrix} \to \frac{N}{\mathrm{n}_T^\infty} \begin{bmatrix} f_C^0 - \phi \\ f_D^0 - \phi \end{bmatrix}. \tag{E8}$$

Therefore, in mean-field approximation, the direction of change from an initial state in linkage equilibrium is given by

$$\frac{d}{dt} \begin{bmatrix} \log\left(\frac{\bar{n}_T N}{\bar{n}_C^0 \bar{n}_T^\infty} \right) \\ \log\left(\frac{\bar{n}_{T'} N}{\bar{n}_D^0 \bar{n}_T^\infty} \right) \end{bmatrix} \equiv \begin{bmatrix} f_T - f_T^\infty - \left(f_C^0 - \phi \right) \\ f_{T'} - f_T^\infty - \left(f_D^0 - \phi \right) \end{bmatrix}$$

[75] Note that opening D may still be maintained by mutation in types where it is paired with recurrent C or D, where it is neutral, and then re-mixed through either recombination or mutations of the recurrent move.

$$\to \left(\frac{N}{\mathrm{n}_T^\infty} - 1\right) \begin{bmatrix} f_C^0 - \phi \\ f_D^0 - \phi \end{bmatrix}$$
$$\to \frac{\mathrm{n}_T^\infty}{N}\left(1 - \frac{\mathrm{n}_T^\infty}{N}\right) \begin{bmatrix} \mathrm{n}_D^0 \\ -\mathrm{n}_C^0 \end{bmatrix} \frac{R-P}{2}. \qquad \text{(E9)}$$

At general interior points $\mathrm{n}_T^\infty \neq 0, N$, starting from any linkage equilibrium, selection favors linkage of first-move C with recurrent T and suppresses linkage of first-move D with recurrent T.

2. Indicator functions and regressions of fitness

We now illustrate the genetic decomposition of fitness, first assuming additivity and noting its limitations, and then showing how interaction terms may be successively introduced until a stable and fully informative regression results. In order to study multilevel selection, we demonstrate cases where higher order interactions than those of the conventional normal-form game are required.

Let $i \in \{C, C', D, D', T, T', \}$ indicate a fully specified agent type. Introduce superscript notation $\{C^0, D^0\}$ for opening moves, and $\{C^\infty, D^\infty, T^\infty\}$ for recurrent moves. Use lowercase Greek indices for opening moves $\alpha, \beta \in \{C^0, D^0\}$, and capital Roman indices for recurrent moves $I, J \in \{C^\infty, D^\infty, T^\infty\}$. A fully specified agent type index corresponds to a concatenated pair of indices for opening and recurrent moves, as $i \leftrightarrow \alpha I$. With these notations we may introduce indicator functions for the sets corresponding to alleles, defined in the preceding subsection.

The Kronecker delta on fully-specified types is denoted $\delta_{\alpha I,i}$. In terms of these elementary indicator functions, the indicator function for an opening move may be written

$$\sigma_{\alpha i}^0 \equiv 1 - \prod_I \left(1 - \delta_{\alpha I,i}\right). \qquad \text{(E10)}$$

The corresponding indicator for a recurrent move is

$$\sigma_{Ii}^\infty \equiv 1 - \prod_\alpha \left(1 - \delta_{\alpha I,i}\right). \qquad \text{(E11)}$$

It is straightforward to check that the counting rules for set unions are satisfied,

$$\sum_i \sigma_{\alpha i}^0 = \mathrm{n}_\alpha^0$$
$$\sum_i \sigma_{Ii}^\infty = \mathrm{n}_I^\infty$$
$$\sum_i \sigma_{\alpha i}^0 \sigma_{Ii}^\infty = \mathrm{n}_{\alpha I}. \qquad \text{(E12)}$$

Because the recurrent move dominates for most types, the simplest regression is on the recurrent-move "gene" in Eq. (19),

$$f_i^{(0)} \approx \sum_j \sum_{IJ} \sigma_{Ii}^\infty \sigma_{Jj}^\infty a_{IJ}$$
$$= \sum_{IJ} \sigma_{Ii}^\infty a_{IJ} n_J^\infty \qquad \text{(E13)}$$

