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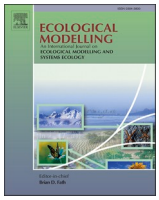

Managing pesticide resistance in Spodoptera frugiperda: A spatially explicit framework for identifying optimal treatment strategies

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ABSTRACT

Pesticide resistance is a major challenge to increasing the resilience and sustainability of current food production systems. Preserving the susceptibility of pest organisms to chemical products is a key factor to optimize a pesticide-based strategy. However, resistance management strategies (RMSs) must consider unique species biologies, multiple resistance mechanisms, environmental factors, and pest management practices, which can make their implementation complex.

Here, we develop a method to help manage this complexity using a grid-based simulation framework for pesticide resistance evolution including population growth and dispersal dynamics. This framework was applied to the fall armyworm, Spodoptera frugiperda, for which resistance evolution is a major concern. We explored the sensitivity of 13 parameters dealing with landscape structure, dispersal rate, chemical treatment protocols, chemical degradation rate, dose-response curves and transition rates (i.e., flux between sub-population driven by the mutation rate). From the sensitivity analysis of simulations, we computed heat maps of the influence of each parameter on a set of variables (total pest population size, fully resistant population size, and resistance frequency).

Assuming a large but realistic range for each parameter, Sobol's sensitivity index showed that resistant transition rate (from phenotypically susceptible to resistant sub-populations) and pesticide properties (in particular, degradation rate and dose-response curve) are more important in the outbreak of resistance compared with resistance ratio (i.e., the benefit of being resistant over susceptible in terms of fitness), chemical application intensity and landscape composition. In addition, using Pareto optimality, we assessed the performance of different pesticide application regimes according to total population size, population size of resistant individuals, the total amount of pesticide, and the total area of host plants suitable for S. frugiperda. Across the wide parameter space explored, we revealed the high volatility of outputs suggesting that the performance of treatment protocols depends on the ecological context. Nevertheless, despite this variability, a "windowing" management strategy consisting of a single pesticide group applied per insect generation, provided the optimal control of S. frugiperda and resistance evolution outcomes.

This work provides a set of tools to test RMS scenarios for the control of S. frugiperda and to understand how variabilities may arise at different management steps and geographical scales.

1. Introduction

Current chemical-based pest control strategies are challenged by sustainability goals (FAO, 2018) and eco-evolutionary responses that lead to increased tolerance and the evolution of pesticide resistance (Dermauw et al., 2018). Globally, agricultural pests have been

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successfully managed for decades using pesticides and this has contributed to an increase by a factor of two to three in global food production throughout the "green revolution" (Evenson and Gollin, 2003). However, there is increasing awareness of the negative side-effects of the widespread application of pesticides, such as impacts to human health, natural ecosystems and biodiversity (Bourguet and Guillemaud 2016), and the evolution of resistant populations requiring even higher chemical application rates (Sparks and Nauen 2015). Despite these issues and the progress that has been made in biological controls and other non-chemical options, synthetic pesticides are expected to play a key role in future control programs targeting agricultural pests.

Resistance, defined here as the heritable decrease in a population's sensitivity to a chemical to which it is exposed over successive generations, remains one of the best documented examples of rapid contemporary evolution (Heckel 2012) and can render chemical applications ineffective in just a few generations (Nauen 2007; Umina et al., 2019a). To control resistance, management programs typically attempt to reduce the intensity of selection for resistance, which can be manipulated in various ways such as alternating chemicals and reducing the frequency and/or intensity of applications. However, these strategies are often only implemented after resistance appears. Sustainable and effective pest management strategies require a deep understanding of the evolutionary and ecological processes underpinning the emergence and spread of pesticide resistance. In the case of pest arthropods, this can be particularly challenging given the large dispersal capacity of many species (Miller and Sappington 2017), often requiring large heterogeneous landscapes to be considered, which may span a variety of climates, plant production industries, and land usages. Globally, more than 580 arthropod species have been reported with resistance to approximately 325 different chemicals (Sparks and Nauen 2015; APRD, 2022) and these numbers continue to grow each year.

Resistance Management Strategies (RMSs) aim to prevent or delay resistance evolving, or to help regain susceptibility in pest populations in which resistance has already arisen. The evolution of resistance is influenced by many factors, ranging from genetic inheritance to ecological factors that affect the life-history of a pest population. Consequently, RMSs are often perceived as part of a broader Integrated Pest Management program (Onstad 2013; Umina et al., 2019a), which employs a package of tactics to reduce pest pressures. Such approaches include: (i) the diversification of causes of mortality so that a pest is not selected by a single mechanism, (ii) reduction of selection pressure for each mortality mechanism, (iii) maintenance of a refuge or immigration to promote mixing of susceptible and resistant individuals, and (iv) the generation of baseline data and implementation of resistance monitoring programs (Onstad 2013; Umina et al., 2019a).

Statistical modelling can also play an important role in understanding resistance risk. For example, models can identify correlations between agricultural factors (e.g., chemical practices, land usage or climatic patterns) and the emergence of resistance (Maino et al., 2018a; Umina et al., 2019b), while simulations of realistic scenarios can be compared with RMSs (REX, 2013; Papaïx et al., 2018). The majority of pesticide resistance simulations explore different treatment protocols, whereby multiple chemical groups are applied with various patterns in space and time: (i) uniform in space but heterogenous in time (e.g., temporal cycles, or a successive application without cycle); (ii) uniform in time but with a mosaic distribution in space (without overlap), or (iii) a combination of variation in both time and space (REX, 2013). These models will often consider complex processes, like the genotype basis of resistance (Sudo et al., 2018), the dispersal of pest populations (Miller and Sappington 2017) and ecological factors (e.g., density-dependency, abiotic factors) (Haridas and Tenhumberg, 2018).

The present study aims to simulate realistic pesticide resistance scenarios by developing a spatially-explicit eco-evolutionary model that explores resistance dynamics of pest populations across heterogeneous environments (i.e., different climates, land use, pest management scenarios, and pesticide exposure). As a case study, we parameterize and apply the model to fall armyworm, *Spodoptera frugiperda* (J. E. Smith) (Lepidoptera: Noctuidae), to help inform RMSs in Australia as farmers adapt to this new pest. *Spodoptera frugiperda* is native to the American continent, where it is a sporadic pest (Bodkin 1913; Sparks, 1979). Its range has been widely extended to other continents: to Africa in 2016, then Asia, and since February 2020 to Australia. *Spodoptera frugiperda* has a permanent range restricted to warmer climates, but has substantial migration capacity and annually invades less suitable climatic regions during warmer periods (Nagoshi et al., 2012). *Spodoptera frugiperda* is highly polyphagous, particularly attacking Poaceae including maize (*Zea mays*), sorghum (*Sorghum* spp.), rice (*Oryza sativa* L.) and various species of pasture grasses, as well as other non-grass crops including cotton (*Gossypium hirsutum* L.) (Casmuz et al., 2010; Montezano et al., 2018).

