SSWD-EvoEpi: A Coupled Eco-Evolutionary Epidemiological Model for Sea Star Wasting Disease in *Pycnopodia helianthoides*

Technical Report — Model Development and Sensitivity Analysis

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Abstract

Sea star wasting disease (SSWD) caused one of the largest wildlife mass mortality events in marine ecosystems, driving the sunflower sea star (*Pycnopodia helianthoides*) to a 90.6% range-wide decline and IUCN Critically Endangered status. The recent identification of *Vibrio pectenicida* strain FHCF-3 as a causative agent, combined with active captive breeding and the first experimental outplanting of captive-bred juveniles, creates an urgent need for quantitative tools to guide recovery. We present SSWD-EvoEpi, an individual-based, spatially explicit eco-evolutionary epidemiological model coupling *V. pectenicida* transmission dynamics with polygenic host evolution under sweepstakes reproductive success. Each agent carries a diploid genotype across 51 loci governing three fitness-related traits — resistance (immune exclusion), tolerance (damage limitation), and recovery (pathogen clearance) — that evolve in response to disease-driven selection. Disease dynamics follow an SEIR compartmental structure with an environmental pathogen reservoir, pathogen evolution along a virulence–transmission tradeoff, and temperature-dependent forcing. Reproduction implements sweepstakes reproductive success with $N_e/N \sim 10^{-3}$, sex-asymmetric spawning induction, and post-spawning immunosuppression. Four rounds of global sensitivity analysis (Morris screening and Sobol variance decomposition) across up to 47 parameters reveal that model behavior is dominated by nonlinear interactions among disease mortality rate, host susceptibility, environmental pathogen pressure, and genetic architecture, with recovery

trait evolution emerging as the fastest adaptive response. The model provides a framework for evaluating captive-bred release strategies, assisted gene flow, and the feasibility of evolutionary rescue on conservation-relevant timescales.

Contents

1 Introduction

1.1 Sea Star Wasting Disease and the Collapse of *Pycnopodia helianthoides*

Sea star wasting disease (SSWD) caused one of the largest documented wildlife mass mortality events in marine ecosystems when it swept through populations of over 20 asteroid species along the northeastern Pacific coast beginning in 2013 [22, 27, 45]. The disease, characterized by behavioral changes (arm twisting, lethargy), loss of turgor, body wall lesions, ray autotomy, and rapid tissue degradation, devastated populations from Baja California to the Gulf of Alaska within months [30, 44]. Among the species affected, the sunflower sea star (*Pycnopodia helianthoides*) suffered the most catastrophic decline, losing an estimated 5.75 billion individuals and experiencing a 90.6% range-wide population reduction based on 61,043 surveys across 31 datasets [18, 23]. Along the outer coast from Washington to Baja California, declines exceeded 97%, with many regions recording zero individuals in subsequent surveys [18, 21]. The species was assessed as Critically Endangered by the IUCN in 2021 [18] and is under consideration for listing as Threatened under the U.S. Endangered Species Act [37].

As a large-bodied, mobile, generalist predator capable of consuming sea urchins at rates sufficient to structure entire subtidal communities, *Pycnopodia helianthoides* functions as a keystone species in northeastern Pacific kelp forest ecosystems [6, 14, 42]. Its precipitous decline has been linked to cascading trophic effects, including sea urchin population explosions and extensive kelp forest deforestation, with northern California losing 96% of its kelp canopy since the 2014 marine heatwave [43, 48]. The loss of this apex predator thus represents not only a conservation crisis for a single species but a destabilization of an entire marine ecosystem [21, 34].

1.2 Etiology: A Decade-Long Mystery Resolved

For over a decade following the initial outbreak, the causative agent of SSWD remained contested. An early hypothesis implicating sea star associated densovirus (SSaDV; Hewson et al. 27) was subsequently retracted after repeated failures to reproduce the original challenge experiments and the discovery that the virus is endemic in healthy echinoderm populations worldwide [28–30]. An alternative hypothesis invoking boundary layer oxygen depletion (BLODL) at the animal–water interface proposed that microbial respiration on sea star surfaces draws down dissolved oxygen, leading to tissue hypoxia [2, 25]. While this mechanism may contribute to disease susceptibility, it did not identify a specific pathogen.

The breakthrough came with Prentice et al. [46], who fulfilled Koch's postulates by demonstrating that *Vibrio pectenicida* strain FHCF-3, a Gram-negative marine bac-

terium, is a causative agent of SSWD in *Pycnopodia helianthoides*. Through seven controlled exposure experiments using captive-bred, quarantined sea stars, the authors showed that injection of cultured *V. pectenicida* FHCF-3 into the coelomic cavity reliably produced disease signs — arm twisting, lesion formation, autotomy, and death within approximately two weeks. Heat-treated and 0.22 µm filtered controls remained healthy, confirming a living bacterial agent. Critically, the pathogen was re-isolated from experimentally infected animals, completing Koch's postulates. Earlier investigations had missed *V. pectenicida* because they sampled body wall tissue rather than coelomic fluid, where the bacterium resides.

However, the etiological picture is not entirely resolved. Hewson [26] demonstrated that *V. pectenicida* FHCF-3 was not consistently detected in non-*Pycnopodia helianthoides* species during the 2013–2014 mass mortality, suggesting it may be specific to *Pycnopodia helianthoides* or may function as an opportunistic pathogen rather than a universal SSWD agent across all affected asteroid taxa. The bacterium also exhibits explosive growth in the presence of decaying echinoderm tissue, raising questions about whether it acts primarily as a pathogen or a saprobe under different conditions [26]. Nonetheless, for *Pycnopodia helianthoides* — the focus of this study — the evidence for *V. pectenicida* as the primary causative agent is robust. The identification of a specific bacterial pathogen with known temperature-dependent growth dynamics [40] provides a mechanistic basis for modeling disease transmission and environmental forcing.

1.3 Conservation Urgency and Active Recovery Efforts

The failure of *Pycnopodia helianthoides* populations to recover naturally in the decade following the initial epizootic — contrasting with partial recovery observed in some co-occurring asteroid species [17] — has motivated intensive conservation action. The species' long generation time (∼30 years), broadcast spawning reproductive strategy, and vulnerability to Allee effects at low density [15, 38] compound the challenge of natural recovery. Historical precedent is sobering: the Caribbean long-spined sea urchin *Diadema antillarum*, which suffered a comparable 93–100% mass mortality in 1983–1984, achieved only ∼12% recovery after three decades [35]. Another asteroid, *Heliaster kubiniji*, has never recovered from a 1975 mass mortality event in the Gulf of California [11].

In response, a coordinated multi-partner recovery effort has emerged. The Association of Zoos and Aquariums (AZA) Saving Animals From Extinction (SAFE) program maintains over 2,500 captive juveniles and 130+ reproductive adults across 17 AZA institutions [5]. The first experimental outplanting of captive-bred *Pycnopodia helianthoides* occurred in December 2025 in Monterey, California, with 47 of 48 juveniles surviving after four weeks [53]. A Roadmap to Recovery developed by over 30 leading experts defines regionally nested recovery objectives, from local demographic benchmarks to range-wide

genetic structure targets [23]. Cryopreservation of gametes has been demonstrated for a congener and is under development for *Pycnopodia helianthoides* to enable assisted gene flow from genetically diverse founders [20, 52]. In 2025, the California Ocean Protection Council approved $630,000 in funding for captive breeding, disease diagnostics, and experimental outplanting [7]. A reference genome has also been published [51], laying the groundwork for genome-wide association studies (GWAS) to identify resistance loci.

These recovery efforts require quantitative predictions: How many captive-bred individuals should be released, where, and when? What are the genetic consequences of releasing animals from a limited captive founder population? Can natural selection drive resistance evolution fast enough to matter on conservation timescales? How do pathogen evolution, environmental change, and spatial structure interact to shape recovery trajectories? Answering these questions demands a modeling framework that integrates disease dynamics with population genetics in an explicitly spatial context.

1.4 The Need for an Eco-Evolutionary Framework

Existing models of SSWD dynamics have focused on either epidemiological or ecological aspects in isolation. Aalto et al. [1] coupled an SIR-type model with ocean circulation to explain the rapid continental-scale spread of SSWD, finding that temperature-dependent mortality best matched observed patterns. Tolimieri [54] conducted a population viability analysis using stage-structured matrix models but did not incorporate disease dynamics or host genetics. Arroyo-Esquivel et al. [4] recently modeled epidemiological consequences of managed reintroduction following disease-driven host decline, but their framework lacks genetic evolution. None of these approaches captures the interplay between disease-driven selection, host genetic adaptation, and demographic recovery that is central to predicting conservation outcomes.