For this approximation the regression coefficients on recurrent moves are the same as those in the CDT game of the text – abusing notation, $a_{IJ} = a_{ij}$, except for the recurrent-T regression. The least-squares regression coefficient may readily be computed to be

$$a_{T^\infty T^\infty} = \left\langle \frac{R\mathrm{n}_T + P\mathrm{n}_{T'}}{n_T^\infty} \right\rangle$$
$$= \frac{R+P}{2} + \frac{R-P}{2}\left\langle \frac{\mathrm{n}_T - \mathrm{n}_{T'}}{n_T^\infty} \right\rangle, \qquad \text{(E14)}$$

where we use $\langle\ \rangle$ as a reminder that these regression coefficients are now functions of the (hidden) dynamical population variables n_T, $\mathrm{n}_{T'}$. The regression (E13) recovers the forms for all f_I^∞ of the preceding subsection, which were computed from the replicator dynamic acting on set unions. It both conflates types T and T', and produces a regression coefficient which may depend sensitively on the samples from the population process used to compute it.

We could try to improve the approximation (E13) with a regression that assumes additive genetic variance, by including an indicator function for the first move (out of many such possibilities, an interaction with opponent first moves is shown here),

$$f_i^{\mathrm{lin}} \approx \sum_j \left(\sum_{IJ} \sigma_{Ii}^\infty \sigma_{Jj}^\infty a_{IJ}^{\mathrm{lin}} + \sum_{\alpha\beta} \sigma_{\alpha i}^0 \sigma_{\beta j}^0 b_{\alpha\beta}^{\mathrm{lin}} \right)$$
$$= \sum_{IJ} \sigma_{Ii}^\infty a_{IJ}^{\mathrm{lin}} \mathrm{n}_J^\infty + \sum_{\alpha\beta} \sigma_{\alpha i}^0 b_{\alpha\beta}^{\mathrm{lin}} \mathrm{n}_\beta^0 \qquad \text{(E15)}$$

No regression of this form can generate coefficients $b_{\alpha\beta}$ large enough to be useful in the general case, because all but the f_T and $f_{T'}$ fitness functions are well-approximated by their f^∞ estimators. Thus even in this simple game additivity is violated at the single-gene level.

Therefore we consider interactions. The simplest interaction is between an agent's first move and recurrent move, still depending on the environment only through the opponent's recurrent move. We write this regression

$$f_i^{(1)} \approx \sum_j \sum_{IJ} \sigma_{Ii}^\infty \sigma_{Jj}^\infty \left(a'_{IJ} + \sum_\alpha \sigma_{\alpha i}^0 a_{\alpha IJ}^{(1,0)} \right)$$
$$= \sum_{IJ} \sigma_{Ii}^\infty \left(a'_{IJ} + \sum_\alpha \sigma_{\alpha i}^0 a_{\alpha IJ}^{(1,0)} \right) \mathrm{n}_J^\infty. \qquad \text{(E16)}$$

Again the recurrent-move regression coefficients almost always equal those in the text – $a'_{IJ} = a_{ij}$ – but now the constant part of the T-T interaction has changed from

the value in Eq. (E14) because an interaction term predicts a separate component of the variance. The constant coefficient

$$a'_{T^\infty T^\infty} = a_{T^\infty T^\infty} - \left(\frac{R-P}{4}\right)\left\langle \frac{\mathrm{n}_T - \mathrm{n}_{T'}}{n_T^\infty}\right\rangle = \frac{R+P}{2} + \frac{R-P}{4}\left\langle \frac{\mathrm{n}_T - \mathrm{n}_{T'}}{n_T^\infty}\right\rangle, \quad \text{(E17)}$$

and the first-order interaction term is now stable,

$$a^{(1,0)}_{C^0T^\infty T^\infty} = -a^{(1,0)}_{D^0T^\infty T^\infty} = \frac{R-P}{4}. \quad \text{(E18)}$$

Eq. (E16) now produces the correct averaged forms for f_T and $f_{T'}$, and half of the correct (constant) difference from Eq. (E6).

