

Trait-Based Specific Protection Goals for Wild Pollinators: A Staged Modelling Framework for Pesticide Risk Assessment

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Abstract

Background

Wild pollinator populations in the European Union lack defined, quantitative Specific Protection Goals (SPGs) for pesticide risk assessment. Without an agreed threshold, lower-tier toxicity data cannot be evaluated for acceptability, nor can higher-tier study results be assessed against a protection goal, leaving the tiered ERA process at an impasse. The existing honey bee surrogate compounds the problem: this was selected for laboratory convenience rather than functional representativeness, and their managed, colonial life history cannot reliably predict outcomes for the diverse life-history strategies of wild pollinators.

Results

This paper presents a pragmatic, staged framework combining trait-based species selection with demographic toxicity assessment. It focuses on Stage 1 (non-spatial) of a the three-stage process, where life-stage-structured population models are implementable now (2026–2027), uses existing data and tools, and generates quantitative, population-level SPG thresholds within the current [1] tiered assessment framework. The framework combines two methodological advances: trait-based species selection identifies functionally vulnerable species by grouping taxa according to exposure- and sensitivity-related traits and population resilience; and demographic toxicity assessment translates individual-level pesticide effects into population-level outcomes through life-stage-structured projection matrices. Parameter space exploration demonstrates that Stage 1 yields defined, quantitative thresholds: a maximum acceptable population decline and a maximum acceptable exceedance risk probability.

Conclusions

Stage 1 resolves the immediate operational constraint by providing provisional, population-level thresholds for regulatory use. A roadmap for adaptive implementation and progressive refinement is provided: Stage 2 (operational 2028–2029) incorporates landscape-scale spatial dynamics, and Stage 3 (operational 2030+) implements full agent-based modelling using the ALMaSS system. The approach is demonstrated for bees (bumble bees and solitary bees), but the trait-based framework is designed to be flexible enough to generalise across hoverflies, butterflies, moths, and other pollinating taxa.

1. Introduction

1.1 The Regulatory Impasse and Objectives

Protecting wild pollinators from pesticide exposure poses a significant challenge in contemporary Environmental Risk Assessment (ERA). The European Food Safety Authority (EFSA) published a revised bee guidance in 2023 [1]. Risk managers from EU Member States suggested a Specific Protection Goal (SPG) of a 10% maximum colony size reduction threshold for honey bees to result from the application of a pesticide under field conditions. This SPG emerged from a process involving the European Commission, Member States and stakeholders, where EFSA supported by exploring the consequences of selecting different magnitudes for the SPG [1–3], including the 7% proposed to represent negligible effects in the 2013 version of the bee guidance [4]. The 10% thresholds suggested by risk managers weighed statistical detectability, field study practicality, and economic considerations [2]. For other bee species (bumblebees and solitary bees), however, Member States suggested an “undefined” SPG threshold following consultations by the European Commission in 2022 [5]. Whilst a majority of Member States supported the eventual establishment of defined SPGs [6], the lack of data on population-related attributes for wild bees, i.e. colony strength and population abundance [7], prevented the setting of a defined value at this point in time. As a consequence, a tiered ERA approach for wild bees could not be established, as it depends on a defined SPG that sets trigger values indicating when tests must be escalated to a higher tier. Therefore, the regulatory default requires higher-tier studies (cage, semi-field, and/or field studies) for nearly all pesticide applications where wild bee exposure cannot be excluded, unless lower-tier assessments for honey bees and non-target arthropods show no unacceptable effects, or laboratory studies demonstrate very low toxicity ($LD_{50} > 100 \mu\text{g}/\text{bumble bee}$). If the acceptability of the effects is determined on the basis of SPGs that are defined for honey bees or non-target arthropods, this could entail a risk that the ERA is not protective to wild bee species that may be more sensitive to the pesticide in question and/or require a more conservative SPG due to their specific population dynamics [cf. 8].

This approach, founded on an absence of defined SPGs, presents several compounding problems. The higher-tier-by-default requirement creates assessment bottlenecks as the volume of active substances to be evaluated grows. Field studies cannot efficiently address population-level resilience across diverse landscapes and multiple stressor combinations, and without a defined threshold, their results cannot be evaluated against any agreed protection goal. The approach also conflates hazard with risk by using toxicity-based triggers that do not account for exposure.

To address this impasse, we propose a staged framework integrating trait-based species selection with demographic population modelling. The three stages differ in ecological realism, spatial complexity, and the infrastructure required to implement them:

- **Stage 1 (non-spatial, implementable now):** Life-stage-structured Lefkovitch population models parameterised with existing data and tools. Stage 1 defines operational SPG thresholds through parameter space exploration and integrates directly with the [1] tiered assessment framework without requiring new regulatory infrastructure. Stage 1 can be deployed within 12–18 months and is the primary focus of this paper.

- **Stage 2 (landscape-explicit, operational 2028–2029):** Population models incorporating spatial structure, dispersal, and habitat heterogeneity, integrated with EFSA Tier 2 exposure assessment. Stage 2 reduces the conservatism of Stage 1 by replacing worst-case homogeneous exposure assumptions with realistic landscape-scale scenarios.
- **Stage 3 (agent-based systems models, operational 2030+):** Full individual-based simulation using the ALMaSS framework, incorporating individual-level behaviours, multiple stressors, and community-level interactions for complex assessment scenarios, fully including density-dependent factors.

Stage 1 resolves the immediate regulatory constraint. Stages 2 and 3 provide a pathway towards progressively greater ecological realism, with each stage building on validated outputs from the previous one.

Defining quantitative SPG thresholds for individual pesticide ERAs is a necessary first step: it allows registration dossiers to be evaluated against an agreed population-level standard and enables the tiered assessment process to function as intended. The broader question of how those thresholds should be calibrated and coordinated across policy sectors to meet landscape- and societal-scale biodiversity obligations (including those under the Nature Restoration Regulation ((EU) 2024/1991, NRR) and the Pesticide Regulation (EC 1107/2009) is an important downstream challenge that this framework is designed to support, and is addressed in Section 4.6.

The following sections demonstrate why current approaches fail (Sections 1.2–1.3) and how demographic toxicity and trait-based approaches resolve these failures (Section 1.3).

1.2 Why Current Risk Assessment Approaches Fail for Wild Pollinators

Current SPGs for terrestrial arthropods in the EU focus on short-term, statistically detectable effects. These thresholds were established primarily on the basis of what could be measured in field trials rather than ecological relevance [1]. As Stark [9] articulates in his comprehensive critique of current pesticide risk assessment, this individual-level, short-term approach suffers from fundamental conceptual problems when applied to population persistence.

The core problem is a disconnect between individual-level effects (colony-level effects in case of social insects like honey bees and bumblebees) and population outcomes. Stark [9] documents through experimental studies both catastrophic underestimation of risk (populations declining at concentrations 60-fold below protective thresholds) and over-protection by factors of six or more. Several mechanisms explain this unpredictability.

Multiple toxic effects and population structure. Pesticides cause both mortality and sublethal effects (impaired foraging, navigation, learning, reproduction, and communication or immunosuppression), yet current mortality-focused approaches fail to capture this total effect [10, 11]. Simultaneously, population

structure significantly influences susceptibility [12]: for univoltine solitary bees, timing of exposure relative to life stage is a critical determinant of outcome, because larval exposure to contaminated pollen provisions can cascade across development to dramatically reduce adult emergence and subsequently depress population growth in the following season [13–15].