The theoretical motivation for coupling these processes is compelling. Mass mortality events impose intense directional selection on host populations [49], and in *Pisaster ochraceus* — a co-occurring sea star affected by SSWD — rapid allele frequency shifts ($\Delta q \approx 0.08$–0.15 at outlier loci) were detected within a single generation of the epizootic, with geographic consistency across sites indicating selection rather than drift [49]. However, in broadcast-spawning marine invertebrates, the genetic consequences of mass mortality are filtered through sweepstakes reproductive success (SRS), whereby variance in individual reproductive success is so large that effective population size (N_e) is orders of magnitude smaller than census size ($N_e/N \sim 10^{-3}$; Árnason et al. 3, Hedgecock and Pudovkin 24). SRS amplifies genetic drift on ecological timescales [55], can facilitate rapid adaptation when coupled with bottlenecks [13], and generates chaotic genetic patchiness that confounds simple predictions of evolutionary trajectories. Any model of evolutionary rescue in *Pycnopodia helianthoides* must therefore account for this fundamental feature

of marine broadcast spawner genetics.

The closest methodological precedent is the eco-evolutionary individual-based model (IBM) developed by Clement et al. [9] for coevolution between Tasmanian devils (*Sarcophilus harrisii*) and devil facial tumour disease (DFTD). That model coupled an SEI epidemiological framework with polygenic quantitative genetics, parameterized from two decades of field data and GWAS results, and found a high probability of host persistence over 50 generations through coevolutionary dynamics. Our model extends this approach to a marine system with fundamentally different reproductive biology — broadcast spawning with sweepstakes reproductive success, external fertilization subject to Allee effects, and a pelagic larval phase mediating spatial connectivity — challenges that no existing eco-evolutionary disease model has addressed.

1.5 Model Overview

We present SSWD-EvoEpi, an individual-based, spatially explicit, eco-evolutionary epidemiological model designed to simulate SSWD dynamics and evolutionary responses in *Pycnopodia helianthoides* metapopulations across the northeastern Pacific. The model tracks individual sea stars as agents within a network of habitat nodes connected by larval dispersal and pathogen transport. Each agent carries a diploid genotype across 51 loci governing three fitness-related traits: resistance (r_i, 17 loci; immune exclusion reducing infection probability), tolerance (t_i, 17 loci; damage limitation extending survival during late-stage infection), and recovery (c_i, 17 loci; pathogen clearance enabling transition from infected to recovered states). Per-locus allele frequencies are drawn from a Beta(2,8) distribution, reflecting polygenic architecture with most loci at low frequency [32].

Disease dynamics follow an SEIR-type compartmental structure with exposed (E), early infected (I_1), and late infected (I_2) stages, coupled with an environmental pathogen reservoir (P) whose dynamics are temperature-dependent [16, 40]. Pathogen evolution is modeled through a heritable virulence phenotype that evolves along a mechanistic tradeoff curve linking shedding rate to host survival duration. Reproduction incorporates sweepstakes reproductive success via a heavy-tailed offspring distribution producing N_e/N ratios consistent with empirical estimates for marine broadcast spawners [24], with sex-asymmetric spawning induction and post-spawning immunosuppression derived from species-specific observations. Spatial connectivity is implemented through distinct larval exchange and pathogen dispersal matrices computed from overwater distances across the model domain.

The model is implemented in Python with NumPy-vectorized agent operations, achieving sufficient performance for large-scale sensitivity analysis and calibration (75,000 agents across 150 nodes in $\sim$72 s). Four rounds of sensitivity analysis using Morris screening and Sobol variance decomposition across up to 47 parameters have identified the key drivers of

model behavior, revealing strong nonlinear interactions and highlighting priority targets for empirical calibration.

1.6 Paper Outline

The remainder of this paper is organized as follows. Section 2 describes the overall model architecture, agent representation, and simulation flow. Sections 3–6 detail the disease, genetics, population dynamics, and spatial modules, respectively. Section 7 presents four rounds of global sensitivity analysis, identifying the parameters with greatest influence on epidemiological, demographic, and evolutionary outcomes. Section 8 describes model validation against available empirical data. Section 9 synthesizes findings, discusses limitations, and outlines the path toward calibrated conservation scenario evaluation. Parameter tables and supplementary analyses are provided in Appendix A.

2 Model Architecture

SSWD-EvoEpi is an individual-based model (IBM) that couples epidemiological, demographic, genetic, and spatial dynamics to simulate the eco-evolutionary consequences of sea star wasting disease in *Pycnopodia helianthoides*. Each agent represents a single sea star tracked through its complete life history, carrying a diploid genotype at 51 loci that determines three quantitative defense traits against *Vibrio pectenicida*. We chose an individual-based approach over compartmental (ODE/PDE) models because SSWD dynamics depend critically on individual-level heterogeneity in genetic resistance, body size, spatial position, and disease stage—features that compartmental models cannot represent without substantial loss of biological realism [10, 19].

2.1 Agent Representation

Each individual is represented as a record in a NumPy structured array (`AGENT_DTYPE`) comprising approximately 59 bytes per agent. Table 1 summarizes the principal state variables grouped by functional module.

Table 1: Agent state variables in SSWD-EvoEpi.

Module	Field	Description
Spatial	`x`, `y`	Position within node habitat (m)
	`heading`	Movement heading (rad)
	`speed`	Instantaneous speed ($\mathrm{m\,min^{-1}}$)
	`node_id`	Home node index
Life history	`size`	Arm-tip diameter (mm)
	`age`	Age (years, fractional)
	`stage`	Life stage (0–4; Table 2)
	`sex`	Sex (0 = female, 1 = male)
Disease	`disease_state`	Compartment ($\mathrm{S/E/I_1/I_2/D/R}$)
	`disease_timer`	Days remaining in current disease stage
Genetics	`resistance`	Resistance score $r_i \in [0, 1]$
	`tolerance`	Tolerance score $t_i \in [0, 1]$
	`recovery_ability`	Recovery/clearance score $c_i \in [0, 1]$
Spawning	`has_spawned`	Bout count this season
	`immunosuppression_timer`	Post-spawning immunosuppression (days)
Administrative	`alive`	Active flag
	`origin`	Wild / captive-bred / AGF / wild-source
	`pathogen_virulence`	Virulence of infecting strain v_i

Genotypes are stored in a separate array of shape $(N_{\max}, 51, 2)$ with `int8` entries, where axis 1 indexes loci and axis 2 indexes the two allele copies (diploid). This separation from the agent record improves cache performance during non-genetic operations (disease transmission, movement), which need not touch the genotype array.

Table 2: Life stages and size thresholds for *Pycnopodia helianthoides*.

Index	Stage	Size threshold (mm)	Reproductive
0	Egg/Larva	—	No
1	Settler	Settlement	No
2	Juvenile	≥ 10	No
3	Subadult	≥ 150	No
4	Adult	≥ 400	Yes

2.2 Node Structure

The spatial domain is represented as a metapopulation network of K discrete habitat nodes. Each node encapsulates:

- A population of agents (structured array + genotype array), initialized at local carrying capacity;
- Environmental state: sea surface temperature $T(t)$ (sinusoidal annual cycle with warming trend), salinity S, and tidal flushing rate ϕ_k;
- A local Vibrio concentration $P_k(t)$ (bacteria mL^{-1});
- Node metadata: latitude, habitat area, fjord classification.

Inter-node coupling occurs through two connectivity matrices:

1. **Pathogen dispersal matrix D**: governs daily exchange of waterborne *Vibrio pectenicida* between nodes, parameterized with an exponential distance kernel (scale $D_P = 15\,\mathrm{km}$);
2. **Larval connectivity matrix C**: governs annual dispersal of competent larvae among nodes, parameterized with a broader kernel (scale $D_L = 400\,\mathrm{km}$) reflecting the extended pelagic larval duration of *Pycnopodia helianthoides*.

