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Towards a system-level causative knowledge of pollinator communities

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Pollination plays a central role in both crop production and maintaining biodiversity. However, habitat loss, pesticides, invasive species and larger environmental fluctuations are contributing to a dramatic decline of pollinators worldwide. Different management solutions require knowledge of how ecological communities will respond following interventions. Yet, anticipating the response of these systems to interventions remains extremely challenging due to the unpredictable nature of ecological communities, whose nonlinear behaviour depends on the specific details of species interactions and the various unknown or unmeasured confounding factors. Here, we propose that this knowledge can be derived by following a probabilistic systems analysis rooted on non-parametric causal inference. The main outcome of this analysis is to estimate the extent to which a hypothesized cause can increase or decrease the probability that a given effect happens without making assumptions about the form of the cause–effect relationship. We discuss a road map for how this analysis can be accomplished with the aim of increasing our system-level causative knowledge of natural communities.

This article is part of the theme issue 'Natural processes influencing pollinator health: from chemistry to landscapes'.

1. Introduction

Pollinators comprise a highly diverse group of species including bees, flies, butterflies, beetles and some vertebrates [1]. They all have in common a shared interest in visiting flowers to extract resources, collectively and indirectly mediating the reproduction of most of the worldwide plant species [2] and maximizing crop production for 75% of cultivated crops [3]. Hence, pollination is now recognized not only as a key ecosystem function, but also as a key ecosystem service contributing to human food security. However, human-induced rapid environmental change has been threatening most of these pollinators [4]. On the one hand, habitat destruction and modification are reducing the populations of many pollinator species, often leading to local extirpation. On the other hand, some other species can thrive in human-modified ecosystems, but those often face extra pressures such as pesticide exposure, exotic species, or pathogens. On top of that, climate change is altering species' physiological responses, distribution and activity periods [5]. Overall, we are assisting in a rapid loss of pollinator communities worldwide, where their relative abundance, composition and ecological interactions are being restructured, with hard-to-predict consequences for their health.

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These multi-factorial pressures on pollinator communities have increased the need for human interventions to protect the composition, functioning and stability of pollinators and their interactions [6]. These interventions include from well-established practices such as habitat protection, to more complex actions such as the addition or removal of particular species and their interactions [7]. For instance, planting field margins [8] or adding managed pollinators [9] have become, respectively, popular restoration practices in agricultural systems to increase resources for pollinators or supplement crop pollination. However, anticipating the response of these systems to interventions remains extremely challenging due to the unpredictable nature of ecological communities, whose nonlinear behaviour depends on the specific details of species interactions and the various unknown or unmeasured confounding factors [10,11]. For example, these practices often ignore side effects, such as the effects that field margins can have by altering micro-climate conditions, which in turn can change pollinators' occupancy patterns [12,13] or the co-lateral effects of introducing managed species on pollinator health. This is particularly important since it has been shown how managed pollinator densities not only increase competition among pollinators [14] but also increase parasite loads [15], which can spill over to other species. Yet, as of today, we lack a community-wide framework to guide interventions beyond single species. Indeed, it has been shown that even small local interventions (i.e. at the species level) can have heterogeneous and arbitrary cascading effects across entire communities [16]. This has emphasized the dire need to establish a system-level causative knowledge of natural communities.

To address the challenge above, ideally, we need to establish well-defined experiments eliminating all sources of bias (e.g. using randomized controlled trials) and test the effectiveness of a given intervention [17]. However, those sources of bias become extremely difficult to identify and measure, especially considering the nonlinear dynamics of natural communities conformed by several co-occurring and interacting species [18]. Moreover, many of these interventions may not be ethical (e.g. species removal) or feasible to perform because species move freely and are difficult to track. This implies that it is necessary to obtain interventional knowledge (i.e. the cause–effect relationship) from observational data. However, these observational data (that record, for example, the observed presence/absence of pollinators) differ from fully controlled studies (e.g. removing or adding pollinators) in the sense that observational variables are the result of what is perceived by the investigator and not of their intervention. In this line, causal inference tools, such as path analysis or structural equation modelling, have been developed to obtain information about causes from observations [19]. While extremely useful, these tools assume linearity or monotonicity in all the relationships, but often this can be difficult to prove [17,20]. Hence, to move towards a more general approach, we propose to follow a probabilistic systems analysis using current advancements on non-parametric causal inference [17,20]. Instead of aiming to predict exactly what would happen under an intervention—something that may be impossible to generalize [11]—we propose to focus on how much a likely cause can affect the probability that a given effect happens. In the following, we discuss a road map for how this probabilistic systems analysis can be accomplished and illustrate it with a case study.