Life history and compensatory dynamics. Species with different life histories recover at vastly different rates from the same proportional mortality [e.g. 8], yet current risk assessment treats all species identically after applying generic uncertainty factors [9]. Conversely, thinned populations may compensate through density-dependent mechanisms [16], producing non-linear responses that individual-level assessments cannot capture. Experimental studies confirm that even closely related species exposed to identical stress levels recover at very different rates [17].

The surrogate species problem. Honey bees (**Apis mellifera*) are mainly managed (although wild honey bees can also be found in nature), live in large perennial colonies (20,000–80,000 individuals), and, when managed, benefit from beekeeper care. Wild pollinators exhibit fundamentally different life histories: many are solitary or form small annual colonies, are univoltine with no within-season recovery opportunity, nest in soil or cavities, and may be floral specialists [14, 18]. Others are multivoltine or nest in leaves or plant fibres. These differences influence exposure routes, population resilience, and recovery dynamics following pesticide stress [8, 19]. In addition, substantial interspecific variation in pesticide sensitivity has been documented among bee species, indicating that taxonomic relatedness alone does not reliably predict surrogate suitability [20]. The conceptual mismatch is fundamental, and not simply a matter of insufficient data or statistical power. Population-level assessment for wild pollinators is a basic reframing, not an optional refinement.

1.3 Methodological Solutions: Demographic Toxicity and Trait-Based Approaches

To address the aforementioned issues, two complementary methodological advancements are necessary. The concept of demographic toxicity [9] provides a pragmatic and implementable approach to help bridge the gap between individual and population levels by examining how factors such as age, sex, genetics, and environment influence chemical responses. This approach goes beyond the basic dose-response model to assess risks for specific population groups. Importantly, integrating demographic toxicity data with population modelling enables predictions of when and whether populations will recover from pesticide exposure, as well as the likelihood of extinction, which is the ultimate protection goal.

Demographic toxicity principles alone cannot, however, address the surrogate species problem. Galic *et al.* [21] demonstrate that trait-based approaches can help solve this by identifying functionally vulnerable species based on life-history characteristics rather than taxonomic convenience, enabling systematic selection of representative species spanning trait space, with model parameterisation using trait databases rather than exhaustive species-specific testing. Similar arguments have been made for the systematic definition of environmental scenarios for representative aquatic species [22].

The integration of these two approaches is timely. EFSA's 2023 bee guidance establishes a 10% maximum colony size reduction threshold for honey bees, reached through risk manager decision rather than derived from colony modelling [1]. That even this threshold for the best-studied pollinator lacks a rigorous ecological foundation reinforces the need for the framework proposed here. Williams *et al.* [23] advocate population-level assessment for wild pollinators, whilst Nicholson *et al.* [24] provide landscape-scale empirical evidence supporting this approach. Our framework operationalises this regulatory momentum by combining demographic toxicity assessment with trait-based species selection to provide readily implementable SPG thresholds for wild pollinators.

2. Methods

2.1 Proposed Specific Protection Goal Framework

We propose an SPG framework consisting of two complementary dimensions that together ensure population resilience.

Magnitude Dimension: Acceptable Population Decline. Maximum acceptable reduction in local population abundance over a defined assessment period relative to untreated baseline. This is a population-level threshold framed in terms of proportional decline that provides a direct link between individual-level toxicity data and demographic consequences. The specific threshold value is not fixed here; it is determined through exploration of model behaviour across representative life-history configurations (Section 2.4) and established through consultation with risk managers, following the precedent suggested for honey bees related to the EFSA bee guidance [1].

Persistence Dimension: Exceedance Risk. The maximum acceptable probability that a stochastically projected exposed population falls below the protective decline threshold, relative to the deterministic control trajectory, in the evaluation year. This dimension quantifies the likelihood that population recovery is not achieved under demographic stochasticity, thereby complementing the deterministic magnitude criterion with a probabilistic measure of persistence risk. A 10-year assessment timeframe is proposed as a starting point, spanning multiple generations for focal species whilst aligning with policy cycles; in practice, for Stage 1 models with a growth rate $\lambda > 1$, the primary evaluation year is year 3 (see Section 2.4.2).

An optional service linkage dimension may be specified for pollination-dependent crops: maximum acceptable reduction in pollination service delivery, measured through crop-specific indicators such as seed set, fruit yield, quality, or yield stability. This dimension should remain supplementary rather than primary, because pollination-service delivery depends not only on pollinator abundance but also on crop dependence, landscape context, managed pollinators, weather, and farm-management responses. Nevertheless, making this linkage explicit would allow population-level SPGs to be connected in future work to bioeconomic farm models, cost-effectiveness analysis of restoration measures, and estimates of the marginal or shadow value of pollinator populations.

Recovery is implicit in the persistence dimension: populations facing recurring annual pesticide exposure must recover between exposure events to persist, so explicit recovery criteria are redundant. Stage 1 models assume local populations without immigration, appropriate for fragmented landscapes; Stages 2 and 3 incorporate spatial dynamics for refined assessments.

The methods integrate three core components:

1. trait-based species selection (addressing the surrogate species problem),
2. demographic toxicity assessment (capturing total population-level effects), and
3. population modelling (translating laboratory testing-derived individual-level effects into SPG-relevant outcomes).

Sections 2.2–2.4 address each in turn.

2.2 Trait-Based Species Selection

2.2.1 Rationale

Trait-based approaches offer a systematic solution to the surrogate species problem by functionally grouping species based on life-history, behavioural, and sensitivity characteristics. This allows the identification of vulnerable trait combinations, selection of representative species, and parameterisation of models using existing trait databases [21, 25]. Critically, the framework does not replace one universal surrogate with another: it eliminates the single-surrogate concept entirely. Instead of asking whether a given species protects all wild pollinators, the trait-based approach asks which trait combination is most vulnerable to the specific pesticide and exposure scenario under assessment, and selects the model species accordingly.

Not all traits carry equal weight in determining vulnerability: exposure-related traits (particularly phenological overlap with application windows and larval diet route) typically exert stronger influence on outcomes than resilience traits for acute pesticide scenarios, though the relative importance of trait categories is compound- and scenario-specific. Formal trait weighting schemes represent a research priority for Stage 2 development.

2.2.2 Relevant Trait Categories for Pollinators

Following the vulnerability framework of De Lange *et al.* [26], as reviewed in De Lange *et al.* [27] and extended to bees by Schmolke *et al.* [28], traits governing wild pollinator vulnerability to pesticides fall into three categories.

Exposure-Related Traits

Foraging range (landscape-scale exposure), phenology (temporal overlap with applications), floral specialisation (oligolectic versus polylectic), pollen choices, larval diet preferences in butterflies, moths

and hoverflies, nesting and pupation location and substrate, sociality, and life stage durations [c.f. 19 for bees].

Intrinsic Sensitivity Traits: Body size (correlates with sensitivity [8, 29]) and metabolic rate (affects detoxification capacity). Biochemical traits also belong in this category: detoxification enzyme capacity and the presence of relevant molecular initiating events (MIEs) and adverse outcome pathways (AOPs) determine whether a given compound produces harm at ecologically relevant doses. Direct toxicity data are scarce for most wild pollinator species, necessitating inter-species extrapolation based on physiological traits [30].