2.3 Simulation Loop

The simulation advances in daily timesteps ($\Delta t = 1\,\mathrm{day}$) nested within an annual cycle. At each daily step, the following operations are executed in sequence at every node (Figure **??**):

1. **Environment update.** Compute $T_k(t)$ from a sinusoidal annual SST function with linear warming trend; update flushing rate ϕ_k (seasonally modulated for fjord nodes); salinity is constant per node.
2. **Movement.** Agents execute a correlated random walk (CRW) with 24 hourly substeps per day. Movement speed is modulated by disease state ($\times 0.5$ for I_1, $\times 0.1$ for I_2, $\times 0$ for D). Elastic boundary reflection constrains agents within the habitat.
3. **Disease dynamics.** Vibrio concentration is updated via an Euler step of the pathogen ODE. Susceptible agents are exposed to a force of infection that depends on local pathogen density, individual resistance, salinity, and body size. Infected agents progress through the SEIPD+R compartments with Erlang-distributed stage durations (Section 3).

4. **Pathogen dispersal.** Vibrio is exchanged between neighboring nodes via the **D** matrix, representing waterborne transport.

5. **Settlement.** Larval cohorts whose pelagic larval duration (PLD) has elapsed are settled into the local population via Beverton–Holt density-dependent recruitment, modulated by an adult-presence settlement cue (Allee effect).

6. **Spawning.** During the spawning season (November–July), reproductively mature adults spawn stochastically with daily probability modulated by a seasonal Gaussian envelope centered on the peak spawning day. Female and male multi-bout spawning, sex-asymmetric cascade induction, and post-spawning immunosuppression are modeled explicitly.

7. **Daily demographics.** Natural mortality is applied as a daily probability converted from stage-specific annual survival rates:

$$p_{\text{death,daily}} = 1 - S_{\text{annual}}^{1/365}, \tag{1}$$

with a senescence overlay for individuals exceeding the senescence age ($\tau_{\text{sen}} = 50\,\text{yr}$). Growth follows the von Bertalanffy differential form with daily-scaled stochastic noise; stage transitions are one-directional based on size thresholds (Table 2).

At the end of each simulated year, an annual step performs:

1. **Larval dispersal** via the connectivity matrix **C**: unsettled cohorts from all nodes are pooled, redistributed probabilistically among destination nodes, and settled at receiving nodes or retained in a pending queue for next-year daily settlement.

2. **Disease introduction** (at the designated epidemic year): a fixed number of agents per node are seeded in the Exposed (E) compartment.

3. **Genetic recording**: per-node allele frequencies, additive genetic variance V_A, and trait means are logged annually. Pre- and post-epidemic allele frequency snapshots are captured for calibration against genomic data.

2.4 Design Rationale

Several design choices distinguish SSWD-EvoEpi from previous SSWD models:

Individual-based representation. SSWD mortality is strongly size-dependent [OR = 1.23 per 10 mm; 12], genetically mediated [50], and spatially heterogeneous. A compartmental SIR/SEIR model would require aggregating these axes of variation into homogeneous classes, losing the emergent eco-evolutionary dynamics that arise from individual

heterogeneity in resistance, tolerance, and recovery. Following Clement et al. [8], who demonstrated that individual-based eco-evolutionary models are essential for predicting host–pathogen coevolution in Tasmanian devil facial tumor disease, we track each individual's genotype, phenotype, and infection history explicitly.

Continuous daily demographics. Rather than applying mortality, growth, and reproduction as annual pulses, SSWD-EvoEpi evaluates natural mortality and growth daily (Eq. 1), with spawning resolved to individual daily events across a multi-month season. This avoids artificial synchronization artifacts and allows disease–demography interactions (e.g., post-spawning immunosuppression) to operate on their natural timescales.

Separated genotype storage. The 51-locus diploid genotype array (102 bytes per agent) is stored separately from the agent state record. This ensures that the most frequently accessed fields during daily disease and movement updates (position, disease state, size) occupy contiguous memory, improving CPU cache performance by a factor of $\sim$2–3$\times$ in profiled benchmarks.

Three-trait genetic architecture. The 51 loci are partitioned into three independently segregating trait blocks of 17 loci each, controlling resistance (immune exclusion), tolerance (damage limitation), and recovery (pathogen clearance). This architecture captures the empirical observation that host defense against infectious disease operates through mechanistically distinct pathways that can evolve semi-independently [47].

3 Disease Module

The disease module implements a stochastic, environmentally driven SEIPD+R (Susceptible–Exposed–Infectious$_1$–Infectious$_2$–Dead, plus Recovered) compartmental framework operating at the individual level. Each agent carries its own disease state, countdown timer, genetic defense traits (r_i, t_i, c_i), and (when pathogen evolution is enabled) the virulence phenotype v_i of its infecting strain. Disease dynamics are resolved daily at each spatial node, coupled to the environmental forcing module for temperature-dependent rates and to the genetics module for individual susceptibility.

3.1 Compartmental Structure

The disease pathway consists of five compartments plus a recovery state (Figure **??**):

- **S (Susceptible):** Healthy, at risk of infection.
- **E (Exposed):** Latently infected; not yet shedding pathogen. Duration is Erlang-distributed with shape $k_E = 3$.

- **I_1 (Early infectious):** Pre-symptomatic shedding at rate $\sigma_1(T)$. Duration is Erlang-distributed with shape $k_{I_1} = 2$. Agents with high clearance ability ($c_i > 0.5$) may recover early.

- **I_2 (Late infectious):** Symptomatic wasting with high shedding rate $\sigma_2(T)$. Duration is Erlang-distributed with shape $k_{I_2} = 2$. Agents may recover with probability $p_{\text{rec}} = \rho_{\text{rec}} \times c_i$ per day.

- **D (Dead from disease):** Carcass continues to shed pathogen saprophytically for a 3-day window at rate σ_D.

- **R (Recovered):** Immune; functionally equivalent to S for demographics but not susceptible to reinfection.

3.1.1 Erlang-Distributed Stage Durations

Durations in compartments E, I_1, and I_2 are drawn from Erlang distributions rather than geometric (exponential) distributions. The Erlang distribution with shape parameter k and rate parameter $k\mu$ has mean $1/\mu$ and coefficient of variation $\text{CV} = 1/\sqrt{k}$, producing more realistic, peaked duration distributions compared to the memoryless exponential [57]. For each individual entering a compartment, a duration is sampled as:

$$\tau \sim \text{Erlang}(k,\ k\mu(T)), \quad \text{rounded to} \quad \max(1,\ \text{round}(\tau))\ \text{days}, \tag{2}$$

where $\mu(T)$ is the temperature-dependent transition rate at the current SST (Section 3.3.2). The shape parameters are:

$$k_E = 3\ \ (\text{CV} = 0.58), \quad k_{I_1} = 2\ \ (\text{CV} = 0.71), \quad k_{I_2} = 2\ \ (\text{CV} = 0.71). \tag{3}$$

Timers count down by one each day; when the timer reaches zero, the agent transitions to the next compartment.

3.2 Force of Infection

The per-individual instantaneous hazard rate of infection is:

$$\lambda_i = a_{\text{exp}} \underbrace{\frac{P_k}{K_{1/2} + P_k}}_{\text{dose–response}} \underbrace{(1 - r_i)}_{\text{resistance}} \underbrace{S_{\text{sal}}}_{\text{salinity}} \underbrace{f_{\text{size}}(L_i)}_{\text{size}}, \tag{4}$$

where:

- $a_{\text{exp}} = 0.75\,\text{d}^{-1}$ is the baseline exposure rate;

- P_k is the local Vibrio concentration (bacteria mL^{-1}) at node k;
- $K_{1/2} = 87{,}000\,\text{bacteria}\,\mathrm{mL}^{-1}$ is the half-infective dose (Michaelis–Menten dose–response);
- $r_i \in [0, 1]$ is the individual's resistance score (immune exclusion; Section 4);
- S_{sal} is the salinity modifier (Section 3.2.2);
- $f_{\text{size}}(L_i)$ is the size-dependent susceptibility modifier (Section 3.2.3).

The discrete daily probability of infection is:

$$p_{\text{inf}} = 1 - \exp(-\lambda_i \, \Delta t) \,, \quad \Delta t = 1 \text{ day}. \tag{5}$$

3.2.1 Dose–Response Function

Pathogen exposure follows a Michaelis–Menten (saturating) dose–response:

$$D(P_k) = \frac{P_k}{K_{1/2} + P_k}. \tag{6}$$

At low concentrations ($P_k \ll K_{1/2}$), infection probability scales linearly with pathogen density; at high concentrations ($P_k \gg K_{1/2}$), it saturates at $D \to 1$, reflecting physiological limits on pathogen uptake.