2. Observational data

Under a lack of systematically controlled experiments, observational data from field studies or quasi-controlled experiments (where few factors may be controlled) can provide the raw material to understand the behaviour of a community. This behaviour comes in the form of a joint probability distribution $P_{\mathbf{V}}$ over a set of relevant variables $\mathbf{V}$. For example, studies may record any aspect of community composition as a function of a set of semi-controlled variables such as the presence (or density) of specific pollinators [21], their floral resources including both the identity of interacting plant species [22,23] and plant chemical composition [24–26], top–down regulators including pathogen [27] and predators [28], as well as several environmental variables such as temperature [13,29,30] or pesticide exposure [31,32]. These observational studies can be either for a specific period of time across different locations or measure pollinator communities repeatedly over time in order to capture a wider range of temporal conditions affecting pollinators' population trajectories [33,34]. These relevant variables form the set $\mathbf{V}$ and their simultaneous behaviour (values associated with such variables) forms $P_{\mathbf{V}}$.

While observational data are designed to predict likely mechanisms affecting pollinator communities, they cannot establish cause–effect relationships by themselves, only associations [17,19]. That is, following Reichenbach's principle [35], if two variables (X, Y) are statistically related, then there exists a third variable or set of variables (Z) that causally influenced both (known as confounding effect: $X \leftarrow Z \rightarrow Y$). In some situations, Z coincides with either X or Y (i.e. $Z = X$ or $Z = Y$), establishing a causal link between X and Y (i.e. $X \rightarrow Y$ or $Y \rightarrow X$). But without knowledge of Z (or when this unknown effect cannot be blocked from the analysis), we cannot safely conclude cause–effect relationships [17]. In other words, conditional distributions (e.g. $P_{Y|X}$) derived from observational data can coincide with causal mechanisms (e.g. $X \rightarrow Y$), but not necessarily. Similarly, two variables (X, Y) may be statistically related if both are the common causes of a given effect Z (i.e. $X \rightarrow Z \leftarrow Y$: known as collider in the causal-inference literature [17]) and we condition by Z, i.e. $X \perp\!\!\!\perp Y \mid Z$, but $X \not\perp\!\!\!\perp Y \mid \{\emptyset\}$ ($\not\perp\!\!\!\perp$ $\perp\!\!\!\perp$ $\not\perp\!\!\!\perp$ $\perp\!\!\!\perp$ and denote dependence and independence, respectively). Moreover, in a multivariate system, the sources of bias can be originated from direct and indirect common causes and effects. These properties make extremely problematic the interpretation of relationships derived from multivariate regression and meta-analysis that are not structured by a causal hypothesis [20].

For example, let us assume that pollinator abundance is caused by flower abundance, temperature and some unknown factors. Similarly, let us assume that flower abundance is caused by water availability, temperature and a subset of the same unknown factors. Then, in a multivariate regression model that includes all factors (except for the unknown) as likely explanations of pollinator abundance, it is likely that water availability will have a strong explanatory effect over pollinator abundance (even though we are conditioning over flower abundance). This happens for the reason that flower abundance introduces a bias (collider) between water and the unknown factors, which then gets propagated to pollinator abundance following the cause–effect relationships. Note that flower abundance cannot be

eliminated from the regression model either, because it is needed to partially block the path between water availability and pollinator abundance. This type of example also illustrates that prediction is different from explanation [36]. Therefore, to infer cause–effect relationships in this example, it is necessary to have more information about the underlying causal story and the corresponding unknown confounding factors.

3. Causal inference: a probabilistic systems analysis

While observational data *per se* are not enough to obtain a causative knowledge about pollinator communities, they can be translated into interventional distributions using non-parametric causal-inference techniques to then provide an estimate of the extent to which a likely cause can affect the probability that a given effect happens—known as average causal effect (ACE) [17,19]. Recently, promising non-parametric causal-inference methods have been developed, such as inverse modelling approaches [37,38] or empirical dynamical modelling [39], but these methods require large amounts of data that for several reasons can be difficult to obtain. However, non-parametric causal inference based on probabilistic systems analysis as used in economics, social science and medicine [17] can be a good candidate for inferring interventional distributions and consequently for identifying the casual drivers of pollinator communities.

First and foremost, causal inference requires a causal graph involving the set of relevant variables (nodes) $\mathbf{V}$ (e.g. $\mathbf{V} = \{X, Y\}$, $X \rightarrow Y$) upon which to test causal relationships (directed edges) [17]. These graphs serve as a guideline (testable hypothesis) to understand the likely paths linking causes and effects, which should be studied in order to eliminate spurious associations (e.g. due to confounding or sampling bias). In general, causal graphs should be drawn based on expert knowledge or intuition about how the world works, although different algorithms have been proposed for use as guidelines for creating causal graphs [17]. These graphs can be used after identifying and corroborating their testable implications expressed as unconditional and conditional independencies between variables (in causal-inference analysis, this is called *d*-separation of variables [17]). That is, a statistical dependence between two connected variables (X, Y) does not corroborate a causal graph since we cannot be sure of having all the likely confounders. Similarly, a lack of correlation between two connected variables does not immediately invalidate a causal graph since we cannot be sure of having sampled all possible values within the sample space. However, independency (*d*-separation) between two disconnected variables (X, Y) does support the hypothesized causal graph. Thus, causal graphs inform about both the likely dependencies and hypothesized independencies between variables. The more testable are the conditions (*d*-separations), the stronger the support for the causal story. If the data do not corroborate the causal graph, then a new causal story must be drawn and tested.