Population Resilience Traits

Voltinism, generation time (governs recovery rate following disturbance [31]), fecundity, dispersal capacity, mating system (haplodiploidy creates sex-ratio vulnerabilities), colony size, and overwintering stage.

EFSA *et al.* [1] already employs body size as a correction factor for interspecific extrapolation using allometric scaling [1, Section 6.5]). This is a useful first step, but body size alone cannot account for differential toxicodynamics, life-history-driven differences in recovery capacity, or exposure route variation between species. The full trait-based framework proposed here addresses all three trait categories simultaneously.

2.3 Demographic Toxicity Assessment

2.3.1 Data Sources for Demographic Parameterisation

Demographic parameters are compiled from published trait databases [21], published life table studies, and species-specific literature. Key sources include solitary bee demographic studies [14, 32], trait compilations for European pollinators [33], and existing population models [34].

2.3.2 Incorporating Pesticide Effects into Demographic Parameters

Pesticide exposure modifies demographic parameters through three primary mechanisms [see also 35].

Direct mortality

Acute and chronic toxicity reduce stage-specific survival probabilities. Dose-response relationships (LC₅₀, LD₅₀) from standard tests (OECD 2013/2014) are used to calculate proportional mortality at given exposure concentrations.

Sublethal effects on reproduction

Reduced fecundity, impaired mating success, impaired gamete performance, or decreased offspring viability reduce stage-specific fecundity values. While for acute toxicity testing, guidelines exist for honeybee, bumblebee and mason bee, i.e. guidelines 213–214 + 237 (HB) 246–247 (BB), 254 (Osmia), chronic tests are only standardised for honeybees, i.e. 245. However, none of these latter guidelines help in assessing reprotoxicity in adults, despite new proposals existing [36].

Sublethal effects on development

Delayed development, increased stage duration, or developmental abnormalities alter transition probabilities, and may be captured in brood tests (OECD 239) but are rarely incorporated into risk assessment.

For Stage 1 models, conservative exposure assumptions are applied throughout: 100% of the foraging area treated (worst-case spatial exposure); peak residue concentrations (worst-case temporal exposure); no spatial refuges or untreated areas; and continuous exposure throughout the active season. These assumptions ensure Stage 1 screening is protective, with refinement possible in Stages 2 and 3.

2.4 Matrix Population Model Construction

Matrix population models use standard linear algebra [37] and have been widely applied in ecology, conservation biology, and ecotoxicology [38]. The Leslie (1945) matrix framework projects age-structured populations through discrete time steps; where populations are better characterised by life stage than by age, the stage-structured extension introduced by Lefkovich [39] is more appropriate. The population growth rate (λ , the dominant eigenvalue of the projection matrix) indicates the trajectory: $\lambda > 1$ indicates growth, $\lambda = 1$ indicates stability, and $\lambda < 1$ indicates decline.

For univoltine wild pollinators, we construct a three-stage annual projection matrix with stages corresponding to distinct annual checkpoints: overwintered individuals (C), emerged adult females (A_e), and reproductively active adult females (A_r). This structure, with an annual timestep, correctly reflects the biology: all juvenile development (egg hatching, larval feeding, pupation) is completed within a single season, so each matrix transition spans one full year. Separating emerged and reproductive adults allows independent application of pesticide effects on adult survival versus fecundity, essential for demographic toxicity assessment [9]. It should be noted that voltinism is not fixed: several species are univoltine at northern latitudes but become multivoltine further south, requiring adaptation of the matrix structure and timestep for southern assessments.

The framework initialises models with stable stage distributions (eigenvector method) to avoid transient dynamics, ensuring observed deviations are attributable to pesticide effects rather than initialisation artefacts. Projections run for 10 years (10 generations for univoltine species). Models yield three key metrics: (1) population growth rate λ ; (2) deterministic population trajectories over time; and (3) stochastic projections using Monte Carlo methods (1,000 + iterations), generating distributions of outcomes, extinction probabilities, and confidence intervals.

2.5 Deriving SPG Thresholds Through Parameter Space Exploration

2.5.1 Generic Solitary Bee Model Structure

To demonstrate Stage 1 feasibility and derive indicative SPG thresholds, we developed a generic stage-structured Lefkovitch population model for univoltine solitary bees using a three-stage annual projection matrix parameterised using published data for *Osmia bicornis* [4]. *O. bicornis* is used here as a demonstration species representing the univoltine, cavity-nesting solitary bee trait profile, not as a proposed universal surrogate for all wild pollinators. In a full trait-based assessment, the choice of model species would depend on which trait profile is identified as most vulnerable to the pesticide and exposure scenario under evaluation. Initial conditions: Populations are initialised using the stable stage distribution derived from the control (unexposed) matrix. This distribution is represented by the right eigenvector corresponding to the dominant eigenvalue λ , normalised to a total population size of 500 individuals [$C = 300$, $A_e = 133$, $A_r = 67$]. This ensures control populations exhibit smooth exponential growth from year 0 rather than artificial transient oscillations.

Parameter ranges from literature: Based on published studies of *Osmia bicornis* and related cavity-nesting species [32, 40, 41]:

- F_{net} : 5.5–11.0 daughters (as overwintered individuals) per reproductive female per year; derived as $total_eggs \times sex_ratio \times P_{in-nest}$, with total brood cells 15–36 (Bosch and Kemp 2001), sex ratio 0.40–0.50 female, and in-nest egg-to-cocoon survival 0.70–0.90;
- P_{ow} : 0.60–0.72 (combined overwintering, eclosion survival and establishment rate [32, 41, 42];
- P_{AS} : 0.75–0.92 (adult survival through the foraging period; inferred from adult mortality studies).

The Lefkovitch matrix for the described stages is defined by

$$A = \begin{pmatrix} 0 & 0 & F_{net} \\ P_{ow} & 0 & 0 \\ 0 & P_{AS} & 0 \end{pmatrix}$$

The central parameter scenario ($F_{net} = 8.10$, $P_{ow} = 0.68$, $P_{AS} = 0.90$) is calculated by

$\lambda = (F_{net} \times P_{ow} \times P_{AS})^{(1/3)} = (8.10 \times 0.68 \times 0.90)^{(1/3)} = 1.71$, resulting in the dominant eigenvalue $\lambda = 1.71$, that indicates a population increase by 71% per year under unexposed conditions. This follows directly from the three-stage annual structure.

Across the parameter space, baseline λ ranges from 1.4 (slow life history) to 1.94 (fast life history), capturing variation in solitary bee demographic strategies within empirically documented bounds.

2.5.2 Pesticide Effect Scenarios

Pesticide exposure was modelled as a proportional reduction in demographic parameters, representing combined lethal and sublethal effects. Following Stark [9] demographic toxicity framework, we separated mortality effects (affecting adult survival) from fecundity effects (affecting reproductive output).

Adult mortality (m_A): Reduction in adult survival: $P_{AS, \text{exposed}} = P_{AS, \text{baseline}} \times (1 - m_A)$.

Fecundity reduction (r_F): Sublethal effects on reproductive output: $F_{\text{exposed}} = F_{\text{baseline}} \times (1 - r_F)$.