2. Methods

We use a spatially explicit approach that includes ecological and evolutionary components, leveraging a recent simulation study on the establishment and spread potential of *S. frugiperda* in Australia (Maino et al., 2021). The choice of a population-based model is particularly efficient when dealing with high spatial resolutions and to explore a large range of parameters for sensitivity analysis. Within this spatially-explicit population-based framework, we implement compartmental models where we define sub-populations of *S. frugiperda* according to their resistance to pesticides.

The implementation of the model framework is generic (multi-species, multi-pesticide) (Schouten et al., 2022; Github: cesaraustralia/PopulationResistanceFramework), but in the present study we reduce the application to two generic pesticides with different Modes of Action (MoA) assuming different mechanisms that lead to the acquisition of resistance. For realism of the pesticide properties (e.g., degradation rate, dose response, resistance ratio) we parameterized the model on chlorantraniliprole (MoA group 28) and indoxacarb (MoA group 22A), which are denoted respectively as 'C' and 'I' in mathematical variables and parameters herein. These two chemicals are among the most important pesticides used to manage *S. frugiperda* globally, have different levels of toxicity and varying levels of field resistance (Bird et al., 2022).

The model does not consider the genetic basis of resistance but focusses on phenotype, which has two benefits. First, this approach relates directly to how resistance is normally measured (at the phenotypic level); most field resistance data are phenotypic and report the mean of survival endpoints over populations, and then assess the 'resistance' by comparing these to phenotypic endpoints for each population, giving a resistance ratio (Gutiérrez-Moreno et al., 2019). Second, the approach can account for multiple resistances, and in the present situation where we consider two pesticides and one species, this leads to only four phenotypes: 'P_{CI}' for individuals resistant to chlorantraniliprole and to indoxacarb; 'P_C' for individuals resistant to chlorantraniliprole but susceptible to indoxacarb; 'P_I' for individuals resistant to indoxacarb but susceptible to chlorantraniliprole; and 'P_S' for individuals susceptible to both pesticides.

The evolutionary response of a population in the model is supported by three mechanisms. Considering that a local population at a simulation step t_n, denote S_n, is described by the size of the population for each phenotype: $S_n = \{N_S, N_C, N_I, N_{CI}\}_n$. At the next time step, t_{n+1}, the structure of the population S_{n+1} results from three processes:

1 The addition of individuals appearing within each phenotype category and the removal of those dying. Only the mortality rate includes the effect of pesticides (denoted μ_C and μ_I in Table 2), since we do not account for other pesticide effects (e.g., reduced reproduction).

2 The results of dispersal for each phenotype between individuals that are removed because they move away from this local population and the addition of individuals from other locations.
3 The transition process accounting for genetic changes resulting in the introduction of resistant phenotypes from susceptible parents. This could involve a new dominant mutation appearing in the offspring, the expression of resistance through offspring inheriting recessive alleles from both parents, or (in the case of polygenic resistance) recombination events that result in resistance alleles being accumulated in some offspring that results in resistance even if both parents are susceptible. Based on the four phenotype categories, we consider only four possible transitions: P_S to P_C, P_S to P_I, P_C to P_{CI}, and P_I to P_{CI}; with rates respectively denoted $m_{S\to C}$, $m_{S\to I}$, $m_{C\to CI}$, $m_{I\to CI}$.

For this latter transition process, there are two additional assumptions. Firstly, we do not consider a multi-resistant transition, like for instance from a full susceptible, P_S, to a full-resistant phenotype, P_{CI}, because under the assumption that 'single-resistant' transitions are independent events, the following law would apply: $m_{S\to CI} = m_{S\to C} \times m_{C\to CI}$, and therefore there is no need to define multi-resistant transitions as $m_{S\to CI}$. Note however that $m_{S\to C}$ is not a probability, but a rate, as it can theoretically be negative, but we do not explore negative transition rates. In the same vein, and this is the second assumption, we only assume that if a transition occurs, it's only with a gain of resistance:, and never a gain of susceptibility.

The model presented in Fig. 1 depicts the succession of variables and the rules of our approach, with quantitative details provided in the sections below. The simulation was initialized with a random landscape composed of three types of habitat cells (see section "2.1 Landscape structure"). Every cell of the grid is defined by the landscape with a realistic temperature time series that reflects North-East Australia, (lon 140°E-153°E; lat 20°S-30°S), where *S. frugiperda* is currently causing economic impact (among other subtropical regions) (Plant Health Australia, 2020). The establishment of susceptible *S. frugiperda* individuals is computed based on population growth rates calculated from climatic and environmental data from SMAP (Soil Moisture Active Passive) at the 9 km resolution every 3 h (surface temperature and wilting based on soil moisture) (Reichle et al., 2017) and within suitable habitats. Population dispersal is based on the seasonal population development of *S. frugiperda* and the suitability of habitat at the landscape scale. Simultaneously, the exposure profiles of the two pesticides are computed based on the landscape mosaic of crop fields and chemical treatment protocols (see section "2.3.1 Treatment protocols"). Population mortality is estimated from pesticide dose-response curves and resistance profiles (see section "2.3 Exposure and Toxicity").

The model is mechanistic in the sense that two runs with exactly the same parameterization would provide exactly the same output. However, for every simulation, we randomly selected 19 values (see Tables 1 &2) giving 4500 unique outputs. All code required to replicate the analysis is provided in the online repository (Github: cesaraustralia/PopulationResistanceFramework).

2.1. Landscape structure

We consider a local landscape with a grid of 200×200 pixels with 1 pixel representative of 1 ha (i.e., a grid of 20×20 square km) with 3 land categories (see Fig. 2): (i) cropping areas where the crop grown is suitable for *S. frugiperda* development (named "crop habitat"), (ii) non-cropping areas that contain plant hosts that are suitable for *S. frugiperda* development (named "refuge habitat"), and (iii) non-cropping areas that do not contain any plants suitable for *S. frugiperda* development (named "no habitat"). For every simulation, a new landscape was generated based on a random set of proportions for each category.