Importantly, under a corroborated causal graph, likely causes can be further defined as potential or genuine [17]). A potential cause between X and Y exists if (i) X and Y are statistically dependent in every context and (ii) there exists a set of variables Z (including the empty set) and context S such that: X and Z are independent given S, and Z and Y are statistically dependent given S. Note that a context is defined as a set of variables tied to specific values. In turn, a genuine cause between X and Y exists if (i) X and Y are statistically dependent in every context and (ii) there exists a set of variables Z and context S such that: Z is a potential cause of X; Z and Y are statistically dependent given S; and Z and Y are independent given S and X. The hierarchy of likely, potential and genuine causation can then serve as a guideline for constructing causal graphs when one is interested in a particular cause–effect relationship.

Causal graphs are non-parametric by construction since they do not depend on the specific form of causal relationships, they only specify the existence or lack of a causal relationship between variables. While most of the standard work on causal inference has been developed for directed acyclic graphs (DAGs), cyclic graphs (subject to mutual causality or feedback processes) can also be analysed, especially under equilibrium conditions [40]. Typically, DAGs are assumed to be Markovian, meaning that measured variables (nodes) are affected by mutually independent, unknown, random variables (typically not drawn in the causal graph) [17]. If unknown common factors are not mutually independent, then they need to be represented in the causal graph as nodes. In some situations, the likely confounding effects of non-mutually exclusive unknown factors can be eliminated using standard causal-inference techniques (e.g. using the so-called front-door and back-door criteria [17]).

Then, non-parametric knowledge of interventional distributions (our goal) by using observational distributions (our data) can be performed with *do*-calculus [17], which are the rules for moving from interventions to observations using the causal graph. That is, causal inference moves (whenever identifiable) from the probabilistic causal association $P(y \mid do(x))$ to the probabilistic observational association $P(y \mid x)$, where y is the value of the likely effect Y and x is the value taken after the intervention on the inferred cause X. The nomenclature $do(x)$ implies that we are not just merely observing X to take the value of x (hence performing a traditional condition), but we need to make all the observations of X to have the value of x (hence performing an intervention). This action is then represented in a modified causal graph by eliminating all the incoming edges (causes) from an intervened variable (since its value is no longer dependent on mechanisms, but on a given action; see figure 1). This implies working with a modified version of the causal graph (i.e. $P(y \mid do(x)) = P_m(y \mid x)$). It is typically assumed that mechanisms $P(y \mid do(x))$ are independent from each other, invariant, and follow the arrow of time (i.e. causes before effects), allowing the application of probabilistic Markov properties (i.e. each variable is independent from its non-causal variables—known as ancestors—given its causes—known as parents [17]). Consequently, the Markov property allows us to use product decomposition to simplify conditional probabilities as much as possible, and to use the observed probability distributions (i.e. $P(y \mid x)$) whenever $P_m = P$. That is, we substitute modified probabilities (P_m) by observational probabilities (P) if and only if they are both equivalent in the modified and original graphs.

For a given DAG G and disjoint variables X, Y, Z and W (these variables can also be empty sets), the translation methodology of *do*-calculus mentioned above can be summarized under three rules (figure 1) [17]: (1) insertion/deletion of observations: $P(y \mid do(x), z, w) = P(y \mid do(x), w)$ if