Combined effects: Both applied simultaneously, capturing the “total effect” described by Stark [9], who demonstrated that combined mortality and fecundity reductions produce more than additive population-level consequences: survivors produce fewer offspring, whilst those offspring face the same mortality risk in subsequent generations.

We explored effects from 0% to 50% for both parameters. For each parameter combination, the analysis:

1) Recalculated λ for the exposed population; 2) Projected population size deterministically over 10 years; 3) Calculated proportional population decline at year 3: $(N_{\text{control}} - N_{\text{exposed}}) / N_{\text{control}}$; (4) Ran 1,000 stochastic simulations incorporating demographic stochasticity (Poisson sampling of the fecundity transition $A_r \rightarrow C$; binomial sampling of survival transitions $C \rightarrow A_e$ and $A_e \rightarrow A_r$); and (5) calculated exceedance risk, i.e. the proportion of stochastic simulations in which the exposed population at year 3 falls below 90% of the deterministic control at year 3, i.e. the probability of breaching the 10% decline threshold under stochastic conditions. This metric is more appropriate than classical quasi-extinction probability for growing populations ($\lambda > 1$), where demographic stochasticity alone rarely drives extinction over decadal timescales.

Note that in ‘3’, Year 3 is used as the primary evaluation point because it corresponds to three complete annual reproductive cycles from the stable-stage initial conditions, capturing the first full expression of differential λ within a regulatory-relevant timeframe. Longer horizons amplify even small λ differences without bound. For example, a 5% reduction in adult survival produces a 5% decline at year 3 but a 15% decline at year 10, so the threshold value and evaluation year cannot be chosen independently. Year 3 provides a proportionate, interpretable assessment window in which a given percentage decline corresponds directly to a similar percentage effect on the relevant vital rate.

3. Results

This section demonstrates that quantitative SPG thresholds can be derived from demographic toxicity principles using a univoltine solitary bee as an example. The analysis uses two provisional threshold criteria, a protective decline threshold and an SPG persistence threshold. For demonstration, values of 10% population decline at year 3 and 90% persistence probability ($\leq 10\%$ exceedance risk) are used, respectively. The 10% magnitude threshold aligns with the existing regulatory trigger applied to *Apis mellifera* forager populations under the tiered assessment [1], providing a consistent protection standard across managed and wild bee taxa. These values are not prescriptive and should be adjusted/validated

through consultation with risk managers. The methodology is threshold-agnostic, allowing regulators to explore the consequences of different protection levels systematically.

3.1 Threshold Derivation

Table 3

Population-level effects of pesticide-induced mortality and fecundity reduction Results based on central parameter scenario (baseline $\lambda = 1.71$, derived from $F_{net} = 8.10$, $P_{ow} = 0.68$, $P_{AS} = 0.90$; [32, 40, 42]. Starting population: stable stage distribution totalling 500 individuals [$C = 300$, $A_e = 133$, $A_r = 67$]. Decline and exceedance risk evaluated at year 3. Exceedance risk = proportion of 1,000 stochastic simulations in which the exposed population at year 3 falls below 90% of the deterministic control (i.e. proportion breaching the 10% decline threshold). SPG compliance requires % Decline $\leq 10\%$ AND Exceedance Risk $\leq 10\%$.

Adult mortality (%)	Fecundity reduction (%)	λ (exposed)	Population Year 3	Decline Year 3 (%)	Exceedance risk (%)
Single effect on adult mortality					
0	0	1.71	2479	0	0.6
5	0	1.68	2355	5	8.1
10	0	1.65	2231	10	55.7
15	0	1.62	2107	15	93.2
Single effect on fecundity reduction					
0	10	1.65	2231	10	52.8
0	15	1.62	2107	15	96.6
Interaction between adult mortality and fecundity reduction					
5	5	1.65	2237	9.8	52.1
10	10	1.59	2008	19	99.7
15	15	1.53	1791	27.7	100

Full parameter space exploration included 81 scenarios (9 mortality levels $\times$ 9 fecundity levels; Fig. 1). Representative results are shown.

Key observations:

- Non-linear interaction between mortality and fecundity: Combined effects exceed either effect alone, but are less than additive. A population experiencing 5% mortality plus 5% fecundity reduction shows $\lambda = 1.65$ and 9.8% decline at year 3, compared with 5% decline from either effect individually. At 10% mortality combined with 10% fecundity reduction, the decline reaches 19%, nearly double that of either effect alone. This interaction arises because each vital rate constrains population growth independently, so reductions in P_{AS} and F_{net} compound across the annual cycle,

reflecting the "total effect". The combined effect is less than additive on population decline because the two reductions act multiplicatively on λ : a 5% reduction in each vital rate yields a combined λ ratio of $0.95 \times 0.95 = 0.9025$, marginally above the purely additive expectation of 0.90, so the resulting population decline falls slightly below the sum of the two individual declines. This compounding is also visible when mortality and fecundity reductions are expressed as the combined pesticide effect, with λ reduction increasing across the effect gradient (Fig. 2).

- **Threshold correspondence and the role of the persistence criterion:** The 10% protective decline threshold corresponds to approximately 10% adult mortality or 10% fecundity reduction individually. However, the persistence criterion is the binding constraint for borderline scenarios. At 10% adult mortality, the deterministic decline exactly meets the 10% magnitude threshold, but exceedance risk is 55.7%: the persistence criterion fails because stochastic variation frequently pushes the population over the threshold. The effective SPG boundary, therefore, sits between 5% and 6% for single effects, with the dual-criterion framework providing a self-consistent protection standard that is more conservative than either criterion alone. From a decision-analytic perspective, the proposed SPG can be interpreted as a two-constraint rule: a deterministic population-impact constraint combined with a probabilistic exceedance constraint. This is analogous to a chance-constrained decision framework, in which compliance is assessed not only by the expected magnitude of impact but also by the probability of breaching the protection threshold under stochastic demographic conditions. Representative deterministic trajectories for the control and selected exposure scenarios illustrate how these threshold outcomes develop over time (Fig. 3).
- **Life-history dependency:** The 10% magnitude threshold is insensitive to life history: a given proportional effect on a vital rate produces the same proportional decline regardless of baseline λ . By contrast, the exceedance risk criterion is life-history dependent: slow life histories (baseline $\lambda \approx 1.4$) 15.9% fail the persistence criterion even at 5% mortality, reflecting greater demographic fragility. Fast life histories (baseline $\lambda \approx 1.94$) pass at 5% mortality with lower exceedance (7.7%), as higher intrinsic growth provides greater stochastic resilience. This life-history dependency directly supports the trait-based species selection approach described in Section 2.3.
- **Alignment with the *Apis mellifera* threshold:** The 10% magnitude threshold aligns with the EFSA (2023) honey bee trigger level for forager population loss; however, the final SPG for wild bees would probably need to be more conservative because they are not human-managed. The population-level metric used here is a proportional decline in total population at year 3 relative to an unexposed control. This is arguably more conservative than the forager-count metric for honey bees, since it integrates effects across all life stages and captures demographic consequences over the full annual cycle rather than a single functional class.

3.2 Implementation Readiness

This analysis demonstrates that Stage 1 assessment can commence immediately using: existing demographic data from literature; standard toxicity test data (OECD guidelines 245, 246, 247 for bumble bees, and 74 for *Osmia*); readily available population modelling tools (see Supplementary Materials for

Python code); and provisional SPG thresholds grounded in population dynamics, adjustable through consultation with risk managers.

