Sobol Sensitivity Analysis (Round 4): Three-Trait SSWD-EvoEpi Model

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Abstract

We present the results of a variance-based Sobol sensitivity analysis (Round 4) for the SSWD-EvoEpi agent-based model. This analysis evaluates the influence of 47 parameters across 23 output metrics using $N = 512$ base samples (25,088 total model evaluations) on an 11-node stepping-stone spatial network. The model incorporates three-trait genetic architecture (resistance, tolerance, recovery), pathogen virulence evolution, and satellite-derived SST forcing. Key findings: the disease transmission cluster—`K_half` ($S_T = 0.456$), `a_exposure` ($S_T = 0.337$), `P_env_max` ($S_T = 0.251$), and `sigma_2_eff` ($S_T = 0.232$)—dominates population crash outcomes, collectively accounting for over 70% of total-order sensitivity. Strong parameter interactions pervade the model ($\sum S_T / \sum S_1 = 3.37$ for population crash), confirming that calibration must account for correlated parameter effects. We provide prioritized calibration recommendations for subsequent ABC-SMC inference.

Contents

1 Executive Summary

This report presents the Sobol sensitivity analysis (Round 4) of the SSWD-EvoEpi model—an agent-based simulation of Sea Star Wasting Disease incorporating host evolution and pathogen adaptation across a spatially explicit 11-node stepping-stone network.

Top 5 Most Influential Parameters (Population Crash)

1. `K_half` ($S_T = 0.456 \pm 0.128$): Half-saturation constant for dose-response. Controls the threshold pathogen load for infection. Dominates both main effects and interactions.
2. `a_exposure` ($S_T = 0.337 \pm 0.097$): Exposure rate exponent. Governs nonlinear transmission scaling with environmental pathogen concentration.
3. `P_env_max` ($S_T = 0.251 \pm 0.079$): Maximum environmental pathogen load. Sets the ceiling for pathogen accumulation in the water column.
4. `sigma_2_eff` ($S_T = 0.232 \pm 0.083$): Late-stage (I_2) shedding rate. Controls how much pathogen severely infected individuals release.
5. `sigma_D` ($S_T = 0.141 \pm 0.066$): Dead-animal shedding rate. Determines post-mortem pathogen contribution to the environmental reservoir.

Main Conclusions

- **Disease transmission dominates:** All top-5 parameters belong to the disease module. The transmission pathway—from environmental reservoir (`P_env_max`), through dose-response (`K_half`, `a_exposure`), to shedding feedback (`sigma_2_eff`, `sigma_D`)—is the primary driver of population outcomes.
- **Pervasive interactions:** The ratio $\sum S_T / \sum S_1 = 3.37$ indicates that parameter interactions account for roughly two-thirds of explained variance. No parameter acts purely additively.
- **Genetics matters for evolutionary metrics:** While genetic parameters rank mid-pack for population crash, they dominate evolutionary outcomes (resistance/tolerance/recovery shifts). `n_resistance` ($S_T = 0.020$) and `target_mean_r` ($S_T = 0.021$) are key.
- **Morris–Sobol agreement is partial:** Morris R4 ranked `rho_rec` as #1, but Sobol places it at #32. This discrepancy reveals that `rho_rec`'s Morris μ^* was inflated by interaction effects that Sobol decomposes properly.
- **Bottom quartile can be fixed:** Parameters ranked 35–47 by S_T all have $S_T < 0.004$, contributing negligibly. These can be fixed at literature values for calibration.

2 Methods

2.1 Model Configuration

The SSWD-EvoEpi model (Round 4) represents the complete three-trait architecture:

- **Spatial network:** 11-node stepping-stone chain spanning the latitudinal range of *Pycnopodia helianthoides* habitat (Alaska to southern California). Nodes include both fjord-type (high self-retention) and open-coast (lower retention, higher dispersal) sites.
- **Genetic architecture:** Three heritable quantitative traits—resistance (reduces infection probability), tolerance (extends survival while infected), and recovery (increases clearance rate). Each trait is controlled by a configurable number of additive loci ($n_{\text{resistance}}$, $n_{\text{tolerance}}$).
- **Pathogen evolution:** Virulence evolves via mutation, with trade-offs between transmission (shedding), virulence (kill rate), and progression rate controlled by α_{kill}, α_{shed}, and α_{prog}.
- **Disease dynamics:** Prentice (2025) disease rates with $R \to S$ immunity loss. Environmental pathogen reservoir with temperature-dependent VBNC dynamics.
- **Temperature forcing:** Satellite-derived SST profiles for each node, capturing the latitudinal temperature gradient.

2.2 Sensitivity Analysis Design

- **Method:** Saltelli sampling scheme for Sobol indices (SALib), `calc_second_order=False`
- **Parameters:** 47 (Table **??** in Appendix)
- **Metrics:** 23 output summary statistics (population, disease, evolutionary, spatial)
- **Sample size:** $N = 512$ base samples $\times$ $(47 + 2) = 25{,}088$ total runs
- **Computation:** 20.4 wall-clock hours on Intel Xeon W-3365 (48 cores)
- **Base seed:** 88888
- **Indices computed:** First-order (S_1) and total-order (S_T) with bootstrap confidence intervals

The first-order index S_1 measures the direct (additive) contribution of a parameter to output variance. The total-order index S_T captures both direct effects and all interactions involving that parameter. Their difference $S_T - S_1$ quantifies the contribution of parameter interactions.

2.3 Output Metrics

The 23 metrics span four domains:

- **Disease dynamics (7):** `pop_crash_pct`, `peak_mortality`, `time_to_nadir`, `disease_death_fraction`, `total_disease_deaths`, `extinction`, `recovery`
- **Evolutionary (7):** `resistance_shift_mean`, `resistance_shift_max`, `tolerance_shift_mean`, `recovery_shift_mean`, `va_retention_mean`, `evolutionary_rescue_index`, `total_recovery_events`
- **Ecological (5):** `final_pop_frac`, `n_extinct_nodes`, `mean_recruitment_rate`, `spawning_participati` `recovery_rate`

- **Spatial (4):** `north_south_mortality_gradient`, `fjord_protection_effect`, `mean_final_virulence`, `virulence_shift`

3 Results

3.1 Global Parameter Ranking (Population Crash)

Figure 1 presents the total-order Sobol indices (S_T) for all 47 parameters with respect to `pop_crash_pct`. The distribution is highly skewed: four parameters have $S_T > 0.14$, while 32 parameters have $S_T < 0.01$.