Table 1
Variable descriptions and symbols used in the model equations.

Variable	Symbol
Total population density per cell	N
Sub-population of susceptible to both pesticides	P_S
Sub-population of resistant to chlorantraniliprole	P_C
Sub-population of resistant to indoxacarb	P_I
Sub-population of resistant to both chlorantraniliprole and indoxacarb	P_{CI}
Population density per cell	N_S, N_C, N_I, N_{CI}

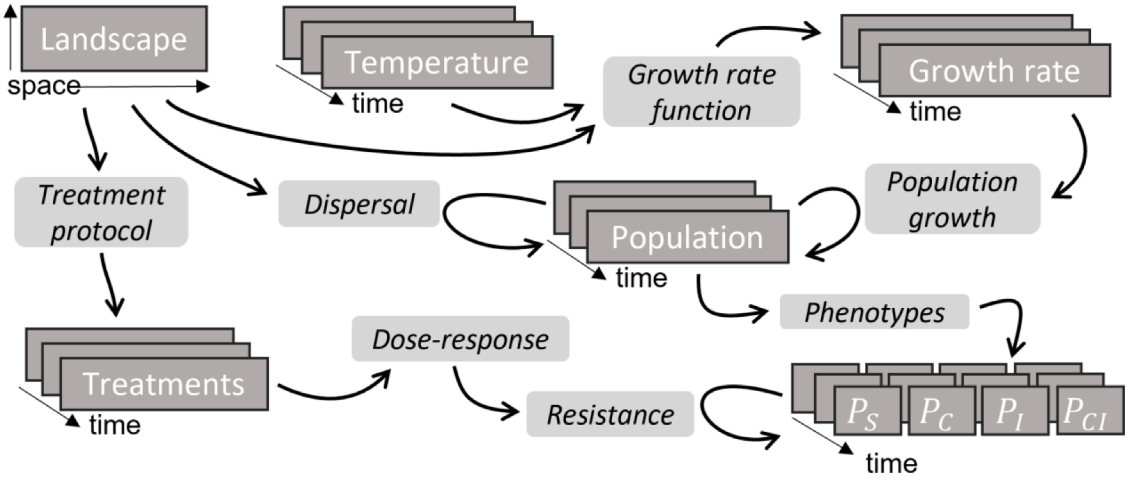

Fig. 1. Schematic of the statistical model used in this study. Dark grey boxes are layers representing spatialized data, while light grey rounded boxes are rules using the data-layers. The "Landscape" box is represents a spatial grid with non-time-variable land composition (i.e., "crop habitat", "refuge habitat", or "no-habitat") on which chemical treatment is applied. The "Treatment protocol" rule depends on landscape composition and the Mode of action (MoA) of the chemical. The "Treatments" box represents the sequence of treatment layer applied on the landscape. "Temperature" is a grid time series of temperature. The "Growth rate function" rule parameterized with "Landscape" and "Temperature" layers results in the "Growth rate" grid time-series. "Growth rate" and "Dispersal" rules drive the population dynamic given by the "Population" grid-time series. Finally, "Dose-response" and "Resistance" rules control the sub-population dynamics based on the phenotypic sub-division of the population.

Table 2
Parameters of the statistical model used in this study.

Parameter	Symbol	Units	Fixed or range values
Grid size	–	px	200×200 (1 pixel = 1 ha) = 20×20 km^2
Proportion of land 'crop habitat'	p_{crop}	–	Uniform [min = 0.1, max = 0.9]
Initial population density	N_0	n/px	$N_S = 10^5$; $N_I = N_C = N_{CI} = 0$
Carrying capacity	K	n/px	10^6 / pixel
Growth rate	r	d^{-1}	Follow Temperature Model
"Natural" death rate	m_0	d^{-1}	Log-Uniform [min = 0.01, max = 0.99]
Dispersal rate	λ	px/d	Log-Uniform [min = 0.001, max = 0.4]
Amount of indoxacarb	X_I	g a.i./px	Uniform [min = 30, max = 60]
Amount of chlorantraniliprole	X_C	g a.i./px	Uniform [min = 15, max = 45]
Degradation rate of chemical	d	d^{-1}	Log-Uniform [min = 0.1, max = 20]
Hill coefficient (slope)	β	–	Log-Uniform [min = 0.5, max = 10]
Resistance ratio	R	–	Log-Uniform [min = 1, max = 10^4]
Transition rate (P_S to P_C, P_S to P_I, P_C to P_{CI}, and P_I to P_{CI})	$m_{S\to C}$, $m_{S\to I}$, $m_{C\to CI}$, $m_{I\to CI}$,	d^{-1}	Log-Uniform [min = 10^−4, max = 10]

2.2. Population dynamics

2.2.1. Population growth

The growth of populations follows a logistic growth rate within each cell with N the total population and N_i the size of sub-population group (i.e., N_S: susceptible to both pesticides, N_{CI}: resistant to both pesticides or N_C and N_I: resistant to a single pesticide – see Table 1 for further details). The carrying capacity is denoted K and is assumed to be the same for each cell (and we assume a symmetric effect of the carrying capacity on each group) and the growth rate r which is assumed the same for all sub populations (i.e., no cost of resistance, same fitness value), meaning that only differences in pesticide mortality responses distinguish the sub-populations. The growth rate r was estimated from data generated under optimized laboratory conditions (see Tables 1 & 2), therefore, to take into account external disturbance on populations that may mitigate this optimized laboratory growth rate (e.g., predation or disease), we assume the possibility of an additional mortality rate linear with the population density denoted m_0, which can be around 50% (Varella et al., 2015). Calibrations of all parameters are given in Table 1 & Table 2.

$$\frac{dN_i}{dt} = rN_i\left(1-\frac{N}{K}\right) - m_0 N_i$$

Fig. 2 provides an example of the initial population densities on cells. Since a different landscape is created at every simulation, a new initial population is also generated following landscape properties: "crop habitat", "refuge habitat" and "no-habitat". To account for cooler climates, we assumed a population growth period to be from August to March, with a non-growing period from April to July (see Fig. 3).

