Sex in Space-Time: How Discrete Interactions Favor Sexual Reproduction

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Abstract

The prevalence of sexual reproduction in nature remains an evolutionary paradox, given its substantial costs compared to asexual alternatives. While genetic explanations such as the Red Queen hypothesis emphasize coevolution with parasites, the role of spatiotemporal discreteness and population structure has been underexplored. Spatial structure matters because real populations are finite, interact locally, and are subject to demographic noise, factors that can fundamentally alter evolutionary outcomes. Here, we show that the discrete, nonlinear character of interactions between hosts and pathogens, when combined with spatial diffusion in low-dimensional habitats, naturally favors sexual reproduction. Using stochastic reaction-diffusion models and renormalization group analysis, we demonstrate that asexual populations face certain extinction in dimensions $d \leq 2$ due to discreteness-induced extinction (DIE), whereas sexual populations can persist and form stabilizing clusters. The mechanism hinges on the recurrence of random walks in low dimensions, which ensures pathogen-host encounters for asexuals but promotes protective clustering for sexuals. Our results suggest that the advantage of sex may emerge from purely physical and demographic constraints, independent of genetic mechanisms, thereby offering a new perspective on this classic evolutionary puzzle.

Keywords: | Paradox of sex | Red Queen theory | Stochastic processes | Complex Systems | Spatial Patterns

1 Introduction

Sex is an evolutionary puzzle [1, 2]. In several ways, sexual reproduction is less efficient when compared with the asexual method [3]. All offspring produced by asexual individuals will be able to reproduce, whereas sexual beings need to spend energy on creating males and females that do not reproduce separately [4]. Hence the resources spent on producing sons are a cost of sexual reproduction and asexual species economize on males. John Maynard Smith [5] summarized this argument as follows:

> *"Suppose a population consists of a mixture of sexual and parthenogenetic females, the former producing equal numbers of male and (sexual) female offspring, and the latter only parthenogenetic females like themselves. If the two kinds of female lay equal numbers of eggs, and if survival probabilities are equal, then the parthenogenetic type will have a twofold selective advantage, and will increase in frequency very rapidly. Sexual reproduction means that a female wastes half her energy producing males."*

He also noted that a sexual individual uses only half of its genetic material on its descendants, while an asexual individual uses all their genes. That is, in the evolutionary race where passing on genes to the next generation is one of the greatest goals, sexual organ-

isms start with a disadvantage of almost 50%, which is known as the *cost of meiosis* [3]. There is also the risk of infection by sexually transmitted diseases, the fact that sexual reproduction is often slower than asexual reproduction, and that during mating, individuals are typically less able to gather resources and evade predators [3, 6]. In addition to these disadvantages, and perhaps more important, is the cost of having to find a mate [6]. If insects are excluded, approximately one-third of animal species are hermaphrodites [7]. Hermaphroditism is even more widespread in plants. The difficulty of finding mates is widely implicated in the evolution of hermaphroditism, so its widespread occurrence suggests that sexual organisms incur significant costs to locate mates [8].

While many models have focused on genetic mechanisms such as mutational load or host-parasite coevolution [9], fewer have addressed the role of spatial stochasticity and finite population effects. Yet, real populations are finite, spatially structured, and subject to demographic noise, factors known to profoundly alter evolutionary dynamics [10]. In particular, the discrete nature of individual interactions and the dimensionality of the environment can lead to emergent behaviors not captured by mean-field models [11]. For host-parasite systems, spatial structure can modulate encounter rates, create refuges, and influence local adaptation, all of which may tip the balance between reproductive strategies. This motivates our investigation into whether spatial discreteness alone—absent genetic detail—can confer an advantage to sexual reproduction.

Sex, therefore, seems to be a luxury that should not exist. Consequently, many works about its evolution look for its compensatory benefits [12].

Since sexual reproduction exists, biologists seek to identify the significant benefit it confers. Maynard Smith argued that sex could only have evolved if this benefit outweighed the substantial cost of meiosis. An interesting theory addressing this question was proposed in [13]. According to this work, the parasites *are everywhere and will always seek, by their nature, to exploit their hosts.* As the generation time of parasites is many times smaller than that of hosts, and their evolutionary rates therefore many times higher, the only way out for the hosts is to produce offspring with greater genetic variability through sexual reproduction. Therefore, competition with parasites that evolve quickly [14] favors sexual reproduction, which enables more rapid genetic adaptation [15, 16, 2].

The world in which this model is inserted became known as the *Red Queen world*, a name given in [17] in reference to a passage in the fable Alice in the mirrors [18]. In this passage, Alice flees the army (of cards) of the Red Queen, but can not distance herself from her pursuers. The Red Queen then says: "Now, here, you see, it takes all the running you can do, to keep in the same place". Alice would be caught only if she stopped running. Things have to change to remain the same.

According to [13], an arms race has been underway between hosts and parasites since life appeared on Earth. The parasites are always breaking the defensive barriers imposed by the host's genotype, while the host, with the help of sex, continually creates new defenses. In the absence of sex, the hosts would remain essentially the same, while the parasites would accumulate adaptations that would enable them to break all the defensive systems of the former. Sooner or later, the hosts would be virtually devoured from the inside out. To escape the parasite army besieging them, the only remaining option is to just keep running. The coevolutionary cycle of parasites and hosts reflects this eternal pursuit. For criticism to the Red Queen theory, see [19, 20].

In recent years, there has been a resurgence of interest in understanding how spatial structure, demographic stochasticity, and the discrete nature of individuals shape the evolution of reproductive strategies. Experimental evolution studies using microbial systems have demonstrated that spatial structure can dramatically alter the relative fitness of sexual and asexual populations [21, 22], while theoretical work has begun to explore the interplay between dispersal, local adaptation, and mating systems [10, 23]. The application of statistical physics tools to ecological and evolutionary questions has also gained momentum, with studies investigating noise-induced transitions [24], critical phenomena in population dynamics [11], and the role of dimensionality in spatial epidemics [25]. More recently, the effects of demographic stochasticity on the maintenance of sex have been examined through individual-based models [26, 27, 28], highlighting the importance of finite population effects. Specifically, Silva et al. [28] demonstrated that the interplay between Allee effects and environmental stress determines the relative success of sexual versus asexual strategies during range expansion into marginal habitats. Orive et al. [29] showed that mixed reproductive strategies (partial clonality) can facilitate adaptation to spatially heterogeneous environments, a finding that resonates with the clustering dynamics observed in our model. Concurrently, mathematical refinements of the Red Queen hypothesis continue to emerge; Alfaro et al. [30] developed a phenotypically-structured PDE model demonstrating that perpetual evolutionary

chases between hosts and pathogens can arise without external forcing. Thompson et al. [31] further established that the discrete nature of individual organisms fundamentally alters spatial coexistence mechanisms compared to continuous-density approximations, directly motivating the field-theoretic approach we adopt here. Finally, Kafri and Fisher [32] demonstrated that Red Queen phases of continual evolution can emerge from purely ecological interactions without host-pathogen arms races, suggesting that perpetual evolutionary dynamics may be a generic property of diverse ecological systems. Collectively, these recent developments underscore the need for systematic analysis of how the discrete, spatial nature of host-pathogen interactions can alone favor sexual reproduction — independent of genetic mechanisms — a gap that the present work aims to fill.