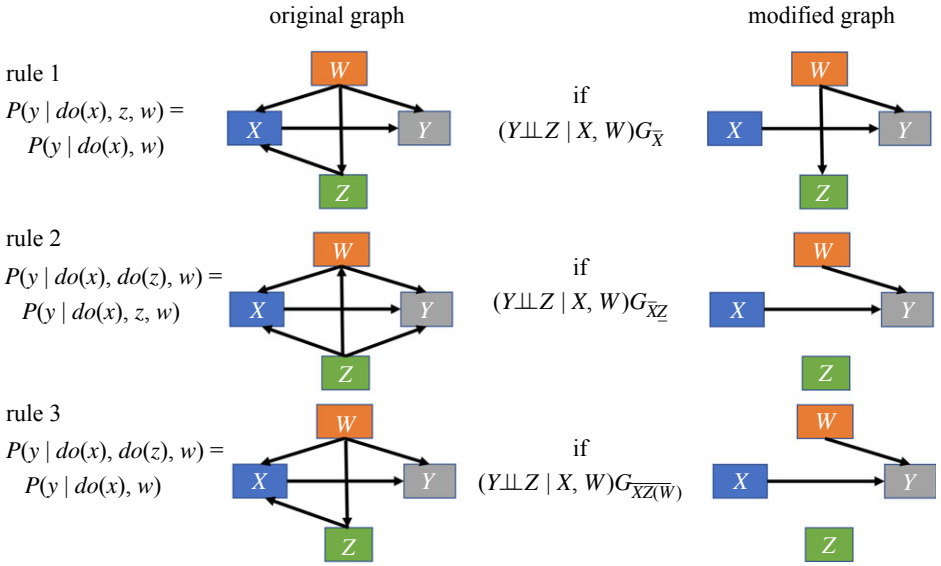

Figure 1. *do*-calculus. The translation from interventional $P(do(x))$ to observational $P(x)$ distributions can be achieved following the rules of *do*-calculus [17]. The figure depicts the three *do*-calculus rules on a graph G with disjoint variables X, Y, Z and W (see main text). Rule 1 is used for insertion/deletion of observations. Rule 2 is used for action/observation exchange. Rule 3 is used for insertion/deletion of actions. Here, $G_{\overline{X}}$ is graph G after the removal of all the incoming edges to X, $G_{\overline{X}\underline{Z}}$ is graph $G_{\overline{X}}$ after the removal of all the outgoing edges from Z and $Z(W)$ is the set of Z-variables that are not ancestors of any W-variable in $G_{\overline{X}}$. Note that ⫫ and | denote independence and conditional on, respectively. The graphs in the left column vary for illustration purposes of each rule. (Online version in colour.)

$(Y \perp\!\!\!\perp Z \mid X, W)_{G_{\overline{X}}}$, where $G_{\overline{X}}$ is graph G after the removal of all the incoming edges to X. This rule establishes the conditions under which it is possible to remove conditional variables from the analysis. (2) Action/observation exchange: $P(y \mid do(x), do(z), w) = P(y \mid do(x), z, w)$ if $(Y \perp\!\!\!\perp Z \mid X, W)_{G_{\overline{X}\underline{Z}}}$, where $G_{\overline{X}\underline{Z}}$ is graph $G_{\overline{X}}$ after the removal of all the outgoing edges from Z. This rule establishes the conditions under which it is possible to replace additional actions (acting as confounders) with observational data. (3) Insertion/deletion of actions: $P(y \mid do(x), do(z), w) = P(y \mid do(x), w)$ if $(Y \perp\!\!\!\perp Z \mid X, W)_{G_{\overline{XZ(W)}}}$, where $Z(W)$ is the set of Z-variables that are not ancestors of any W-variable in $G_{\overline{X}}$. This rule establishes the conditions under which it is possible to remove additional actions (acting as confounders) from the analysis.

Following *do*-calculus, it can be possible to estimate the ACE between two variables (X, Y) having a directed path in the causal graph [17]. Note that the statistical significance of an ACE can be obtained using an independence test. Specifically, the ACE is given by $\partial/\partial x E[Y \mid do(x)]$ and represents the expected increase in the value of Y per unit of X. If variables are binary, the ACE is simplified to $P(y = 1 \mid do(x) = 1) - P(y = 1 \mid do(x) = 0)$, corresponding to the change in the probability that Y happens if X also happens. This calculation provides the total ACE between two variables (X, Y), i.e. considering the direct and all possible indirect paths (mediators) through which the cause X can change the effect Y. If we are interested in estimating a direct effect only, then it is necessary to intervene on the mediators Z, i.e. $P(y \mid do(x), do(z))$ for all the values of Z [17] and again translate interventional to observational distributions. It is worth noticing that while non-parametric tools provide generality, their application to continuous data can be rather challenging. Thus, whenever possible, the data can be discretized [17]. For example, one can use the median of each variable as a cut-off value: values higher that the median are considered one, otherwise zero. While this may be perceived as a disadvantageous simplification, it allows us to calculate the extent to which the occurrence of an intervention can increase or decrease the probability of observing a given effect.

4. Case study

First, let us establish a phenomenon of potential practical importance. While it is commonly known that the richness of plants can be proportionally beneficial to the richness of pollinators, it has remained unclear whether some factors can counterbalance this relationship [34]—specifically, whether regardless of plant richness, controlling the ratio between the numbers of plant and pollinator species can additionally affect pollinator richness. Note that plant–pollinator ratio has been found to have a strong association with the structure of plant-interaction networks and their robustness to perturbations [41–44]. This knowledge can be important for increasing the success of restoration and conservation practices. However, predictions concerning such causal interventions from purely observational models can only be reliably obtained by first proposing and testing cause–effect linkages between the corresponding variables in the form of a causal graph, and then using non-parametric causal inference (e.g. *do*-calculus) to deduce such knowledge.

To illustrate the process above, we used data on the within-season dynamics (phenology) of a pollinator community [34,45]. These publicly available data record co-occurrence of pollinators and flowering plants on a daily basis (whenever weather allowed) in a high-arctic site during the springs of 1996 and 1997 [34,45]. Figure 2 depicts our hypothetical DAG. Specifically, we hypothesized that pollinator richness (number of pollinator species) is affected by plant richness (number of flowering species) and plant–pollinator ratio. Additionally, we hypothesized that plant–pollinator ratio is affected by plant richness and the mean humidity during a day. These hypotheses follow results from previous work [34,45]. Because observations are made