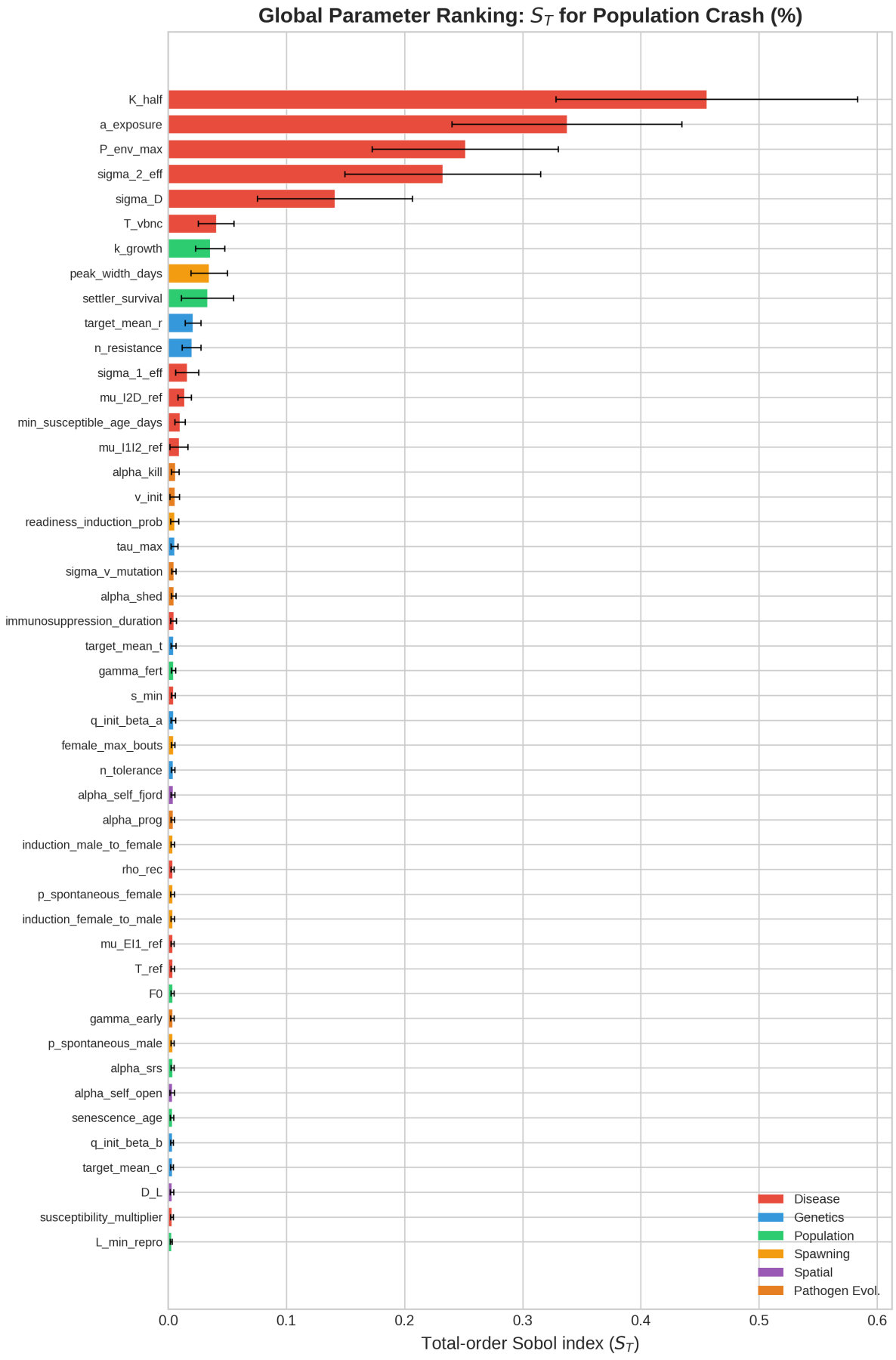

Figure 1: Global parameter ranking by total-order Sobol index (S_T) for population crash percentage. Bars are colored by parameter group. Error bars show 95% bootstrap confidence intervals. The disease transmission cluster (`K_half`, `a_exposure`, `P_env_max`, `sigma_2_eff`) dominates.

3.2 Interaction Structure

Figure 2 reveals the interaction structure. Points above the diagonal line $S_1 = S_T$ indicate parameters whose influence is amplified by interactions with other parameters.

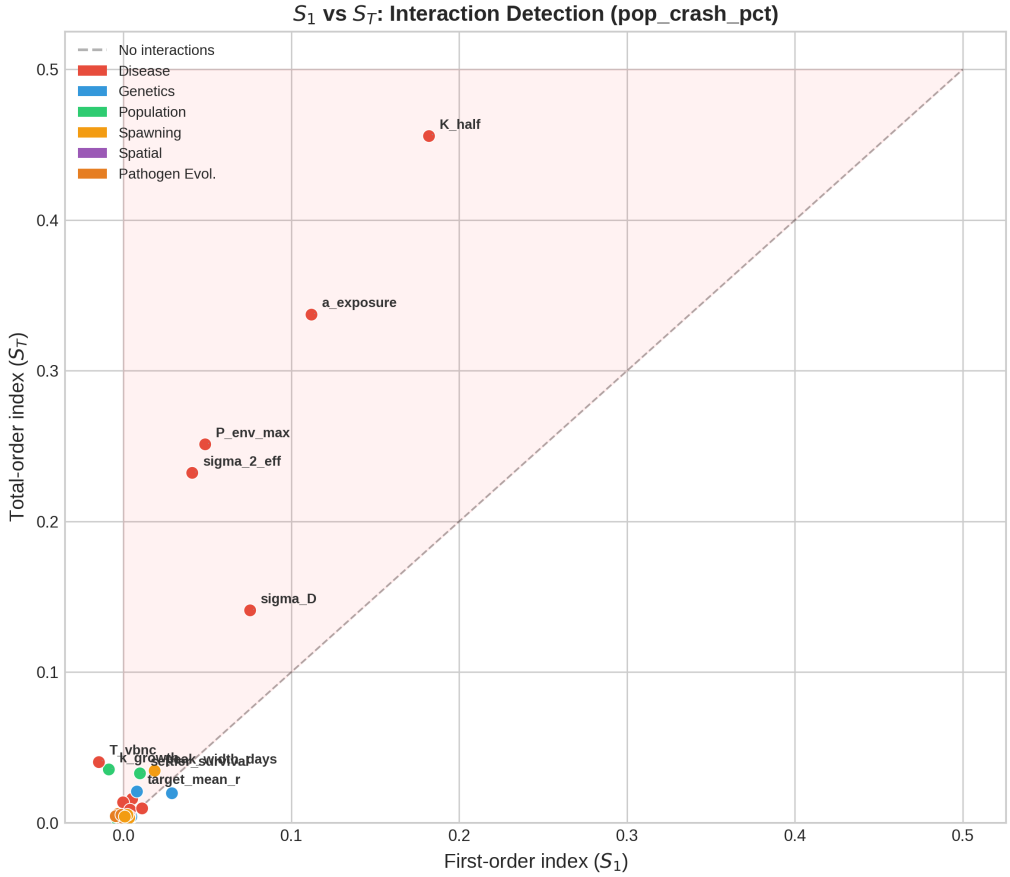

Figure 2: First-order (S_1) vs. total-order (S_T) indices for `pop_crash_pct`. Points above the diagonal indicate parameter interactions. The top-4 disease parameters show large gaps between S_1 and S_T, indicating they interact strongly with each other.

For `pop_crash_pct`, $\sum S_1 = 0.527$ and $\sum S_T = 1.775$, giving a ratio of 3.37. This means that interaction effects are responsible for approximately $1 - (0.527/1.775) = 70\%$ of the total sensitivity. The disease transmission parameters (`K_half`, `a_exposure`, `P_env_max`, `sigma_2_eff`) form a tightly coupled cluster where the effect of each parameter depends on the values of the others.

3.3 Multi-Metric Sensitivity Landscape

Figure 3 shows the S_T values for the top 20 parameters across all 23 metrics, revealing which parameters matter for which outcomes.