The aim of this paper is to investigate the prevalence of sexual reproduction observed in nature through simple models of reaction-diffusion inspired by the Red Queen theory, but *not* fully equivalent to it. Unlike the Red Queen, our model excludes genetic variation and host-parasite coevolution*; instead, it isolates the role of spatial discreteness and demographic noise in shaping reproductive outcomes. The role of the parasite may be replaced by any pathogen which diffuses through space and fatally harms the species. There is also no need for any aspect related to genetics [33, 2].

Over the years, evolutionary theory has shown that the answer to the paradox of sex is more elusive than we initially thought. While most biologists are comfortable with the idea that sex exists to provide genetic variability [2], several mathematical models have shown that this comfort is unjustified. According to [34], *"...the theory of population genetics, as complete as it may be in itself, fails to deal with many problems of primary importance for an understanding of evolution."* After all, it is risky to produce offspring by randomly mixing genes with those of another individual. In the words of [35]:

> *"... sex need not increase variability, variability need not be beneficial and evolution need not favor sex, even when it does increase variability and variability is beneficial."*

*We consider a lethal pathogen (or parasite) that diffuses randomly and kills hosts upon encounter. While the Red Queen hypothesis traditionally emphasizes parasitic coevolution, our model simplifies this interaction to a fatal encounter, making "pathogen" the more accurate term. Nevertheless, we use both terms interchangeably to maintain connection with the biological literature.

Our approach complements these genetic perspectives by focusing on the spatiotemporal stochasticity inherent to finite populations. We build upon a growing body of work that applies tools from statistical physics to ecological and evolutionary dynamics [36, 37, 38]. In particular, the phenomena of discreteness-induced extinction and coexistence we describe are closely related to noise-induced transitions in population models [24], and our spatial analysis aligns with studies of epidemic spreading and evolutionary games on structured populations [39, 40].

There is also a well-documented pattern of high frequency of sex in undisturbed, biologically complex habitats [41, 9, 42, 43] where disease and other "natural enemies" are prevalent [41, 9, 44]. The recognition of the predominance of sex in stable communities has devalued models hypothesizing that sex is common because it provides a selective advantage when abiotic conditions are unpredictable [9, 45, 46, 47].

Moreover, the need to take into account aspects of complexity that are routinely neglected is widely recognized [35]. One reason that an answer to the paradox of sex has been so elusive is that many mathematical models have focused on populations that are infinite in size, unstructured, and isolated from other species. Satisfactory explanations for the paradox of sex should consider *finite* populations of agents that interact in an environment where *structure* and *complexity* are able to *emerge*.

To incorporate a few of these elements, we take into account the *discrete* nature of the species interactions with itself and with its pathogens/parasites, in a $d-$dimensional space. Discreteness of the interactions is a consequence of the finiteness of the populations, interactions with parasites characterize the non-insulation explicitly, and a $d-$dimensional space enables structure and complexity to emerge.

We know that if we want information about the emergent aggregate macroscopic behavior of complex systems, we must consider the discrete, particle-like nature of interacting individuals [48]. We will achieve this goal by employing dynamic renormalization group (DRG) theory to obtain the renormalization group (RG) flow in the parameter space [11, 49]. Starting from the microscopic formulation of the model described by reactions, this RG flow will allow us to understand *how the model parameters scale in space and time*. In turn, this information will be helpful in determining the final equilibrium state of the aggregates of interacting species. Such aggregates imply the advantage of sex on the population - or group-level [50, 51, 52, 53]. This approach is consistent with the perspective of extensive time and space scales taken by

macro-ecology and biogeography [54, 55, 56]. Large-scale spatial correlations are likely to be important for understanding the evolutionary influence of predator and prey or host and pathogen ecology [57].

It is our intention to call attention to an intrinsic feature of sexual reproduction that's been neglected so far. The longevity of the problem indicates that the solution requires a variety of causal effects, and what we propose here could be just one of these effects.

In spatial stochastic systems, two key phenomena arise from discrete interactions: *discreteness-induced coexistence* (DIC), where local clustering enables global persistence, and *discreteness-induced extinction* (DIE), where local fluctuations drive populations to extinction. While our previous work established the general occurrence of DIC [58] and DIE [59], the present study represents a significant conceptual advance by applying this framework to resolve a long-standing evolutionary paradox. We demonstrate how the same physical principles simultaneously explain both the inevitable extinction of asexual populations and the persistence of sexual ones in low-dimensional environments, where these effects are especially pronounced due to the recurrent nature of random walks, thereby unifying these seemingly contradictory outcomes under a single mechanistic explanation.

In this paper, we bridge this gap by combining elements of the Red Queen theory with tools from nonequilibrium statistical mechanics. We ask: Can the discrete, spatial nature of host-pathogen interactions alone confer an advantage to sexual reproduction? To answer this, we introduce minimal stochastic models that exclude genetics but include spatial structure, discreteness, and diffusion-limited interactions. Using field-theoretic renormalization group techniques, we show that the scaling behavior of model parameters under spatial coarse-graining reveals a strong dependence on spatial dimension, with sexual reproduction favored in low-dimensional settings.

Building on our previous discoveries of discreteness-induced coexistence (DIC) and extinction (DIE) as separate phenomena, we now demonstrate their unification to explain the evolutionary stability of sexual reproduction. This reveals a fundamental connection between spatial stochasticity and one of biology's oldest puzzles.

1.1 Models

In this subsection we present the two models used. They are simplified models that attempt to capture only the essential aspects of population dynamics. The first refers to the competition between an asexual species and a pathogen that can harm it, eventually inducing death. The second is the analogous model for the sexual species. The incorporation of the pathogen in interactions was inspired by the Red Queen theory regarding the host's parasites. In principle, however, any other death-inducing agent can be imagined. The models are as follows:

Asexual species model

The model for asexual species is described by the following reactions, occurring in a $d-$dimensional lattice:

$$A \xrightarrow{\lambda} 2A \qquad\qquad A + B \xrightarrow{\mu} B \tag{1}$$

The first reaction describes the reproduction of species A, which occurs at rate λ per time unit at a given site. The second reaction describes the fatal attack of pathogen B on host A: when an A and a B occupy the same site, A is annihilated at rate μ. The pathogen B is assumed to diffuse randomly but neither reproduces nor dies; this reflects a scenario inspired by the Red Queen world where pathogens are ubiquitous and long-lived, continually seeking hosts. This diffusion can be represented as $B + \emptyset \to \emptyset + B$, where $\emptyset$ denotes an empty site.