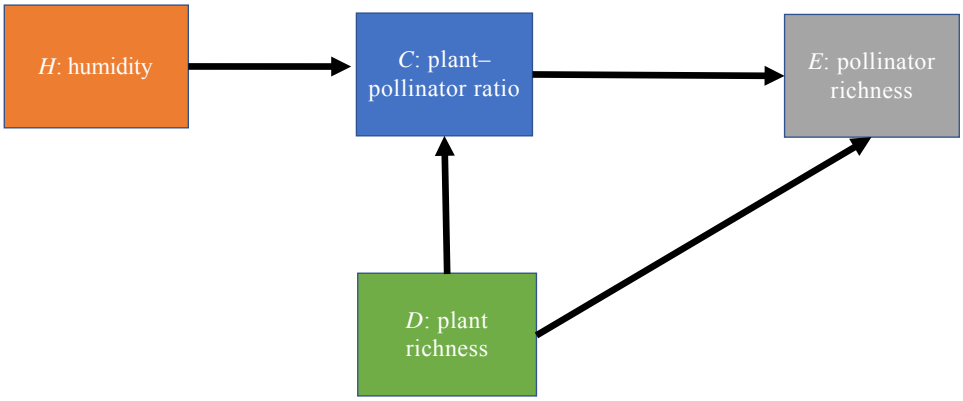

Figure 2. Illustrative example of a hypothesized directed acyclic graph (DAG) to study cause–effect relationships. Each box (node) corresponds to a random variable, and each directed edge corresponds to a direct causal effect. We consider that each causal relationship is autonomous and independent from the others. It is also assumed that mutually independent random noise affects each node (Markovian graph). The variables in this graph should not be equated to the variables in figure 1. That is, each variable can take the role of any of the variables in figure 1, depending on the studied cause-effect relationship. (Online version in colour.)

on a daily basis, for each variable, we captured the dynamics by quantifying changes from time $t-1$ to time t and established a value of 1 if there is a positive change, 0 otherwise. This provided us with four variables with 48 paired observations in total.

The hypothesized DAG has two testable implications (assumed d-separations: no direct path between variables). This test implies finding the conditional independencies (i.e. $X \perp\!\!\!\perp Y \mid Z$) (1) between humidity and pollinator richness conditional on plant–pollinator ratio and (2) between humidity and plant richness conditional on the empty set (note that we cannot condition on plant–pollinator ratio since it is a collider between humidity and plant richness). Using a G^2-test (χ^2-test can also be used for binary data or permutation tests [17,19]), we found no statistical relationship between humidity and pollinator richness (p-value = 0.68, power = 0.78), nor between humidity and plant richness (p-value = 0.56, power = 0.99)—corroborating our causal graph. Note that if the hypothesis had not been supported by the d-separations, then a new causal graph would have to have been drawn and tested.

While our causal graph has only two testable implications (the more the better), we found that plant–pollinator ratio in our DAG is a genuine cause of pollinator richness. That is, plant–pollinator ratio and pollinator richness are unconditionally statistically dependent (G^2-test: p-value = 0.001, power = 0.99), plant–pollinator ratio and pollinator richness are statistically dependent conditional on all other variables (G^2-test: p-value = 0.001, power = 0.99), humidity potentially has an effect on the plant–pollinator ratio under the context defined by the null set, humidity and pollinator richness are unconditionally statistically dependent (G^2-test: p-value = 0.012, power = 0.99), but humidity and pollinator richness are statistically independent conditional on plant–pollinator ratio (G^2-test: p-value = 0.68, power = 0.99)—fulfilling the criteria of genuine cause (see previous section). Note that, in principle, we can also estimate the effect of plant richness on pollinator richness. However, in our DAG, this effect cannot be defined as either potential or genuine, it can only remain at a likely level (see §3).

After corroborating our DAG, we focused on finding the ACE (i.e. average causal effect) of plant–pollinator ratio and pollinator richness. That is, we calculated the probability of observing an increase in pollination richness (E) if the plant–pollinator ratio (C) were to be increased by a given intervention such as preventing grazing or supplementing seeds. This ACE can be written as $P(e=1 \mid do(c)=1) - P(e=1 \mid do(c)=0)$, where the interventional distribution $P(e \mid do(c))$ needs to be translated to observational distribution (the format of our data) following do-calculus. Recall that $P(e \mid do(c)) = P_m(e \mid c)$, where $P_m(e \mid c)$ is the modified DAG (removing the incoming links to plant–pollinator ratio). Using marginalization over humidity (H) and plant richness (D), $P_m(e \mid c) = \sum_h \sum_d P_m(h, d, e \mid c)$. Using the definition of conditional probability, $\sum_h \sum_d P_m(h, d, e \mid c) = \sum_h \sum_d P_m(e \mid c, h, d) P_m(h \mid c, d) P_m(d \mid c) = \sum_d P_m(e \mid c, d) P_m(d)$. Then, we can see that $\sum_d P_m(e \mid c, d) P_m(d) = \sum_d P(e \mid c, d) P(d) = P(e \mid do(c))$. The same translation can be obtained by following the simplified rules of do-calculus (figure 1). That is, starting from $\sum_h \sum_d P(e \mid do(c), h, d) P(h, d \mid do(c))$, we can apply rule 1 to delete humidity entirely from the calculation, and then apply rule 3 to delete the conditional action from plant richness (second term). Using our data, $\mathrm{ACE} = \sum_d P(e=1 \mid c=1, d) P(d) - \sum_d P(e=1 \mid c=0, d) P(d) = -0.53$. As mentioned before, the effect of plant richness on pollinator richness (i.e. $P(e \mid do(d))$) can be estimated only at a likely level. Because the modified graph is the same as the original graph (i.e. there are no incoming links to plant richness), the interventional distribution is exactly the same as the observational distribution. Using our data, $\mathrm{ACE} = P(e=1 \mid d=1) - P(e=1 \mid d=0) = 0.62$, as can be expected.