3.2.2 Salinity Modifier

Vibrio viability is suppressed at low salinities, providing a mechanistic basis for the reduced SSWD prevalence observed in fjord systems:

$$S_{\text{sal}} = \begin{cases} 0 & \text{if } S \le S_{\min} = 10 \text{ psu}, \\ \left(\dfrac{S - S_{\min}}{S_{\text{full}} - S_{\min}} \right)^{\eta} & \text{if } S_{\min} < S < S_{\text{full}}, \\ 1 & \text{if } S \ge S_{\text{full}} = 28 \text{ psu}, \end{cases} \tag{7}$$

where $\eta = 2$ produces a convex response (low salinity is strongly protective).

3.2.3 Size-Dependent Susceptibility

Larger *Pycnopodia helianthoides* are more susceptible to SSWD, consistent with the empirical finding of Eisenlord et al. [12] (odds ratio 1.23 per 10 mm increase in radius). The size modifier is:

$$f_{\text{size}}(L_i) = \exp\!\left(\beta_L \, \frac{L_i - \bar{L}}{\sigma_L} \right), \tag{8}$$

where $\beta_L = 0.021\,\text{mm}^{-1}$ ($= \ln 1.23/10$), $\bar{L} = 300\,\text{mm}$ is the reference size, and $\sigma_L = 100\,\text{mm}$ normalizes the deviation. An individual of diameter $L_i = 500\,\text{mm}$ has $\sim$1.5$\times$ the infection hazard of a 300 mm individual.

3.2.4 Post-Spawning Immunosuppression

Spawning imposes a transient immune cost. Following each spawning event, an individual enters a 28-day immunosuppression window during which its effective resistance is reduced:

$$r_{i,\text{eff}} = \frac{r_i}{\psi_{\text{spawn}}}, \quad \psi_{\text{spawn}} = 2.0, \tag{9}$$

clamped to $[0, 1]$. This halves effective resistance during the immunosuppressed period, creating an evolutionary coupling between reproductive investment and disease vulnerability.

3.3 Disease Progression and Recovery

Disease progression rates are temperature-dependent via an Arrhenius function (Section 3.3.2). At each daily step, disease timers are decremented; when a timer reaches zero, the agent transitions to the next state. Recovery can occur before timer expiry.

3.3.1 Transition Rates

The base progression rates at reference temperature $T_{\text{ref}} = 20\,^\circ\text{C}$ are:

$$\mu_{E\to I_1} = 0.57\ \text{d}^{-1} \qquad (E_a/R = 4{,}000\ \text{K}), \tag{10}$$

$$\mu_{I_1\to I_2} = 0.40\ \text{d}^{-1} \qquad (E_a/R = 5{,}000\ \text{K}), \tag{11}$$

$$\mu_{I_2\to D} = 0.173\ \text{d}^{-1} \qquad (E_a/R = 2{,}000\ \text{K}). \tag{12}$$

The activation energy for $\text{I}_2 \to \text{D}$ is notably lower ($E_a/R = 2{,}000\,\text{K}$ vs. 5,000–6,000 K for other transitions), reflecting evidence that terminal wasting is less temperature-sensitive than earlier disease stages (Errata E1).

3.3.2 Temperature Scaling (Arrhenius)

All temperature-dependent rates are scaled via the Arrhenius equation:

$$k(T) = k_{\text{ref}} \exp\left[\frac{E_a}{R}\left(\frac{1}{T_{\text{ref}}} - \frac{1}{T}\right)\right], \tag{13}$$

where $T_{\text{ref}} = 293.15\,\text{K}$ (20 °C) is the reference temperature corresponding to the *Vibrio pectenicida* thermal optimum [33], and E_a/R is the activation energy divided by the gas constant. The Arrhenius formulation ensures that colder temperatures slow disease

progression (longer E, I_1, I_2 durations) and reduce shedding rates, consistent with the observed latitudinal gradient in SSWD severity.

3.3.3 Tolerance: Extending I_2 Duration

The tolerance trait t_i operates as a damage-limitation mechanism that reduces the effective $I_2 \to D$ mortality rate, extending survival time while infected:

$$\mu_{I_2 \to D,\text{eff}} = \mu_{I_2 \to D}(T) \times (1 - t_i \, \tau_{\max}), \quad \text{floored at } 0.05 \times \mu_{I_2 \to D}(T), \tag{14}$$

where $\tau_{\max} = 0.85$ is the maximum mortality reduction at $t_i = 1$. The floor prevents biologically implausible indefinite survival. The effective rate is used when sampling the I_2 timer (Eq. 2), so tolerant individuals spend longer in I_2— which may prolong both recovery opportunity and pathogen shedding.

3.3.4 Recovery

Recovery from infection proceeds via the clearance trait c_i, which represents the host's capacity for pathogen elimination.

Recovery from I_2. Each day, an I_2 individual has probability:

$$p_{\text{rec},I_2} = \rho_{\text{rec}} \times c_i, \quad \rho_{\text{rec}} = 0.05 \text{ d}^{-1}, \tag{15}$$

of transitioning to the R compartment. At $c_i = 0$ (no clearance ability), recovery is impossible; at $c_i = 1$, the daily recovery probability is 5%.

Early recovery from I_1. Individuals with exceptionally high clearance ability ($c_i > 0.5$) can recover during the pre-symptomatic stage:

$$p_{\text{rec},I_1} = \begin{cases} 0 & \text{if } c_i \leq 0.5, \\ \rho_{\text{rec}} \times 2\,(c_i - 0.5) & \text{if } c_i > 0.5. \end{cases} \tag{16}$$

At $c_i = 1.0$, the early recovery probability equals ρ_{rec}, identical to I_2 recovery at maximum clearance. The threshold at $c_i = 0.5$ ensures that only rare, high-clearance individuals can clear infection before progressing to the symptomatic stage.

3.4 Vibrio Dynamics

The concentration of waterborne *Vibrio pectenicida* at node k evolves according to:

$$\frac{dP_k}{dt} = \underbrace{\sigma_1(T)\, n_{I_1} + \sigma_2(T)\, n_{I_2} + \sigma_D\, n_{D,\text{fresh}}}_{\text{shedding}} - \underbrace{\xi(T)\, P_k}_{\text{decay}} - \underbrace{\phi_k\, P_k}_{\text{flushing}} + \underbrace{P_{\text{env}}(T,S)}_{\text{reservoir}} + \underbrace{\textstyle\sum_j d_{jk}\, P_j}_{\text{dispersal}}, \tag{17}$$

integrated via forward Euler with $\Delta t = 1\,\text{day}$, subject to $P_k \geq 0$.

3.4.1 Shedding

Pathogen shedding from live infectious hosts is temperature-dependent:

$$\sigma_1(T) = 5.0 \times \text{Arr}(T) \quad (\text{I}_1\text{: pre-symptomatic}), \tag{18}$$

$$\sigma_2(T) = 50.0 \times \text{Arr}(T) \quad (\text{I}_2\text{: symptomatic}), \tag{19}$$

where $\text{Arr}(T)$ denotes the Arrhenius factor (Eq. 13) with $E_a/R = 5{,}000\,\text{K}$. The 10-fold difference between early and late shedding reflects the dramatic increase in tissue degradation and pathogen release during the wasting phase. Rates are given in bacteria $\text{mL}^{-1}\,\text{d}^{-1}\,\text{host}^{-1}$ and represent field-effective values (Errata E2).

3.4.2 Carcass Shedding

Dead individuals (D compartment) continue to shed pathogen saprophytically for a 3-day window at a constant rate $\sigma_D = 15\,\text{bacteria}\,\text{mL}^{-1}\,\text{d}^{-1}\,\text{carcass}^{-1}$ (field-effective; Code Errata CE-6). A ring buffer of daily disease death counts over the most recent 3 days tracks the number of "fresh" carcasses contributing to shedding:

$$n_{D,\text{fresh}}(t) = \sum_{\tau=0}^{2} \text{deaths}(t-\tau). \tag{20}$$

3.4.3 Vibrio Decay

Vibrio pectenicida survives longer in warmer water. The natural decay rate $\xi(T)$ is interpolated log-linearly between empirical estimates:

$$\xi(T) = \begin{cases} 1.0\ \text{d}^{-1} & T \leq 10\,^\circ\text{C} \ \ (\text{half-life} \approx 0.7\ \text{d}), \\ 0.33\ \text{d}^{-1} & T \geq 20\,^\circ\text{C} \ \ (\text{half-life} \approx 2.1\ \text{d}), \\ \exp\!\big[(1-f)\ln\xi_{10} + f\ln\xi_{20}\big] & \text{otherwise}, \end{cases} \tag{21}$$

where $f = (T-10)/10$ and values are clamped outside the 10–20 °C range. This counter-intuitive pattern (faster decay at cold temperatures) reflects the environmental Vibrio

literature [39].