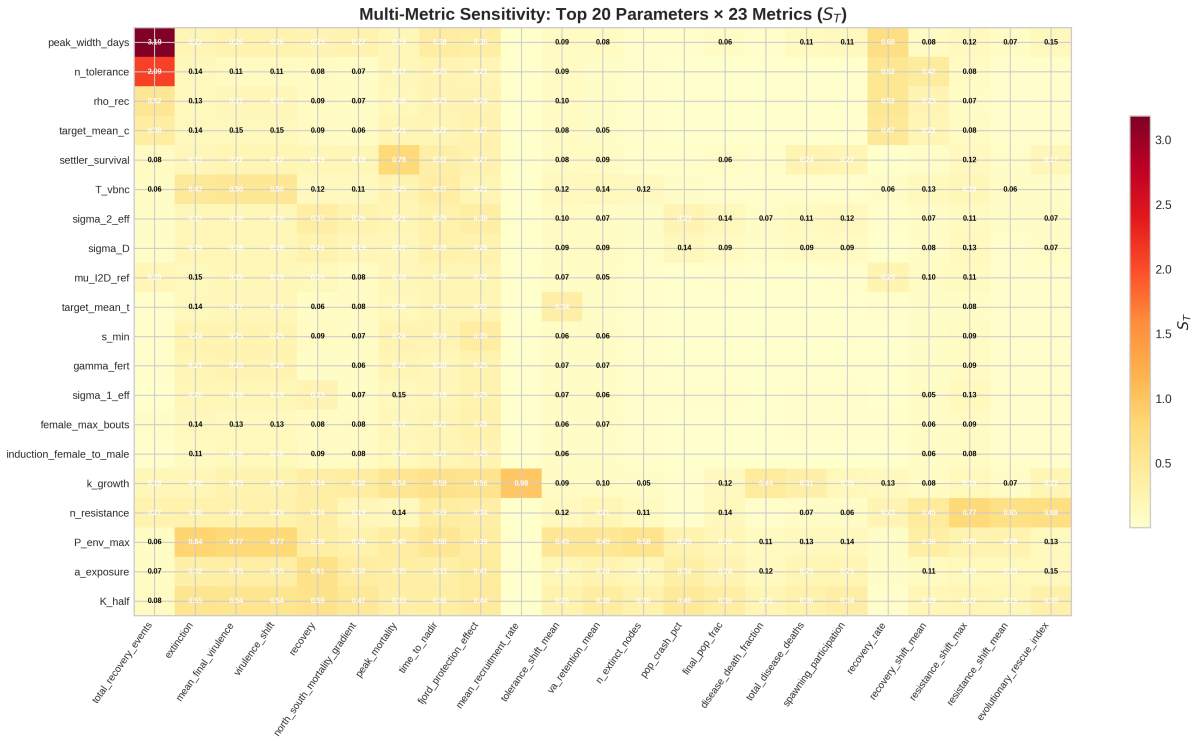

Figure 3: Multi-metric heatmap of S_T values. Rows = top 20 parameters (by maximum S_T across any metric), columns = 23 output metrics. Both axes are hierarchically clustered. The disease transmission cluster (top rows) dominates population/disease metrics, while genetic parameters emerge for evolutionary metrics.

Key patterns in the heatmap:

- **Disease transmission cluster** (`K_half`, `a_exposure`, `P_env_max`, `sigma_2_eff`): High S_T across population crash, disease deaths, extinction, and peak mortality metrics.
- **Metric clustering:** Disease outcome metrics cluster together, as do evolutionary trait shift metrics, confirming that different parameter subsets drive different outcome domains.
- **Parameter specificity:** Some parameters (like `n_resistance`) have modest S_T for population crash but high S_T for `resistance_shift_mean`, indicating domain-specific influence.

3.4 Main Effects vs. Interactions

Figure 4 decomposes the total-order index into main effects (S_1) and interaction components ($S_T - S_1$) for the top 15 parameters.

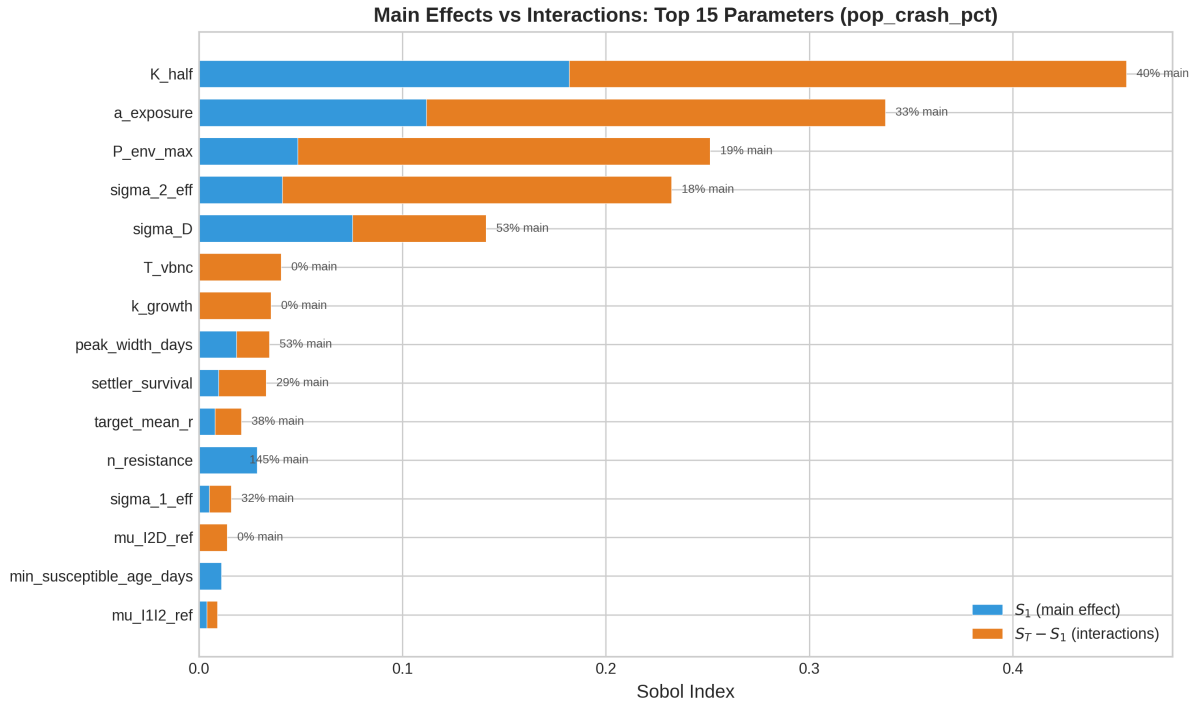

Figure 4: Decomposition of S_T into main effect (S_1, blue) and interaction ($S_T - S_1$, orange) components for the top 15 parameters. Percentages show the fraction attributable to main effects. `K_half` has the highest main-effect fraction (∼40%), while most parameters are interaction-dominated.

3.5 Parameter Group Contributions

Figure 5 summarizes sensitivity by parameter group.

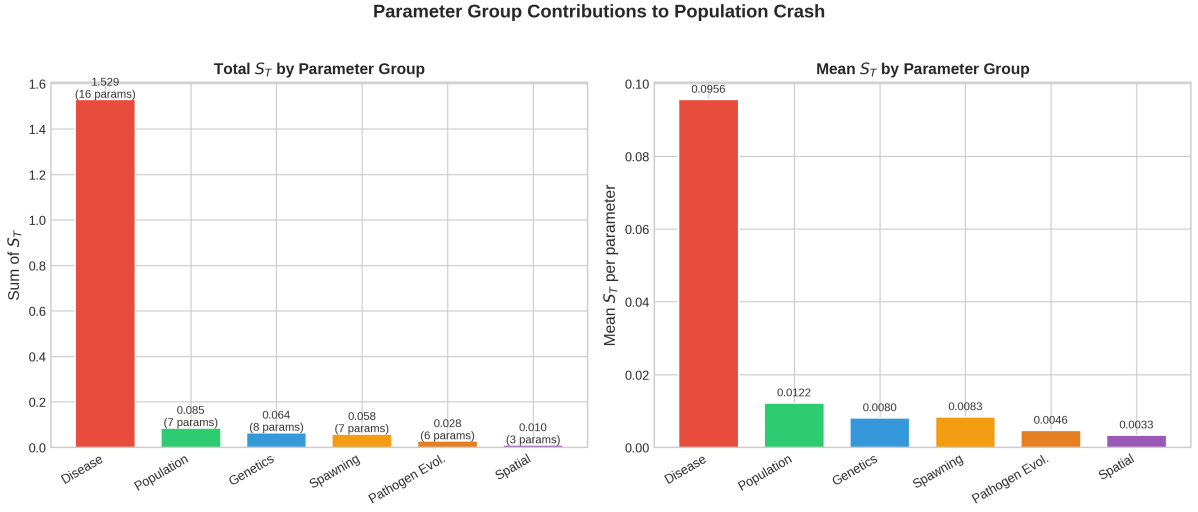

Figure 5: Left: Total S_T summed across all parameters in each group. Right: Mean S_T per parameter in each group. The disease module dominates both total and per-parameter sensitivity for `pop_crash_pct`.

The disease module contributes the most total S_T (16 parameters), but also has the highest per-parameter mean—confirming that disease transmission parameters are genuinely more influential, not just more numerous.

3.6 Evolutionary Metrics

Figure 6 shows the top 10 parameters for each evolutionary outcome metric.