Sexual species model

The model for sexual species is described by the following reactions:

$$2A \xrightarrow{\lambda} 3A \qquad\qquad A + B \xrightarrow{\mu} B \tag{2}$$

The first reaction describes species A's reproduction that occurs at rate λ per time unit per population size unit, always at a given site of the lattice. Because two agents are required to reproduce a third, this reaction captures the cost of finding a mate. Everything else is as in the previous model.

The quadratic reproduction term $2A \xrightarrow{\lambda} 3A$ is the minimal nonlinearity that captures the Allee effect [60] arising from obligate pairing. It is a standard approximation in spatial population models for sexual reproduction [61, 62] and deliberately excludes genetic mixing to isolate demographic effects.

This formulation captures the essential difficulty of mate-finding in sexual populations: reproduction requires the co-location of two individuals, inducing an Allee effect. While it omits genetic recombination, it isolates the demographic consequence of obligate pairing in a spatial context.

This is not the first time that a model of this type is proposed for population dynamics incorporating the

Allee effect[†] [63, 64, 65] on the lattice. For recent references, see [66, 67, 60]. In the process known as the quadratic contact process (QCP) [68], we also have similar reactions. QCP is sometimes called the process of sexual reproduction [61].

It is very important to keep in mind that we are considering the critical situation where the concentration of species in the lattice is initially very small and the dynamics is dominated by diffusion [69]. The average number of agents per site is initially much lower than 1 and sexual reproduction is therefore penalized. Only in this critical regime does it make sense to use the renormalization group techniques. This critical regime is particularly relevant for understanding how sexual populations can recover from bottlenecks or colonize new habitats, scenarios where the twofold cost of sex is most severe.

Our models belong to a broader class of stochastic spatial processes studied in ecology and statistical physics [70]. The asexual model is a variant of the contact process, while the sexual model incorporates an Allee effect via the $2A \longrightarrow 3A$ reaction, analogous to the quadratic contact process (QCP) [62]. Similar formulations have been used to study mating systems [67] and the origin of life [46], but here we apply them to the paradox of sex.

2 Results

To compare the fates of asexual and sexual populations in spatially structured environments, we first analyze mean-field predictions and then incorporate spatial stochasticity through renormalization group analysis. We begin with the simplest analytical approximation. In the following subsection (2.1) we consider A or B as the *average* density of agents in the sites of the lattice.

2.1 Mean-field theory

Asexual model

Using the law of mass action, we obtain the differential equations for asexual species: $\dot{A} = D_A\nabla^2 A + \lambda A - \mu AB$ and $\dot{B} = D_B\nabla^2 B$ with D_A and D_B being diffusion coefficients. The diffusion processes only tend to homogenize populations in space and nabla operators will be neglected from now on in this subsection. The B population is a constant on average denoted by $\langle N_B\rangle$ and therefore $\dot{A} = (\lambda - \mu\langle N_B\rangle)A \equiv mA$, which defines $m = \lambda - \mu\langle N_B\rangle$. We see that if $\mu\langle N_B\rangle < \lambda$, $m > 0$ and the A population tends exponentially to infinity. Otherwise, $m < 0$ and the A population becomes extinct. If $m = 0$, the A population remains constant.

Sexual model

In this case, the equation for population dynamics already disregards diffusion terms and settings $\kappa \equiv \mu\langle N_B\rangle$ is $\dot{A} = \lambda A^2 - \kappa A \equiv -dV/dA$ with $V \equiv -\lambda A^3/3 + \kappa A^2/2$. V is an effective potential that allows a pictorial view of the dynamics, as illustrated in Figure (1). The point P on the potential maximum has coordinates $(A_{\max}, V_{\max}) = (\kappa/\lambda, \kappa^3/6\lambda^2)$. For any initial population $A(0) < \kappa/\lambda$, the population tends to be extinct. This fact is illustrated in figure (1) by the tendency of the red ball to move down the curve to the origin, characterizing the aforementioned Allee effect. If $A(0) > \kappa/\lambda$, population tends to infinity, a fact represented by the tendency of the green ball to get lost in the bottomless potential hole.

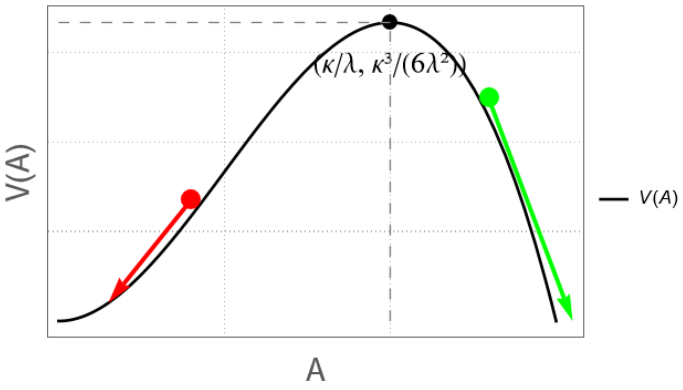


Figure 1: **Schematic representation of the effective potential** $V(A)$. The black point P has coordinates $(A_{\max}, V_{\max}) = (\kappa/\lambda, \kappa^3/6\lambda^2)$.

In the next subsections we will see how the κ, λ and μ parameters change with successive renormalization group iterations, or, in other words, how the discrete nonlinear species interactions in space-time induce variations in the numerical parameter values. These changes can transform very significantly the potential barrier to be overcome (given by $\kappa^3/6\lambda^2$) by the population.

2.2 Statistical Field Theory

Asexual model

We are interested in the way the parameters λ and m vary when we increase the observation scales of time and space. Using the results of statistical field theory

[†]For all sexual populations, there is a density threshold below which the probability of finding a mate is too low to ensure sufficient reproduction for the population to remain viable.

[71, 72, 73], we get the following flow equations for these parameters in the asexual model (see [74]):

$$\frac{d\mu}{dl} = \epsilon\mu + \frac{\mu^2}{2\pi\bar{D}} \tag{3a}$$

$$\frac{dm}{dl} = 2m - \frac{\langle N_B\rangle\mu^2}{2\pi\bar{D}}, \tag{3b}$$

where $\epsilon = 2 - d$ (d is the dimension of space), $l = \ln(s)$ (s is the scale parameter, see [74]), and $\bar{D} \equiv (D_A + D_B)/2$.