The analysis was implemented in Python (NumPy, Pandas) with modular components for Lefkovitch matrix operations, parameter space exploration, and visualisation, facilitating extension and adaptation for regulatory use.

Integration with EFSA Tiered Assessment. Our trait-based population models integrate directly with EFSA (2023) tiered risk assessment, resolving the tension between the need for defined thresholds and compatibility with existing regulatory structures. In Tier 1 (Screening), population models extrapolate individual-level effects; benchmark effect levels derived from trait-space exploration replace generic uncertainty factors. When Tier 1 triggers concern, refined exposure estimates replace conservative assumptions and population models are re-run with more targeted exposure scenarios. Field studies remain an option but are no longer the default requirement. Refinements are conducted by applicants using pre-run lookup tables, ensuring expedient regulatory implementation and comparability across dossiers.

The decision framework is as follows:

- **Pass** – population-level effects below magnitude threshold AND exceedance risk below persistence threshold → substance approved;
- **Trigger** – effects exceed threshold under conservative assumptions → proceed to higher-tier assessment;
- **Fail** – effects exceed threshold even with refined exposure → substance refused or use restrictions applied.

4. Discussion

Stage 1 models can derive quantitative SPG thresholds using existing data and tools, providing immediate regulatory value and a foundation for future development. Here we consider implementation aspects, maintaining focus on immediate deployability whilst indicating paths for progressive improvement.

4.1 Data Availability and Model Validation

4.1.1 Current Data Landscape

Stage 1 implementation relies on three data categories: demographic parameters (survival, fecundity), toxicity endpoints (acute and chronic), and trait classifications. Published studies of cavity-nesting solitary bees [14, 32] and existing trait databases [21] provide sufficient coverage for initial parameterisation, though data gaps exist for ground-nesting species, spring-active species, and multivoltine life histories. The framework is not restricted to bees: existing ALMaSS models for

hoverflies, butterflies, and moths show that the same population modelling approach generalises across pollinator groups [43–47]. Standard toxicity test protocols (OECD 245, 246, 247) generate the required endpoints, though chronic exposure data for solitary bees remain limited, although these are primarily designed for collecting mortality data.

The implementation strategy accepts these data limitations by using conservative assumptions where empirical data are sparse, prioritising regulatory action now over perfect data later, with targeted research addressing identified gaps in parallel with implementation.

4.1.2 Validation Strategy and Uncertainty Quantification

The parameter space exploration constitutes a sensitivity analysis, demonstrating threshold robustness to demographic parameter uncertainty. Primary validation requires empirical verification of input parameters under field conditions: baseline demographic rates for focal species in agricultural landscapes, pesticide exposure levels experienced by foraging adults, and dose-response relationships linking exposure to demographic effects. Laboratory-derived parameters may not reflect field realities due to multiple stressor interactions or behavioural compensation.

Secondary validation tests whether models accurately predict population outcomes through: (1) retrospective comparison with existing field studies [24]; (2) analysis of documented pesticide incidents; and (3) prospective monitoring programmes. Concrete validation opportunities exist within the author team: semi-field and field population data for *Osmia* spp. from controlled pesticide exposure studies [42, 48] and multi-species monitoring data from the PoshBee project [19, 49, 50] provide early retrospective test cases. The growth rate generated by the central parameter scenario ($\lambda = 1.71$) is consistent with the reported net population increase for the control *Osmia cornuta* in Albacete et al. 2024a (approximately 1.7 ± 0.4). This provides a useful plausibility check on the baseline parameterisation. Practical validation would begin with pilot studies on 2–3 focal species representing distinct trait combinations, measuring demographic parameters and population trajectories in treated and control landscapes over 3–5 years.

A further practical constraint on field-based verification of SPG thresholds is the magnitude of natural population variability in wild pollinators. Multi-year community monitoring studies document inter-annual coefficients of variation of 15–50% at the community level [51–53], with higher variability at individual sites [53–55]. SPG thresholds derived from population modelling are necessarily well below this range of background fluctuation, meaning that effects considered unacceptable under the SPG would be undetectable against field noise even in well-designed monitoring programmes. However, this is not a weakness of the framework; it is a structural argument for why model-based assessment should complement higher-tier field-based empirical detection as the primary lower-tier regulatory tool. Exposure and effect monitoring programmes remain valuable for validation and trend detection at the landscape scale, but cannot serve as the sole operational basis for SPG compliance assessment.

4.2 Comparison with Alternative Approaches

4.2.1 Species Sensitivity Distributions

Species Sensitivity Distributions (SSDs) derive protective concentrations from acute toxicity data, assuming the 5th percentile protects 95% of species. Whilst computationally simple, SSDs suffer from three limitations that our framework addresses. First, SSDs rely on individual-level endpoints that may fail to predict population outcomes. Second, SSDs assume taxonomic diversity equates to functional diversity, ignoring life history variation [56]. Third, SSDs cannot distinguish between sensitive-but-resilient species (high reproductive capacity) and moderately sensitive-but-vulnerable species (low reproductive capacity). Our approach explicitly links intrinsic sensitivity, exposure, and population resilience.

4.2.2 Uncertainty Factor Methods

Current risk assessment applies generic uncertainty factors (typically 10×) to laboratory toxicity data. However, as Stark [9] demonstrated, no single uncertainty factor can account for the mechanistic diversity of population responses to toxicants. Population modelling replaces arbitrary uncertainty factors with mechanistically derived safety margins specific to the life history strategy being protected.

4.3 Assumptions, Limitations, and Boundary Conditions

4.3.1 Trait-Based Extrapolation: When Does Functional Similarity Break Down?

Species with similar measurable traits may possess critical unmeasured differences, such as detoxification enzyme systems [57], or specific sensitivities as documented for the leafcutter bee *Megachile rotundata* and flupyradifurone [58], or other molecular differences such as in pesticide receptors [59]. Context-dependent trait expression and non-linear trait interactions can cause functional similarity to break down under stress.

Decision rule: Stage 1 models should not be applied when: (1) target species possess known unique physiological characteristics; (2) chemical mode of action suggests taxon-specific sensitivity; or (3) preliminary toxicity screening reveals sensitivity deviating substantially ($> 10\times$) from trait-based predictions. In such cases, proceed directly to higher-tier species-specific testing. Inter-species sensitivity varies substantially between compounds; trait-based extrapolation reduces but does not eliminate the need for compound-specific empirical calibration, particularly for novel modes of action.

Nicholson *et al.* [24] demonstrated that landscape context modifies pesticide effects on bumble bee colonies, with negative effects strongest in simple landscapes. Trait-based models assuming homogeneous exposure would fail to predict this interaction, which Stage 2 landscape-explicit models would address.

The framework focuses primarily on wild bees because demographic data, toxicity protocols, and trait databases are most developed for this group [23]. However, the trait-based population modelling approach is deliberately generalisable: the same three-category trait framework (exposure, sensitivity, resilience) applies to hoverflies, moths, and other pollinating taxa. Extending coverage beyond bees represents a priority research need.