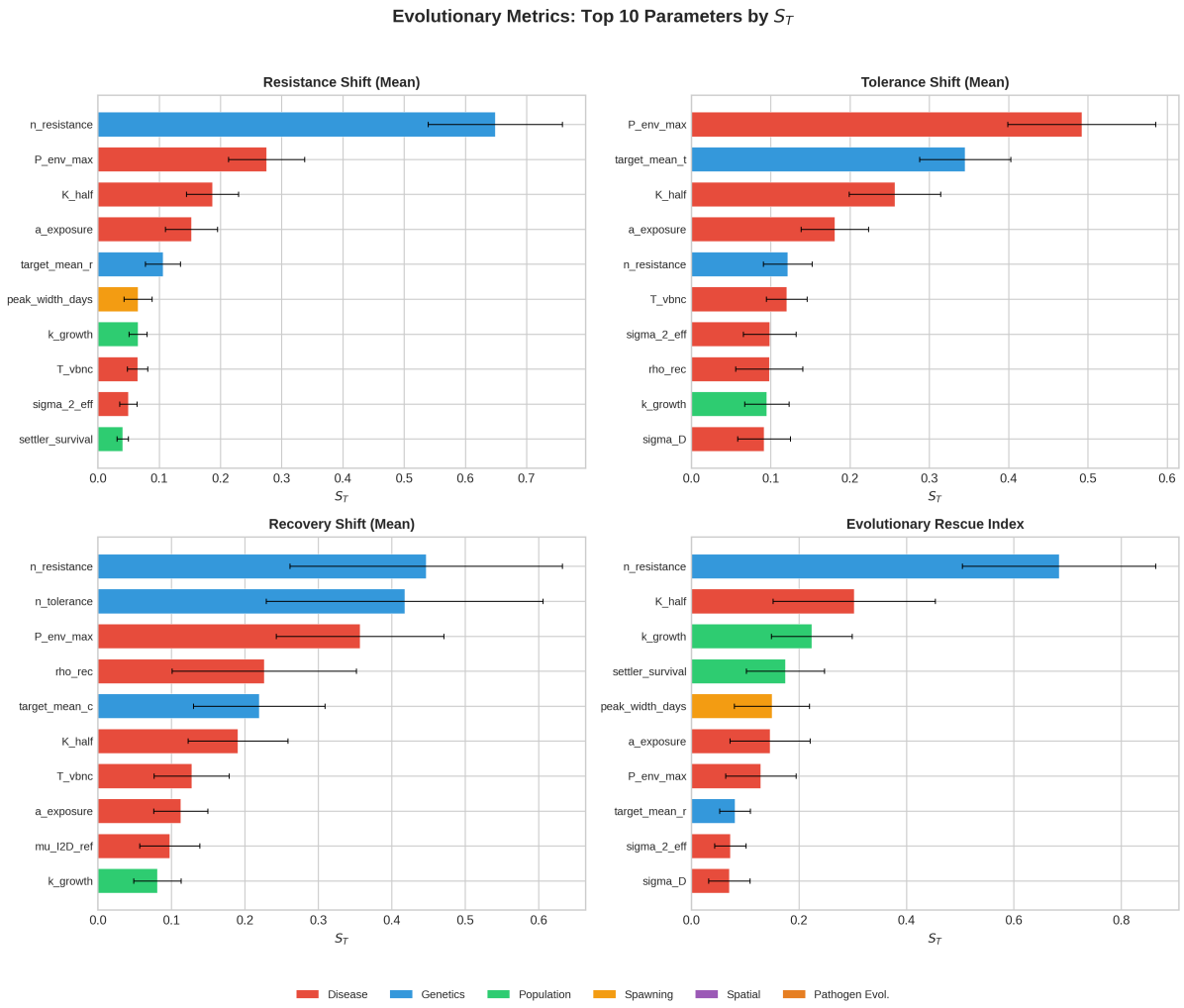

Figure 6: Top 10 parameters by S_T for evolutionary metrics: resistance shift, tolerance shift, recovery shift, and evolutionary rescue index. Genetic parameters (blue) dominate trait shift metrics, while disease parameters (red) control the selection pressure that drives evolution.

3.7 Disease Dynamics

Figure 7 shows disease-related outcome metrics.

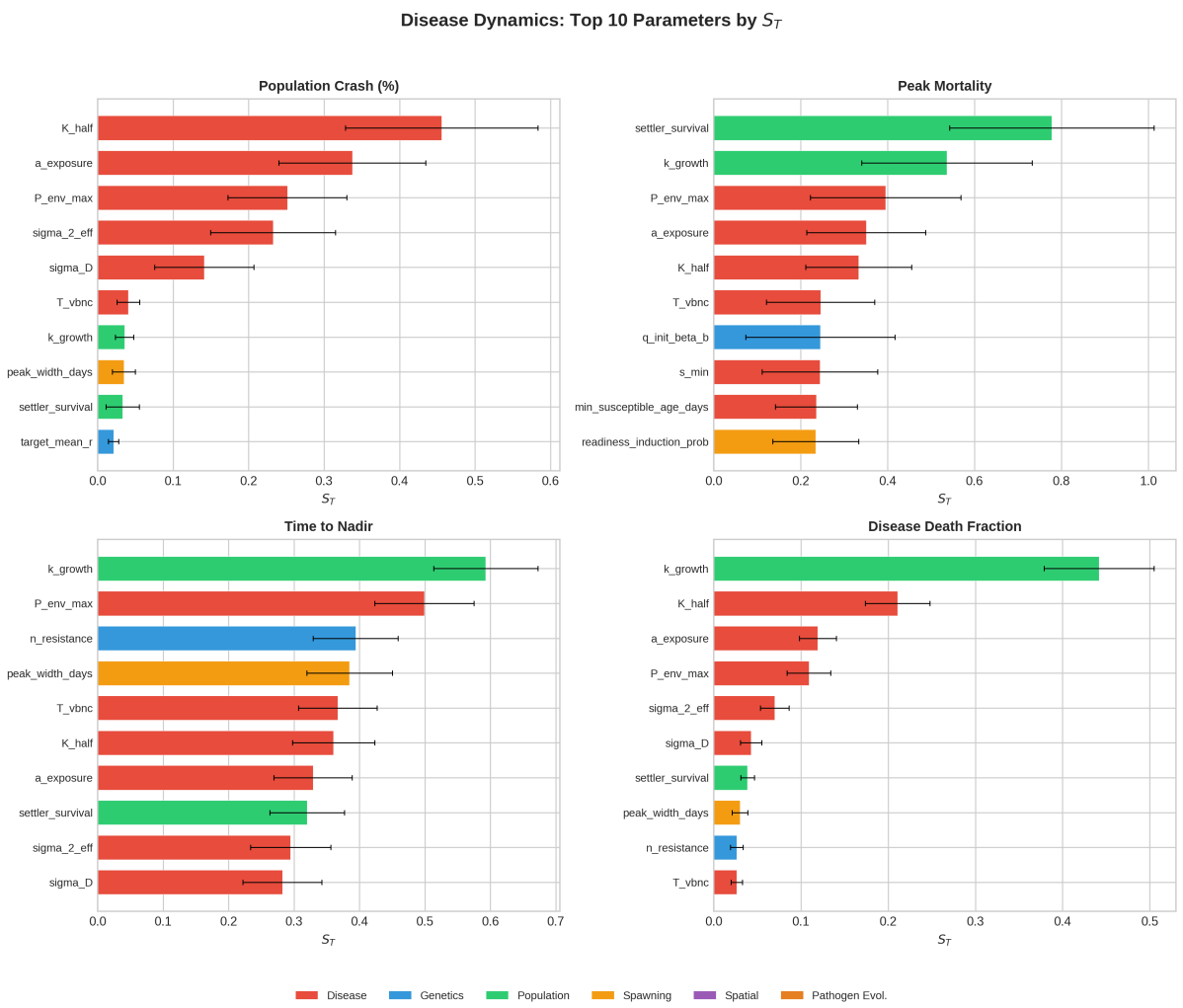

Figure 7: Top 10 parameters by S_T for disease dynamics metrics. The same disease transmission cluster dominates across all four panels, with consistent ranking.

3.8 Ecological Metrics

Figure 8 shows ecological outcome metrics.