3.4.4 Environmental Reservoir

In the ubiquitous scenario (default), *Vibrio pectenicida* is assumed to persist in the sediment as viable-but-non-culturable (VBNC) cells that resuscitate when SST exceeds a threshold. The background input rate is:

$$P_{\text{env}}(T,S) = P_{\text{env,max}} \underbrace{\frac{1}{1+e^{-\kappa_{\text{VBNC}}\,(T-T_{\text{VBNC}})}}}_{\text{VBNC sigmoid}} \underbrace{g_{\text{peak}}(T)}_{\text{thermal performance}} \underbrace{S_{\text{sal}}}_{\text{salinity}}, \tag{22}$$

where:

- $P_{\text{env,max}} = 500\,\text{bacteria}\,\text{mL}^{-1}\,\text{d}^{-1}$ is the maximum input rate;
- $\kappa_{\text{VBNC}} = 1.0\,^{\circ}\text{C}^{-1}$ controls the steepness of VBNC resuscitation;
- $T_{\text{VBNC}} = 12\,^{\circ}\text{C}$ is the midpoint temperature;
- $g_{\text{peak}}(T)$ is a thermal performance curve with Arrhenius increase below $T_{\text{opt}} = 20\,^{\circ}\text{C}$ and quadratic decline above, reaching zero at $T_{\text{max}} = 30\,^{\circ}\text{C}$.

In the invasion scenario, $P_{\text{env}} = 0$ everywhere until the pathogen is explicitly introduced.

3.5 Pathogen Evolution

When pathogen evolution is enabled, each infectious agent carries a continuous virulence phenotype v_i that modulates disease rates via mechanistic tradeoff functions.

3.5.1 Virulence–Tradeoff Curves

More virulent strains kill faster, shed more, and progress more rapidly, but also remove themselves from the host population sooner:

$$\sigma_{1,v}(T) = \sigma_1(T) \times \exp\big(\alpha_{\text{shed}}\,\gamma_{\text{early}}\,(v - v^*)\big), \tag{23}$$

$$\sigma_{2,v}(T) = \sigma_2(T) \times \exp\big(\alpha_{\text{shed}}\,(v - v^*)\big), \tag{24}$$

$$\mu_{I_1\to I_2,v}(T) = \mu_{I_1\to I_2}(T) \times \exp\big(\alpha_{\text{prog}}\,(v - v^*)\big), \tag{25}$$

$$\mu_{I_2\to D,v}(T) = \mu_{I_2\to D}(T) \times \exp\big(\alpha_{\text{kill}}\,(v - v^*)\big), \tag{26}$$

where $v^* = 0.5$ is the ancestral virulence (identity point), $\alpha_{\text{shed}} = 1.5$, $\alpha_{\text{prog}} = 1.0$, $\alpha_{\text{kill}} = 2.0$, and $\gamma_{\text{early}} = 0.3$ attenuates the shedding effect in the pre-symptomatic stage.

3.5.2 Transmission and Mutation

When a new infection occurs, the infecting strain is inherited either from a shedding individual (weighted by shedding rate) or from the environmental reservoir (with virulence $v_{\text{env}} = 0.5$). The probability of inheriting from a shedder is proportional to the total host-derived shedding relative to total pathogen input:

$$P(\text{from shedder}) = \frac{\sum_j \sigma_j(v_j, T)}{\sum_j \sigma_j(v_j, T) + P_{\text{env}}(T, S)}. \tag{27}$$

The inherited virulence is then subject to mutation:

$$v_{\text{new}} = \text{clip}\big(v_{\text{parent}} + \mathcal{N}(0,\ \sigma^2_{v,\text{mut}}),\ v_{\min},\ v_{\max}\big)\,, \tag{28}$$

with $\sigma_{v,\text{mut}} = 0.02$, $v_{\min} = 0$, $v_{\max} = 1$.

3.6 Basic Reproduction Number

The basic reproduction number provides a summary measure of epidemic potential at a node:

$$R_0 = \frac{a_{\text{exp}}\, S_0\,(1-\bar{r})\, S_{\text{sal}}}{K_{1/2}\,(\xi(T) + \phi_k)} \left[\frac{\sigma_1(T)}{\mu_{I_1 \to I_2}(T)} + \frac{\sigma_2(T)}{\mu_{I_2 \to D,\text{eff}}(T) + \rho_{\text{rec}}\,\bar{c}} + \sigma_D\,\tau_D\right], \tag{29}$$

where S_0 is the number of susceptibles, $\bar{r}$ and $\bar{c}$ are population-mean resistance and recovery scores, $\mu_{I_2 \to D,\text{eff}}$ incorporates population-mean tolerance (Eq. 14), $\rho_{\text{rec}}\,\bar{c}$ adds the recovery exit rate from I_2, and $\tau_D = 3\,\text{days}$ is the carcass shedding duration. The three bracketed terms represent the pathogen contribution from each infectious compartment (I_1, I_2, and D carcasses, respectively).

3.7 Daily Update Sequence

Within each daily timestep, the disease module executes the following steps in order:

1. **Update Vibrio concentration** via Euler integration of Eq. 17, using current compartment counts and environmental conditions.

2. **Transmission (S → E):** For each susceptible agent, compute the force of infection λ_i (Eq. 4), convert to daily probability (Eq. 5), and draw a Bernoulli infection event. Newly exposed agents receive an Erlang-sampled E-stage timer. When pathogen evolution is active, the infecting strain is inherited and mutated (Section 3.5.2).

3. **Disease progression:** Decrement all disease timers. For agents with expired timers: E → I_1, I_1 → I_2 (with tolerance-adjusted timer), I_2 → D. For agents with

active timers: check recovery from I_2 (Eq. 15) and early recovery from I_1 (Eq. 16).

4. **Carcass tracking:** Record today's disease deaths in the 3-day ring buffer for saprophytic shedding.

5. **Update diagnostics:** Recount compartments, update cumulative statistics (total infections, deaths, recoveries), track peak prevalence and peak Vibrio.

All operations are vectorized using NumPy batch sampling and array-level random draws for computational efficiency, achieving $O(N)$ scaling in population size.

4 Genetics Module

The genetics module tracks a diploid genotype at 51 biallelic loci for every individual, partitioned into three quantitative defense traits: *resistance*, *tolerance*, and *recovery*. Genotypes are transmitted via Mendelian inheritance with free recombination, mutated at a per-allele rate $\mu = 10^{-8}$ per generation [41], and subject to natural selection through the coupling of trait scores to disease dynamics (Section 3). The module additionally implements sweepstakes reproductive success (SRS) to capture the extreme reproductive variance characteristic of broadcast-spawning marine invertebrates [24].

4.1 Three-Trait Architecture

Each individual carries a (51×2) genotype array of `int8` alleles, where the 51 loci are partitioned into three contiguous blocks:

Table 3: Three-trait genetic architecture. The partition is configurable (constraint: $n_R + n_T + n_C = 51$); the default 17/17/17 split is used in all analyses reported here.

Trait	**Symbol**	**Loci**	**Indices**	**Mechanistic role**
Resistance	r_i	$n_R = 17$	0–16	Immune exclusion: reduces probability of S $\to$ E transition
Tolerance	t_i	$n_T = 17$	17–33	Damage limitation: extends I_2 survival via mortality rate reduction
Recovery	c_i	$n_C = 17$	34–50	Pathogen clearance: daily probability of $I_1/I_2 \to$ R transition

These three traits represent biologically distinct immune strategies with different epidemiological consequences [47]:

- **Resistance** (r_i) acts *before* infection via receptor polymorphisms, barrier defenses, and innate pathogen recognition. Resistant individuals reduce pathogen pressure on the population by preventing shedding entirely.

- **Tolerance** (t_i) acts *during* infection via tissue repair, anti-inflammatory regulation, and metabolic compensation. Tolerant hosts survive longer while infected but continue to shed pathogen—they are epidemiological "silent spreaders" that maintain transmission pressure while saving themselves.