The flow of μ to large values under RG iterations indicates that the effective predation pressure increases at larger scales, leading to the extinction of asexual populations for $d \leq 2$. This phenomenon, which we term Discreteness-Induced Extinction (DIE), is a consequence of the recurrence property of random walks in low dimensions, which ensures that pathogens eventually locate all hosts. This interpretation is further elaborated in Section 3.

Derivation details. The derivation of the flow equations follows the Doi-Peliti field-theoretic formalism combined with dynamic renormalization group techniques. A detailed step-by-step derivation, including the mapping from the stochastic lattice model to the field theory, the Feynman diagram expansion, and the calculation of the renormalization group β-functions, is provided in the *Supplementary Material* [74]. Here we present only the final flow equations and their physical interpretation.

Equations (3) are identical to those obtained in [48, 75] for the reactions $B \xrightarrow{\mu} \emptyset$, and $A + B \xrightarrow{\lambda} 2B + A$, with bare mass $m_{AB} \equiv \mu - \lambda\langle N_A\rangle$, with $\langle N_A\rangle$ representing the average number of A. This model is known as the AB model [76]. It has been originally proposed in [48, 77, 76, 78, 75] to discuss the origin of life in terms of auto-catalysis, and it has been applied in some research areas such as ecology and economy [79, 80, 81]. The spatial version of this model shows that self-replication can be locally maintained with B growing exponentially, even when average A concentration would not be sufficient to sustain growth in a homogeneous vessel. This fact is a consequence of the tendency of μ to grow with the scale s, as shown by the equations (3), and from the definition of m_{AB}. Exactly the opposite will occur in the case of the model with asexual population, since in this case $m = \lambda - \mu\langle N_B\rangle$ and therefore μ is subtracted rather than added.

Figures (2a) and (2b) show the RG flow diagrams associated with equations (3) for $\mu \geq 0$. In the top panel (Fig. 2a), we show the case $\epsilon < 0$ ($d > 2$). The black dot marks the nontrivial fixed point $(\mu^*, m^*) = (2\pi\bar{D}|\epsilon|, \bar{D}\pi\epsilon^2\langle N_B\rangle)$. The horizontal dotted line represents $m = 0$. The green and red curves separate the vector field into distinct dynamical regimes. **Above the separating curve and for** $m > 0$, the RG flow tends to take m to *infinity*. In this case the asexual species population explodes. This happens for a sufficiently small μ. The opposite occurs below the separating curve (*i.e.*, for sufficiently large μ), with the RG flow inducing m to negative values, inducing the population to extinction, even with the mean-field theory indicating explosion. We term this phenomenon *Discreteness-Inducing Extinction* (DIE).

More striking is the bottom panel (Fig. 2b), where the DIE phenomenon is certain across the entire parameter space (for $\mu > 0$) for $d \leq 2$ (or $\epsilon \geq 0$). *In two-dimensional habitats (on surfaces), asexual species always die.*

Sexual model

Having established that asexual populations face certain extinction in low dimensions due to DIE, we now examine whether sexual reproduction can overcome this limitation.

Let's compare the effects caused by the discrete character of the interactions in asexual and sexual reproduction. For the model of sexual reproduction, there is one more parameter (λ) flowing due to the iterations of the renormalization group. This is due to the non-linearity of the process $2A \xrightarrow{\lambda} 3A$. The flow equations are:

$$\frac{d\mu}{dl} = \epsilon\mu + \frac{\mu^2}{2\pi\bar{D}} \tag{4a}$$

$$\frac{d\kappa}{dl} = 2\kappa - \frac{\langle N_B\rangle\mu^2}{2\pi\bar{D}} \tag{4b}$$

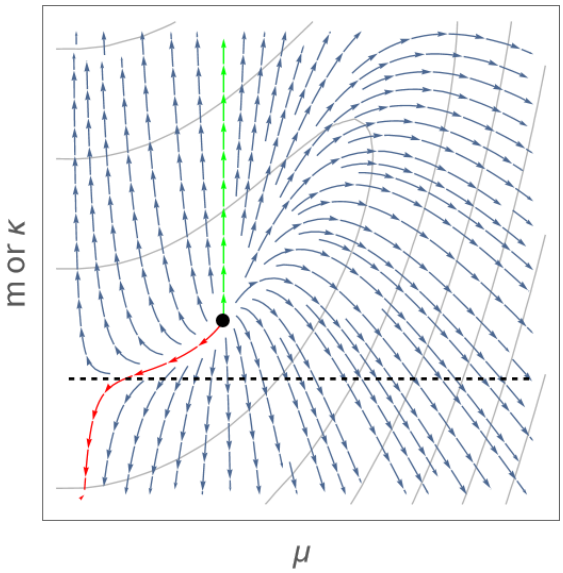
$$\frac{d\lambda}{dl} = \epsilon\lambda + \frac{\lambda^2}{\pi\bar{D}} \tag{4c}$$

where $\epsilon = 2 - d$, $l = \ln(s)$, and $\bar{D} \equiv (D_A + D_B)/2$.

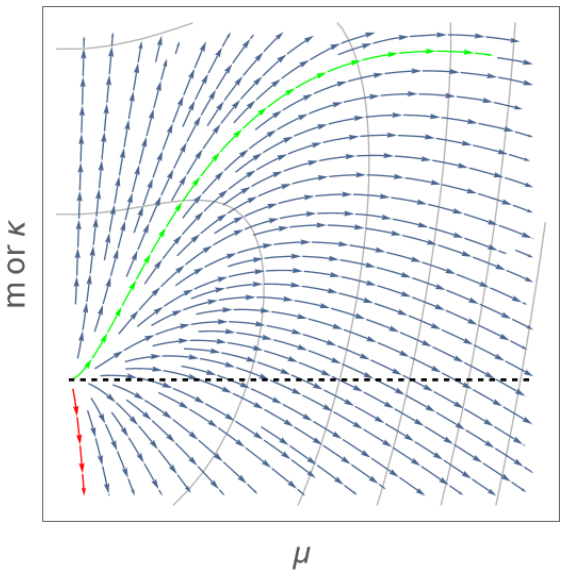
In contrast, the sexual model exhibits a competing nonlinearity: while μ grows, so does λ, due to the $2A \to 3A$ reaction. This effectively lowers the potential barrier $V_{max} = \kappa^3/6\lambda^2$, facilitating population growth. The aggregation of sexual individuals into clusters further amplifies local reproduction rates, a form of emergent self-facilitation [82].