2.2.2. Population dispersal

Population dispersal followed an exponential dispersal kernel (Nathan et al., 2012) with parameter λ varying from 0.001 to 0.4 on a log-scale having a mean distance dispersal by time unit (i.e., days) of 2λ. Based on our understanding of *Spodoptera frugiperda* biology in North-East Australia, we assume *S. frugiperda* only disperse in suitable habitats, and only adult individuals can disperse long distances, so we distinguish between a stationary period and a dispersal period, and we fix the period of dispersion to be in October-November and February-March (Fig. 3). This allowed us to consider two distinct populations of *S. frugiperda* per year in order to better understand the role of each period, stationary and dispersal. Over the whole Australia, the difference in climatic conditions and in particular of temperature exhibit more complex population dynamics and treatment protocols (Maino et al., 2018a), as for instance more generation in warmer regions likely related to a more frequent use of pesticide. We finally consider no dispersal during the cooler conditions experienced between April and July and during stationary life-stages (i.e., eggs, larvae, pupae), assumed to be in August–September and December–January.

We assumed chemical selection only happens during stationary periods (where pesticides are used). While largely reflective of the situation in the field, we acknowledge adult *S. frugiperda* moths will be exposed to chemical applications (albeit at lower frequencies than stationary life-stages). The synchronicity of dispersal with pesticide-selection would be better suited with individual based models, since we would have to track the amount of pesticide collected by each individual during its dispersal journey to apply a dose-response relationship.

2.3. Pesticide toxicity and exposure

2.3.1. Treatment protocols

We considered two pesticides, chlorantraniliprole and indoxacarb, with applications in two periods (August–September and

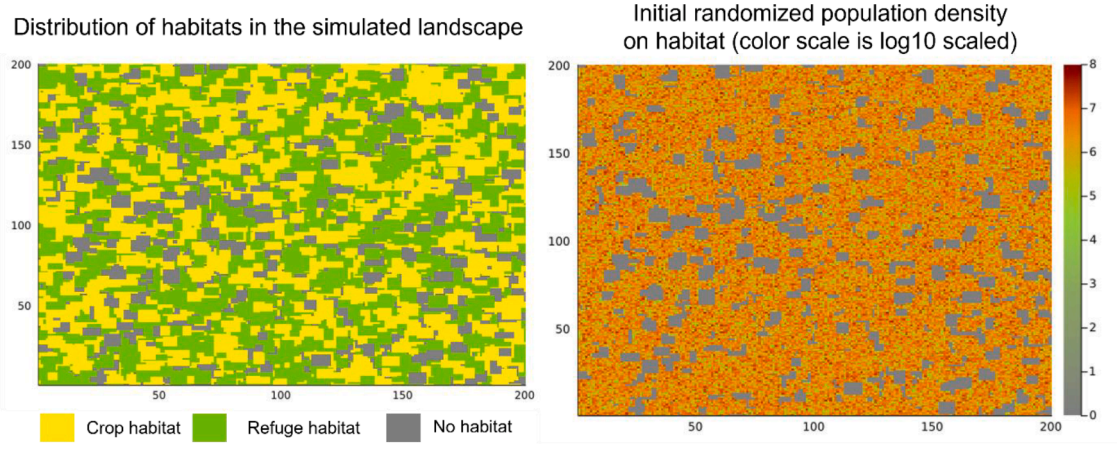

Fig. 2. Example of a simulated landscape with (left) a random distribution of habitats suitable for S. frugiperda development (i.e., "crop habitat" and "refuge habitat") and areas unsuitable for their development ("no habitat"), and (right) initial simulated population densities of S. frugiperda within the landscape shown in the left panel. Landscape is defined as a grid of 200×200 cells of 1 ha. For every simulation, a new landscape and initial population density is generated.

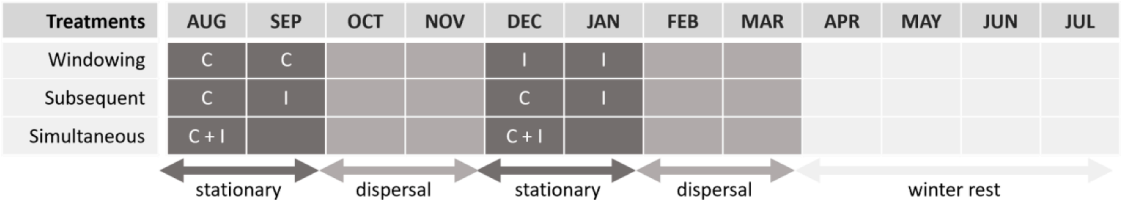

Fig. 3. Calendar of treatment protocols simulated in this study. "C" represents chlorantraniliprole and "I" represents indoxacarb. Three periods are considered: (i) a treatment period in dark-grey where S. frugiperda is considered to be stationary (i.e., eggs, larvae and pupae), (ii) a dispersal period (dark-grey) where only adults move over the landscape area, and (iii) a cool climatic period without growth and dispersal population dynamics. The selection processes happen during the stationary life-stages, allowing dispersed only for selected individuals, and overcoming the need to consider exposure of each individual during dispersal.

December–January) of each year which correspond to stationary periods of *S. frugiperda* in northern Australia (see Fig. 3). Three pest management strategies were implemented: (i) a "windowing" strategy, where a single pesticide is applied to each generation (chlorantraniliprole on the first generation during the August-September period and indoxacarb on the second generation during the December-January period); (ii) a "subsequent" strategy where both pesticides are applied subsequently on each generation with a month delay; and (iii) a "simultaneous" strategy, where both pesticides are applied at the same time and on both the first and second generations. These strategies were chosen based on industry consultation and reflect the range of management options presently being used and/or being considered by Australian farmers to control *S. frugiperda* in grain crops.

We assumed a spike of pesticide on the day of application and then an exponential degradation of treatment (*i.e.*, $\frac{dX_{i(t)}}{dt} = -\ kX_{i(t)}$, with X_i being either $X_C$or $X_I$as defined in Table 2) (EFSA, 2014). The amount of pesticide per ha was randomly selected within a range following permitted chemical application rates, which is 70-90 g/ha for chlorantraniliprole (350 g/kg) (permit number – PER89366) and 400-500 mL/ha for indoxacarb (150g/L) (permit number – PER89530).