The result above implies that by increasing the plant–pollinator ratio in the community, we would decrease by 53% the chance that pollinator richness increases on a given day. This suggests that there can be, in fact, limits to the positive relationship between plant and pollinator richness. This relationship may be attributed either to a set of plants that do not directly affect pollinator richness or to how this ratio modulates species interactions, and consequently, the community dynamics [34]. It is worth recalling that this case study is not intended to demonstrate a general effect, but to serve only for illustration purposes and as a potential guideline for future studies. For example, we try to explain a fairly simple community metric and many more variables can be explicitly taken into account, such as abundance of pathogens, herbivores, chemical compounds, temperature, etc. But we hope future work can build on these methodologies to establish causal knowledge of pollinator communities.

5. Final thoughts

It has long been recognized that causation does not always coincide with correlation. This premise has been extensively applied when studying the behaviour of complex natural systems, where multiple factors can be responsible for the patterns observed in nature. Investigation of pollinator communities has shown that they are no exception to this. As a consequence, the majority of work has carefully stated correlations, which corresponds to what do we see in nature. Indeed, causal statements have long been prevented by the dominance of multivariate regressions and meta-analyses that do test causal hypotheses [20]. However, in the face of rapid environmental change, we need to undertake bolder research programs and answer the questions of how and why the behaviour of pollinator communities is affected. These goals can be achieved by conducting experimental studies. Nevertheless, manipulating all factors related to the behaviour of entire pollination communities can be unrealistic. Instead, these goals can be achieved by using causal-inference techniques.

While not exhaustive, here we have provided a brief overview of how to follow a probabilistic systems analysis using non-parametric causal inference. The main goal is for us to embrace the unpredictable behaviour of ecological communities and to speak a probabilistic causal language. Note that while path analysis or structural equation modelling [19] has many parallels with *do*-calculus, only the latter is a non-parametric framework that can be used without the need for linearity assumptions. For example, in linear regression (or Pearson correlation), it is assumed that the effects are linear and monotonic, and noise is Gaussian. Spearman rank correlations can be used if at least monotonicity is achieved. Instead, non-parametric tools can be used whether or not these assumptions above are fulfilled. Also note that many studies have investigated the problem of inferring missing and spurious interactions in the area of network science [46,47], whose parametric tools can be potentially extended to infer cause–effect relationships in ecological communities. As an example, the non-parametric causal inference approach presented here can help to distinguish whether a positive interaction between two species is either due to a mutualistic effect or indirectly due to the fact that both species respond similarly to an external environmental factor. By taking this perspective across all potential sets of interactions, we can be ready to distinguish which specific interactions among species drive the dynamics of entire pollinator communities. Yet, it is worth emphasizing that ultimate confidence in the use of causal inference is entirely dependent on our confidence in the proposed causal hypothesis, unless we also have independent experimental evidence after performing such causal interventions that show an agreement with our predictions.

Data accessibility. The data used in this manuscript were published in [45] and compiled in [34].

Authors' contributions. S.S.: conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, writing—original draft, writing—review and editing; I.B.: conceptualization, funding acquisition, investigation, writing—review and editing; O.G.: conceptualization, funding acquisition, investigation, writing—review and editing; R.P.R.: conceptualization, funding acquisition, investigation, writing—review and editing; P.Z.: conceptualization, funding acquisition, investigation, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Competing interests. We declare we have no competing interests.