The new λ RG flow does not influence κ and therefore RG flows involving κ and μ are as shown in the vertical panels of Fig. (2) by replacing m with κ on the vertical axes. The same separating curves (green/red) apply, now dividing the regimes for κ.

We can now examine how the potential barrier (given by $V_{\max} = \kappa^3/6\lambda^2$) in figure (1) varies when the



(a) **RG flow for $\epsilon < 0$.**



(b) **RG flow for $\epsilon \geq 0$.**

Figure 2: **RG flow for the asexual (m) / sexual (κ) models. (a) For $d > 2$ ($\epsilon < 0$), a finite fixed point (black dot) exists; populations may survive or go extinct depending on initial parameters. The green and red curves separate the regimes of explosion (above, for $m > 0$) and extinction (below). (b) For $d \leq 2$ ($\epsilon \geq 0$), μ flows to infinity, driving m negative and guaranteeing extinction (DIE). Vector arrows indicate the direction of RG flow.**

parameters are renormalized. We see that on the surface (or in smaller dimensions: $\epsilon \geq 0$), this potential barrier disappears quickly, *favoring* the sexual species. This happens because of how quickly λ approaches infinity (with decreasing κ), making $V_{\max} \to 0$. The barrier that prevented the sexual population's development is increasingly transposable if there is enough space and time for the interactions to occur. And this fact arises from the interaction's discreteness. This is *the importance of being discrete in sex*. Furthermore, according to our simplified models, the only chance for an asexual species to exist in nature is in a three-dimensional space. This finding leads to the conjecture that most extant asexual species live in the oceans, air, or other effectively three-dimensional media.

2.3 Numerical Validation

To test the predictions of the renormalization group analysis, we performed stochastic lattice simulations following the microscopic rules defined in Section 1.1.

Simulation details. Simulations were performed using a discrete-time stochastic simulation algorithm with a fixed time step $\Delta t = 0.01$. This approach approximates the continuous-time dynamics via a tau-leaping-type update [83] and is chosen such that reaction probabilities per site per step are small ($\ll 1$), ensuring an accurate representation of the underlying continuous-time Markov process.

Notation. To avoid confusion with matrix or adjacency notation, we use $A_{i,j}(t)$ in two dimensions (and $A_{i,j,k}(t)$ in three dimensions) to denote the integer number of host individuals at the lattice site with discrete coordinates i, j (and k) at time t. The same convention applies to the pathogen count B.

Let $A_{i,j}(t)$ and $B_{i,j}(t)$ denote the *integer* number of host and pathogen individuals, respectively, at lattice site (i, j) at time t. The initial host density $\rho_0 = 0.5$ was implemented by drawing $A_{i,j}(0)$ from a Poisson distribution with mean ρ_0, allowing multiple individuals per site. Pathogens were initialized with $B_{i,j}(0)$ drawn from a Poisson distribution with mean $\langle N_B \rangle = 0.95$, and they diffused via nearest-neighbor hopping (von Neumann neighborhood) with rate $D_B = 1.0$. Reproduction and death rates were set to $\lambda = \mu = 0.15$.

At each time step, events were processed in the following order:

1. **Reproduction:** Asexual: $A \to 2A$ with probability $\lambda A_{i,j} \Delta t$; Sexual: $2A \to 3A$ with probability $\lambda \mathbf{1}_{[A_{i,j} \geq 2]} \Delta t$.

2. **Death:** At each site (i, j) where $A_{i,j} \geq 1$ and $B_{i,j} \geq 1$, one host individual dies with probability $\mu\, A_{i,j} B_{i,j}\, \Delta t$, implemented as $A_{i,j} \to A_{i,j} - 1$. This rule follows directly from the mass-action law $A + B \xrightarrow{\mu} B$.

3. **Diffusion:** Each pathogen moves to a randomly chosen nearest-neighbor site with periodic boundary conditions.

Relation to field theory. The microscopic rule for sexual reproduction uses a threshold condition ($A_{i,j} \geq 2$) rather than a literal binary collision. Near the extinction threshold, processes of this type are expected to belong to the same universality class as the genuine pair reaction $2A \longrightarrow 3A$ used in the field-theoretic derivation. The reason is that the macroscopic dynamics is controlled by the density dependence of the rates and by the absorbing-state symmetry, not by the microscopic details of the reaction kernel [84, 85]. In particular, the conditional rule introduces an effective quadratic density dependence at the coarse-grained level, leaving the critical exponents unchanged. The renormalization group analysis therefore captures the universal, coarse-grained physics that the simulations are designed to probe.

Each simulation ran for $t_{\max} = 500$ time units or until host extinction, whichever occurred first. Results were averaged over $N_{\text{real}} = 15$ independent realizations.

This parameter regime places the mean-field growth rate of the asexual population at $m = \lambda - \mu\langle N_B\rangle = 0.15 - 0.15\times 0.95 = 0.0075 > 0$, yet the asexual model is predicted to go extinct in $d = 2$ due to discreteness-induced extinction (DIE). For the sexual model, the mean-field Allee threshold is $\rho_c = \mu\langle N_B\rangle/\lambda = 0.95$, well above the initial density $\rho_0 = 0.5$. Thus, any observed persistence of the sexual population cannot be attributed to mean-field growth but must arise from spatial stochasticity and clustering — the hallmark of discreteness-induced coexistence (DIC).

Figure 3 displays the mean population trajectories. The asexual population (red curve) declines toward extinction in all realizations, consistent with the renormalization group prediction that the effective mass parameter becomes negative under coarse-graining—the signature of discreteness-induced extinction (DIE) in dimensions $d \leq 2$. We emphasize that "extinction" in the field-theoretic sense refers to the asymptotic flow of the mass parameter, which drives the system toward the absorbing state on sufficiently large spatiotemporal scales; in finite simulations, a small survival probability may persist for long times, although our trajectories show a clear monotonic decay. In striking contrast, the sexual population (blue curve) exhibits sustained growth despite starting below the Allee threshold, demonstrating the phenomenon of discreteness-induced coexistence (DIC). This biphasic behavior — an initial decline due to the difficulty of finding mates at low densities, followed by recovery and subsequent accelerated growth — is the hallmark of clustering dynamics. As stochastic fluctuations give rise to local aggregates, individuals within these clusters become effectively shielded from diffusing pathogens, allowing the quadratic reproduction term ($2A \longrightarrow 3A$) to dominate over pathogen-induced mortality.

