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RESEARCH ARTICLE

Landscapes of risk: A comparative analysis of landscape metrics for the ecotoxicological assessment of pesticide risk to bees

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Abstract

1. Pesticide use in agricultural landscapes creates environmental contamination that is heterogenous in space and time. Mobile organisms, such as bees, are exposed to multiple contamination sources when visiting patches that vary in the amount, timing and toxicity of pesticides used. Yet, environmental risk assessments (ERA) typically fail to consider this heterogeneity, in part because of the complexities of estimating exposure to different pesticides, and subsequent risk at organism-relevant scales.
2. We use pesticide assays of 269 bee-collected pollen samples to understand the spatiotemporal variability of risk across a network of 41 field sites in southern Sweden. Observed bee pesticide risk is calculated based on compound-specific residue quantifications in pollen and standardized toxicity data. We then compare the ability of three classes of landscape-scale variables to predict this risk: (1) landscape composition and configuration metrics, (2) landscape load based on national pesticide use data and (3) predictions from a newly developed bee pesticide exposure model.
3. Based on use data, 10 crops account for 81% of the total risk. We detected 49 pesticide compounds in bee-collected pollen. Although herbicides and fungicides constitute the bulk of detected pesticides, both in frequency and amount quantified, unsurprisingly, insecticides contribute the most to risk.
4. Landscape composition and configuration metrics did not predict observed pesticide risk, and interactions with bee species indicate taxa-dependency in predictions. Landscape load predicted observed risk consistently between taxa. Risk estimates from our exposure model were strongly predictive but only

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when considering realized risk (i.e., risk estimates based on prior pesticide use information).

5. *Synthesis and applications.* Predicting pesticide risk based on landscape patterns could enable landscape-scale ERA. However, simple metrics of landscape pattern, such as proportion of agricultural land, are not sufficient. We found that risk observed in bee-collected pollen was best predicted when integrating spatialized pesticide use in the pesticide exposure model, underscoring the importance of such data for research, monitoring and mitigation. Further, we propose a guidance framework for future landscape ecotoxicological risk analyses that clarifies data needs relative to risk prediction goals.

KEYWORDS

Apis, bee, biomonitoring, *Bombus*, environmental risk assessment, landscape ecotoxicology, *Osmia*, pesticide

1 | INTRODUCTION

Exposure to agricultural pesticides threatens human health (Larsen et al., 2017) and biodiversity (Beketov et al., 2013; Rigal et al., 2023). Preventing the social, ecological, and economic consequences of these non-target effects are goals of environmental risk assessment (ERA), pesticide reduction strategies (e.g., integrated pest management) and associated policies (e.g., EU 50% reduction in use and risk by 2030). Spatiotemporal patterns of pesticide exposure can critically inform these efforts by identifying where greatest potential impact occurs and therefore opportunities for risk mitigation. This exposure is influenced by ecotoxicity, life history and the spatiotemporal distribution of both pesticide uses and non-target organisms (Cairns & Niederlehner, 1996). However, most ERAs are based on often poorly validated exposure assumptions or models that do not account for chemical and organismal traits and largely ignore spatial and temporal variability (Price & Thorbek, 2014).

Landscape ecology has long provided a theoretical framework for analysing the responses to and effects of spatial pattern and scale, yet ecotoxicology is only recently incorporating landscape structure, function and change into replicated assessments of pollutants in the environment (Beketov & Liess, 2012; Schäfer, 2014). Studies have shown that landscape composition and configuration can affect pollutant transfer in food webs (Fritsch et al., 2011; Vermeulen et al., 2009), pollutant impacts on stream macroinvertebrate abundance (Schriever et al., 2007), water quality (Collins et al., 2019; Liess & Von Der Ohe, 2005) and pesticide use at large scales (Meehan et al., 2011; Nicholson & Williams, 2021; Paredes et al., 2021). Pesticide application rates result in landscape patches that vary in the amount of compounds applied, hereafter pesticide load (Box 1). The underlying processes that create heterogenous exposure are that exposure frequency, duration and intensity varies according to landscape pattern because habitat size, suitability and spatial arrangement affect the way organisms exploit, and are exposed to their environment (Fahrig & Freemark, 1994; Fritsch et al., 2012). These processes result in landscape conditioned exposure to organisms, hereafter landscape exposure (Box 1).

Landscape exposure to pesticides is of high relevance for bees because they forage on crop and non-crop resources over large areas that are directly treated or unintentionally contaminated with pesticides (Knapp et al., 2023; Nicholson et al., 2023; Végh et al., 2022). Bees, and other mobile organisms with large foraging ranges, can integrate multiple sources of exposure by visiting spatially separated patches that vary in the amount, timing and toxicity of contamination. In agricultural landscapes, this results in two key patterns: (1) spatiotemporal heterogeneity of exposure (Sponsler et al., 2019) and (2) exposure to multiple toxic compounds (Lonsdorf et al., 2024; Rundlöf et al., 2022). Indeed, recent biomonitoring of bee pesticide exposure demonstrates the presence of multiple compounds in bee food and nesting material (e.g., flowers—Bloom et al., 2021; Graham et al., 2022, pollen—Calatayud-Vernich et al., 2018; Knapp et al., 2023; McArt et al., 2017; Tosi et al., 2018, nectar—Knapp et al., 2023, colony structures—Mullin et al., 2010; Ravoet et al., 2015), and with both crop (Favaro et al., 2019; McArt et al., 2017; Végh et al., 2022) and non-crop (Graham et al., 2022; Wood et al., 2019) sources of contamination. Recently, we found that bees' exposure, and subsequent risk, was influenced by the proportion of agricultural land, and that this landscape effect varied in strength with the foraging traits of different bee species (Knapp et al., 2023). Yet, exploring landscape exposure across many exposed species and landscape contexts through field-based biomonitoring is a major challenge given costs, potential for interactive effects, and number of combinations between pesticides and non-target organisms.