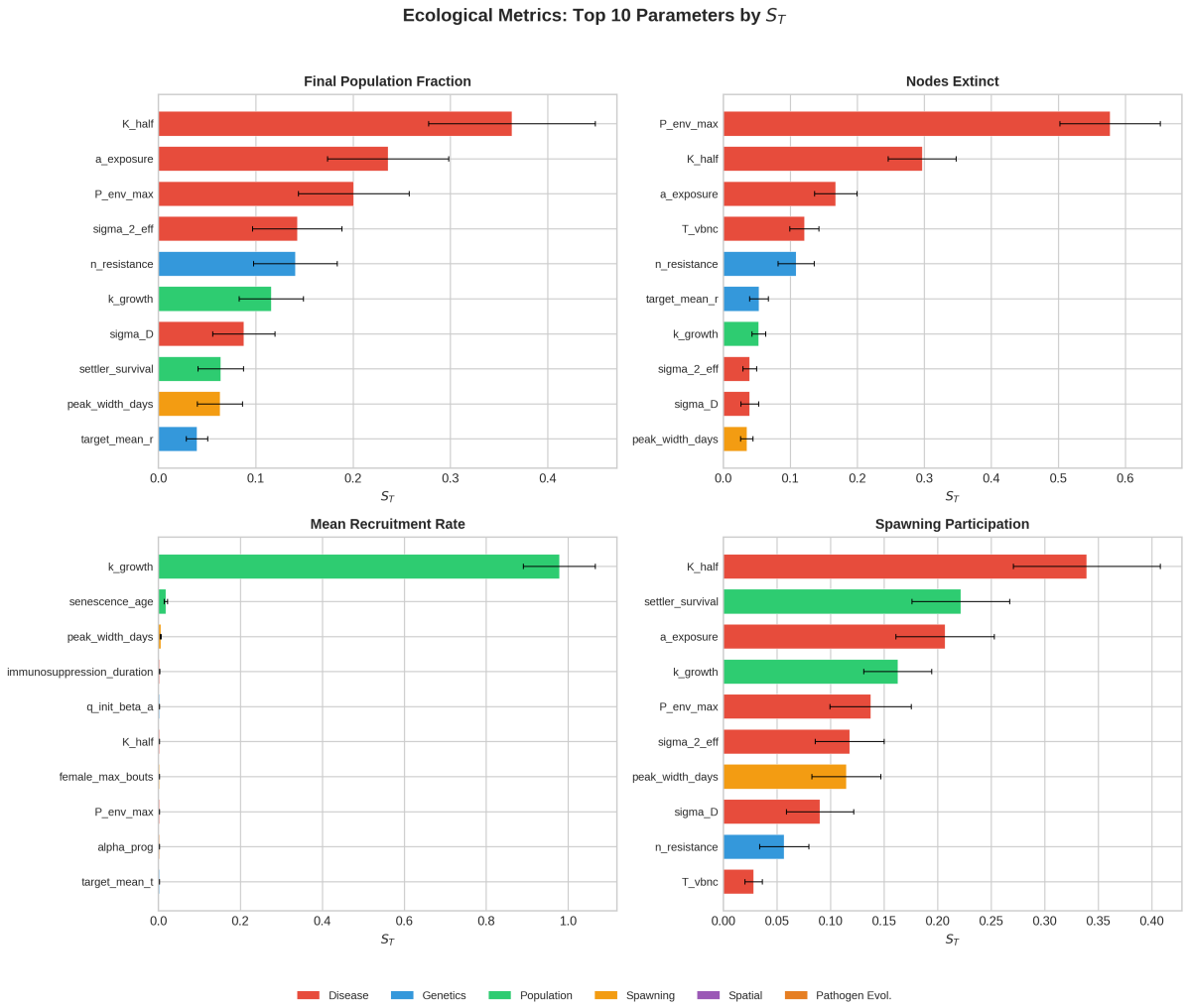

Figure 8: Top 10 parameters by S_T for ecological metrics. Population and spawning parameters become more prominent for recruitment and spawning participation, though disease parameters remain influential.

3.9 Spatial Metrics

Figure 9 shows the spatial outcome metrics.

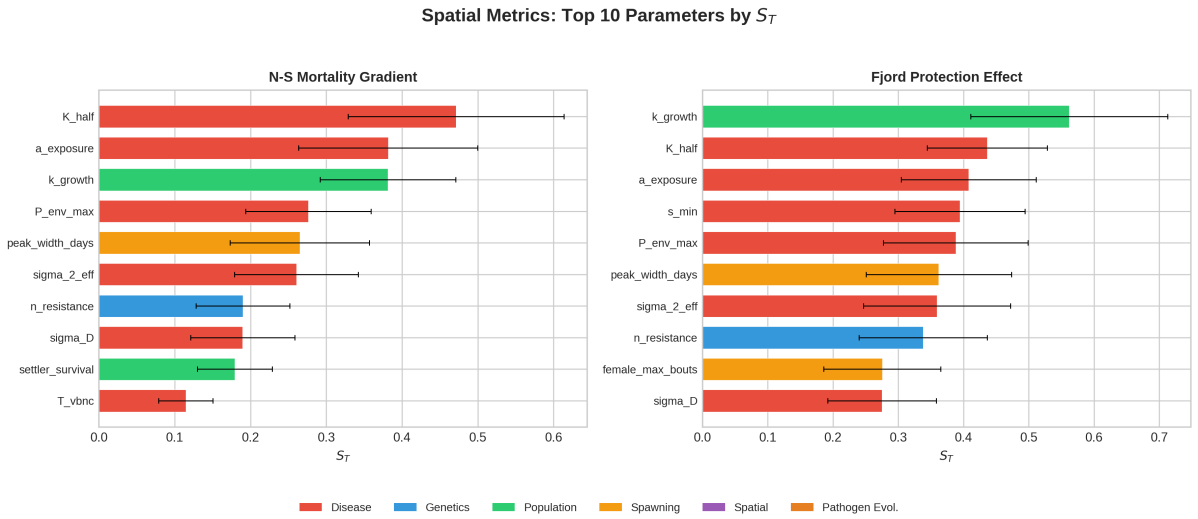

Figure 9: Top 10 parameters by S_T for spatial metrics: north-south mortality gradient and fjord protection effect. Spatial parameters (purple) appear in the top 10 for the first time, confirming the 11-node network resolves spatial dynamics.

3.10 Morris vs. Sobol Comparison

Figure 10 compares the parameter rankings from Morris screening (R4) with the Sobol analysis.