To obtain stable regression coefficients, we must introduce a second-order interaction. The relevant regression is written

$$f_i^{(2)} \approx \sum_j \sum_{IJ} \sigma^\infty_{Ii}\sigma^\infty_{Jj}\left(a''_{IJ} + \sum_{\alpha\beta}\sigma^0_{\alpha i}a^{(1,1)}_{\alpha I\beta J}\sigma^0_{\beta j}\right) = \sum_{IJ}\sigma^\infty_{Ii}\left(a''_{IJ}\mathrm{n}^\infty_J + \sum_{\alpha\beta}\sigma^0_{\alpha i}a^{(1,1)}_{\alpha I\beta J}\mathrm{n}_{\beta J}\right). \quad \text{(E19)}$$

Now the constant coefficient is stable at value

$$a''_{T^\infty T^\infty} = \left(\frac{R+P}{2}\right). \quad \text{(E20)}$$

For the simple example we have used to illustrate, with $R+P-S-T=0$, only an antisymmetric interaction between self- and opponent-first-moves is required,

$$a^{(1,1)}_{C^0T^\infty C^0T^\infty} = -a^{(1,1)}_{D^0T^\infty D^0T^\infty} = \frac{R-P}{2}. \quad \text{(E21)}$$

The regression coefficients in Eq. (E19) with second-order interactions in both opening and recurrent moves are thus needed to reconstruct the extensive-form game in a "genetic" description.

THERE WERE TWO SUBSUBS HERE; COME BACK AND DECIDE WHETHER TO KEEP THEM.

Appendix F: The replicator Prisoners' Dilemma in the predictive variables of the canonical Folk Theorems

In this appendix we map the move profiles generated by the evolutionary repeated Prisoners' Dilemma (RPD) to their counterparts in a strategic repeated game. The mapping will require a modification of the usual association between evolutionary and strategic games: that homogeneous populations map to pure strategies (for a population of cognitively identical strategic agents) while heterogeneous populations map to mixed strategies. In order to produce a faithful mapping between the evolutionary and strategic formulations of repeated play, it will be necessary to map a heterogeneous evolving population to two different concepts: first, a continuous-valued public signal outside the players' control, and second, a pair of mixed strategies that players may use, conditional on the presence or absence of the public signal. The fact that evolutionary RPD maps to this two-dimensional space could have been anticipated, on the grounds that the simplex in a three-type space (ALLC, ALLD, TFT) with fixed player number N is two-dimensional, while any mixed strategy between instantaneous moves C and D is only one-dimensional. The partition between the components of population composition that represent public signals versus mixed strategies will be related to the partition between memory-zero and memory-one strategy types.

We then show how the choice of the type space, together with the payoff coefficients (R,S,T,P), constructs a particular solution as the "target solution" which is supposed to result from prior agreement in the strategic-game Folk Theorems. The evolutionary dynamic will map – as it is expected to – onto the optimizing behavior of agents, while the dynamic within the repeated game will map onto the "punishment" strategies assumed in the strategic-game Folk Theorems.

1. Strict individual rationality maps without difficulty

As in the text, we consider the model with types ALLC, ALLD, and TFT, and deterministic moves. The move profiles produced by any interactions among these map directly to either persistent cooperation or the "trigger" strategies used to prove the Folk Theorem for Nash equilibria of the normal form for the repeated game [56, 57]. These Folk Theorems are therefore the appropriate versions for comparison.

The neutrality structure of RPD ensures that strict individual rationality poses no difficulty in the map from evolutionary to strategic solution concepts. ALLC and TFT are neutral with each other in any combination of play between the two, while ALLD and TFT are neutral when played against ALLD. Thus "punishment" is simply denying ALLD the gains of exploitation, and is an instantaneous best response even in the stage game. It is therefore not necessary to invoke more complex finite-term punishment strategies or indefinite play as are required by subgame-perfect Folk Theorems.