Alongside field-based biomonitoring of pesticides, progress has been made simultaneously in the development of spatially explicit ecotoxicological models (Wickwire et al., 2011)—partly out of necessity, given complexities of landscape exposure assessments (Morrissey et al., 2023). Model scenarios and predictions that help characterize landscape variables that influence exposure, coupled

BOX 1 A landscape ecotoxicology glossary for bees and pesticides

Active ingredient: A compound in a pesticide product that is intended to control pests.

Application rate: The amount of an active ingredient or pesticide product over a given unit, usually area (e.g., g/ha).

Exposure: The level (e.g., concentration, amount and intensity) of a compound that reaches organisms through one or several sources (e.g., soil, air and food) and routes (e.g., contact, ingestion and dietary).

Toxicity: The capacity of a compound to cause adverse effects on organisms at a given exposure level.

Risk: The potential for adverse effects to organisms given exposure and toxicity.

Landscape load: The amount of a compound applied in the patches of a landscape, standardized by total landscape area. *Total landscape load* summarizes *landscape load* across all compounds.

Landscape toxic load: The toxicity-weighted amount of a compound in a landscape. Calculated as the ratio of landscape load to compound-specific toxicity. *Total landscape toxic load* summarizes *landscape toxic load* across all compounds.

Landscape exposure: The level (e.g., concentration, amount and intensity) of a compound that reaches organisms in the landscape as a result of the way organisms interact with landscape patterns.

Landscape risk: The toxicity-weighted *landscape exposure*. Calculated as the ratio of landscape exposure to compound-specific toxicity. *Total landscape risk* summarizes *landscape risk* across all compounds.

Potential versus realized load, exposure and risk: The detection, or not, of compounds depends on different and compounding factors among load, exposure and risk. *Potential landscape load* are compounds applied in a landscape, while *realized landscape load* is what is measurable given analytical methods (e.g., detection limits) and compound environmental fate. *Potential landscape exposure* is potential load but considering organisms' interaction with landscape patterns, while *realized landscape exposure* considers that organisms may be differently active in patches of a landscape. *Potential landscape risk* is potential exposure considering toxicity, while *realized landscape risk* considers that toxic compounds may kill organisms before they can be monitored.

with strategically planned biomonitoring of pesticides, would aid higher tier ERA goals of evaluating spatially explicit exposure patterns over time (Simon-Delso et al., 2017; Sponsler et al., 2019; Topping et al., 2020). To this end, we have developed a detailed predictive framework (Lonsdorf et al., 2024) that links the spatial and temporal mosaic of bee habitat, bee foraging movement and pesticide use to generate spatially explicit patterns of pesticide exposure to bees. Apart from complementing biomonitoring, such predictive models can help identify when and where to implement mitigation strategies to prevent pesticide impacts on non-target bees and other pollinators (Baveco et al., 2016). This spatial planning is a critical component of IPM strategies that aim to integrate pollinator health (i.e., IPPM) through efficient landscape management (Lundin et al., 2021). However, understanding the spatiotemporal heterogeneity of pesticide use, let alone predicting exposure, is often challenging due to a lack of data on pesticide use. Although some places contain rich information on the use of pesticides (e.g., California's Pesticide Use Reports), typically this information is unavailable or at coarse levels of aggregation (e.g., Douglas et al., 2022). Thus, a point of practical importance is whether more readily available measures of land use can be used to predict pesticide risk.

We address this question through comparative analysis of three classes of landscape variables on observed pesticide risk based on pesticide residue quantification in pollen samples ($N = 269$) from a network of 41 sites distributed across southern Sweden (Figure 1a). Our first class of variables are metrics of landscape composition and configuration typical to landscape ecology studies. Our second class are metrics of landscape-level pesticide use. Our third class are predictions from a bee exposure model that integrates landscape-level pesticide use with the foraging ecology of bees. Using compound-specific toxicity values, we calculate pesticide risk to bees for these last two classes (Box 1). We expected that (1) observed pesticide risk would increase with greater dominance of agricultural land (Knapp et al., 2023), (2) incorporating information on the spatiotemporal pattern of pesticide use would improve predictions (Nicholson & Williams, 2021) and (3) the fit between observed and predicted pesticide risk would be improved when accounting for bee foraging ecology (Lonsdorf et al., 2024).

2 | MATERIALS AND METHODS

2.1 | Field biomonitoring network, sentinel bees and pollen pesticide residues

Our biomonitoring network ($N = 57$ site-years) consists of three species (*Apis mellifera* L., *Bombus terrestris* L. and *Osmia bicornis* L.) of sentinel bee colonies and nests distributed across 41 sites in southern Sweden from 2019 through 2021 (Figure 1a; site network centroid: 55°43′09.2″ N, 13°47′12.2″ E; site network extent: 7768 km^2). These sites form the basis for completed (Jonsson et al., 2022; Knapp et al., 2022, 2023) and ongoing projects (Rundlöf & Andersson unpublished data; see Supporting Information, Methods). From each site's set-up, we collected pollen, froze samples (−20°C) and