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References

1. Winfree R, Bartomeus I, Cariveau DP. 2011 Native pollinators in anthropogenic habitats. *Annu. Rev. Ecol.* **42**, 1–22. (doi:10.1146/annurev-ecolsys-102710-145042)
2. Ollerton J, Winfree R, Tarrant S. 2011 How many flowering plants are pollinated by animals? *Oikos* **120**, 321–326. (doi:10.1111/j.1600-0706.2010.18644.x)
3. Klein AM, Vaissiere BE, Cane JH, Steffan-Dewenter I, Cunningham SA, Kremen C, Tscharntke T. 2007 Importance of pollinators in changing landscapes for world crops. *Proc. R. Soc. B* **274**, 303–313. (doi:10.1098/rspb.2006.3721)
4. Potts SG *et al.* 2016 Safeguarding pollinators and their values to human well-being. *Nature* **540**, 220–229. (doi:10.1038/nature20588)
5. Goulson D, Nicholls E, Botías C, Rotheray EL. 2015 Bee declines driven by combined stress from parasites, pesticides, and lack of flowers. *Science* **347**, 1255957. (doi:10.1126/science.1255957)
6. Garibaldi LA, Gemmill-Herren B, D'Annolfo R, Graeub BE, Cunningham SA, Breeze TD. 2017 Farming approaches for greater biodiversity, livelihoods, and food security. *Trends Ecol. Evol.* **32**, 68–80. (doi:10.1016/j.tree.2016.10.001)
7. Menz MH, Phillips RD, Winfree R, Kremen C, Aizen MA, Johnson SD, Dixon KW. 2011 Reconnecting plants and pollinators: challenges in the restoration of pollination mutualisms. *Trends Plant Sci.* **16**, 4–12. (doi:10.1016/j.tplants.2010.09.006)
8. Scheper J *et al.* 2015 Local and landscape-level floral resources explain effects of wildflower strips on wild bees across four European countries. *J. Appl. Ecol.* **52**, 1165–1175. (doi:10.1111/1365-2664.12479)
9. Klein AM, Boreux V, Fornoff F, Mupepele AC, Pufal G. 2018 Relevance of wild and managed bees for human well-being. *Curr. Opin. Insect Sci.* **26**, 82–88. (doi:10.1016/j.cois.2018.02.011)
10. Saavedra S, Medeiros LP, AlAdwani M. 2020 Structural forecasting of species persistence under changing environments. *Ecol. Lett.* **23**, 1511–1521. (doi:10.1111/ele.13582)
11. Deng J, Angulo MT, Saavedra S. 2021 Generalizing game-changing species across microbial communities. *ISME Commun.* **1**, 1–8. (doi:10.1038/s43705-021-00022-2)
12. Soroye P, Newbold T, Kerr J. 2020 Climate change contributes to widespread declines among bumble bees across continents. *Science* **367**, 685–688. (doi:10.1126/science.aax8591)
13. Tabi A *et al.* 2020 Species multidimensional effects explain idiosyncratic responses of communities to environmental change. *Nat. Ecol. Evol.* **4**, 1036–1043. (doi:10.1038/s41559-020-1206-6)
14. Henry M, Rodet G. 2018 Controlling the impact of the managed honeybee on wild bees in protected areas. *Sci. Rep.* **8**, 9308. (doi:10.1038/s41598-018-27591-y)
15. Dynes TL, Berry JA, Delaplane KS, Brosi BJ, de Roode JC. 2019 Reduced density and visually

complex apiaries reduce parasite load and promote honey production and overwintering survival in honey bees. *PLoS ONE* **14**, e0216286. (doi:10.1371/journal.pone.0216286)