2. The map from heterogeneous populations to mixed strategies with either cost-free signaling or public signaling

We may convert the probabilities of the four recurrent-stage outcomes, given in terms of population numbers

in the replicator dynamic, in two forms which separate purely mixed from coordinated strategies:

$$\begin{aligned}
\begin{bmatrix} P_{\mathrm{C/D}} \\ P_{\mathrm{D/C}} \\ P_{\mathrm{D/D}} \\ P_{\mathrm{C/C}} \end{bmatrix} &= \frac{1}{N^2} \begin{bmatrix} \mathrm{n}_C \mathrm{n}_D \\ \mathrm{n}_D \mathrm{n}_C \\ \mathrm{n}_D^2 + 2\mathrm{n}_D \mathrm{n}_T \\ \left(\mathrm{n}_C + \mathrm{n}_T\right)^2 \end{bmatrix} \\
&= (1-\varphi)^2 \begin{bmatrix} \tilde{p}\,(1-\tilde{p}) \\ (1-\tilde{p})\,\tilde{p} \\ (1-\tilde{p})^2 \\ \tilde{p}^2 \end{bmatrix} + 2\varphi\,(1-\varphi) \begin{bmatrix} 0 \\ 0 \\ 1-\tilde{p} \\ \tilde{p} \end{bmatrix} + \varphi^2 \begin{bmatrix} 0 \\ 0 \\ 0 \\ 1 \end{bmatrix} \\
&= (1-\Phi) \begin{bmatrix} p\,(1-p) \\ (1-p)\,p \\ (1-p)^2 \\ p^2 \end{bmatrix} + \Phi \begin{bmatrix} 0 \\ 0 \\ 1-p^2 \\ p^2 \end{bmatrix}
\end{aligned} \tag{F1}$$

The parameters in the second line of Eq. (F1) are defined as

$$\begin{aligned}
\varphi &\equiv \frac{\mathrm{n}_T}{N} \\
\tilde{p} &\equiv \frac{\mathrm{n}_C}{\mathrm{n}_C + \mathrm{n}_D},
\end{aligned} \tag{F2}$$

while those in the third line are defined as

$$\begin{aligned}
\Phi &\equiv \frac{\mathrm{n}_T}{\mathrm{n}_C + \mathrm{n}_T} \\
p &\equiv \frac{\mathrm{n}_C + \mathrm{n}_T}{N}.
\end{aligned} \tag{F3}$$

We may re-formulate the interpretation of mixed play from a population-dynamic model into an equivalent model of a single type of optimizing player, able to use mixed strategies, and also endowed with knowledge of either the opponent's state or of a global signal.

The interpretation of the second line in Eq. (F1) is a literal translation of the effect that memory-one strategies have on the long-term payoff of the repeated game. $\varphi = \mathrm{n}_T/N$ is a probability that a player has "sight", and $1-\varphi$ is the probability that he is "blind". Sight partitions types in the original population between n_T and anything else, and the residual $\{\mathrm{n}_C, \mathrm{n}_D\}$ identify a single type of blind player who plays a mixed strategy C/D with weights $(\tilde{p}, 1-\tilde{p})$. Sight is local in that it may be treated as a binary variable sampled with probabilities $(\varphi, 1-\varphi)$ for each player at the time of play. Thus the idea that an individual may freely vary φ in the same way as $\tilde{p}$ maps the mutation-selection dynamic in a natural way onto the strategic concept of individual optimization. Mechanistically, Eq. (F1) says that blind players, when paired, mix independently over C/D; a sighted player paired with a blind player coordinates on the blind player's strategy; two sighted players paired with each other not only coordinate but optimize on the Pareto-superior coordinated strategy.