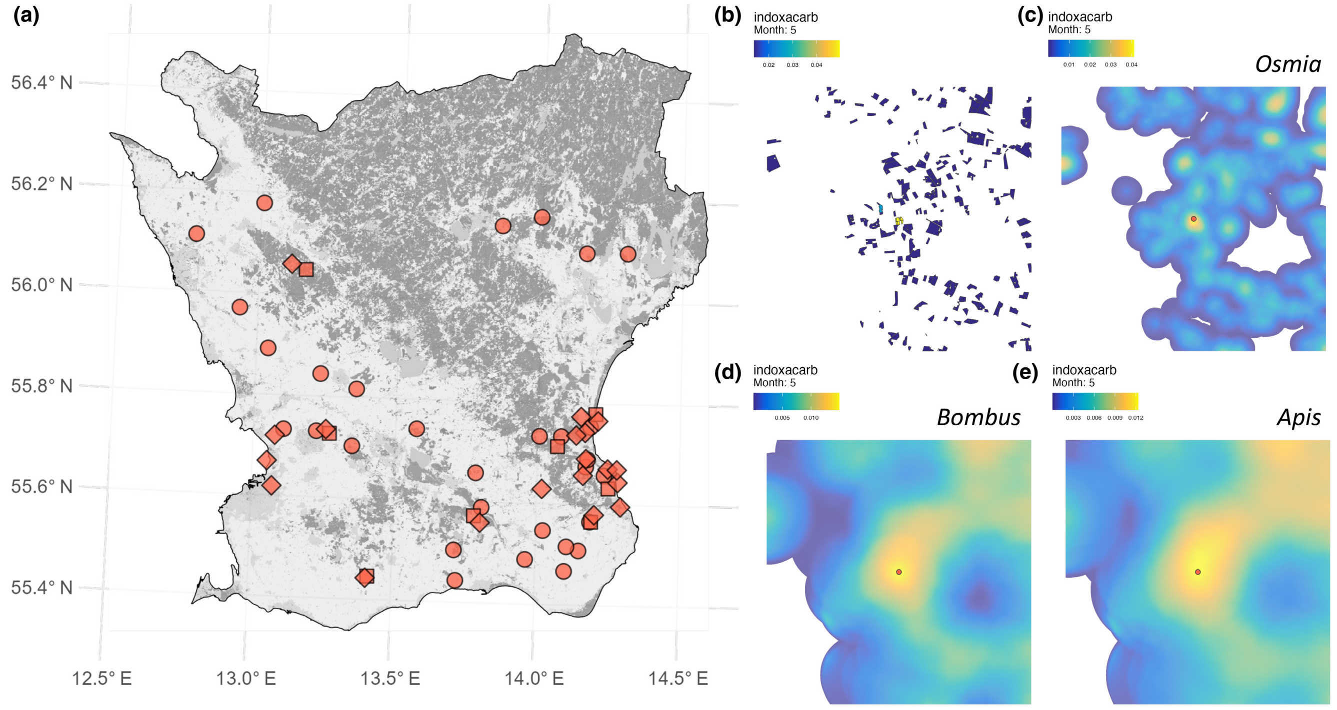

FIGURE 1 Southern Sweden biomonitoring network of 41 sites (a) sampled variously in 3 years (circles: 2019, squares: 2020, diamonds: 2021). At each of these sites we calculate landscape metrics of composition, configuration and pesticide use. We spatialized a national dataset of pesticide use records to generate maps of landscape load (b). We applied our pesticide exposure model to these data for three representative bee species with different foraging ranges: *Osmia bicornis* (500 m), *Bombus terrestris* (1500 m), and *Apis mellifera* (2000 m). The output of this model provides exposure predictions that are species-, compound-, month- and site-specific (c–e). The colour bar in the example pesticide landscape load map (b) depict compound application rate (g/100 m^2). The colour bars in example landscape exposure model outputs (c–e) depict estimated exposure (unitless).

screened each for pesticides (see Supporting Information, Methods). Fieldwork was done with the approval of landowners and managers, and no permits or ethical approval was needed.

2.2 | Pesticide use data

We acquired pesticide use data from Statistics Sweden (SCB), which conducts national surveys to collect differentiated statistics of pesticide use in crops to track use over space and time (see Supporting Information, Methods). Following Douglas et al. (2022), we applied a 'downscaling' approach to match crop- and month-specific use averages to land cover. Specifically, we used 78,111 application records from the two most recent surveys (2017 and 2021) to calculate monthly crop-specific application rates for all active ingredients (AI) by averaging the amount (g) of AIs in each product applied over a reported crop area (deciares). Based on crop identity, we joined these estimated pesticide application rates (g/100m^2) to national agricultural parcel data provided by the Swedish Board of Agriculture (IACS Spatial Data Layer). We used this spatialized pesticide information (Figure 1b) to calculate landscape-level metrics of pesticide use (Box 1) and as input for our exposure model (see below).

2.3 | Landscape data

We analysed the landscape surrounding our sites at three spatial scales according to the average foraging range for our three genera (*Osmia*: 500; *Bombus*: 1500; and *Apis*: 2000 m; Kendall et al., 2022). Using the annually updated IACS Spatial Data Layer, we classified land cover categories into agricultural land (i.e., annual crops, orchards and leys) and calculated the proportional coverage within circular buffers with radii corresponding to the three foraging ranges. Using the pesticide use data, we classified landcover types based on whether they received pesticide application and calculated the proportion of pesticide-treated area in the buffers. We calculated landscape crop diversity with a Shannon index of crop types (richness) and their coverage (evenness). As a measure of landscape configuration, we calculated the interspersion and juxtaposition index (IJI) for each landscape (McGarigal & Marks, 1995). We chose IJI because it was less correlated with the proportion of agricultural land compared to other configuration metrics (e.g., edge density, mean patch area; Figure S1). Lastly, we calculated total landscape pesticide load using the spatialized pesticide use data as:

$$\mathrm{Load}_t = \sum_{i=1}^{n} \sum_{j=1}^{n} \frac{r_{it} a_j}{A},$$

where we summed monthly compound application rates (r_{it}) for compound i multiplied by area (a_j) of crop j divided by the foraging range-defined buffer area (A) around a site.