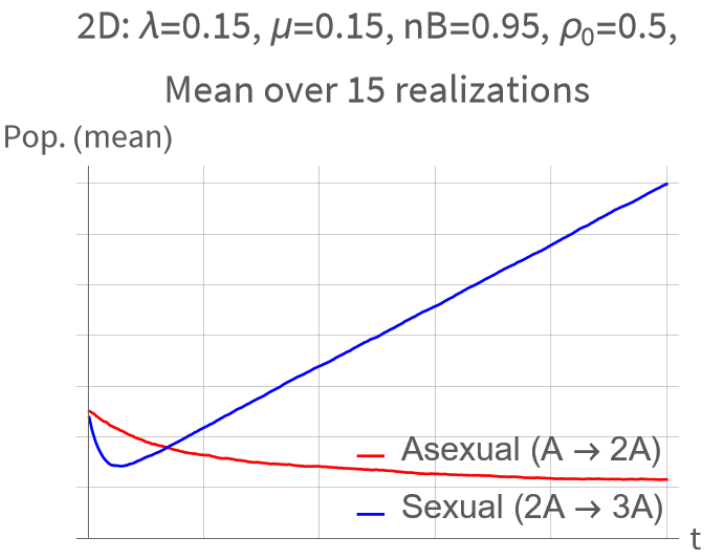


Figure 3: **Mean population trajectories for asexual and sexual reproduction in two dimensions.** Stochastic lattice simulations on a 50×50 lattice with $\lambda = \mu = 0.15$, $\langle N_B\rangle = 0.95$, and initial density $\rho_0 = 0.5$ (implemented via a Poisson distribution). Mean-field theory predicts slow positive growth for the asexual population ($m = \lambda - \mu\langle N_B\rangle = 0.0075 > 0$) but extinction for the sexual population, since the initial density lies below the Allee threshold ($\rho_0 = 0.5 < \rho_c = \mu\langle N_B\rangle/\lambda = 0.95$). However, the discrete and stochastic nature of interactions leads to drastically different outcomes. The asexual model ($A \longrightarrow 2A$, red curve) declines toward extinction, consistent with the asymptotic prediction of *discreteness-induced extinction* (DIE) for $d \leq 2$. Conversely, the sexual model ($2A \longrightarrow 3A$, blue curve) exhibits persistent clustering and sustained population growth, demonstrating *discreteness-induced coexistence* (DIC) and the selective advantage conferred by sexual reproduction. Curves represent averages over 15 independent realizations. These numerical results corroborate the renormalization group analysis, confirming that spatial discreteness and demographic noise favor sexual reproduction in low-dimensional habitats.

One-dimensional simulations. We have also performed simulations on one-dimensional lattices (not shown). The results are qualitatively identical to the two-dimensional case: asexual populations decline toward extinction, consistent with the discreteness-induced extinction (DIE) mechanism, while sexual populations persist and form clusters via discreteness-induced coexistence (DIC). In fact, the decline toward extinction is even more rapid in 1D than in 2D, con-

sistent with the recurrence property of random walks being stronger in lower dimensions. These findings confirm that the advantage of sexual reproduction is not limited to two-dimensional habitats but extends to all low-dimensional environments ($d \leq 2$).

Three-dimensional simulations

To verify the dimensional crossover predicted by the renormalization group analysis, we performed simulations on a three-dimensional lattice ($L = 10 \times 10 \times 10$, periodic boundary conditions) using the same reaction rates ($\lambda = 0.1$, $\mu = 0.2$) and initial host density ($\rho_0 = 1.5$) for both reproductive strategies. The time step was $\Delta t = 0.05$, and the total simulation time was $t_{\max} = 500$. Diffusion was implemented as a particle-wise hopping process with probability $p_A = p_B = 10^{-4}$ per individual per time step, ensuring that movements are rare and independent. Results were averaged over $N_{\text{real}} = 10$ independent realizations.

For the asexual model, the pathogen density was set to $\langle N_B \rangle = 0.95$, yielding a mean-field growth rate $m = \lambda - \mu\langle N_B \rangle = 0.1 - 0.2 \times 0.95 = -0.09 < 0$. According to the mean-field prediction, this would imply extinction; however, the discrete and stochastic nature of the interactions can still sustain growth in higher dimensions where the Gaussian fixed point is stable.

For the sexual model, the pathogen density was reduced to $\langle N_B \rangle = 0.5$, giving an Allee threshold $\rho_c = \mu\langle N_B \rangle / \lambda = 1.0$. Since the initial density $\rho_0 = 1.5$ exceeds this threshold, the population is expected to grow provided that the mean-field conditions are favorable. In $d = 3$, fluctuations are too weak to nucleate protective clusters from densities below the Allee threshold (the discreteness-induced coexistence mechanism is inoperative), so survival depends solely on the deterministic condition $\rho_0 > \rho_c$.

Figure 4 displays the mean population trajectories. In clear contrast to the two-dimensional case, the asexual population no longer goes extinct but grows steadily, confirming that discreteness-induced extinction (DIE) is absent for $d > 2$. The sexual population also grows, demonstrating that sexual reproduction can be viable in three dimensions when the initial density lies above the Allee threshold. We emphasize that these three-dimensional simulations serve primarily as illustrative support for the theoretical prediction that DIE disappears above $d = 2$, rather than as an exhaustive parameter scan. A systematic exploration of lattice sizes and rate parameters is left for future work. This confirms the dimensional selectivity of the DIE and DIC mechanisms: only in low dimensions ($d \leq 2$) can spatial fluctuations alone rescue sexual populations from below the Allee threshold and, simultaneously, drive asexual populations to extinction.

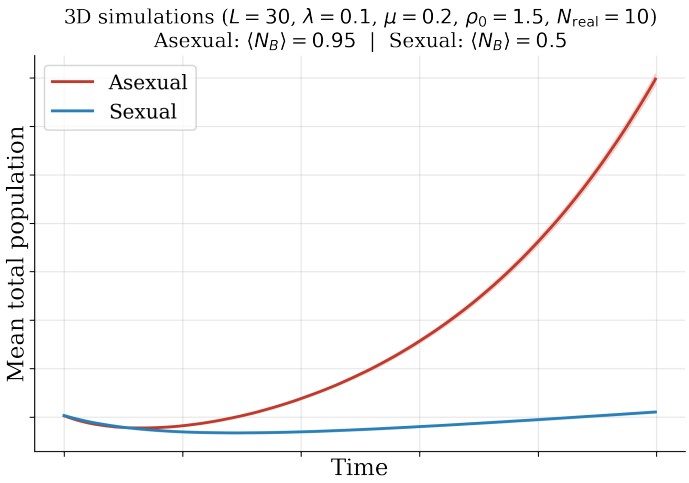


Figure 4: **Mean population trajectories for asexual and sexual reproduction in three dimensions.** Stochastic lattice simulations on a $10 \times 10 \times 10$ lattice with $\lambda = 0.1$, $\mu = 0.2$, initial host density $\rho_0 = 1.5$, and $N_{\text{real}} = 10$. For the asexual model ($\langle N_B \rangle = 0.95$, red curve), the population grows steadily, in stark contrast to the extinction observed in $d = 2$. For the sexual model ($\langle N_B \rangle = 0.5$, blue curve), the initial density exceeds the Allee threshold ($\rho_c = 1.0$), and the population also grows. Error bands denote ± 1 standard deviation. These results confirm that discreteness-induced extinction (DIE) is absent for $d > 2$, and that the advantage of sex in low dimensions is genuinely due to strong spatial fluctuations.