This partition of the population gives the opening move in the three composite strategies the interpretation of a cost-free signal, which may be used to coordinate the subsequent recurrent behavior that determines fitness. It cannot be included in a single-round 2×2 stage game, and indeed requires repetition over a sufficient number of rounds that the cost $\propto 1/N_{\mathrm{ply}}$ may be made as small as necessary to be regarded as cost-free. These conditions could also be interpreted as measures of the "cognitive complexity" of signaling strategies, which, if it is ruled out, requires the non-cooperative solution concept for the stage-game normal form.

The interpretation of the third line in Eq. (F1) replaces local sight with sight of a global signal, provided exogenously. Φ is the probability that the signal is provided in any single instantiation of the stage game, and $1-\Phi$ is the probability that it is absent. All players simultaneously know whether the global signal exists or not, and if not, they independently mix over C/D with probabilities $(p, 1-p)$, corresponding to the frequencies of the opening move in the population. In order to produce outcomes with the probabilities of the population model, we must suppose that if the signal exists, it takes some form such as a random variable uniformly distributed in the unit interval. Players coordinate on move C if the variable is above p^2, and move D otherwise. This "threshold" use of the global signal remains defined in terms of the same parameter as the independent mixed strategies, so that players have a single parameter to optimize. The dynamics of the probability Φ must be translated from the population model in some other terms, as a property of the game rather than an individual optimization parameter. While this interpretation requires greater complexity of the stage game, and assumes a rather arbitrary use of the mixing probabilities, it has the feature of expanding the feasible set to include coordinated strategies with one-stage non-cooperative solution concepts, and is therefore

within the class conventionally used to define the feasible set in strategic game theory.

The feasible set defined by the range of values $\Phi \in [0, 1]$ is shown in Fig. 26. Optimizers responsible only for p would move along the boundary $\Phi = 0$ away from C/C and toward D/D, as all mixed strategies are dominated by the pure-strategy Nash equilibrium of the stage game. Along the fully-coordinated boundary $\Phi = 1$ motion is away from D/D and toward C/C, because these are effectively coalitional strategies ordered by Pareto dominance. The payoff parameters (R, S, T, P), in a population with these three strategies, define a limit point of zero mutation rate along the C-T axis, which we associate with a value $\Phi_{\text{pop-dyn}}$. The map to the strategic-game formulation requires us to regard Φ as an externally controlled signal frequency, and p as a variable optimized by the players. Along each interior contour of fixed $\Phi \in (0, \Phi_{\text{pop-dyn}})$, one or more stable interior optima exist for p in the repeated game.[76] This optimum converges to $p \to 1$ for all $\Phi \geq \Phi_{\text{pop-dyn}}$.

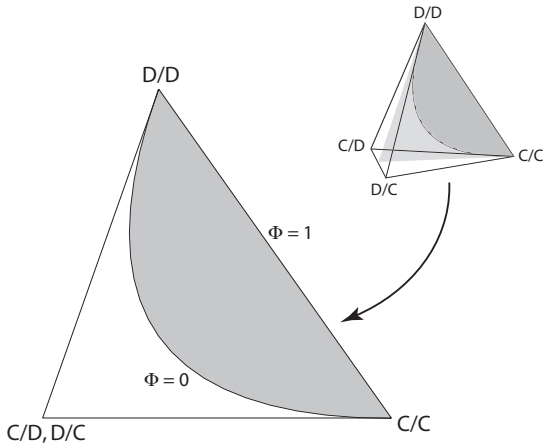

FIG. 26. The four possible recurrent move profiles in RPD are C/C, D/D, C/D, D/C. All possible matchings of types produce frequencies for these move profiles shown in the 3-dimensional probability simplex, inset. The set of role-symmetric feasible strategies is contained within the vertical plane through this simplex at $P_{\text{C/D}} = P_{\text{D/C}}$, shaded in the inset, and plotted by projecting the full simplex onto this plane in the main panel. Without a public signal to enable coordination, the feasible set is the one-dimensional set of products of mixed strategies, indicated by the heavy curve $\Phi = 0$. When players can use a public signal, the feasible set expands to the shaded lens between $\Phi = 0$ (no signal) and $\Phi = 1$ (signal present in every round). The contour $\Phi = 0$ maps in Fig. 19 onto population configurations on the lower axis between ALLD and ALLC, while the contour $\Phi = 1$ maps onto the upper-left axis between ALLD and TFT.