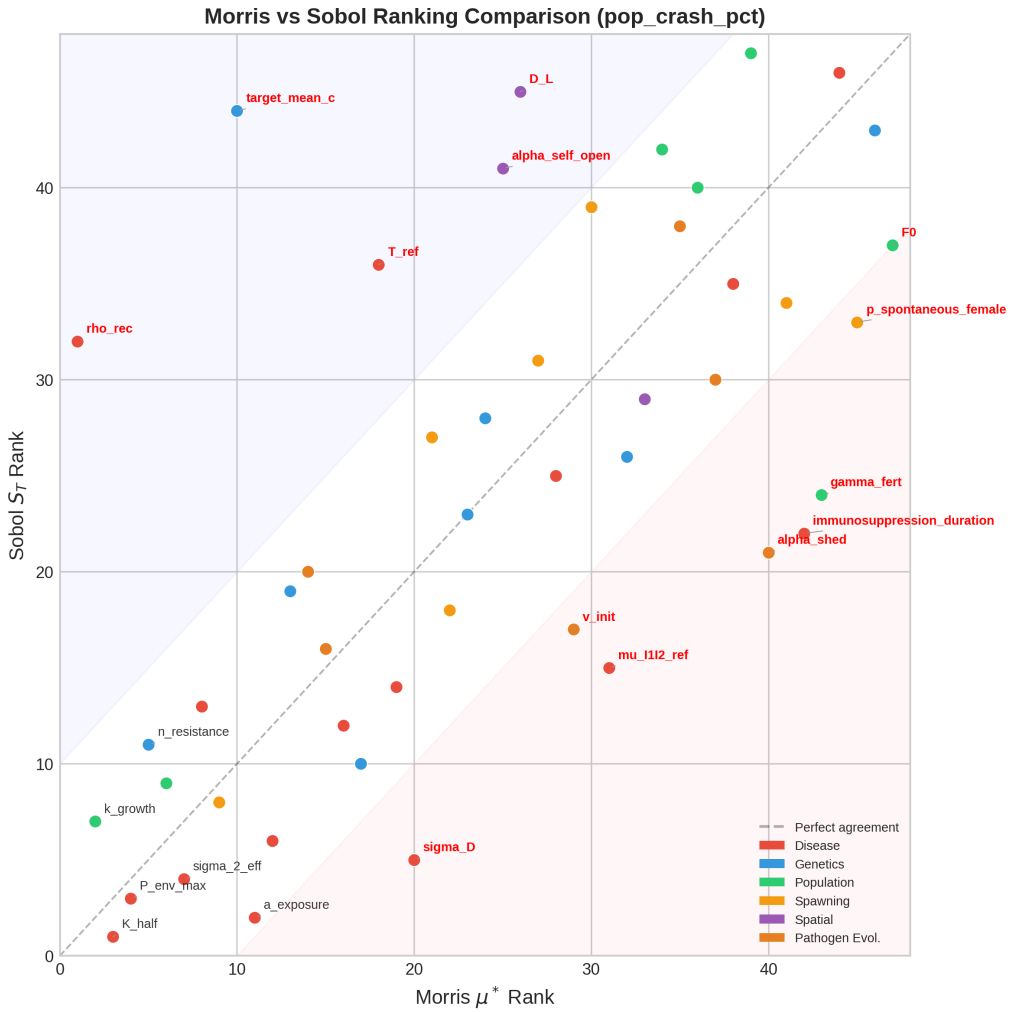

Figure 10: Morris μ^* rank vs. Sobol S_T rank for `pop_crash_pct`. Points on the diagonal indicate perfect agreement. Red-labeled parameters show rank changes ≥ 10 positions. Notable: `rho_rec` drops from Morris #1 to Sobol #32; `sigma_D` jumps from Morris #20 to Sobol #5.

Major rank discrepancies between Morris and Sobol:

- **`rho_rec`** (Morris #1 $\rightarrow$ Sobol #32): Morris's OAT perturbations captured strong local effects of recovery rate, but Sobol reveals these are absorbed by interactions with other parameters in the global variance decomposition.
- **`k_growth`** (Morris #2 $\rightarrow$ Sobol #7): Slight drop; population growth is important but less dominant than disease transmission in variance decomposition.
- **`sigma_D`** (Morris #20 $\rightarrow$ Sobol #5): Dead-animal shedding is underestimated by Morris because its effect is highly nonlinear and interaction-dependent—exactly what Sobol captures.
- **Top-3 agreement:** Both methods agree that `K_half` and `a_exposure` are among the most important parameters.

3.11 Confidence Intervals

Figure 11 shows the statistical precision of the Sobol estimates for the top 15 parameters.

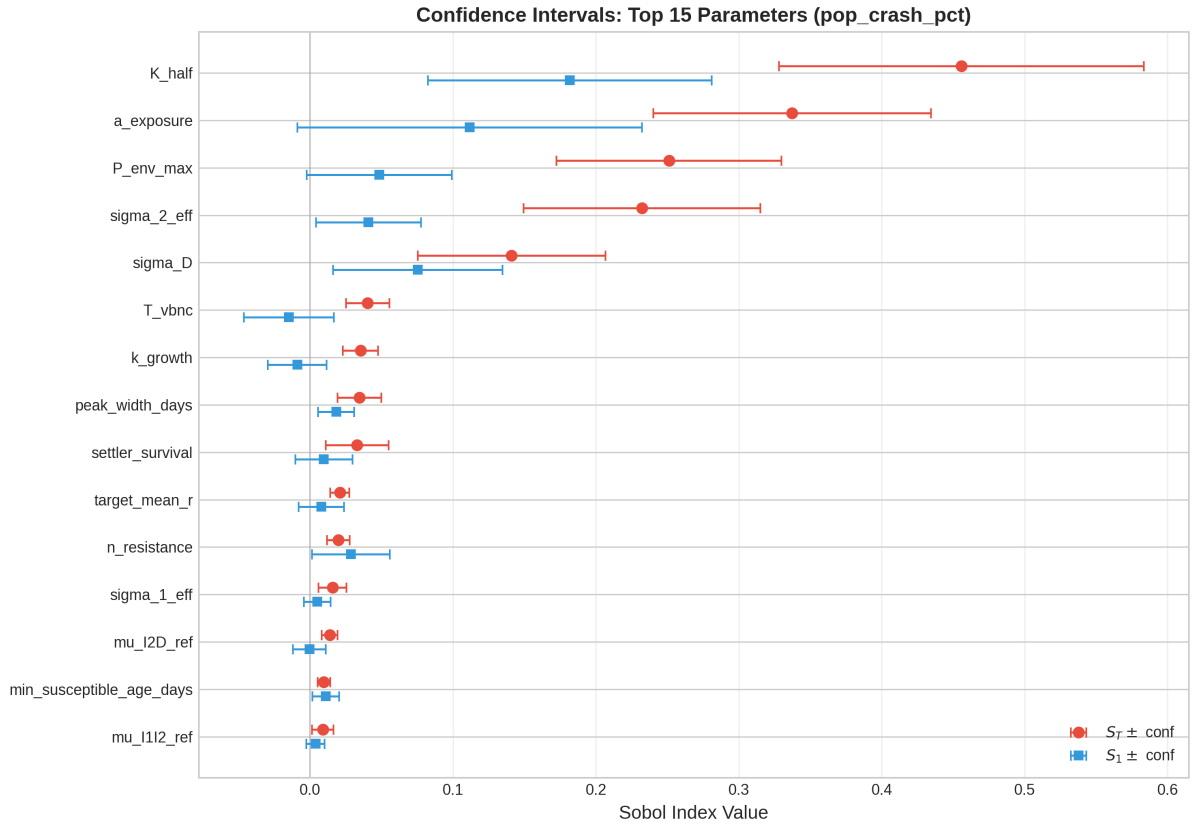

Figure 11: Sobol indices with bootstrap confidence intervals for the top 15 parameters (`pop_crash_pct`). Red circles: S_T; blue squares: S_1. The top-4 parameters have well-separated confidence intervals, confirming their ranking is robust. Parameters ranked 6+ have overlapping intervals, making their relative ordering uncertain.

The confidence intervals reveal:

- The top-4 parameters (`K_half`, `a_exposure`, `P_env_max`, `sigma_2_eff`) are clearly separated from the rest—their ranking is statistically robust.
- Parameters ranked 5–9 have overlapping confidence intervals, forming a "second tier" whose internal ordering is uncertain.
- Some S_1 values are slightly negative (within confidence of zero), a known artifact of Monte Carlo estimation at limited sample sizes.

3.12 Calibration Priority Matrix

Figure 12 maps each parameter's sensitivity importance against its current prior range width.