- **Recovery** (c_i) acts *during late infection* via coelomocyte phagocytosis and immune effector mobilization. Recovering hosts actively clear the pathogen and transition to an immune state (R), removing a shedding host from the population.

The locus count of 51 is motivated by Schiebelhut et al. [49], who identified ∼51 loci under selection in *Pisaster ochraceus* SSWD survivors. No genome-wide association study (GWAS) data currently distinguish resistance, tolerance, and recovery loci in *P. helianthoides*; the equal 17/17/17 partition is a simplifying assumption whose sensitivity is explored via the n_R parameter in the global sensitivity analysis (Section 7). A reference genome for *P. helianthoides* is now available [51], enabling future empirical partitioning.

Removal of EF1A overdominant locus. An earlier model version included a 52nd locus representing the EF1A elongation factor with overdominant fitness effects, based on Wares and Schiebelhut [56] who documented allele frequency shifts at this locus in *Pisaster ochraceus* following SSWD. We removed this locus because (1) the EF1A finding is specific to *Pisaster* with no evidence of overdominance in *P. helianthoides*, and (2) a single overdominant locus imposed a hard floor on heterozygosity loss that was biologically unjustified for our focal species.

4.2 Trait Score Computation

At each locus ℓ, an individual carries two alleles $g_{\ell,0}, g_{\ell,1} \in \{0, 1\}$, where 1 denotes the derived (protective) allele and 0 the ancestral allele. Each locus within a trait block has a fixed effect size $e_\ell > 0$, and an individual's trait score is the effect-weighted mean allele dosage:

$$\theta_i = \sum_{\ell \in \mathcal{L}_\theta} e_\ell \, \frac{g_{\ell,0} + g_{\ell,1}}{2} \tag{30}$$

where $\mathcal{L}_\theta$ denotes the locus set for trait $\theta \in \{r, t, c\}$ and $\theta_i \in [0, \sum e_\ell]$. Effect sizes within each trait block are normalized so $\sum_{\ell \in \mathcal{L}_\theta} e_\ell = 1$, bounding all trait scores to $[0, 1]$.

4.2.1 Effect Size Distribution

Per-locus effect sizes are drawn from an exponential distribution $e_\ell \sim \text{Exp}(\lambda = 1)$, normalized to sum to 1.0 within each trait, and sorted in descending order. This produces a distribution where a few loci have large effects and the remainder have small effects,

consistent with empirical QTL architectures for disease resistance traits [36]. A fixed seed ensures identical effect sizes across simulation runs. Each trait block receives independently drawn effect sizes.

4.2.2 Coupling to Disease Dynamics

The three traits feed into the disease module (Section 3) as follows:

1. **Resistance** reduces the per-individual force of infection:

$$\lambda_i = a \cdot \frac{P}{K_{1/2} + P} \cdot (1 - r_i) \cdot S_{\text{sal}} \cdot f_L(L_i) \tag{31}$$

   where a is the exposure rate, P the local *Vibrio pectenicida* concentration, $K_{1/2}$ the half-infective dose, S_{sal} the salinity modifier, and $f_L(L_i)$ the size-dependent susceptibility factor.

2. **Tolerance** reduces the $\text{I}_2 \to \text{D}$ transition rate via a timer-scaling mechanism:

$$\mu^{\text{eff}}_{\text{I}_2\text{D},i} = \mu_{\text{I}_2\text{D}}(T) \cdot \big(1 - t_i \cdot \tau_{\max}\big) \tag{32}$$

   where $\tau_{\max} = 0.85$ is the maximum mortality reduction achievable at $t_i = 1$. A floor of 5% of the baseline rate prevents complete elimination of disease mortality. Tolerant individuals survive longer while infected but continue shedding, creating a selective conflict between individual and population-level fitness.

3. **Recovery** determines the daily clearance probability:

$$p_{\text{rec},i} = \rho_{\text{rec}} \times c_i \tag{33}$$

   where $\rho_{\text{rec}} = 0.05\ \text{d}^{-1}$ is the base recovery rate. Recovery from I_1 requires $c_i > 0.5$ (early clearance); recovery from I_2 has no threshold. Successful recovery transitions the agent to the R (recovered, immune) compartment.

4.3 Genotype Initialization

Initial allele frequencies are drawn independently for each locus from a Beta distribution:

$$q_\ell \sim \text{Beta}(a, b) \quad (\text{default } a = 2,\ b = 8) \tag{34}$$

producing a right-skewed frequency spectrum where most protective alleles are rare ($\mathbb{E}[q] = a/(a+b) = 0.2$), consistent with standing variation in immune genes maintained by mutation–selection balance. The raw frequencies are then rescaled per-trait so that the expected population-mean trait score equals a configurable target:

Table 4: Default target population-mean trait scores at initialization.

Trait	**Target mean**	**Rationale**
Resistance (r_i)	0.15	Pre-epidemic standing variation
Tolerance (t_i)	0.10	Moderate damage limitation
Recovery (c_i)	0.02	Rare standing variation for clearance

Recovery is initialized with the lowest mean because active pathogen clearance is assumed to be the rarest phenotype prior to epidemic exposure. Per-locus frequencies are clipped to $[0.001, 0.5]$ to prevent fixation at initialization while ensuring the derived allele never begins at majority frequency. Genotypes are then sampled assuming Hardy–Weinberg equilibrium at each locus: each allele copy is independently drawn as a Bernoulli trial with probability q_ℓ.

4.4 Mendelian Inheritance and Mutation

At reproduction, each offspring inherits one randomly chosen allele from each parent at every locus (independent assortment, no linkage). The vectorized implementation draws allele choices for all $n_{\text{offspring}} \times 51 \times 2$ positions simultaneously, then indexes into parental genotype arrays.

Mutations are applied to offspring genotypes at rate $\mu = 10^{-8}$ per allele per generation [41]. The total number of mutations per cohort is drawn from a Poisson distribution: $n_{\text{mut}} \sim \text{Pois}(\mu \times n_{\text{offspring}} \times 51 \times 2)$. Each mutation flips the allele at a randomly chosen position ($0 \to 1$ or $1 \to 0$), providing bidirectional mutational pressure. At the default mutation rate, mutations are negligible within the 20–100 year simulation horizon (expected $\sim 10^{-6}$ mutations per offspring), and evolution proceeds primarily through selection on standing variation.

4.5 Sweepstakes Reproductive Success

Broadcast-spawning marine invertebrates exhibit sweepstakes reproductive success (SRS): a tiny fraction of adults contribute the majority of surviving offspring in any given cohort [24]. This phenomenon produces N_e/N ratios on the order of 10^{-3} in empirical observations [3] and dramatically amplifies genetic drift while simultaneously accelerating the fixation of favorable alleles in post-epidemic populations [13].

SSWD-EvoEpi implements SRS via a Pareto-weighted reproductive lottery. Each spawning adult receives a random weight drawn from a Pareto distribution with shape parameter α_{SRS} (default 1.35):

$$w_i \sim \text{Pareto}(\alpha_{\text{SRS}}) + 1 \tag{35}$$

Female weights are additionally multiplied by size-dependent fecundity (Section 5.5), so larger females that win the sweepstakes lottery contribute disproportionately:

$$\tilde{w}_{i,\text{female}} = w_i \times \left(\frac{L_i}{L_{\text{ref}}}\right)^b \tag{36}$$

where $b = 2.5$ is the fecundity allometric exponent and $L_{\text{ref}} = 500$ mm. Male weights use the raw Pareto draw without fecundity modulation. Parents are then sampled with replacement from the normalized weight distributions, and offspring receive Mendelian-inherited genotypes.

The Pareto shape $\alpha_{\text{SRS}} = 1.35$ was chosen to produce N_e/N ratios consistent with empirical estimates of $\sim 10^{-3}$ in marine broadcast spawners [3, 24]. A small annual variation in α (drawn from $\mathcal{N}(\alpha_{\text{SRS}}, \sigma_\alpha^2)$ with $\sigma_\alpha = 0.10$) produces temporal fluctuation in the variance of reproductive success across cohorts.

Effective population size. N_e is computed from the realized offspring distribution using the standard formula [24]:

$$N_e = \frac{4N - 2}{V_k + 2} \tag{37}$$

where N is the number of spawning parents and V_k is the variance in offspring number. Sex-specific N_e values are computed for females and males separately, then combined via harmonic mean: $N_e = 4N_{e,f}N_{e,m}/(N_{e,f} + N_{e,m})$.