2.3.2. Exposure-response relationship

The relationship between pesticide exposure and survival follows a classical sigmoidal shape based on data for the two chemical compounds against *S. frugiperda* (Hardke et al. 2011; Belay, Huckaba, and Foster 2012). The sigmoid shape is modeled with a log-logistic function given by:

$$f(x) = \frac{x}{1 + (x/LC_{50})^{-\beta}}$$

where x is the pesticide concentration, LC_{50} is the lethal concentration for 50% of the population and β the Hill coefficient defining the shape, or the slope, of the curve.

For chlorantraniliprole, Hardke et al. (2011) estimated an LC_{50} value (and 95% Confidence Intervals - C.I.s) after 72 h of 0.068 (0.060 – 0.077) ppm, with a slope (and s.e.) of 2.55 (± 0.23), while Deshmukh et al. (2020) found results with an LC_{50} value of 0.0129 (0.0092–0.0229) ppm and a slope of 0.9 (± 0.1) at 24 h after exposure. A different study found 25%, 50% and 85% mortality of *S. frugiperda* after 16 h, 48 h and 96 h, respectively when chlorantraniliprole was applied in the field at a rate of 85 g/a (Belay et al., 2012), while an average LC_{50} value (and 95% C.I.s) of 0.055 (0.052- 0.058) ppm with a slope (and s.e.) of 2.4 (± 0.1)(Bird et al., 2022) was estimated after an exposure period of 7 days.

For indoxacarb, an LC_{50} value was estimated at 0.392 (0.317–0.481) ppm after 72 h, with a slope of 2.35 (± 0.25) (Hardke et al., 2011), and 0.290 (0.145–0.435) ppm, with a slope of 0.6 (± 0.1) after 24 h (Deshmukh et al., 2020). When applied in the field at a rate of 85 g/a, mortality of *S. frugiperda* was measured to be 15%, 20% and 80% after 16 h, 48 h and 96 h, respectively (Belay et al., 2012).

While bioassay methods are different, we observe a great variability of toxicological values. In order to explore the largest range of possibility in the sensibility analysis, we considered a large range of toxicological value with an LC_{50} value ranging between 0.01 and 0.99 and a slope from 0.5 to 10 for both pesticides and distinguished these based on the amount of pesticide applied (see Table 2).

2.3.3. Pesticide resistance ratios

One study (Gutiérrez-Moreno et al., 2019) provides resistance ratios for chlorantraniliprole, denoted R, calculated by dividing the LC_{50} or LC_{90} values of field populations of *S. frugiperda* by the LC_{50} or LC_{90} values of a laboratory insecticide-susceptible colony. The resistance ratio for chlorantraniliprole was 160 when using LC_{50} values and 500 when using LC_{90} values. Other studies have shown far lower levels of field resistance, for instance a resistance ratio of 1–2 (Zhang et al., 2021). There have been no reported cases of field resistance to indoxacarb in *S. frugiperda* (e.g. Zhang et al., 2021). Additionally, Yu and McCord (2007) found no evidence for resistance to indoxacarb after 20 generations of exposure and selection in the laboratory.

2.4. Sensitivity analysis

A sensitivity analysis was conducted to identify the key factors and their interactions driving population dynamics in the stochastic spatio-temporal model. Our objective being to find how each of the parameters (and their combinations) influence the emergence of fully resistant populations of *S. frugiperda* at the landscape scale. We simulated 4500 scenarios, each corresponding to a duration of 4 years.

The sensitivity analysis consisted of four key steps:

1 Definition of the target parameters and their respective ranges of interest (Table 2).
2 Definition of the target simulation outcome statistic on which the sensitivity analysis is applied. Here, we computed the mean size of fully resistant populations, N_{CI} over the landscape for every time step over the final simulation year.
3 Sampling parameter values from an appropriate distribution defined by the parameter range (Table 2).
4 Computation of sensitivity indices (see below).

Sobol's index is a variance-based measure of sensitivity providing the sensitivity of an output variable to a selected input parameter. To compute Sobol's sensitivity index, 4500 simulations were conducted across "gridded-parameter space". The first order Sobol's index quantifies the contribution of the regression $E[Y|X_i]$ to the variance of Y (Saltelli et al., 2008).

$$Sobol_i = \frac{Var[E[Y|X_i]]}{Var[Y]}$$

In parallel with the Sobol's index, we computed the Pareto's frontier in order to find the best trade-off between resistance increase, pest density and chemical inputs. The Pareto's frontier is the set of parameterization having the same and maximal Pareto optimality considering a set of variables, that is the best trade-off where we cannot increase one variable without reducing at least one of the others (Roocks 2016).

3. Results

3.1. Response of resistance evolution to parameter variation

To better understand the correlation between variables, we computed pair-plots (Figure 4), which represent the mean amount of each sub-populations (*i.e.*, P_{CI}, P_C, P_I, and P_S) summed over the last year of the simulation, following: $\sum_{last\ year}(Mean_{landscape}(N_i))$. This was performed for 13 parameters. For each pair-plot representing pairwise plot of *sub-population density vs. parameter*, one black point corresponds to one simulation.

We then computed heatmaps, which correspond to the distribution density of the simulation endpoints for each pair-plot (Fig. 4). The heatmap allows for comparisons of parameters, for instance the amounts of chlorantraniliprole, X_C, and indoxacarb, X_I, exhibit a very similar pattern because of their identical role in the model. The pesticide killing rate, μ, also has the same pattern as the amount of pesticide, while the degradation rate, d, mirrors these patterns.

We used classical regression models applied over scatter plots (i.e., linear model, square model, second order polynomial model) to capture the trend of repartition of the endpoints. The graphics show that linear regressions are unable to capture the large variability in simulation results. It illustrates the non-linearity of density responses to changes in parameters, except, eventually, for the transition rates between phenotypes. While the three parameters driving transition rate have a strong influence on simulated resistance outcomes, all other parameters are much more difficult to interpret (Fig. 4).

3.2. Sensitivity indices: Sobol's index

The computation of sensitivity indices (Sobol's index and Pareto optimality) helps to provide another perspective. Fig. 5 presents the first-order Sobol's indices for the 13 parameters on the mean number of fully resistant populations to chlorantraniliprole and indoxacarb. As expected from the regression analysis and pair-plots shown in Fig. 4, transition rates between phenotypic states showed the strongest influence on densities of *S. frugiperda*. Unsurprisingly, $m_{S\to C}$ and $m_{S\to I}$ rates had a strong influence on the susceptible and mono-resistance populations while $m_{C/I\to CI}$ had a strong influence on the bi-resistant population (P_{CI}).