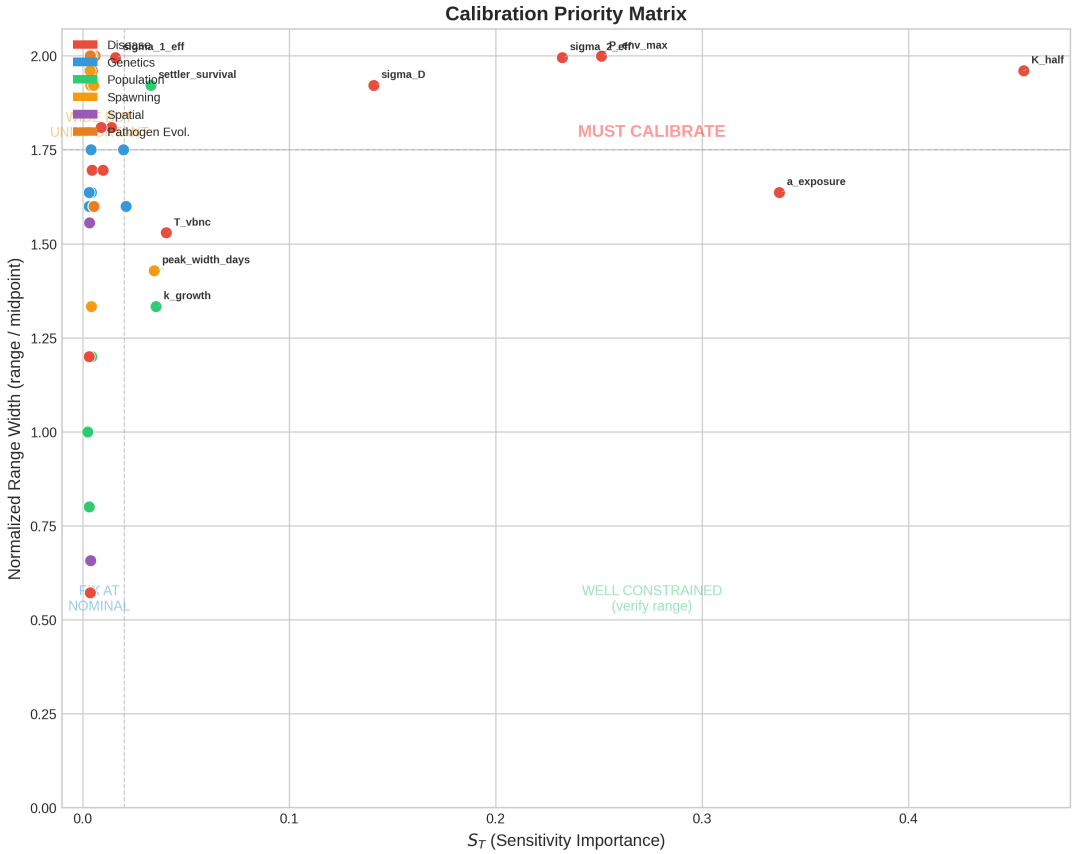

Figure 12: Calibration priority matrix. X-axis: S_T (importance); Y-axis: normalized range width. Upper-right quadrant = high importance with wide range ("MUST CALIBRATE"). Lower-right = high importance with narrow range ("WELL CONSTRAINED"). Lower-left = low importance with narrow range ("FIX AT NOMINAL").

4 Discussion

4.1 Parameter Importance Hierarchy

The Sobol analysis reveals a steep importance hierarchy for population crash outcomes. Four disease transmission parameters collectively explain the vast majority of variance:

1. **Dose-response parameters** (`K_half`, `a_exposure`): These define the shape of the infection probability curve as a function of environmental pathogen exposure. Together they account for $S_T \approx 0.79$, meaning population crash is primarily controlled by *how efficiently pathogen converts to infection*.

2. **Pathogen loading parameters** (`P_env_max`, `sigma_2_eff`): These control the environmental pathogen pool. `P_env_max` sets the ceiling; `sigma_2_eff` controls the dominant input from severely infected animals.

3. **Post-mortem shedding** (`sigma_D`): A surprise entry at #5 that Morris missed. Dead animals continue releasing pathogen, creating a positive feedback loop that Sobol detects through its global variance decomposition.

Below the top 5, a "second tier" of 4 parameters ($S_T \approx 0.03$–0.04) includes demographic (`k_growth`, `settler_survival`), spawning (`peak_width_days`), and reservoir (`T_vbnc`) parameters. These modulate the population's ability to absorb and recover from disease-induced mortality.

4.2 Interaction Structure

The ratio $\sum S_T / \sum S_1$ quantifies the overall interaction intensity:

Metric	$\sum S_1$	$\sum S_T$
`pop_crash_pct`	0.527	1.775
`final_pop_frac`	0.429	1.856
`resistance_shift_mean`	1.034	2.653
`peak_mortality`	1.232	10.019

`peak_mortality` shows extreme interactions ($\sum S_T = 10.0$), meaning this metric is determined almost entirely by parameter combinations rather than individual parameters. This makes it a poor target for univariate calibration but an excellent diagnostic for detecting model structural issues.

4.3 Disease Transmission Cluster

The four core disease parameters (`K_half`, `a_exposure`, `P_env_max`, `sigma_2_eff`) form a tightly coupled transmission cluster. Their interaction structure suggests:

- `K_half` and `a_exposure` jointly define the dose-response curve: changing one shifts the effective threshold, and the other controls the steepness. Their interaction is mechanistically inevitable.

- `P_env_max` interacts with the dose-response pair because it sets the maximum exposure level—if the ceiling is low, the dose-response shape matters less.

- `sigma_2_eff` creates a feedback loop: more shedding → more environmental pathogen → more infection → more shedding. This amplification means its effect depends on the dose-response parameters.

Implication: These four parameters cannot be calibrated independently. ABC-SMC should sample them jointly and use summary statistics that constrain their combined effect (e.g., disease prevalence time series, environmental pathogen concentrations).

4.4 Genetics Cluster

For evolutionary metrics, genetic parameters dominate:

- `n_resistance` is the primary determinant of resistance evolution speed (controls genetic variance).
- `target_mean_r` sets the initial resistance level, determining how far the population must evolve.
- For tolerance and recovery shifts, the corresponding trait-specific parameters (`target_mean_t`, `target_mean_c`, `tau_max`) are most influential.

The genetics cluster has *low* influence on population crash ($S_T < 0.03$ for all genetic parameters) but *high* influence on trait dynamics. This separation suggests that calibrating evolutionary parameters can proceed semi-independently from disease transmission calibration.

4.5 Parameters That Don't Matter

The bottom quartile (ranks 35–47) includes:

- `alpha_self_open`, `senescence_age`, `q_init_beta_b`, `target_mean_c`, `D_L`, `susceptibility_multiplie` `L_min_repro` (all $S_T < 0.004$)

These parameters contribute negligibly to output variance and can be safely fixed at their nominal (literature) values during calibration. Notably, `susceptibility_multiplier`—which was the #1 parameter in Round 1 Sobol—is now ranked #46. This dramatic decline occurred because explicit genetic resistance mechanics (`n_resistance`, `target_mean_r`) now capture the biology that the multiplier previously approximated.

4.6 Comparison with Morris R4

The Morris–Sobol rank correlation is moderate, with several instructive discrepancies:

- **`rho_rec` collapse** (Morris #1 → Sobol #32): Morris's one-at-a-time perturbations revealed that changing recovery rate dramatically affects outcomes. However, Sobol shows this effect is almost entirely interaction-dependent—`rho_rec`'s *direct* contribution to variance is negligible. Its effect is mediated through the disease transmission cluster.
- **`sigma_D` rise** (Morris #20 → Sobol #5): Post-mortem shedding creates a nonlinear feedback that Morris's linear derivative approximation underestimates. Sobol's global decomposition captures this correctly.
- **Core agreement:** Both methods identify `K_half`, `a_exposure`, `P_env_max`, and `sigma_2_eff` as top-tier parameters, validating the screening approach for identifying the parameter "neighborhood" that matters.

Lesson: Morris screening is effective for identifying the top ∼10 parameters but unreliable for precise ranking. For calibration prioritization, Sobol indices are essential.

5 Calibration Recommendations

Based on the Sobol analysis, we recommend a three-tier calibration strategy:

5.1 Priority 1: Must Calibrate

These parameters have high S_T and wide prior ranges. Inaccurate values will propagate large errors into model predictions.

Parameter	Module	S_T	Rationale
`K_half`	Disease	0.456	Dose-response threshold; most influential
`a_exposure`	Disease	0.337	Transmission nonlinearity exponent
`P_env_max`	Disease	0.251	Environmental reservoir ceiling
`sigma_2_eff`	Disease	0.232	Late-stage shedding rate
`sigma_D`	Disease	0.141	Dead-animal shedding feedback

5.2 Priority 2: Should Calibrate

These parameters have moderate S_T and contribute to secondary outcomes (recovery, recruitment, spatial patterns).