2.4 | Modelling bee pesticide exposure

We used a previously developed and validated mechanistic model (Lonsdorf et al., 2024) to predict pesticide exposure to bees in the landscape. We calculated exposure at nest site i from forage site j in month t, $E_{C_{ijt}}$, as:

$$E_{C_{ijt}} = \left(z_c P_{cjt}\right)\left(\frac{F_{jt} e^{-\frac{D_{ij}}{\gamma}}}{\sum_{j=1}^{n} F_{jt} e^{-\frac{D_{ij}}{\gamma}}}\right),$$

where the first term represents the amount of compound C available during the visit to site j in month t and the second term represents the probability of foraging in site j from nest site i. In the first term, the scalar, z_c, describes the proportion of compound C that would be collected given how much is available on site j at time t, here always equal to one. In the second term, the model assumes that the likelihood bees will travel from nest site i to forage in site j depends on the distance, D, between them and on the floral quality, F, of the site—as D_{ij} increases and F_{jt} decreases, the likelihood of visiting the site declines (Figure 1c–e). The parameter, γ, represents the average foraging distance a bee would travel from its nest (i.e., 500, 1500 or 2000 m). As inputs, the model requires spatial information on pesticide loads, bee nest locations and floral resources (see Supporting Information, Methods).

2.5 | Risk calculations

We calculated toxicity-weighted exposure (TWE) to estimate the direct pesticide risk to bees (Knapp et al., 2023; Rundlöf et al., 2022), where the TWE for each compound (TWE_i) is the ratio of its exposure value (c_i) and its respective acute toxicity endpoint (LD_{50i}). Following the recommended concentration addition approach (EFSA Scientific Committee, 2019), we summed TWE_is to calculate risk (see Supporting Information, Methods). Using this approach, we calculate pesticide risk from different information sources. From pollen samples, c_i are the detected pesticide residues (ng/g). From the spatialized pesticide use data, c_i are AI-specific loads (i.e., $r_{it}a_j$). From the bee exposure model, c_i are predicted exposure values (i.e., $E_{C_{ijt}}$). When summarized across AIs, these provide measures of observed risk, total toxic landscape load and total landscape risk (Box 1), respectively. Finally, to facilitate comparisons of risk between AIs (Table 1; Table S2), we quantified compound-specific risk (PRQ) as the average of concentrations across samples for a given AI divided by its respective average LD_{50} and multiplied by its site detection frequency (Sanchez-Bayo & Goka, 2014).

2.6 | Potential versus realized risk and load

Our predictors of load and risk will depend on the type and number of AIs included in risk summaries. Recognizing that in many situations, the identities of pesticides used are unknown, we calculate *potential* risk and load based on the common set of screened AIs, and the recorded applications in our pesticide use data. These 'screen lists' are based on the Swedish national monitoring scheme (Boye et al., 2019) but differed slightly between years and contained between 66 and 72 compounds. Thus, *potential landscape load* would, for example, summarize load based on area-weighted application rates (i.e., $r_{it}a_j$) of all compounds that *could be* detected. We contrast these 'potential' measures with metrics of *realized* risk and load, which use the residue information to identify a subset of detected AIs across which to summarize. Thus, *realized total landscape risk* would, for example, estimate risk based on exposure predictions

TABLE 1 Compound-specific pesticide risk based on the relevant detection rate and concentration. Pesticide identity, type (I, insecticide; F, fungicide; H, herbicide; N, nematicide), compound group, toxicity (average acute and contact LD_{50} for *Apis mellifera* adults, μg per bee), method detection limit (μg kg^{-1}), frequency of detection, concentration (average, μg kg^{-1}) and compound-specific risk (PRQ) of the 10 riskiest compounds (for all compounds see Table S2). These 10 compounds were detected in all sentinel bee species (*A. mellifera*, *Bombus terrestris*, *Osmia bicornis*) and years (2019–2021).

AI	Compound group	LD50 mean	Detection limit	Detection frequency	Concentration average	PRQ
Indoxacarb (I)	Oxadiazine	0.156	1.00	47 (17%)	268	300
Acetamiprid (I)	Neonicotinoid	11.3	0.0500	128 (48%)	29.5	1.24
Penconazole (F)	Triazole	7.10[a]	0.500	24 (9%)	85.9	1.08
Imidacloprid (I)	Neonicotinoid	0.0420	0.500	22 (8%)	0.515	1.00
Thiacloprid (I)	Neonicotinoid	28.1	0.0100	148 (55%)	22.2	0.436
Azoxystrobin (F)	Strobilurin	113[a]	0.500	105 (39%)	67.3	0.234
Tebuconazole (F)	Triazole	142[a]	1.00	34 (13%)	227	0.203
Prosulfocarb (H)	Thiocarbamate	91.7	0.300	61 (23%)	46.9	0.116
Fluopyram (F)	Benzamide	101[a]	0.0500	153 (57%)	17.2	0.0967
Metamitron (H)	Triazinone	98.6[a]	0.100	72 (27%)	17.7	0.0481

[a] LD_{50} based on limit tests.

(i.e., $E_{c_{ijt}}$) of all compounds that were detected in the pollen sample for that site.

2.7 | Statistical analyses

We tested whether observed pesticide risk can be predicted by: (i) metrics of landscape composition and configuration (proportion agricultural land, proportion treated agriculture land, crop diversity, IJI), (ii) metrics of landscape-level pesticide use (total landscape load, total landscape toxic load) and (iii) total landscape risk based on predictions from the exposure model.