[76] Usually this stable optimum is unique. The reader may check from the graphs that along a narrow range of Φ, for the parameters used in the examples, this solution splits into an unstable rest point and two stable rest points at high and low p.

Appendix G: Variation in the linked-heterosis model, and the heritable component of fitness

In this appendix we present a stochastic differential equation for the linked-heterosis model of Sec. IV H, and derive the effective fitness that results from the positive selection for population covariance.

Consider the role of fluctuations due to random pairing, about a background in which $\langle \sigma^k \rangle_{\text{n}} = 0$, $\forall k$, so that the population is evenly mixed on average for L and R alleles at each locus, and the only remaining characteristic to be selected is correlation between loci. Then $f_i = 0$, $\forall i$ and $\phi = 0$ as well. We will make the further simplifying assumption that $\text{n}_i = \text{n}_{-i}$, for all pairs $(i, -i)$ which differ by sign change of each σ_i^m, because such symmetrized pairs share all fluctuation properties, and our goal is to understand how fluctuations affect such pairs as a result of their correlation structures.

1. Langevin equation to capture the fitness consequences of random pairing of players

Let the consequences of random pairing be represented by a Langevin field, so that in the notation of the Price equation, the number of descendents from individuals of type i is a random variable written

$$\text{n}_i' = (1 + \xi_i)\, \text{n}_i. \tag{G1}$$

By Eq. (63), the expectation $\langle \xi_i \rangle_t = \Delta t\, (f_i - \phi) = 0$, where $\langle\ \rangle_t$ denotes the average over pairings in some interval Δt starting from time t. Then, by Eq. (64) at $f_i = 0$, the variance in ξ_i is given by

$$\langle \xi_i^2 \rangle_t = \frac{(\Delta t)^2}{\text{n}_i} \sum_j a_{ij}^2 \frac{\text{n}_j}{N} \tag{G2}$$

To conserve total number, it is necessary that $\sum_j \text{n}_j \xi_j \equiv 0$ in each realization of the variables ξ, leading to the requirement that in correlations

$$\sum_j \text{n}_j \langle \xi_i \xi_j \rangle_t \equiv 0,\ \forall i. \tag{G3}$$

Now we consider the fluctuations in a succeeding interval Δt, starting at a time $t' \equiv t + \Delta t$. Putting aside mutation or crossover effects, which we will assume occur at a low rate, the number of second-generation descendants is

$$\text{n}_i'' = (1 + \xi_i')\, (1 + \xi_i)\, \text{n}_i. \tag{G4}$$

In the context of the fluctuation at t, the Langevin field ξ_i' now has a nonzero expectation at t' due to fitness change in the population,

$$\langle \xi_i' \rangle_{t'} = \Delta t\, (f_i' - \phi'), \tag{G5}$$

in which

$$f_i' = f_i + \sum_j a_{ij} \frac{\mathrm{n}_j}{N} \xi_j, \tag{G6}$$

and $\phi' = \sum_i \mathrm{n}_i f_i'/N$ as usual. We assume that apart from the mean value (G5), fluctuations ξ_i' are uncorrelated with fluctuations ξ_i.