3 Discussion

Our RG analysis reveals a fundamental asymmetry between reproductive strategies: while discreteness guarantees extinction for asexual populations in low dimensions (DIE), it simultaneously enables persistence and clustering for sexual ones. This dimensional dependence has broad implications for evolutionary theory and ecology, which we explore below.

3.1 Relation to Existing Theories

Our results complement genetic explanations for the maintenance of sex. While the Red Queen hypothesis emphasizes allele-frequency dynamics [14], our model highlights how spatial structure and discreteness can modulate the host-pathogen arms race itself. Specifically, in low-dimensional habitats (characteristic of many stable, complex environments like forest floors

or benthic zones), the recurrence of random walks ensures persistent pathogen encounters. This persistent pressure makes the discreteness-induced extinction (DIE) mechanism particularly potent against asexual populations, while sexual populations can exploit the same spatial constraints to form protective clusters. This provides a mechanistic explanation for the empirical observation that sex is more common in stable, complex environments where pathogen pressure is high [86]. Moreover, our findings resonate with ideas that sex may be favored in spatially heterogeneous environments [21], but we propose a causal inversion: sex can *generate* such heterogeneity through clustering, rather than merely responding to pre-existing environmental patchiness.

We emphasize that our mechanism is not alternative but complementary to genetic theories like the Red Queen. While the Red Queen operates at the genetic level (e.g., through rare-allele advantage), our model operates at the demographic level, showing how the very same host-parasite interaction, when viewed through the lens of spatial stochasticity, naturally favors sexual reproduction. The two levels are likely synergistic in nature.

3.2 Biological Implications and the Dimensionality of Habitat

If spatial discreteness favors sex in low-dimensional habitats, this could explain the prevalence of sex in many marine and terrestrial environments that are effectively 2D. Our conjecture that asexual species may persist primarily in three-dimensional environments like oceans finds resonance in stochastic models developed for phytoplankton dynamics in marine ecosystems [87], where fluid mixing and spatial structure play key roles in population persistence. It also suggests that asexual lineages may thrive only in high-dimensional or well-mixed settings, a prediction that could be tested using microcosm experiments with controlled spatial structure [21]. The clustering of sexual agents, an emergent phenomenon in our model, helps to explain the group-level advantage of sex [59]. This prediction aligns with experimental evolution studies using microbial systems, where sexual reproduction is favored in spatially structured environments, but not in well-mixed liquid cultures [21]. The crucial role of spatial dimension in our model echoes fundamental results in low-dimensional transport phenomena, where diffusion exhibits anomalous scaling and recurrence properties markedly different from higher dimensions [88].

This dimensional hypothesis is consistent with the broader literature on geographical parthenogenesis [89], which documents that asexual taxa tend to occur in three-dimensionally complex or well-mixed environments, while sexuals dominate stable, effectively two-dimensional habitats. Moreover, experimental evolution studies have directly shown that spatial structure favors higher rates of sex [26], providing empirical validation of the principle that dimensionality matters.

3.3 Limitations and Future Directions

Our model intentionally omits genetic detail to isolate the role of spatial stochasticity, and it assumes a constant, non-reproducing pathogen population. This simplification corresponds to ecological scenarios where pathogens are generalist, long-lived, or externally maintained in environmental reservoirs such as soil or water [90, 91]. In such systems, pathogen pressure remains effectively constant, decoupled from host density, allowing us to isolate the spatial stochasticity mechanism. Relaxing this assumption to allow pathogen dynamics to depend on host density could modulate the quantitative outcomes but is unlikely to eliminate the low-dimensional advantage of sex, because the recurrence of random walks remains the fundamental physical constraint [92]. The recurrence of random walks in low dimensions ensures persistent pathogen-host encounters, a consequence of the spatial structure that can be analyzed through network centrality measures [93]. This persistent encountering underlies the DIE phenomenon for asexual populations.

Our results lead to a testable ecological prediction: sexual reproduction should be favored in low dimensional habitats (effectively one- or two dimensional environments), while asexual reproduction may persist in three dimensional settings where spatial refuges are more accessible. This hypothesis finds support in the ecological literature. Many asexual taxa are disproportionately represented in marine environments and the open ocean — effectively three-dimensional habitats — including bdelloid rotifers [94, 95], ostracods [96], and various benthic invertebrates [97]. Conversely, sexual reproduction predominates in structurally complex, effectively two-dimensional environments such as tropical forest floors [9], leaf litter [42], and coral reef surfaces [98], where pathogen pressure is high and spatial structure limits escape. The recognition that sex is more common in stable, biologically complex habitats [41, 43] aligns with our dimensional hypothesis, as these environments are often effectively two-dimensional.

Future work should integrate genetic variability and coevolutionary dynamics within a spatial frame-

work [27, 99] to assess potential synergies between the physical mechanism proposed here and traditional genetic explanations such as the Red Queen hypothesis [15]. Additionally, while we focus on low-density regimes where renormalization group techniques are applicable, the transition to high-density populations — where carrying capacity, competition, and saturation effects become relevant — warrants further investigation. Such studies could bridge the critical regime analyzed here with the well-understood dynamics of saturated populations [68].

Extensions to more complex spatial structures, including networks with fractal dimensionality [100] or empirical landscape topologies [101], could reveal how habitat geometry influences the evolution of reproductive strategies. Experimental microcosm studies using spatially structured environments [22, 21] could directly test the dimensional prediction by comparing asexual and sexual populations in effectively one-, two-, and three-dimensional arenas. Such experiments would provide a powerful empirical complement to the theoretical framework developed here.

Finally, we acknowledge that our model represents a minimal abstraction designed to isolate a specific physical mechanism. The deliberate omission of genetic variation, mate choice, and other biological complexities means that our results should be interpreted as demonstrating that spatial stochasticity alone can confer an advantage to sexual reproduction, rather than providing a complete explanation for its prevalence in nature. The evolutionary maintenance of sex likely results from multiple interacting mechanisms operating across genetic, demographic, and spatial scales [19, 102], and our findings suggest that the physical constraints imposed by habitat dimensionality deserve a place alongside these more established explanations.