Given a strong right skew, we log transformed (ln(*x*)) observed risk. Given strong right skews and the presence of zeros, we log-transformed ($\ln(x+c)$) predictor risk and load values, using a AIC-based constant (*c*) selection method (Ekwaru & Veugelers, 2018) for each metric (*c* value range: 1e-07–1e-02). To allow comparison of regression coefficients, all predictors were standardized (*Z*-scores). For each predictor, we specified separate linear mixed effect models (LMM) with month, year, and that predictor in interaction with bee species as fixed effects, and with site as a random effect. We reported marginal R^2 values calculated following the methods of Nakagawa and Schielzeth (2013). We constructed LMMs with the 'lme4' package (Bates et al., 2015). We estimated slopes with the 'emmeans' package (Lenth, 2023) and evaluated models for overdispersion, normality and multicollinearity using diagnostic functions in the 'performance' package (Lüdecke et al., 2021) and the 'DHARMa' package (Hartig, 2022). We performed analyses and data visualization using R version 4.1.1.

3 | RESULTS

3.1 | National pesticide use data

Pesticide use in Sweden is dominated by herbicides, followed by fungicides, applied mainly May–October (Figure 2; Figure S2). A total of 143 AIs were reported across 44 crops, including 62 herbicides, 51 fungicides, 21 insecticides, eight plant growth regulators and one molluscicide. Despite the comparatively small amount of insecticides used (6% of reported pesticide uses), these compounds contribute most to total risk for bees (Figure 2; Figure S2).

Ten crops account for 81% of total risk from reported pesticide uses (Figure 2a), with the risk posed by these 10 crops attributable to a few, broadly used insecticides (Figure 2b). High-risk insecticides include pyrethroids (cyflutrin, alpha-cypermethrin), as well as the oxadiazine indoxacarb (Figure 2b).

3.2 | Pesticide residues in pollen

A total of 49 AIs were detected in pollen samples across the three bee species (*A. mellifera*, *B. terrestris* and *O. bicornis*), including 27 fungicides, 15 herbicides, 5 insecticides, 1 acaracide and 1 plant growth regulator. Most pollen samples contained multiple pesticides (96% with >1 compound; median: 6; range: 1–19). The compounds with the greatest compound-specific risk were insecticides (Table 1) but some herbicides (e.g., prosulfocarb and metamitron) and fungicides (e.g., penconazole and azoxystrobin) also ranked highly. As with the pesticide use data, while herbicides and fungicides comprised the bulk of total pollen detections (75%) and residues (58%, in $\mu g\,kg^{-1}$), insecticides generated most (>99%) of the compound-specific risk (Table S2). These high-risk insecticide AIs include neonicotinoids (acetamiprid, imidacloprid and thiacloprid), as well as an oxadiazine (indoxacarb) (Table 1).

3.3 | Landscape predictors of observed pesticide risk

Observed pesticide risk was best explained by landscape metrics based on pesticide information: total landscape risk, total landscape toxic load, and total landscape load (Figure 3; Table S5). We did not detect an interaction between these metrics and bee species (all $p>0.05$), and thus, we report coefficient estimates with this interaction removed. Both fit (standardized coefficient estimates) and explained variance (marginal R^2) depended on whether our risk predictor was constructed from the list of compounds that were screened (i.e., *potential* risk) or the list of compounds that were detected (i.e., *realized* risk). If considering realized risk (Figures 3 and 4 circles), the landscape risk predictions from the exposure model explained substantial variation in observed risk ($R^2_m=0.64$, Figures 3 and 4). However, if considering potential risk (Figures 3 and 4, squares), landscape metrics based on pesticide information performed similarly (R^2_m: 0.19–0.27, Figure 3).

The proportion of agricultural land explained observed risk but depended on bee species ($F_{2,246.3}=3.379$, $p=0.04$, Figure 4; Table S3), with risk tending to increase with the proportion of agricultural land for *O. bicornis* and *B. terrestris*, although individual slopes did not differ significantly from zero. We also detected an interaction between bee species and landscape crop diversity ($F_{2,244.5}=4.118$, $p=0.017$, Figure S3; Table S3), with risk decreasing significantly with increasing crop diversity for *A. mellifera* (slope: −0.86 [95% CI: −1.61, −0.10]). We did not detect positive taxa-consistent responses of observed risk to landscape juxtaposition (IJI) or to proportion of treated agriculture (Table S3). Year and month were significant fixed effects in most models (Table S3).

4 | DISCUSSION

Mobile organisms, such as bees, experience exposure from multiple, spatially separated and heterogenous sources. Given the potential for this landscape-scale exposure, ecotoxicological evaluations must be informed by analysis of broader landscape patterns. Commonly available information of landscape pattern could be a valuable risk