4.3.2 Ecological Processes Not Captured in Stage 1 Models

Stage 1 models intentionally omit: (1) spatial population structure and rescue effects, conservatively assuming no immigration from refuge populations; (2) behavioural plasticity and pesticide avoidance; (3) interspecific interactions and community dynamics; and (4) evolutionary responses, relevant only for multi-generational assessments exceeding 10 years.

Decision rule

Stage 1 models are most reliable for relatively isolated populations with invariant behaviour, weak community effects (interspecific competition for nesting or floral resources, parasitism), and assessment timeframes ≤ 10 years. When populations are demonstrably strongly spatially structured, when pesticides trigger documented behavioural avoidance, or when community interactions are tight (obligate mutualisms), proceed directly to Stage 2. For example, a solitary bee population with documented immigration rates $> 20\%$ per generation violates Stage 1's isolation assumption, and Stage 2 spatial models incorporating dispersal kernels become necessary.

4.3.3 Density-Dependence and Compensatory Dynamics

Strong density-dependence can lead to overcompensation, where pesticide-induced mortality releases survivors from competition, increasing per capita reproduction [16]. This could cause Stage 1 models to overestimate pesticide impact. Conversely, Allee effects at low densities can cause populations to decline even when models predict persistence [60].

Decision rule

Stage 1 models are unreliable for populations at extreme densities (documented densities $> 2\times$ or $< 0.5\times$ long-term mean). This bidirectional uncertainty justifies conservative threshold setting, and field validation is essential to characterise species-specific density-dependence functions.

4.3.4 Temporal Dynamics: Exposure Variability and Phenological Mismatches

Pesticide exposure consequences for univoltine species depend heavily on timing relative to life-stage transitions. Worst-case exposure timing provides conservative screening but may substantially overestimate risk for pesticides applied outside critical windows.

Decision rule

When application timing demonstrably mismatches peak pollinator activity (> 4 weeks separation), refine Stage 1 scenarios to reflect realistic temporal exposure patterns. For multivoltine species or pesticides

with delayed effects, Stage 1 oversimplification may underestimate risk, requiring refined temporal resolution in higher-tier assessments.

4.4 Trade-offs: Ecological Realism Versus Regulatory Tractability

Stage 1 models make pragmatic simplifications: homogeneous environment (conservative, assuming worst-case uniform exposure); fixed vital rates (ignoring density-dependence); single-species focus (omitting community interactions such as competition and parasitism); and linear extrapolation of pesticide effects across trait space. Stage 1 accepts false positives (predicting decline when real populations persist due to spatial refuges or compensation) to avoid false negatives. Stage 1's conservative assumptions will generate false positives at an unquantified rate pending Stage 2 validation, which requires future evaluation when Stage 2 models are available.

Decision framework for tier escalation

Substances exceeding the protective decline threshold by < 20% warrant refinement with realistic exposure scenarios. Substances exceeding the threshold by > 50% are unlikely to pass higher tiers and should proceed directly to risk management decisions. This prevents unnecessary refinement effort whilst allowing borderline cases to benefit from reduced conservatism.

4.5 Implementation Roadmap and Research Priorities

Stage 1 deployment requires three parallel tracks.

Regulatory track (2026–2027)

Stakeholder consultation establishing agreed parameter ranges, exposure scenarios, and provisional threshold values. Pilot implementation with 3–5 exemplary pesticide-species combinations to test workflow integration with existing EFSA procedures. Regulatory guidance document development specifying data requirements, modelling protocols, and decision criteria.

Validation track (2026–2027)

Retrospective comparison against existing field studies to quantify model prediction accuracy. Prospective monitoring programmes for new pesticide registrations, tracking predicted versus observed population trajectories. Database compilation of validation results to refine parameter ranges and identify systematic biases.

Development track (2027–2030+)

Stage 2 landscape-explicit models incorporating spatial population structure, dispersal, and habitat heterogeneity, integrated with EFSA Tier 2 exposure assessment. Stage 3 agent-based models (ALMaSS

framework) incorporating individual-level behaviours, multiple stressors, and systems-level assessment for complex scenarios.

The timeline reflects a stepwise transition strategy in which implementable components are introduced early, whilst supporting infrastructure is successively provided by pilots [23, 61–65]. Priority research needs to include demographic data for under-represented taxa (ground-nesting bees, spring-active species), chronic toxicity data for sublethal effects, phenological data linking pesticide application windows to pollinator life stages, and empirical density-dependence relationships from multi-year monitoring. Bereswill *et al.* [66] recommended using *O. bicornis* with appropriate safety factors as an interim measure pending data on more sensitive focal species. The trait-based framework presented here supersedes this stopgap: rather than applying safety factors to a single surrogate, it identifies which trait combination is most vulnerable for the compound and scenario under assessment and selects the model species accordingly. Stage 1 uses conservative assumptions and documents the extrapolation domain; Stage 2 ensures adaptive implementation with updated model parameters and decision thresholds, enabling action rather than postponing implementation.

Success criteria for stage transitions: Stage 1 → Stage 2 requires demonstrated Stage 1 prediction accuracy > 75% for validation case studies and regulatory acceptance of Stage 1 thresholds. Stage 2 → Stage 3 requires validated landscape models and computational infrastructure supporting spatially-explicit assessment at scale.

4.6 Implications Beyond Risk Assessment

Adopting population-level SPGs influences pesticide product development, landscape management, and monitoring programmes. Stage 1's continuous exposure assumption incentivises developing rapidly degrading compounds, aligning environmental goals with commercial innovation. Population models can inform agri-environment schemes and other habitat restoration actions by quantifying how habitat provision (nesting sites, floral resources) interacts with pesticide risk, enabling landscape design that enhances pollinator population and pollination service resilience. In future applications, the outputs of such models could also be linked to bioeconomic and economy-wide models to assess how changes in pollinator populations affect crop yields, land allocation, input demand, farm income, consumer prices, and broader welfare outcomes. This would make it possible to compare pesticide-risk mitigation and pollinator-restoration strategies not only in ecological terms, but also in terms of their marginal economic benefits, opportunity costs, and shadow values.

The trait-based resilience framework presented here is not inherently restricted to pollinators: the same three-category vulnerability approach (exposure traits, sensitivity traits, and population resilience traits) could, in principle, be extended to other non-target organism groups currently assessed through recovery-rate proxies, offering a more ecologically grounded basis for SPG derivation across ERA schemes.

The framework supports adaptive management through monitoring designs that test model predictions, creating a feedback loop between assessment and empirical reality. More broadly, the framework directly supports Member State obligations under the NRR, whose Article 10 requires the reversal of pollinator population declines by 2030. Defined, quantitative SPG thresholds are a prerequisite for demonstrating that pesticide approvals are compatible with this obligation.

5. Conclusions

This framework provides quantitative SPG thresholds grounded in population resilience principles, using trait-based approaches for species selection and demographic toxicity assessment for total population-level effects. It fits seamlessly within EFSA's tiered assessment structure and offers a progressive implementation pathway from immediate Stage 1 deployment through future landscape-explicit refinements.

The regulatory constraint created by undefined wild pollinator SPGs has forced higher-tier studies by default, creating assessment bottlenecks whilst providing limited population-level insight, since no objective threshold exists against which results can be evaluated. Currently, higher-tier field study outcomes are assessed through statistical equivalence to control treatments rather than against a population-level protection goal; this is a methodological proxy that does not directly address the question of population persistence. Our framework resolves this: Stage 1 provides defined thresholds that are implementable within 12–18 months using existing tools and data, whilst Stages 2 and 3 establish a pathway for adaptive implementation towards increasingly sophisticated, ecologically grounded assessment.