16. Bartomeus I, Saavedra S, Rohr RP, Godoy O. 2021 Experimental evidence of the importance of multitrophic structure for species persistence. *Proc. Natl Acad. Sci. USA* **118**, e2023872118. (doi:10.1073/pnas.2023872118)
17. Pearl J. 2009 *Causality*. Cambridge, UK: Cambridge University Press.
18. Kimmel K, Dee LE, Avolio ML, Ferraro PJ. 2021 Causal assumptions and causal inference in ecological experiments. *Trends Ecol. Evol.* **36**, 1141–1152. (doi:10.1016/j.tree.2021.08.008)
19. Shipley B. 2016 *Cause and correlation in biology*. Cambridge, UK: Cambridge University Press.
20. Bareinboim E, Pearl J. 2016 Causal inference and the data-fusion problem. *Proc. Natl Acad. Sci. USA* **113**, 7345–7352. (doi:10.1073/pnas.1510507113)
21. Brosi BJ, Briggs HM. 2013 Single pollinator species losses reduce floral fidelity and plant reproductive function. *Proc. Natl Acad. Sci. USA* **110**, 13 044–13 048. (doi:10.1073/pnas.1307438110)
22. Biella P, Akter A, Ollerton J, Tarrant S, Janecek Š, Jersáková J, Klecka J. 2019 Experimental loss of generalist plants reveals alterations in plant-pollinator interactions and a constrained flexibility of foraging. *Sci. Rep.* **9**, 7376. (doi:10.1038/s41598-019-43553-4)
23. Lanuza JB, Bartomeus I, Godoy O. 2018 Opposing effects of floral visitors and soil conditions on the determinants of competitive outcomes maintain species diversity in heterogeneous landscapes. *Ecol. Lett.* **21**, 865–874. (doi:10.1111/ele.12954)
24. Zu P, Boege K, Del-Val E, Schuman MC, Stevenson PC, Zaldivar-Riveró A, Saavedra S. 2020 Information arms race explains plant-herbivore chemical communication in ecological communities. *Science* **368**, 1377–1381. (doi:10.1126/science.aba2965)
25. Zu P *et al.* 2021 Pollen sterols are associated with phylogeny and environment but not with pollinator guilds. *New Phytol.* **230**, 1169–1184. (doi:10.1111/nph.17227)
26. Kantsa A, Raguso RA, Dyer AG, Sgardelis SP, Olesen JM, Petanidou T. 2017 Community-wide integration of floral colour and scent in a mediterranean scrubland. *Nat. Ecol. Evol.* **1**, 1502–1510. (doi:10.1038/s41559-017-0298-0)
27. Adler LS, Barber NA, Biller OM, Irwin RE. 2020 Flowering plant composition shapes pathogen infection intensity and reproduction in bumble bee colonies. *Proc. Natl Acad. Sci. USA* **117**, 11 559–11 565. (doi:10.1073/pnas.2000074117)
28. Dukas R, Morse DH. 2003 Crab spiders affect flower visitation by bees. *Oikos* **101**, 157–163. (doi:10.1034/j.1600-0706.2003.12143.x)
29. Frund J, Zieger SL, Tscharntke T. 2013 Response diversity of wild bees to overwintering temperatures. *Oecologia* **173**, 1639–1648. (doi:10.1007/s00442-013-2729-1)
30. Zaragoza-Trello C, Vilá M, Botías C, Bartomeus I. 2020 Interactions among global change pressures act in a non-additive way on bumblebee individuals and colonies. *Funct. Ecol.* **35**, 420–434. (doi:10.1111/1365-2435.13703)
31. Rundlof M *et al.* 2015 Seed coating with a neonicotinoid insecticide negatively affects wild bees. *Nature* **521**, 77–80. (doi:10.1038/nature14420)
32. Zaragoza-Trello C, Vilá M, Bartomeus I. 2021 Interaction between warming and landscape foraging resource availability on solitary bee reproduction. *J. Anim. Ecol.* **90**, 2536–2546. (doi:10.1111/1365-2656.13559)
33. Clark T, Luis AD. 2020 Nonlinear population dynamics are ubiquitous in animals. *Nat. Ecol. Evol.* **4**, 75–81. (doi:10.1038/s41559-019-1052-6)
34. Song C, Saavedra S. 2018 Structural stability as a consistent predictor of phenological events. *Proc. R. Soc. B* **285**, 20180767. (doi:10.1098/rspb.2018.0767)
35. Reichenbach H. 1956 *The direction of time*. Berkeley/Los Angeles, CA: The University of California Press.
36. Shmueli G. 2010 To explain or to predict? *Stat. Sci.* **25**, 289–310. (doi:10.1214/10-STS330)
37. Ives AR, Dennis B, Cottingham K, Carpenter S. 2003 Estimating community stability and ecological interactions from time-series data. *Ecol. Monogr.* **73**, 301–330. (doi:10.1890/0012-9615(2003)073[0301:ECSAEI]2.0.CO;2)
38. Almaraz P, Oro D. 2011 Size-mediated non-trophic interactions and stochastic predation drive assembly and dynamics in a seabird community. *Ecology* **92**, 1948–1958. (doi:10.1890/11-0181.1)
39. Sugihara G, May R, Ye H, Hsieh CH, Deyle E, Fogarty M, Munch S. 2012 Detecting causality in complex ecosystems. *Science* **338**, 496–500. (doi:10.1126/science.1227079)
40. Mooij JM, Janzing D, Scholkopf B. 2013 From ordinary differential equations to structural causal models: the deterministic case. In *UAI'13: Proc. of the Twenty-Ninth Conf. on Uncertainty in Artificial Intelligence*, pp. 440–448. Arlington, VA: AUAI Press.
41. Bascompte J, Jordano P, Melián CJ, Olesen JM. 2003 The nested assembly of plant–animal mutualistic networks. *Proc. Natl Acad. Sci. USA* **100**, 9383–9387. (doi:10.1073/pnas.1633576100)
42. Rohr RP, Saavedra S, Peralta G, Frost CM, Bersier LF, Bascompte J, Tylianakis JM. 2016 Persist or produce: a community trade-off tuned by species evenness. *Am. Nat.* **188**, 411–422. (doi:10.1086/688046)
43. Song C, Rohr RP, Saavedra S. 2017 Why are some plant–pollinator networks more nested than others? *J. Anim. Ecol.* **86**, 1417–1424. (doi:10.1111/1365-2656.12749)
44. Song C, Saavedra S. 2020 Telling ecological networks apart by their structure: an environment-dependent approach. *PLoS Comput. Biol.* **16**, e1007787. (doi:10.1371/journal.pcbi.1007787)
45. Olesen JM, Stefanescu C, Traveset A. 2011 Strong, long-term temporal dynamics of an ecological network. *PLoS ONE* **6**, e26455. (doi:10.1371/journal.pone.0026455)
46. Clauset A, Moore C, Newman MEJ. 2008 Hierarchical structure and the prediction of missing links in networks. *Nature* **453**, 98–101. (doi:10.1038/nature06830)
47. Guimerà R, Sales-Pardo M. 2009 Missing and spurious interactions and the reconstruction of complex networks. *Proc. Natl Acad. Sci. USA* **106**, 22 073–22 078. (doi:10.1073/pnas.0908366106)