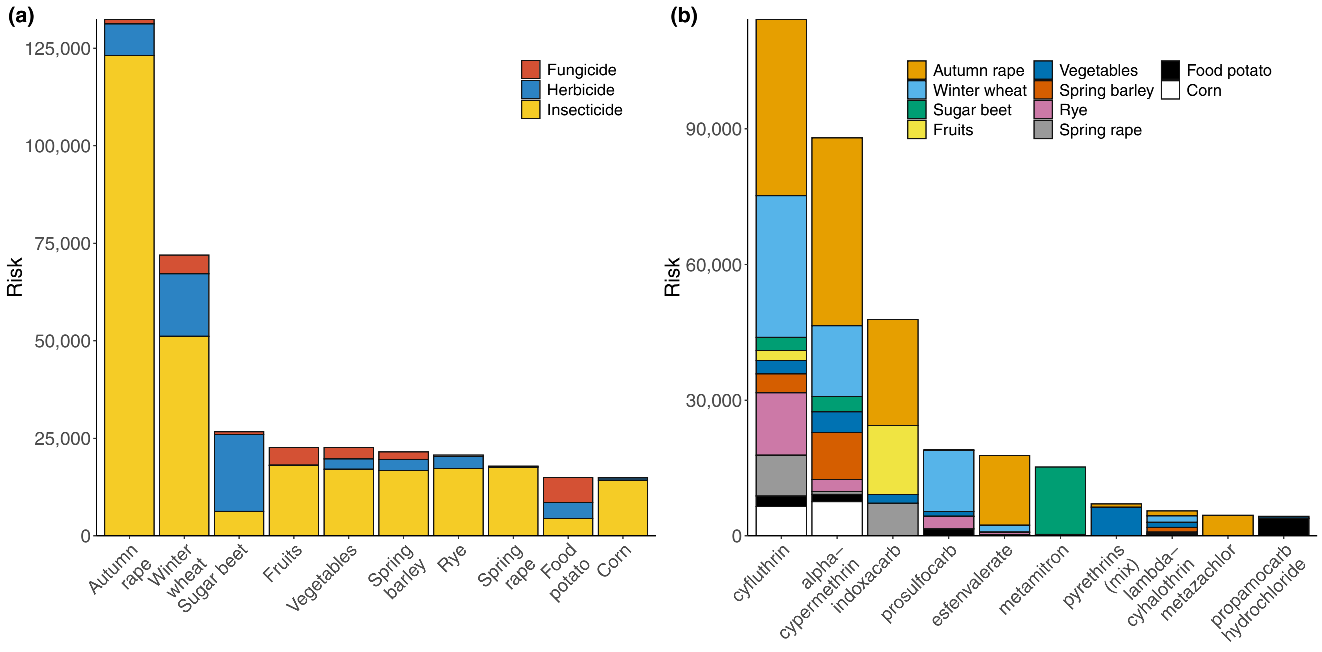

FIGURE 2 Rank pesticide risk based on reported uses in different crops. The top 10 crops comprising the most risk (a), coloured according to pesticide class (Figure 2). For these 10 crops, the top 10 compounds contributing the most to risk are shown (b).

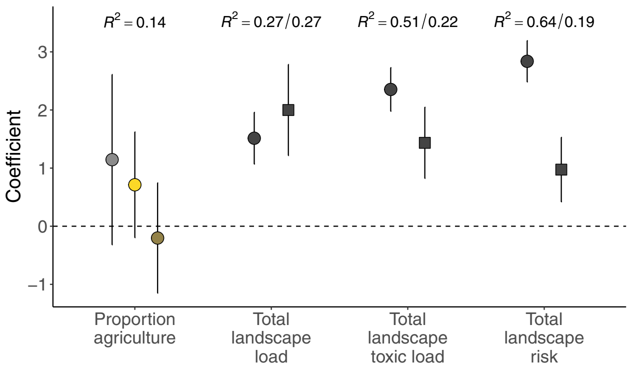

FIGURE 3 Comparative analysis of landscape metrics for the ecotoxicological assessment of pesticide risk to bees. Points depict regression coefficient estimates and their 95% confidence intervals from linear mixed effects models with *z*-score standardized predictors of proportion agriculture, total landscape load, total landscape toxic load, and total landscape risk versus observed risk. Where significant interactions between landscape metric and bee species were detected, we estimate their individual slopes and colour them according to taxa: *Osmia bicornis* (grey), *Bombus terrestris* (yellow), *Apis mellifera* (brown). For landscape metrics based on pesticide use information (total landscape load, total landscape toxic load, total landscape risk), circles correspond to realized risk (left-hand R^2 values) and squares to potential risk (right-hand R^2 values). For additional landscape metrics (Crop diversity, IJI, Proportion treated agriculture), see Figure S3 and Table S5.

index and used to inform ERA (EFSA, 2022). Yet, we found that simple landscape metrics do not consistently predict observed pesticide risk for bees, while landscape metrics based on pesticide use information and bee foraging ecology outperformed those simple landscape metrics. Our exposure model, which incorporates expected patterns of bee foraging, improved predictions of pesticide risk, but only when based on prior knowledge of local pesticide use. This work answers recent calls to integrate ecotoxicology with considerations of spatial pattern and scale (Beketov & Liess, 2012) by including both assessment of exposure to pesticides in a real-world system at large spatial scales and combining these observations with advanced mechanistic modelling.

Recent evidence of spatially heterogenous patterns of pesticide exposure to bees (e.g., Graham et al., 2022; Knapp et al., 2023; McArt et al., 2017) suggests that bees' exposure to pesticides could depend on broader landscape patterns. We believe two mutually inclusive causes could explain this effect. First, landscape pattern could affect pesticide use—the amount, frequency and type of compounds applied. Pesticide use can be greater in simple landscapes (Larsen & Noack, 2017; Nicholson & Williams, 2021), potentially due to increased pest pressure resulting from reduced natural enemy abundance or enhanced pest colonization from greater host crop connectivity (Meehan et al., 2011). Thus, bees may be more exposed in simple landscapes because there are simply more pesticides used. Second, landscape complexity and composition could affect pollinator foraging and subsequent pesticide exposure. Central place foraging theory (Olsson et al., 2008; Schoener, 1971) would predict reduced exposure if high resource patches close to nests, such as semi-natural grasslands or mass flowering crops, are also uncontaminated. Thus, as the dynamics of bee foraging change with landscape pattern, so too will their exposure.