4.6 Genetic Diagnostics and Tracking

The model records a suite of genetic summary statistics at each node at annual intervals:

- **Per-trait means and variances:** $\bar{r}$, $\bar{t}$, $\bar{c}$ and $\text{Var}(r)$, $\text{Var}(t)$, $\text{Var}(c)$.
- **Additive genetic variance** (V_A) per trait:

$$V_{A,\theta} = \sum_{\ell \in \mathcal{L}_\theta} 2\, e_\ell^2\, q_\ell\, (1 - q_\ell) \tag{38}$$

  where q_ℓ is the derived allele frequency at locus ℓ. V_A determines the potential rate of evolutionary response to selection.

- **Heterozygosity:** Observed (H_o) and expected (H_e) heterozygosity averaged across all 51 loci.
- F_{ST}**:** Weir–Cockerham-style F_{ST} across nodes, computed as $F_{ST} = \text{Var}(\bar{q})/[\bar{q}(1-\bar{q})]$ averaged across polymorphic loci.

- **Pre- and post-epidemic allele frequency snapshots:** Full 51-locus allele frequency vectors taken immediately before pathogen introduction and two years after the epidemic onset, enabling direct measurement of allele frequency shifts (Δq) attributable to selection.

No cost of resistance. A cost-of-resistance parameter (fecundity penalty for high r_i) was considered but excluded following discussion with the senior author. No empirical evidence supports a measurable fecundity cost for disease resistance alleles in *P. helianthoides*, and including an unparameterized cost would introduce a free parameter with no calibration target. Fecundity depends solely on body size (Section 5.5).

4.7 Genotype Bank (Tier 2 Nodes)

For Tier 2 spatial nodes that use simplified demographics rather than full agent tracking, the genetics module maintains a *genotype bank* of $N_{\text{bank}} = 100$ representative diploid genotypes with associated frequency weights. The bank is created by random sampling from the alive population and preserves all three trait scores and allele frequencies. When agents migrate from a Tier 2 to a Tier 1 node, genotypes are expanded from the bank using SRS-weighted sampling (Pareto weights $\times$ bank frequency weights) to reconstruct individual-level genetic variation.

5 Population Dynamics

The population dynamics module governs the complete life history of *Pycnopodia helianthoides*: growth, natural mortality, reproduction, larval dispersal, and settlement. All demographic processes operate on a daily timestep, integrated within the master simulation loop described in Section 2. Disease-driven mortality is handled by the disease module (Section 3); coupling occurs through shared access to the agent array.

5.1 Life Stages

Each individual progresses through five life stages defined by size thresholds (Table 5). Stage transitions are unidirectional: agents can only advance, never regress.

Table 5: Life stages and transition thresholds for *P. helianthoides*.

Stage	**Size range**	**Transition at**	**Duration**
Egg/Larva	Planktonic	Settlement event	49–146 days PLD
Settler	0.5–10 mm	≥ 10 mm	$\sim$1 year
Juvenile	10–150 mm	≥ 150 mm	$\sim$1–5 years
Subadult	150–400 mm	≥ 400 mm	$\sim$5–10 years
Adult	>400 mm	—	10–50+ years

5.2 Growth

Individual growth follows the von Bertalanffy (VB) growth model in differential form, resolved daily:

$$L(t + \Delta t) = L_\infty - (L_\infty - L(t)) \cdot \exp(-k_{\text{growth}} \cdot \Delta t) \tag{39}$$

where $L_\infty = 1000$ mm is the asymptotic arm-tip diameter, $k_{\text{growth}} = 0.08$ yr^{-1} is the Brody growth coefficient, and $\Delta t = 1/365$ yr for the daily timestep. Individual growth variation is introduced through a multiplicative log-normal noise term applied to the daily increment:

$$\Delta L_i = (L_{\text{det}} - L_i) \cdot \exp\big(\varepsilon_i\big), \quad \varepsilon_i \sim \mathcal{N}\left(0,\ \frac{\sigma_g}{\sqrt{365}}\right) \tag{40}$$

where $\sigma_g = 2.0$ mm is the annual growth noise scale and the $\sqrt{365}$ scaling preserves the annual CV when integrated over daily steps. Size is constrained to never decrease (no shrinking). Stage transitions are evaluated after each growth step based on the thresholds in Table 5.

Aging proceeds at 1/365 years per day, producing fractional ages that drive size-at-age trajectories and determine eligibility for senescence mortality.

5.3 Natural Mortality

Natural mortality is resolved daily using continuous hazard rates derived from stage-specific annual survival probabilities. The daily death probability for individual i is:

$$p_{\text{death},i} = 1 - \big(1 - m_{\text{annual}}(s_i)\big)^{1/365} \tag{41}$$

where $m_{\text{annual}}(s) = 1 - S_{\text{annual}}(s)$ is the annual mortality rate for stage s. The annual survival schedule (Table 6) produces a type III survivorship curve with high settler/juvenile mortality and low adult mortality, consistent with demographic estimates for long-lived asteroids.

Table 6: Stage-specific annual survival rates.

Stage	**Annual survival (S)**	**Annual mortality**
Settler	0.001	0.999
Juvenile	0.03	0.97
Subadult	0.90	0.10
Adult	0.95	0.05
Senescent	0.98	0.02 (base)

Senescence. Individuals exceeding the senescence age ($a_{\text{sen}} = 50$ yr) accumulate additional mortality linearly:

$$m_{\text{total}}(s_i, a_i) = m_{\text{annual}}(s_i) + m_{\text{sen}} \cdot \frac{a_i - a_{\text{sen}}}{20} \tag{42}$$

where $m_{\text{sen}} = 0.10$ and the divisor of 20 scales the senescence ramp such that a 70-year-old individual experiences an additional 10% annual mortality.

Daily mortality is applied via a single vectorized random draw across all alive agents, converting stage-dependent annual rates to daily hazard probabilities. This continuous approach avoids the artificial synchronization artifacts of annual batch mortality and permits realistic within-year population fluctuations.

5.4 Spawning System

SSWD-EvoEpi implements a biologically detailed spawning system reflecting the extended reproductive season and cascading spawning behavior observed in *P. helianthoides*.

5.4.1 Spawning Season and Phenology

The spawning season extends from day 305 (≈November 1) through day 196 (≈July 15) of the following year, spanning approximately 270 days and wrapping across the calendar year boundary. Spawning intensity follows a Normal envelope centered on a latitude-adjusted peak:

$$P_{\text{season}}(d) = \exp\left(-\frac{(\Delta d)^2}{2\,\sigma_{\text{peak}}^2}\right) \tag{43}$$

where Δd is the shortest circular distance between day d and the peak day (accounting for year wrapping), and $\sigma_{\text{peak}} = 60$ days is the standard deviation of the seasonal peak. The peak day-of-year is latitude-dependent:

$$d_{\text{peak}}(\phi) = d_{\text{peak,base}} + \lceil (\phi - 40°\text{N}) \times 3 \text{ d}/° \rceil \tag{44}$$

where $d_{\text{peak,base}} = 105$ (≈April 15) is the reference peak at 40°N, and higher-latitude populations spawn approximately 3 days later per degree northward.

5.4.2 Spontaneous Spawning

Each day during the spawning season, mature adults (≥400 mm, Susceptible or Recovered disease state) are first evaluated for *readiness*, a stochastic physiological state modulated by the seasonal envelope $P_{\text{season}}(d)$. Once ready, individuals attempt spontaneous spawning with sex-specific daily probabilities:

$$p_{\text{spawn,female}} = 0.012 \tag{45}$$

$$p_{\text{spawn,male}} = 0.0125 \tag{46}$$

These rates were calibrated to produce ≥80% female spawning participation per season and a mean of ∼2.2 male bouts per season, consistent with the observation that males spawn more frequently than females in broadcast-spawning asteroids.

Bout limits and refractory periods. Females are limited to a maximum of 2 spawning bouts per season; males are limited to 3 bouts. Males enter a brief refractory period between bouts (default 0 days, configurable) during which they cannot spawn, reflecting the physiological recovery time for spermatogenesis.