2. Relaxation response to fluctuations, and effective fitness

The apparent fitness of any individual in a type i over two time lags, taking into account the relaxation of the population in response to previous fluctuations, is

$$\begin{aligned} \frac{\langle\langle \mathrm{n}_i'' - \mathrm{n}_i \rangle\rangle_{t',t}}{\mathrm{n}_i} &= \langle \langle \xi_i \rangle_{t'} \xi_i \rangle_t \\ &= \Delta t \langle (f_i' - \phi') \, \xi_i \rangle_t \\ &\equiv 2\Delta t \left(f_i^{\mathrm{eff}} - \phi^{\mathrm{eff}} \right). \end{aligned} \tag{G7}$$

If we choose Δt to represent approximately the time over which fluctuation correlations have delayed, the *effective fitness* f_i^{eff} will correspond to the coefficient obtained by regression of the mean population change among types that differ by their correlations. The quantity determining the effective fitness in Eq. (G7) is the correlation

$$\begin{aligned} \langle f_i' \xi_i \rangle_t &= \sum_j a_{ij} \frac{\mathrm{n}_j}{N} \langle \xi_j \xi_i \rangle_t \\ &= \sum_{j \neq i} (a_{ij} + 1) \frac{\mathrm{n}_j}{N} \langle \xi_j \xi_i \rangle_t. \end{aligned} \tag{G8}$$

In the second line we have used the cancellation of the covariance terms in Eq. (G3), together with the identity $a_{ii} \equiv -1$, $\forall i$.

In order to evaluate Eq. (G8), it is necessary to obtain an estimate for the off-diagonal covariance terms $\langle \xi_j \xi_i \rangle_t$. To obtain this, we consider the off-diagonal counterpart to Eq. (G7)

$$\begin{aligned} \langle \langle \xi_i' \rangle_{t'} \xi_k \rangle_t &= \Delta t \langle (f_i' - \phi') \, \xi_k \rangle_t \\ &= \frac{\Delta t}{N} \left\{ a_{ik} \mathrm{n}_k \langle \xi_k^2 \rangle_t - \mathrm{n}_i \langle \xi_i \xi_k \rangle_t + \sum_{j \neq i,k} \mathrm{n}_j a_{ij} \langle \xi_j \xi_k \rangle_t + \mathcal{O}\left(\frac{1}{N} \right) \right\}, \end{aligned} \tag{G9}$$

in which $\mathcal{O}(1/N)$ stands for terms from $\langle \phi' \xi_k \rangle_t$ which we do not write down.

We now make the self-consistency ansatz that for a suitable choice of Δt, the two terms

$$\langle \langle \xi_i' \rangle_{t'} \xi_k \rangle_t + \langle \langle \xi_k' \rangle_{t'} \xi_i \rangle_t \approx \langle \xi_i \xi_k \rangle_t \tag{G10}$$

in steady state. These two terms are the relaxation responses to primary fluctuations caused by random pairing, during the time interval before the primary correlations have regressed to the mean.

From the self-consistency condition (G10) we obtain a *Dyson equation* for the off-diagonal correlation terms,

$$\left(1 + \Delta t \frac{\mathrm{n}_i + \mathrm{n}_k}{N} \right) \langle \xi_i \xi_k \rangle_t - \frac{\Delta t}{N} \sum_{j \neq i,k} \mathrm{n}_j \left(a_{ij} \langle \xi_j \xi_k \rangle_t + \langle \xi_i \xi_j \rangle_t a_{jk} \right) = \frac{\Delta t}{N} a_{ik} \left(\mathrm{n}_i \langle \xi_i^2 \rangle_t + \mathrm{n}_k \langle \xi_k^2 \rangle_t \right) \tag{G11}$$

We now make use of the assumption that sufficiently many types are populated, that number conservation does not enforce stronger correlations between types than those induced by fluctuating fitness. Under this assumption $\mathrm{n}_i/N \ll 1$, $\forall i$. Then the leading term in the solution to the Dyson equation (G11) is

$$\begin{aligned} \langle \xi_i \xi_k \rangle_t &\approx \frac{\Delta t}{N} a_{ik} \left(\mathrm{n}_i \langle \xi_i^2 \rangle_t + \mathrm{n}_k \langle \xi_k^2 \rangle_t \right) \\ &= \frac{(\Delta t)^3}{N} a_{ik} \sum_j \left(a_{ij}^2 + a_{kj}^2 \right) \frac{\mathrm{n}_j}{N} \end{aligned} \tag{G12}$$

where the second line makes use of Eq. (G2). The important conclusion of Eq. (G12) is that among non-identical types, the equal-time covariance of fluctuations $\langle \xi_i \xi_k \rangle_t$ has the sign of the pair-fitness function a_{ik}, weighted by the rate of primary fluctuation generated for the two types.