The second most influential group of mechanisms impacting the population dynamics of *S. frugiperda* was the properties of each pesticide (i.e., degradation rate, dose response and slope). Surprisingly, the amount of pesticide applied in the model had a lower impact than the pesticide properties. We note that since chlorantraniliprole was applied prior to indoxacarb (which is an artifact of the model), but is also more toxic to *S. frugiperda* as evidenced by published LC_{50} values (Bird et al., 2022), chlorantraniliprole had a stronger effect on population dynamics than indoxacarb. Finally, landscape composition and the dispersal rate of individual *S. frugiperda* had a relatively weak role in the population dynamics in our model.

3.3. Sensitivity indices: Pareto optimality

Representing simulation endpoints according to their Pareto optimality provides a Pareto frontier, which enables each treatment scenario to be ranked, thus indicating the overall best scenario globally. Fig. 6 provides the ranking of Pareto optimality according to different formula: "a" had the formula $low(\mathrm{N_{CI}}) * low(\mathrm{N}) * high(p_{crop}) * low(\mathrm{X_C}) * low(\mathrm{X_I})$, which means that the parameterization was optimized in order to minimize the population density of *S. frugiperda* resistant to both pesticides, minimize the total population size of *S. frugiperda*, obtain a high proportion of land as "crop habitat" and minimize the input of each pesticide; "b" was optimized to minimize the population density of *S. frugiperda* resistant to both pesticides and minimize the input of each pesticide; "c" was only constrained to minimize the population density of *S. frugiperda* resistant to both pesticides; "d" was parameterized to minimize the population density resistant to both pesticides and to minimize the population density of the total *S. frugiperda* population; "e"

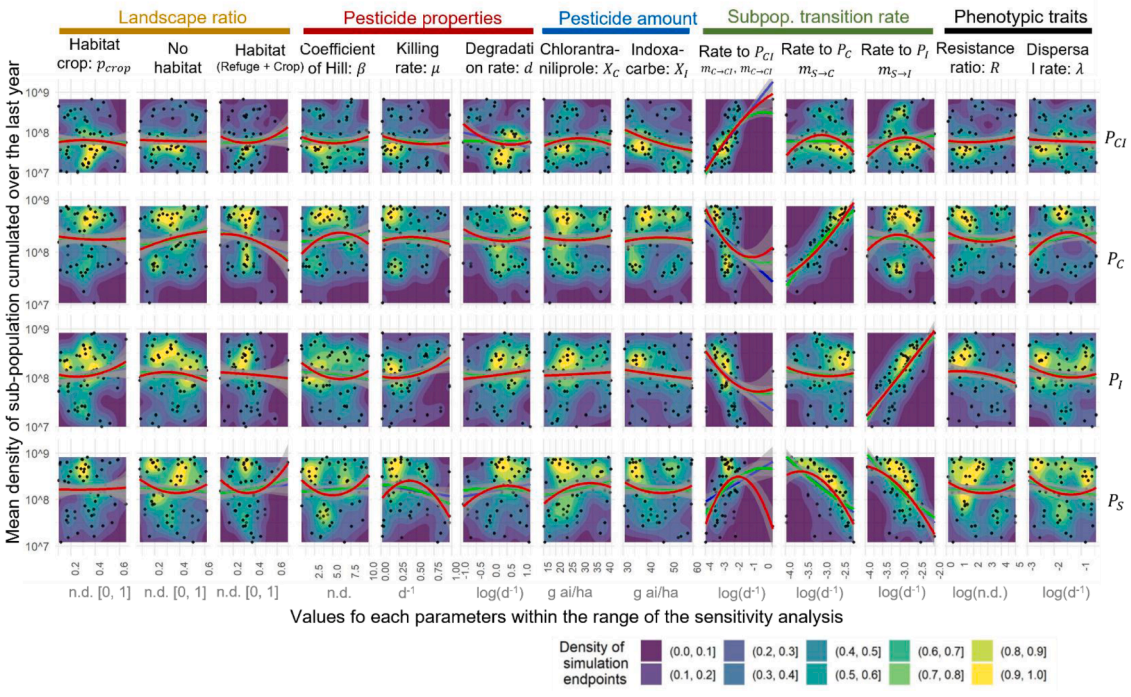

Fig. 4. Two dimensional-density plots, displaying the mean S. frugiperda abundance of each sub-population group ($9km^2$) summed over the final year of the simulations according to the range of parameters used in the sensitivity analysis. From the top row moving downwards P_{CI} is a fully resistant sub-population, P_C is resistant to chlorantraniliprole and susceptible to indoxacarb, P_I is susceptible to chlorantraniliprole and resistant to indoxacarb, and, P_S is a fully susceptible sub-population. Lines corresponds to the regression models: (blue) linear model, (green) square model, (red) second order polynomial model.

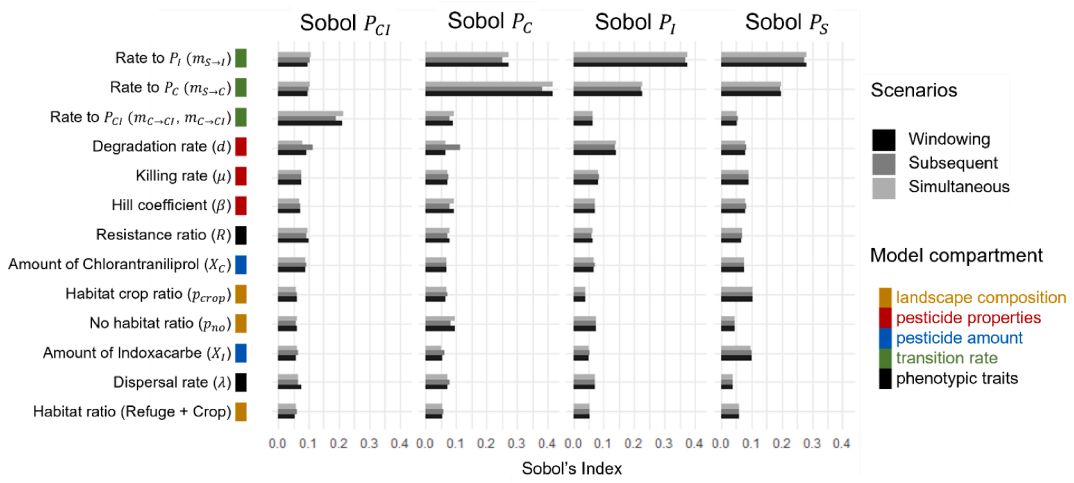

Fig. 5. First-order Sobol's indices for 13 target parameters on the mean number of S. frugiperda in a fully resistant population (P_{CI}), a population resistant to chlorantraniliprole and susceptible to indoxacarb (P_C), a population susceptible to chlorantraniliprole and resistant to indoxacarb (P_I), and a population susceptible to both pesticides (P_S). The grey bars correspond to scenarios of treatment protocols depicted in Figure 3.