We did not find that landscape pattern alone consistently predicted observed bee pesticide risk, and what evidence we did find was taxon-dependent. Like Knapp et al. (2023), we observed an

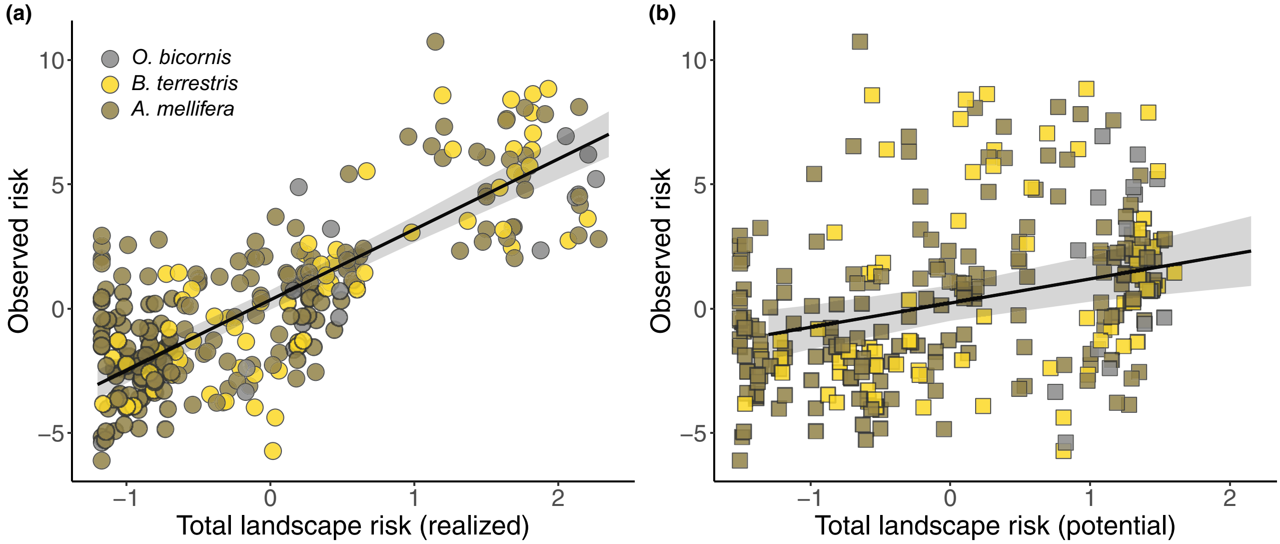

FIGURE 4 Observed pesticide risk in pollen samples was predicted by risk estimate from our exposure model when calculating total landscape risk based on all compounds detected (a; realized risk, cirlces) or risk based on all compounds screened (b; potential risk, squares). Points correspond to site-years ($N = 269$) where pollen was collected from *Apis mellifera* (brown), *Bombus terrestris* (yellow), or *Osmia bicornis* (grey). Fitted lines are estimated based on linear mixed effects models. Shaded areas represent the regression 95% confidence intervals. Results from statistical models are given in Table S3.

interaction between the proportion of agriculture and bee species, with risk marginally increasing (i.e., not significantly at $p < 0.05$) for *Osmia* nests and *Bombus* colonies in landscapes dominated by agricultural land use. Likewise, we observed an interaction between bee species and habitat intermixing (IJI) and landscape crop diversity. For honeybees, we found that risk decreased significantly with increasing crop diversity, while for the other taxa these landscape metrics had no individual effects. On balance, landscape pattern metrics do not explain much variance in observed risk and taxa-dependent effects add complicating context-dependency to risk assessment.

Leveraging information on pesticide use enhanced our ability to predict pesticide risk to bees. By integrating land cover maps with crop-specific data on typical agricultural pesticide use, we could calculate total landscape load and toxicity-weighted load, both of which were predictive and explained substantial variance in observed risk (R^2 range: 0.22–0.51). This result, although perhaps unsurprising, underscores the importance of reliable and readily available pesticide use data for research, monitoring, and mitigation. Data on pesticide use per se—as opposed to information on sales—are typically uncommon or variable in detail and level of information aggregation. For example, in the EU, applicators are usually required to record the location and date of pesticide application. Yet, these data are only periodically consolidated to produce national estimates of pesticide use that are neither crop- nor compound-specific, let alone spatially explicit (Galimberti et al., 2020). Where spatially and temporally granular pesticide use data do exist (e.g., California's Pesticide Use Reports, PUR), they have been used to determine spatiotemporal heterogeneity of multiple ecotoxicological endpoints (Zhang et al., 2018), including honeybee (Mullin et al., 2016) and bumblebee exposure (Lonsdorf et al., 2024). Openly accessible pesticide use data is essential to evaluate the effects of pesticides on human and ecosystem health, support pesticide regulation and improve transparency (Möhring et al., 2020).

The pesticide use data we used are the best available in Sweden, yet still deserve a few caveats. First, the data are not truly spatially explicit, as we did not know the exact field location of application. Second, averaging application rates and spatializing these based on crop identity assumes a level of homogeneity in plant protection practices. Although we captured variation in extent (proportion of fields using a compound), we assumed static values of intensity (amount of compound applied per area). Third, we inherited the biases of the sample survey, so we may lack use information for rare crops or compounds registered for use outside of the survey years. Lastly, these data are not publicly available and require a formal requisition process. In the light of these caveats and our present findings, we echo the recommendations of Mesnage et al. (2021) that pesticide use reporting is: (1) spatially explicit (e.g., individual field level); (2) includes application information (e.g., active ingredients, target crop, rate and timing of application); and (3) frequently reported and openly databased.

If landscape-level pesticide use information is available, is a pollinator exposure model needed to predict risk? It depends on how much is known about actual agricultural pesticide use. In situations where there is spatially explicit information of pesticide use—for example from highly detailed use maps (e.g., California's PUR), grower spray records (e.g., Graham et al., 2022) or from compound detections (e.g., the present study)—the exposure model excels at estimating observed risk. Indeed, when considering *realized risk* (i.e., risk calculated from the subset of detected compounds) our exposure model predictions better fit the data than all other metrics and explained a substantial amount of variance ($R^2 = 0.64$). However, prior