Parameter	Module	S_T	Rationale
`T_vbnc`	Disease	0.040	Pathogen persistence temperature
`k_growth`	Population	0.035	Body growth rate; recovery capacity
`peak_width_days`	Spawning	0.035	Spawning window width
`settler_survival`	Population	0.033	Recruitment success; recovery speed
`target_mean_r`	Genetics	0.021	Initial resistance level
`n_resistance`	Genetics	0.020	Genetic architecture (variance)
`sigma_1_eff`	Disease	0.016	Early-stage shedding rate
`mu_I2D_ref`	Disease	0.014	Stage $I_2 \rightarrow$ Death rate
`min_susceptible_age_days`	Disease	0.010	Age susceptibility threshold
`mu_I1I2_ref`	Disease	0.009	Stage $I_1 \rightarrow I_2$ rate

5.3 Priority 3: Fix at Literature Values

These 32 parameters (ranks 16–47 by S_T) have $S_T < 0.006$ for `pop_crash_pct` and can be fixed at their nominal literature values during calibration. This reduces the calibration problem from 47 to 15 dimensions.

Notable parameters safe to fix:

- All pathogen evolution parameters (α_{kill}, α_{shed}, α_{prog}, γ_{early}, $\sigma_{v,\text{mut}}$, v_{init}): collectively $S_T < 0.006$ each
- All spatial parameters (`D_L`, $\alpha_{\text{self,fjord}}$, $\alpha_{\text{self,open}}$): $S_T < 0.004$ each
- `rho_rec`: Despite Morris ranking it #1, Sobol shows $S_T = 0.004$
- `susceptibility_multiplier`: Former #1 in R1, now superseded by explicit genetics

5.4 Recommended ABC-SMC Target Statistics

For calibrating the Priority 1–2 parameters, we recommend the following summary statistics:

1. **Population crash magnitude** (`pop_crash_pct`): Primary target; most sensitive to the calibration parameters.
2. **Time to population nadir** (`time_to_nadir`): Constrains disease progression speed.
3. **Disease death fraction** (`disease_death_fraction`): Separates disease mortality from demographic effects.
4. **Final population fraction** (`final_pop_frac`): Constrains long-term recovery.
5. **North-south mortality gradient**: Constrains spatial parameters and temperature dependence.
6. **Resistance shift** (`resistance_shift_mean`): Constrains genetic architecture if evolutionary data become available.

Appendix

A. Full Parameter Table: `pop_crash_pct`

Rank	Parameter	S_1	S_1 conf	S_T	S_T conf
1	K_half	0.1819	0.0993	0.4558	0.1276
2	a_exposure	0.1118	0.1205	0.3373	0.0972
3	P_env_max	0.0485	0.0508	0.2512	0.0788
4	sigma_2_eff	0.0410	0.0368	0.2323	0.0829
5	sigma_D	0.0754	0.0593	0.1410	0.0657
6	T_vbnc	−0.0147	0.0316	0.0404	0.0151
7	k_growth	−0.0089	0.0205	0.0354	0.0123
8	peak_width_days	0.0184	0.0125	0.0345	0.0154
9	settler_survival	0.0097	0.0200	0.0330	0.0220
10	target_mean_r	0.0079	0.0158	0.0209	0.0067
11	n_resistance	0.0286	0.0271	0.0197	0.0079
12	sigma_1_eff	0.0051	0.0095	0.0159	0.0098
13	mu_I2D_ref	−0.0003	0.0115	0.0138	0.0056
14	min_susceptible_age_days	0.0111	0.0094	0.0098	0.0044
15	mu_I1I2_ref	0.0038	0.0064	0.0089	0.0075
16	alpha_kill	−0.0034	0.0063	0.0058	0.0034
17	v_init	−0.0014	0.0069	0.0054	0.0040
18	readiness_induction_prob	0.0024	0.0052	0.0052	0.0034
19	tau_max	0.0021	0.0054	0.0052	0.0029
20	sigma_v_mutation	−0.0047	0.0078	0.0046	0.0017
21	alpha_shed	−0.0021	0.0065	0.0045	0.0021
22	immunosuppression_dur.	0.0036	0.0051	0.0045	0.0024
23	target_mean_t	0.0046	0.0058	0.0043	0.0021
24	gamma_fert	0.0012	0.0052	0.0042	0.0018
25	s_min	0.0015	0.0059	0.0042	0.0015
26	q_init_beta_a	0.0003	0.0064	0.0042	0.0019
27	female_max_bouts	0.0008	0.0082	0.0041	0.0015
28	n_tolerance	−0.0021	0.0065	0.0040	0.0016
29	alpha_self_fjord	0.0030	0.0054	0.0038	0.0016
30	alpha_prog	0.0012	0.0066	0.0037	0.0014
31	ind. male_to_female	0.0020	0.0056	0.0037	0.0015
32	rho_rec	−0.0015	0.0048	0.0037	0.0013
33	p_spont. female	−0.0014	0.0059	0.0036	0.0016
34	ind. female_to_male	−0.0026	0.0057	0.0036	0.0015
35	mu_EI1_ref	−0.0027	0.0066	0.0036	0.0014
36	T_ref	−0.0009	0.0064	0.0036	0.0015
37	F0	0.0045	0.0048	0.0035	0.0014
38	gamma_early	−0.0009	0.0055	0.0035	0.0015
39	p_spont. male	0.0032	0.0048	0.0035	0.0014
40	alpha_srs	−0.0015	0.0058	0.0035	0.0013
41	alpha_self_open	0.0012	0.0063	0.0032	0.0019
42	senescence_age	0.0022	0.0060	0.0031	0.0014
43	q_init_beta_b	0.0006	0.0040	0.0031	0.0011
44	target_mean_c	−0.0001	0.0058	0.0031	0.0011
45	D_L	0.0004	0.0044	0.0030	0.0014
46	suscept. multiplier	0.0015	0.0051	0.0030	0.0011
47	L_min_repro	−0.0038	0.0060	0.0024	0.0009

B. Parameter Ranges

Module	Parameter	Lower	Upper	Scale
Disease	a_exposure	0.5	5.0	linear
Disease	K_half	50	5000	log

Module	Parameter	Lower	Upper	Scale
Disease	sigma_1_eff	10^4	10^7	log
Disease	sigma_2_eff	10^5	10^8	log
Disease	sigma_D	0.01	0.5	linear
Disease	rho_rec	0.001	0.05	log
Disease	mu_EI1_ref	0.05	0.5	linear
Disease	mu_I2D_ref	0.005	0.1	log
Disease	P_env_max	10^3	10^7	log
Disease	T_ref	10.0	18.0	linear
Disease	susceptibility_mult.	0.5	2.0	linear
Disease	T_vbnc	2.0	15.0	linear
Disease	s_min	0.01	0.5	linear
Disease	mu_I1I2_ref	0.01	0.2	linear
Disease	immunosupp. dur.	30	365	linear
Disease	min_suscept. age	30	365	linear
Population	F0	10^4	10^6	log
Population	gamma_fert	0.5	2.0	linear
Population	settler_survival	0.001	0.05	log
Population	alpha_srs	0.0001	0.01	log
Population	senescence_age	15	35	linear
Population	k_growth	0.1	0.5	linear
Population	L_min_repro	10	30	linear
Genetics	n_resistance	2	30	linear
Genetics	n_tolerance	2	30	linear
Genetics	target_mean_t	0.1	0.9	linear
Genetics	target_mean_c	0.1	0.9	linear
Genetics	tau_max	0.1	0.9	linear
Genetics	target_mean_r	0.1	0.9	linear
Genetics	q_init_beta_a	0.5	5.0	linear
Genetics	q_init_beta_b	0.5	5.0	linear
Spawning	p_spont. female	0.001	0.1	log
Spawning	ind. female_to_male	0.01	0.5	linear
Spawning	ind. male_to_female	0.01	0.5	linear
Spawning	p_spont. male	0.001	0.1	log
Spawning	peak_width_days	10	60	linear
Spawning	readiness_ind. prob	0.01	0.5	linear
Spawning	female_max_bouts	1	5	linear
Spatial	D_L	1.0	100.0	log
Spatial	alpha_self_fjord	0.5	0.99	linear
Spatial	alpha_self_open	0.1	0.8	linear
Path. Evol.	alpha_kill	0.0	1.0	linear
Path. Evol.	alpha_shed	0.0	1.0	linear
Path. Evol.	alpha_prog	0.0	1.0	linear
Path. Evol.	gamma_early	0.0	0.5	linear
Path. Evol.	sigma_v_mutation	0.001	0.1	log
Path. Evol.	v_init	0.1	0.9	linear