Whilst Stage 1 models require validation of parameters and population predictions, this framework is demonstrably more protective than continuing with honey bees as the default surrogate. Current approaches implicitly assume that protecting honey bees protects wild pollinators, yet this assumption has never been validated and is contradicted by a mechanistic understanding of population dynamics. An imperfect model that addresses the right question (wild pollinator population persistence) provides better protection than a well-characterised model that addresses the wrong question (honey bee colony effects). Regulatory decisions always proceed under uncertainty; the choice is between acknowledging and addressing uncertainty through population modelling or ignoring it through inappropriate surrogacy.

Success requires stakeholder collaboration to establish agreed parameter ranges, exposure scenarios, and threshold values, as well as decision makers willing to implement this in the regulatory process. Existing trait databases, demographic data from published studies, and established population modelling tools provide the foundation. Pilot implementations with model species can validate approaches and build confidence before broader adoption. Regulators can act now with Stage 1 whilst developing enhanced capabilities, providing immediate regulatory value and establishing excellence in population-level pesticide risk assessment aligned with EU biodiversity and restoration obligations. By defining explicit biological constraints that can later be linked to ecosystem-service and economic

models, the framework also provides a foundation for more transparent, cost-effective, and policy-consistent decisions on pesticide use and pollinator restoration.

Abbreviations

Abbreviation

Meaning

ALMaSS

Animal Landscape and Man Simulation System

DG ENV

Directorate-General for Environment (European Commission)

EFSA

European Food Safety Authority

ERA

Environmental Risk Assessment

EU

European Union

LC50

Median lethal concentration

LD50

Median lethal dose

NRR

Nature Restoration Regulation

OECD

Organisation for Economic Cooperation and Development

PEC

Predicted Environmental Concentration

SPG

Specific Protection Goal

SSD

Species Sensitivity Distribution

λ

Population growth rate (dominant eigenvalue of projection matrix)

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Glossary

Ecological concepts

Trait: The definition of “trait” has evolved over time, and no consensus exists in the literature [67]. Here, we use the term to refer to functional traits defined as measurable properties of organisms that

influence their survival, reproduction, and ecological performance. We focus mainly on response traits that mediate how species respond to environmental stressors.

Resilience: The capacity of a population to recover from exposure to a chemical, physical, or biological disturbance and return to its pre-disturbance state in terms of abundance, structure, and function. Resilience encompasses recovery rate, recovery magnitude, persistence capacity, and demographic buffering.

Allee effect: Drake and Kramer [68] defined the Allee effect as a positive density-dependence process in which average individual fitness, or per capita population growth rate, is positively associated with population size over a range of low population densities. A strong Allee effect creates a critical threshold below which the population cannot persist.

Oligolectic versus polylectic: Terms referring to the diet specialisation of pollen-collecting insects. Oligolectic species collect pollen from only a few floral species (specialists); polylectic species collect from a broad range (generalists).

Rescue effect: Brown and Kodric-Brown [69] described the rescue effect as a metapopulation process in which immigration from neighbouring populations reduces local extinction risk.

Ecotoxicological concepts

SPG (Specific Protection Goal): The specific goal of an environmental risk assessment in terms of what to protect, where to protect it, over what time and with what degree of certainty.

LD50 versus LC50: Lethal Dose 50% (LD50) is the amount of a substance that causes death in 50% of a population of test organisms within a certain timeframe. For bees LD50 is expressed as amount of substance per individual. Lethal Concentration 50% (LC50%) is the concentration of a substance that causes death in 50% of a population of test organisms within a certain timeframe. For bees LC50 is expressed as concentration of the substance in the exposure medium. LD50 values allow comparison across species of different sizes, while LC50 values allow comparison across environmental concentrations.

Demographic toxicity: An environmental risk assessment approach that evaluates the adverse effects of a substance on population dynamics, including changes in survival, reproduction, growth, or development, which may alter the size, structure, or long-term viability of a population. Modelling is typically used to translate ecotoxicological endpoints (e.g., survival and fecundity) into population-level impacts.

Benchmark dose methodology: A dose–response modelling approach used to identify the dose of a substance (benchmark dose, BMD) that produces a predefined level of effect compared to a control,

based on the entire dataset. It provides a quantitative reference point, often with a lower confidence limit (BMDL), for use in risk assessment.

PECpollen/PECnectar (Predicted Environmental Concentration): The concentration of a chemical in pollen and nectar as predicted by exposure models based on its application and environmental fate.

Species Sensitivity Distribution (SSD): A statistical method used to assess the effects of toxic substances on entire biological communities. An SSD describes the variation in sensitivity among species to a given chemical, identifying both the most sensitive and the most tolerant species. It is constructed by fitting a cumulative distribution curve to toxicity values (e.g., LD50) estimated for each species in the community. SSDs can be used to characterise the distribution of sensitivities and to estimate the proportion of species potentially affected at a given level of environmental contamination.

Modelling concepts

Agent-based (system) modelling: Agent-based system modelling in environmental risk assessment represents individual organisms as interacting autonomous agents whose local rules and stochastic behaviours generate emergent spatial-temporal patterns that can be analysed to predict and manage system-level exposure, impacts, and uncertainty.

Dominant eigenvalue (λ): The dominant eigenvalue (often denoted λ) of a matrix population model is the **largest real eigenvalue of the projection matrix**, and it determines the asymptotic per-time-step growth rate of the population: if $\lambda > 1$ the population expands, $\lambda = 1$ it remains stable, and $\lambda < 1$ it declines.

Leslie/Lefkovitch matrix: A Lefkovitch matrix captures stage-structured dynamics by allowing individuals to remain in, progress to, or even regress between stages each time step - unlike a Leslie matrix, which forces a strict, one-step-forward progression through age classes.

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Figures

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Figure 1

Year-3 population decline across adult mortality and fecundity-reduction scenarios. Population decline at year 3 is shown for the central solitary-bee parameter scenario across combinations of adult mortality and fecundity reduction. The blue contour indicates the 10% protective decline threshold used for demonstration.