3.4 General Discussion

Addressing a potential objection

A possible objection to the sexual model is that various particles can accumulate in one lattice site, leading to a divergence in the population due to the reaction $2A \xrightarrow{\lambda} 3A$. In principle, the amount of particles per site can be infinite, but for our purposes, it does not matter. One possible argument goes as follows:

If we establish the same initial conditions for the two models, with both species on the verge of extinction, the sexual species will win unequivocally, after a transient. And this occurs twice: once because of the certainty of extinction of the asexual species (at least for $d = 2$) and once because of the trend of sexual species to diverge. It does not matter if ultimately the amount of individuals per site becomes greater than one. From this point on, our minimal model is no longer valid and constraints must be added, but the battle has already been won by the sexual species.

Physical intuition for dimension-dependent behavior

Beyond this specific objection, a more general question arises: Is it possible to understand physically why the description based on differential equations (or mean-field theory) becomes invalid for long time-frames and large scales in lower dimensions? To answer this question, let's first consider a different situation, for the sake of simplicity. Consider a one-dimensional lattice, where nearly all lattice sites are populated by particles of species A, such that the only reaction occurring is the *annihilation* reaction $A + A \xrightarrow{\alpha} \emptyset$. In principle, this reaction can take place everywhere. The initial dynamics is therefore accurately described by the solution of the kinetic rate equation $\frac{\partial \rho}{\partial t} = D_A \nabla^2 \rho - \alpha \rho^2$, where $\rho(x,t)$ is the concentration of A individuals in the lattice and D_A is the diffusion constant. Therefore, initially, $\rho \propto t^{-1}$. At later times, the lattice becomes more and more diluted and the reaction rate is limited by the first passage time of a random walk in $d = 1$, scaling exactly with $t^{-1/2}$. Hence, the long time behavior of ρ is renormalized to $\rho \propto t^{-1}/t^{-1/2} = t^{-1/2}$. This is a simple example of how the reactions limited by diffusion [106] might provide different results from the equivalent models described in terms of differential equations.

The probability of species B (the parasites) finding species A is 1 for $d \leq 2$ for asexual species at the limit of the reaction limited by diffusion because of the recurrence property of random walks in low dimensions. Physically, this means that the diffusing particle will thoroughly sweep its neighborhood. It's therefore highly probable that it will react with another particle in its vicinity. Hence, it is reasonable to expect that in the long run, the system will be in a state where there are almost *no* individuals of species A. This phenomenon no longer occurs in $d > 2$ and asexual species may exist.

In the case of sexual reproduction, two nonlinear phenomena are in competition: $2A \xrightarrow{\lambda} 3A$ and $A + B \xrightarrow{\mu} B$. As we have seen, however, the former prevails over the latter, and the tendency to form clusters of sexual individuals predominates *due to the "shielding" effects that the boundaries of the clusters have on the individuals within it.* It is therefore reasonable to expect that after sufficient time the system will reach a

state where there are many isolated particles.

Emergent heterogeneity and the origin of sex

This leads to the interesting question whether "ancestral" sexed reproductions are responsible for habitat heterogeneity (through the formation of clusters of individuals) rather than the heterogeneity of the environment inducing mating [26, 103, 104, 105]. We are referring to the chicken or the egg causality dilemma. Taking this idea to the extreme, we might ask: is there sex *because* of demographic noise?

3.5 Unifying Discreteness-Induced Coexistence and Extinction

Our results link two phenomena arising from spatial stochasticity: Discreteness-Induced Coexistence (DIC) and Discreteness-Induced Extinction (DIE). Although previously studied separately [59, 58], they emerge here from the same underlying physical principle, the recurrence of random walks in low dimensions, applied to different reproductive strategies.

Discreteness-Induced Extinction manifests for asexual populations. As established in [58], for $d \leq 2$, the recurrence property of random walks ensures that pathogens eventually locate all hosts. The RG flow equations (3) show that the effective predation parameter μ grows indefinitely with scale, driving the mass parameter $m = \lambda - \mu\langle N_B \rangle$ to negative values. This guarantees extinction even when mean-field theory predicts population explosion, precisely the DIE phenomenon. Asexual populations, with their linear reproduction term, cannot overcome this discreteness-induced death spiral.

Conversely, sexual populations exhibit Discreteness-Induced Coexistence. The nonlinear reproduction term $2A \xrightarrow{\lambda} 3A$ introduces a crucial asymmetry. While the death parameter μ still grows under RG flow, so does the reproduction parameter λ (Eq. 4c). This simultaneous renormalization effectively lowers the potential barrier $V_{max} = \kappa^3/(6\lambda^2)$ that sexual populations must overcome. More profoundly, sexual individuals form clusters that shield interior members from pathogen encounters, a spatial organization that leverages the DIC principle where local aggregation enables global persistence [59]. The sexual subpopulations within these clusters effectively "coexist" with their pathogens by reducing encounter probabilities at the cluster boundaries.

The unification is mathematically explicit in the RG flows. Comparing Eqs. (3) and (4) reveals that both models share identical flow equations for μ and m/κ, differing only in the additional equation for λ in the sexual case. This single additional term, originating from the nonlinear reproduction, suffices to transform certain extinction into potential persistence. The dimensional dependence $d \leq 2$ that guarantees DIE for asexuals simultaneously enables DIC for sexuals through the same physical mechanism: the recurrence of random walks in low dimensions.

This unified perspective suggests that what appears as contradictory phenomena, coexistence versus extinction, actually represents different manifestations of the same underlying stochastic physics, with the outcome determined by the nonlinear structure of interactions. The maintenance of sex, therefore, emerges not merely as a genetic advantage but as a spatiotemporal stabilization strategy that harnesses discreteness effects which would otherwise be fatal.

4 Conclusion

The unification of discreteness-induced extinction and coexistence under a single physical mechanism, namely the recurrence of random walks in low dimensions, provides a parsimonious explanation for the evolutionary stability of sex. We have shown that the discrete and spatial character of host-pathogen interactions can, at the level of coarse-grained field theory, account for the evolutionary stability of sexual reproduction in low-dimensional environments. By combining concepts from stochastic processes and renormalization group theory, we identify a physical mechanism, rooted in demographic noise and diffusion-limited encounters, that complements genetic hypotheses. This underscores the value of cross-disciplinary approaches in resolving long-standing biological puzzles.

Our results synthesize insights from both discreteness-induced coexistence and extinction phenomena: sexual populations form clusters that benefit from DIC-like dynamics, while asexual populations suffer DIE due to constant pathogen pressure. This dual perspective reveals how spatial stochasticity creates conditions that naturally maintain sexual reproduction despite its costs.

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