Plugging Eq. (G12) back into the expressions (G7,G8) for the two-timelag effective fitness yields

$$f_i^{\mathrm{eff}} = \frac{1}{2} \langle f_i' \xi_i \rangle_t$$

$$\approx \frac{(\Delta t)^3}{2N} \sum_{k\neq i} \left(a_{ik}^2 + a_{ik}\right) \frac{\mathrm{n}_k}{N} \sum_j \left(a_{ij}^2 + a_{kj}^2\right) \frac{\mathrm{n}_j}{N}$$

$$\approx \frac{(\Delta t)^3}{2N} \sum_{k\neq i} a_{ik}^2 \frac{\mathrm{n}_k}{N} \sum_j \left(a_{ij}^2 + a_{kj}^2\right) \frac{\mathrm{n}_j}{N} \qquad \text{(G13)}$$

The approximation in the second line of Eq. (G13) is that terms at $\mathcal{O}(1/N)$ as well as higher-order terms in the Dyson equation (G12) have been dropped from the expression for $\langle \xi_i \xi_k \rangle_t$. A further approximation at the same order is made in the third line, in which the linear term in a_{ik} sums to $\mathcal{O}(1/N)$. The reason is that, for any two indices k and $-k$, defined so that $\sigma_k^m = -\sigma_{-k}^m$, $\forall m$, $a_{ki} = -a_{-ki}$, $\forall i$. Meanwhile, $a_{kj}^2 = a_{-kj}^2$, so the argument in the sum on k is antisymmetric under $k \to -k$. If the sum were complete, and the approximation (G12) were applicable to $k = i$, then in any background n symmetric under $i \leftrightarrow -i$, the antisymmetric term would vanish entirely. The residual from complete vanishing is $-2\mathrm{n}_i \sum_j a_{ij}^2 \mathrm{n}_j / N^2$, which is $\mathcal{O}(n_i/N)$ relative to the sum on symmetric terms in a_{ik}^2.

3. The heritable component of fitness

We may estimate the result of running a regression on the complicated sum (G13), to obtain the heritable component of the effective fitness. Such estimates are qualitatively coarser than the $\mathcal{O}(1/N)$ approximations that have been made up to now, in that dependencies on the fine structure of the population will be absorbed into overall prefactors.

The overlap function $a_{ik}^2 \sim 1$ for $k \sim i$ and $k \sim -i$, and zero for types with small correlation with i. Meanwhile, $a_{kj}^2 \sim a_{ij}^2$, $\forall j$, for all such k with large overlap. Therefore, an estimate for the effective fitness becomes

$$f_i^{\mathrm{eff}} \sim \frac{(\Delta t)^3}{N} \sum_j \frac{\mathrm{n}_i}{N} \frac{\mathrm{n}_j}{N} a_{ij}^2$$

$$= \frac{(\Delta t)^3 \mathrm{n}_i}{N^2} \frac{1}{4N_{\mathrm{ply}}^2} \sum_{k,k'} \sigma_i^k \sigma_i^{k'} \mathrm{Cov}\Big(\sigma^k, \sigma^{k'}\Big) \,. \qquad \text{(G14)}$$

In the second line we have re-introduced appropriate factors f_i, which were set to zero for the balanced-allelic populations assumed in this analysis, and made use of Eq. (62) in the text to re-express the dependence on population composition $\{\mathrm{n}_j\}$ in terms of the allele covariance. We see that the heritable component of fitness is expected to be $\sim (\Delta t)\, \mathrm{n}_i / N_2$ times the variance in individual reproductive success over pair assignments as given in Eq. (62).

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