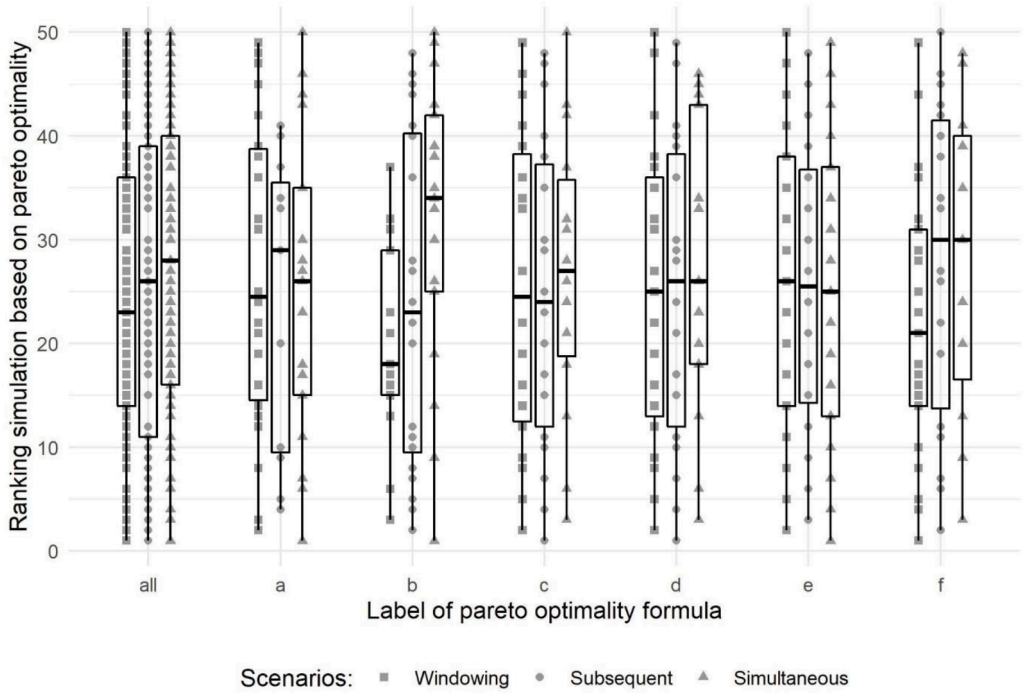

Fig. 6. Ranking of treatment scenarios using Pareto optimality, whereby the formulae used are: (a) $low(N_{CI}) * low(N) * high(p_{crop}) * low(X_C) * low(X_I)$; (b) $low(N_{CI}) * low(X_C) * low(X_I)$; (c) $low(N_{CI})$; (d) $low(N_{CI}) * low(N)$; (e) $low(X_C) * low(X_I)$; and (f) $low(N) * low(X_C) * low(X_I)$. (all) represents a pool of all these rankings into a single group, which is not a Pareto optimality.

" was parameterized to minimize the input of each pesticide; and "f" was parameterized to minimize the input of each pesticide and minimizing the total population size.

The Pareto optimality indicates that on average, the "windowing" treatment scenario was optimal for five of the seven formulae investigated. More specifically, as long as the size of population or sub-population was considered within the Pareto formula, the windowing scenario provided the best fit as indicated by the medians. We also pooled these rankings into a single group ("all"), which although is not a Pareto optimality ranking, it does enable the combination of all these scores to be visualized (Fig. 6).

4. Discussion

The development of RMSs is challenging because of the numerous genetic mechanisms, ecological processes and socio-economical constraints that are involved (REX, 2013; Gould et al., 2018). More specifically, the spatial component in RMSs for insecticide application and population dispersal is a crucial parameter (Yamamura 2021). Here, we explored the sensitivity of 13 parameters dealing with landscape structure, dispersal rate, treatment protocols, chemical degradation rate, dose-response curves and transition rates between sub-populations of the highly invasive global pest, *S. frugiperda*. From sensitivity simulations, we were able to develop a set of heat maps showing the influence of each parameter on a set of variables. The modelling approach we used

is heuristic in the sense that it uncovers the complexity behind resistance management and provides a working example of how spatially explicit mechanistic models can help to tackle this complexity. The purpose is to consider idealized scenarios where underlying constraints can be manipulated to reveal key processes and their measures that could lead to effective resistance management (Storer et al., 2003).

4.1. No isolated drivers of resistant outbreaks

A clear output of the modelling approach is the lack of a single obvious parameter that drives the population dynamics, and consequently the outbreak of resistance within a population. In other words, the challenge facing resistance management is the need to consider multiple disparate processes within the same approach, as previously conveyed by others (e.g. Onstad 2013; Gould et al., 2018). While we reduced the complexity of the system to account for only 13 parameters driving the population dynamics of four phenotypes in *S. frugiperda*, the high variability prevents the identification of any discrete RMS that is optimal in minimizing resistance in this species. But knowing this can work as a "null" model (see Harte 2004), the non-linearity (no identifiability of pattern) between parameters and variables of interest supports the notion that accurate resistance predictions are only possible when considering the whole complexity. That being said, our model is generic and the parameter space we explored was quite large. The failure of our model to identify simple recommendations demonstrates the need to reduce parameter uncertainty with empirical testing in *S. frugiperda* (Harte 2004) and/or to test the sensitivity of inputs on the model outputs.

4.2. Sensitivity indices to optimize control strategies

The exploration of a large range of parameter values illustrates that the emergence of resistant populations is highly dependent on the ecological context. The sensitivity analysis shows that the transition rate between phenotypic states has the strongest influence on the dynamics of *S. frugiperda* populations. This is expected since they account for the introduction of new phenotypes to be selected through other variables (particularly pesticide applications). Then, as a consequence of selection pressure and pesticide properties, such as the degradation rate of the product after spraying and dose-response properties (i.e., killing rate and slope), resistance outbreaks occur. Pesticides are by design a strong selective force that can lead to rapid evolution (Hawkins et al., 2019), which is widely acknowledged to be highly influenced by the properties of the pesticide (e.g., see Bird et al., 2022).