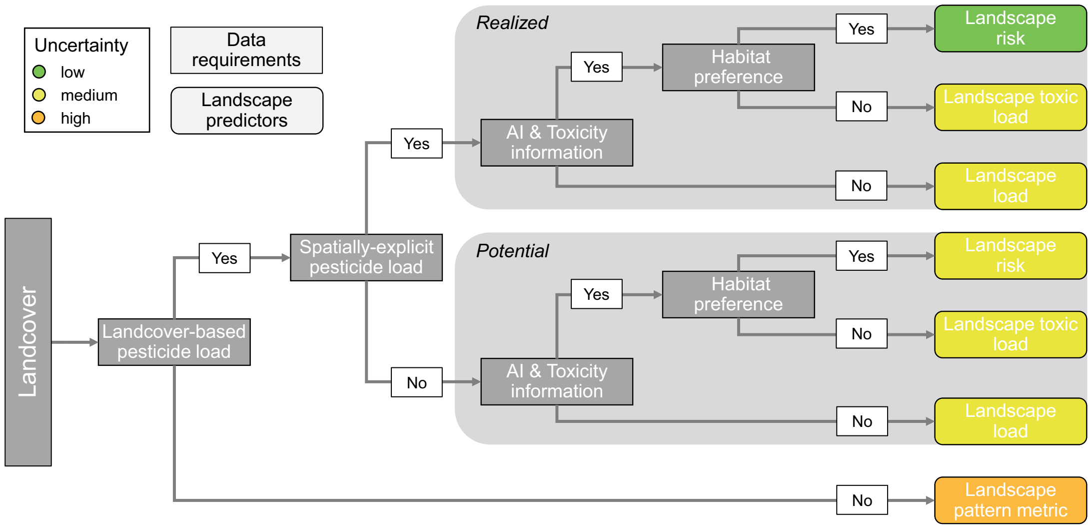

FIGURE 5 A guidance framework of landscape metric selection for the estimation of pesticide risk to bees. Depending on data availability and prediction goals (i.e., uncertainty tolerance), our comparative analysis suggests different landscape level pesticide metrics are suitable.

information of actual pesticide use is often unknown and compounds may go undetected. A pesticide could be absent from pollen if it is not used locally, but non-detection may arise from other factors, including:

- Compound application method. Pesticide application methods (e.g., soil drench of a non-systemic compound) may not result in pollen contamination.
- Compound fate. Pesticides with rapid degradation (e.g., pyrethroids) may go undetected.
- Compound toxicity. Pesticides with high acute toxicity (e.g., neonicotinoids) may go undetected because exposure leads to forager death or disorientation and contaminated pollen never returns to the colony.
- Patch quality. Pesticides applied to patches without floral resources may not result in exposure via pollen or nectar.
- Sample size and design. Pesticides may be missed due to stochasticity and limited sample size, especially those that are rarely used in the landscape or used at low rates.

For these reasons, *potential risk* (i.e., risk calculated from the broader set of all screened compounds) provides a more generalizable, yet overestimation of predicted pesticide risk. It is *generalizable* because it incorporates exposure predictions for all possible compounds that could contribute to risk in a given landscape and is thereby transferable to the common situation where exact pesticide use patterns are unknown. It *overestimates* because it incorporates predictions of compounds that were not detected and thus increases risk prediction uncertainty. When considering *potential risk* our exposure model performed similarly to landscape-level pesticide use metrics. It is worth noting that all metrics based on pesticide use (total landscape load, total landscape toxic load and total landscape risk) had a significantly positive fit with observed exposure for both realized and potential risk. In practice, this means that where actual use is unknown or uncertain a general, albeit imprecise, estimate of risk can be approximated from pesticide load (Figure 5). Conversely, situations calling for precise estimates of risks will require obtaining prior information on actual pesticide use (Figure 5).

5 | CONCLUSIONS

There is a long-standing acknowledged need in ecotoxicology for methods that integrate spatial aspects and context heterogeneity into risk assessment. The discipline of landscape ecotoxicology (Cairns & Niederlehner, 1996) aims to address this need by incorporating principles and methods from landscape ecology (Johnson, 2002; Schäfer, 2014). We advance landscape ecotoxicological research using a multiyear terrestrial biomonitoring network of bee pollen pesticide residues to assess the capacity of different land cover-based metrics to predict bees' pesticide risk. We answer the call (EFSA, 2022; Price & Thorbek, 2014) for the development and use of spatially explicit exposure predictions from mechanistic models at organism-relevant scales. Nonetheless, these models require specialized tools, data, and expertise, which can be cost- and time-intensive. Our comparative analysis (Figure 4) provides a roadmap for landscape-level risk prediction for pollinators that evaluates assessment goals relative to data availability and uncertainty tolerance (Figure 5). Taken together, our biomonitoring network, modelling, comparative analysis, and proposed framework are the next step towards building standardized methods for landscape-level pesticide risk assessment for pollinators.

AUTHOR CONTRIBUTIONS

Charlie C. Nicholson, Eric V. Lonsdorf, Neal M. Williams and Maj Rundlöf were involved in conception. Charlie C. Nicholson, Mikaela

Gönczi, Joachim R. de Miranda and Maj Rundlöf were involved in design and methodology. Georg K. S. Andersson, Jessica L. Knapp, Glen P. Svensson, Mikaela Gönczi, Ove Jonsson, Joachim R. de Miranda and Maj Rundlöf were involved in data acquisition. Charlie C. Nicholson and Ove Jonsson were involved in data analysis. Charlie C. Nicholson and Maj Rundlöf were involved in data interpretation. Charlie C. Nicholson was involved in original draft. All authors contributed critically to the drafts and gave final approval for publication. Our study brings together authors from several different countries, including scientists based in the country where the study was carried out.

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CONFLICT OF INTEREST STATEMENT

No conflicts of interest.

DATA AVAILABILITY STATEMENT

Data are available via figshare https://doi.org/10.6084/m9.figshare.25260232 (Nicholson et al., 2024a). Model code is available on Github https://github.com/CCNicholson/SEMLE and archived on Zenodo https://zenodo.org/records/10066592 (Nicholson et al., 2024b).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

Appendix S1. Supplemental methods, tables and figures.

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