5.4.3 Cascade Induction

Spawning by one individual can trigger spawning in nearby conspecifics via waterborne chemical cues (spawning-induced spawning), producing the synchronous mass spawning events observed in broadcast spawners. Induction operates over a 3-day chemical cue persistence window and is strongly sex-asymmetric:

$$\kappa_{\text{fm}} = 0.80 \quad \text{(female} \rightarrow \text{male induction)} \tag{47}$$

$$\kappa_{\text{mf}} = 0.60 \quad \text{(male} \rightarrow \text{female induction)} \tag{48}$$

where κ_{fm} is the probability that a ready male spawns when a female within the cascade radius (200 m) has spawned within the cue window. The female-to-male asymmetry reflects the stronger spawning trigger provided by egg-associated chemical signals. Readiness induction also operates: individuals not yet physiologically ready can be driven to readiness by nearby spawning activity, with a daily probability of 0.5 when within a 300 m detection radius.

5.4.4 Post-Spawning Immunosuppression

Spawning imposes a 28-day immunosuppression period during which the individual's force of infection is multiplied by a susceptibility factor of 2.0:

$$\lambda_i^{\text{eff}} = \lambda_i \times \begin{cases} \chi_{\text{immuno}} = 2.0 & \text{if immunosuppression timer} > 0 \\ 1.0 & \text{otherwise} \end{cases} \tag{49}$$

This reflects the metabolic cost of gamete production and the documented increase in disease susceptibility following reproductive investment in marine invertebrates. The immunosuppression timer is reset each time an individual spawns, so multiple spawning bouts within a season extend the vulnerability window. Immunosuppression timers are decremented daily regardless of spawning season status.

5.5 Fecundity

Female fecundity follows an allometric relationship with body size:

$$F_i = F_0 \cdot \left(\frac{L_i}{L_{\text{ref}}} \right)^b \tag{50}$$

where $F_0 = 10^7$ eggs is the reference fecundity at $L_{\text{ref}} = 500$ mm and $b = 2.5$ is the allometric exponent. Only females at or above the minimum reproductive size $L_{\min} = 400$ mm produce eggs. No cost-of-resistance penalty is applied to fecundity (Section 4.6).

5.6 Fertilization Kinetics and the Allee Effect

Broadcast spawners face a fertilization Allee effect: at low population density, sperm limitation reduces the fraction of eggs successfully fertilized [15, 38]. We model fertilization success using a mean-field approximation of the Lundquist and Botsford [38] broadcast fertilization model:

$$\mathcal{F}(\rho_m) = 1 - \exp(-\gamma_{\text{fert}} \cdot \rho_{m,\text{eff}}) \tag{51}$$

where $\gamma_{\text{fert}} = 4.5$ m^2 is the sperm contact parameter and $\rho_{m,\text{eff}}$ is the effective male density, potentially enhanced by spawning aggregation behavior. The aggregation factor increases effective local density within spawning clumps above the spatially uniform average when adult count exceeds a threshold.

This produces a quadratic relationship between zygote production and density at low density: zygotes $\propto \rho_f \times \mathcal{F}(\rho_m) \propto \rho^2$ when $\rho \to 0$, creating a strong demographic Allee effect. For high-fecundity broadcast spawners like *P. helianthoides*, the deterministic Allee threshold is near zero density; the practical Allee effect operates through stochastic processes at low N.

5.7 Larval Phase

Fertilized eggs enter a temperature-dependent pelagic larval duration (PLD):

$$\mathrm{PLD}(T) = \mathrm{PLD}_{\mathrm{ref}} \cdot \exp\big(-Q_{\mathrm{dev}} \cdot (T - T_{\mathrm{ref}})\big) \tag{52}$$

where $\mathrm{PLD}_{\mathrm{ref}} = 63$ days at $T_{\mathrm{ref}} = 10.5$°C [31], and $Q_{\mathrm{dev}} = 0.05$ °C^{-1} produces shorter PLD at warmer temperatures. PLD is clamped to $[30, 150]$ days.

Larval survival during the pelagic phase follows a constant daily mortality model:

$$S_{\mathrm{larval}} = \exp(-\mu_{\mathrm{larva}} \cdot \mathrm{PLD}) \tag{53}$$

with $\mu_{\mathrm{larva}} = 0.05$ d^{-1}. At the reference PLD of 63 days, this yields $S_{\mathrm{larval}} \approx 4.3\%$ — high mortality that is compensated by the enormous fecundity of *P. helianthoides*.

Larval cohorts carry genotypes inherited via the SRS lottery (Section 4.5) and are tracked as discrete objects during the pelagic phase. Upon completion of PLD, competent larvae are available for settlement. In the spatial simulation (Section 6), cohorts are dispersed between nodes via the larval connectivity matrix $\mathbf{C}$ before settlement.

5.8 Settlement and Recruitment

Competent larvae settle into the benthic population through a three-stage process:

1. Settlement cue (Allee effect). Settlement success is modulated by the presence of conspecific adults via a Michaelis–Menten function representing biofilm-mediated settlement cues:

$$C_{\mathrm{settle}}(N_{\mathrm{adults}}) = 0.2 + \frac{0.8 \cdot N_{\mathrm{adults}}}{5 + N_{\mathrm{adults}}} \tag{54}$$

where the baseline of 0.2 represents settlement on coralline algae in the absence of adults, and the additional 0.8 reflects enhanced settlement induced by adult biofilm. The half-saturation constant of 5 adults means that even a small remnant population provides strong settlement cues.

2. Density-dependent recruitment (Beverton–Holt). The number of recruits is governed by a standard Beverton–Holt stock-recruitment relationship:

$$R = \frac{K \cdot s_0 \cdot S}{K + s_0 \cdot S} \tag{55}$$

where S is the number of effective settlers (after cue modulation), K is the carrying capacity, and $s_0 = 0.03$ is the density-independent per-settler survival rate. At low S,

$R \approx s_0 S$ (supply-limited); at high S, $R \to K$ (habitat-limited). For broadcast spawners with $S \gg K$, recruitment is typically habitat-limited and population self-regulates.

3. Agent initialization. Recruited settlers are placed in dead agent slots, assigned size 0.5 mm, age 0, Settler stage, random sex (1:1 ratio), Susceptible disease state, and random position within the node's habitat area. Genotypes are copied from the SRS-selected settler genotypes, and all three trait scores (r_i, t_i, c_i) are computed from the inherited genotype.

Juvenile immunity. Newly settled individuals can optionally be granted a juvenile immunity period (configurable, default 0 days) during which they are not susceptible to infection. The settlement day is recorded for each recruit to enable age-dependent susceptibility calculations.

5.9 Continuous Settlement

Rather than settling all larvae in an annual pulse, the model tracks individual larval cohorts and settles them daily as their PLD elapses. Cohorts generated by daily spawning events throughout the extended spawning season (Section 5.4.1) are stored in a per-node pending list sorted by settlement day. Each simulation day, cohorts whose PLD has elapsed are popped from the sorted list front (amortized $O(1)$) and passed through the settlement pipeline. This continuous approach produces realistic seasonal recruitment pulses that peak several months after the spawning peak, consistent with the observed temporal offset between spawning and juvenile recruitment in *P. helianthoides*.

At the annual boundary, any remaining unsettled cohorts from each node are collected for spatial dispersal via the connectivity matrix $\mathbf{C}$ (Section 6), then redistributed to destination nodes where they continue to settle daily as PLD elapses.

5.10 Demographic–Genetic–Epidemiological Coupling

The population dynamics module is bidirectionally coupled to the disease and genetics modules:

- **Disease → demographics:** Disease kills individuals ($I_2 \to D$), reducing population size and altering age/size structure. Post-spawning immunosuppression (Section 5.4.4) increases disease risk for recent spawners, creating a temporal alignment between peak reproductive effort and peak epidemic severity during warm months.
- **Demographics → disease:** Reduced population density lowers contact rates and environmental pathogen concentration. The fertilization Allee effect (Section 5.6)

amplifies population collapse by reducing reproductive output at low density, potentially trapping populations in an extinction vortex.

- **Genetics → demographics:** The SRS lottery (Section 4.5) produces extreme reproductive variance that amplifies genetic drift while accelerating the fixation of resistance, tolerance, and recovery alleles enriched by selection during epidemic episodes.

- **Demographics → genetics:** Population bottlenecks from disease reduce N_e far below census N, compounded by SRS ($N_e/N \sim 10^{-3}$). The interaction of selection with small effective population size determines whether evolutionary rescue is fast enough to prevent extinction.

6 Spatial Module

7 Sensitivity Analysis

8 Validation

9 Discussion

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A Parameter Tables