Conversely, the landscape composition and the dispersal rate of individuals appears to play a relatively small role in the population dynamics of *S. frugiperda*. Mechanistically, from the model structure, (Fig. 1), landscape parameters are the farthest in the chain of events leading to population density and so it may be difficult for sensitivity indices, like Sobol's index, to reveal a non-monotonic (e.g., bell-shaped, concave) link between the model input and output (Allard and Fischer 2009). Looking at the pair-plots (Fig. 4), the dispersal parameter, λ exhibits a concave shape, meaning that the middle of the range is a minimum (P_S and P_I) or a maximum (P_C and P_I). An explanation is that dispersal is required for the invasion of resistance once it emerges, but very high dispersal rates may compromise the emergence of resistant individuals, due to competition with susceptible individuals that invade crop fields after the degradation of pesticides. At extremely high dispersal rates the whole landscape becomes homogeneous, and refuges for susceptible individuals limit the invasion of resistant phenotypes through genetic dilution, fitness costs and competition (Takahashi et al., 2017). The trade-off between competition and dispersal may limit the coexistence of multiple-phenotypes as already demonstrated in heterogeneous landscapes (Snyder and Chesson 2004; North and Ovaskainen 2007).

4.3. Practical implications and limitations

Our model was necessarily simplified compared with the reality experienced in the field, due to several of the assumptions of the underlying processes. While variation in parameter values partially reflects the uncertainties, it can also reflect differences in management practices. For instance, while it is hard to manipulate the transition rate (which includes the genetic resistance mutation rate between subpopulations) or dispersal of pest populations, farmers have influence over the amounts of pesticides used, the treatment protocols followed and the proportion of different crop types grown within a landscape, and agri-chemical companies may change the toxicological properties of their pesticide products. The development of RMSs is ultimately a matter of co-construction between different stakeholders, since each has a manageable variable on which to play (Gould et al., 2018).

The discontinuous fall armyworm activity and insecticide regimes tested in our study do not cover the full spectrum of population dynamics and treatment protocols that are seen in practice across Australia. For example, in warmer regions there will be larger number of generations, more continuous pest activity and a (generally) higher frequency of insecticide use. Under these circumstances it is likely that resistance would develop faster due to the stronger selection pressure. Conversely, in cooler climates, pest activity would be less (low intensity, with more frequent or longer periods of no activity). In these situations, it is likely that resistance would develop more slowly. Indeed, other studies focussing on the climatic effects of resistance evolution support these general relationships (Maino et al., 2018b). In any case, resistance management strategies can be locally designed so they are appropriate to different regional ecological and farm management contexts.

Our modelling suggests a key driver for an increase of resistant in *S. frugiperda* is transition rate, which farmers have little control over (if not by keeping the population size low enough to reduce the chance of mutations arising) highlighting the difficulty of applying pre-emptive RMSs. However, the degradation rate of pesticides, which in part is influenced by the product formulation, also has an important influence on resistant levels and warrants greater attention by field practitioners and others designing RMSs. Interestingly, while degradation rate (i.e., decay rate) of the pesticides seems very important for risk-assessment of non-target species (Ali et al., 2021) and in product formulation and design (Cloyd 2011), to our knowledge, this factor is not well integrated into RMSs although it is a component of some older models (C. E. Taylor and Georghiou 1982; Charles E. Taylor et al., 1983).

4.4. Comparison of treatment protocols

Avoiding the repeated use of the same chemical pesticide, or the same MoA group, is a recommended approach to limit pesticide resistance and has been termed "windowing" (Sparks and Nauen 2015). The two pesticides we consider here, chlorantraniliprole and indoxacarb, have different modes of action, the first one disrupts muscle functioning that leads to paralysis and the second blocks neuronal activities (IRAC - Mode of Action Database 2022). As such, we assume the resistance mechanism for one of these pesticides would not affect resistance to the other one (Bird et al., 2022). Furthermore, our model does not include a consideration of the precise resistance mechanism but assumes independence between selective responses.

In this study, we simulate three strategies: (i) "windowing", which involves the application of a single pesticide at each generation of *S. frugiperda*; (ii) "subsequent", which introduces a weak-windowing strategy as the two generations of *S. frugiperda* receive both pesticides but sequentially, and (iii) "simultaneous", which involves the application of both pesticides at the same time but only once in each generation. The global sensitivity analysis does not reveal a strong difference between treatment protocols in the presence of large uncertainty in other model parameters, but the Pareto optimality ranking indicates that the "windowing" strategy, on average, performed better than the other two.

It is also interesting to see that the "simultaneous" strategy (which involves no windowing of pesticides) was the worst-performing, on average. It is important to note that model assumes a clear separation of generations in the field, which was important to allow for heritable phenotypes. However, *S. frugiperda* can produce multiple and overlapping generations in a given year (Nagoshi et al., 2014), which is likely to reduce the effectiveness of a windowing strategy.

5. Conclusion

The objectives of this study were to (i) implement a generic framework of population resistance at landscape scale to explore a large set of parameters that are known to be important in field resistance, and (ii) use this framework to explore three different control strategies available to farmers ("windowing", "sequential", "simultaneous"). Unfortunately for Australian growers wishing to implement a standardized RMS for *S. frugiperda*, we did not find a universal treatment strategy that was predicted to outperform all other strategies. Furthermore, we only found a strong predictive signal between the emergence of resistance and transition rate (i.e., flux between sub-population driven by the mutation rate), which beyond suppressing the total population size, is not a manageable process. Future research aimed at developing resistance management strategies for fall armyworm should focus on reducing the uncertainty in biological parameters (e.g. through experimental studies), and more carefully consider local ecological and management conditions.

CRediT author statement

Virgile Baudrot: Methodology, Software, Formal analysis, Writing Original Draft and Review. Rafael Schouten: Methodology, Software, Data Curation. Paul A. Umina: Conceptualization, Writing Original Draft and Review, Supervision. Ary A. Hoffmann: Writing Original Draft. Lisa Bird: Data Curation, Writing Original Draft. Melina Miles: Data Curation, Writing Original Draft. James L. Maino: Methodology, Software, Formal analysis, Writing Original Draft and Review, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ecolmodel.2023.110416.

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