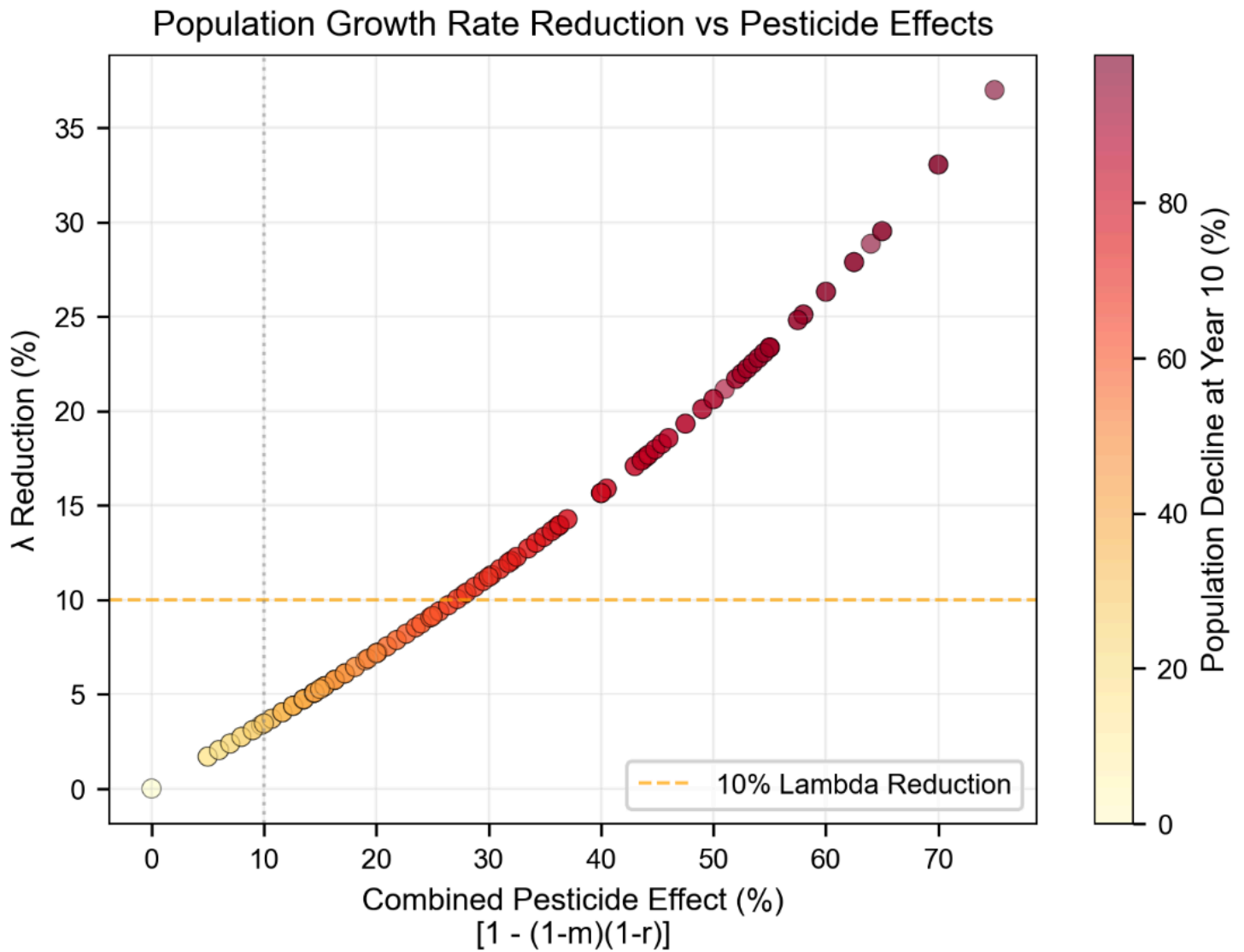


Figure 2

Reduction in population growth rate across combined pesticide-effect scenarios. Reduction in λ is plotted against the combined pesticide effect, calculated as $1 - (1 - m)(1 - r)$, where m is adult mortality and r is fecundity reduction. Point colour indicates projected population decline at year 10, illustrating why evaluation year and threshold magnitude cannot be selected independently.

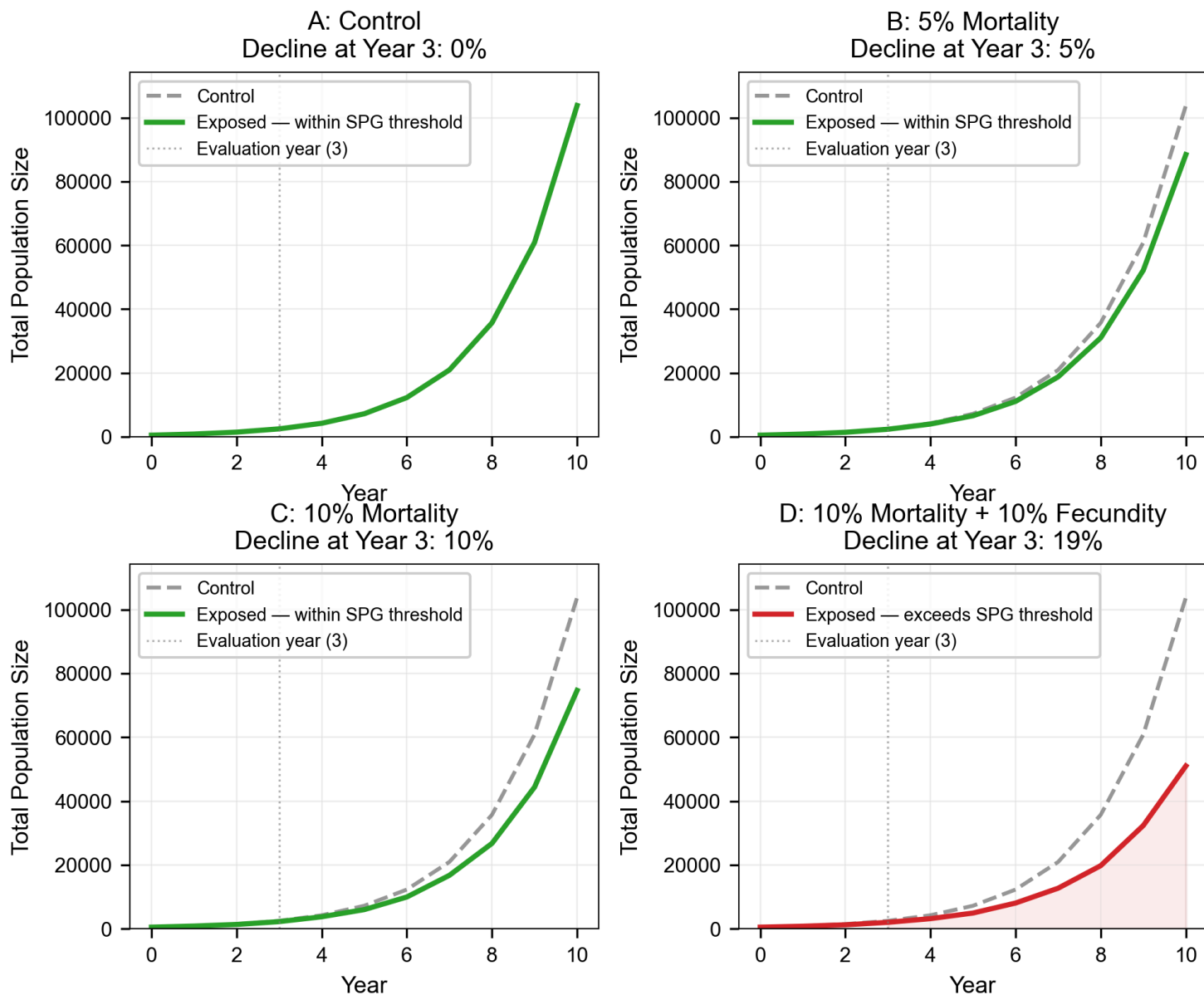


Figure 3

Representative population trajectories under selected pesticide-effect scenarios. Deterministic population trajectories are shown for the central parameter scenario under control conditions and selected adult mortality/fecundity-reduction scenarios. The vertical dotted line marks the year-3 evaluation point used for SPG threshold assessment.