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# Community ecology as a framework for predicting contaminant effects

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**Most ecosystems receive an assortment of anthropogenic chemicals from the thousands possible, making it important to identify a predictive theory for their direct and indirect effects. Here, we propose that the impacts of contaminants can be simplified and unified under the framework of community ecology. This approach offers predictions of the strength and direction of indirect effects, which species are crucial for propagating these effects, which communities will be sensitive to contaminants, and which contaminants will be most insidious to communities. We discuss insights offered by this approach, potential limitations and extensions, outstanding questions, and its value for integrated pest management, ecological risk assessment, and the development of remediation and ecosystem management strategies.**

## Introduction

Chemical contaminants are found in most ecosystems, can be major selective forces, can alter ecosystem functions (e.g. [1]), and are regarded as the second greatest threat to aquatic and amphibious species (behind habitat loss) [2]. Despite past environmental catastrophes associated with the pesticide DDT, the number and extent of pesticide applications has reached unprecedented levels [3]. Even recently, contaminants have been associated with reproductive impairment [4], disease emergence [5] and declines of non-targeted species [6].

One reason why we might still be seeing substantial adverse effects of contaminants is because their indirect effects (see Glossary) are often overlooked [7–10] [Also see special issues in *Environmental Toxicology and Chemistry* (1996) and *Ecological Applications* (1997) on indirect effects of contaminants.] Indeed, pesticide and industrial chemical registration decisions in the USA are based largely on tests that cannot directly detect indirect or population-level effects [11]. This is despite continued pleas to shift from the predominantly individual-based approach to toxicology to tests on higher levels of biological organization (reviewed in [9]).

Although the field of ecotoxicology has progressed substantially since the 1962 release of Rachel Carson's seminal book *Silent Spring*, one statement often still rings true: 'Chemicals are pre-tested against a few individuals, but not against living communities' [12]. This is disconcerting because increasing evidence suggests that the indirect effects of pesticides are more common and complex than are their direct effects [7–9,13,14]. Furthermore, recent legislation, such as the US Food Quality Protection Act, and a greater emphasis on integrated pest management (IPM) practices are reducing applications of broad-spectrum pesticides and accelerating the development and use of more precisely targeted toxins (e.g. reduced-risk pesticide) [15]. Hence, as direct effects on non-targeted organisms are reduced, indirect effects of pesticides are only expected to become proportionally more common, placing even greater value and urgency for a framework to predict indirect effects of contaminants.

We propose here that community ecology theory can serve as this framework. In particular, because community ecology has a long history of studying indirect effects, generalities have emerged on factors that influence their direction and magnitude. We thus use concepts in community ecology to provide new insights on indirect contaminant effects. We discuss support for, and limitations, extensions and applications of, this community ecology approach, and, in addition, address outstanding questions in ecotoxicology.

## A brief historical perspective on ecotoxicology

Early pesticides (e.g. many organochlorine insecticides) had strong, negative, direct impacts on a broad range of species, both target and non-target taxa. Relative to these early pesticides, 'modern' pesticides at environmentally common concentrations typically have shorter half-lives, less biomagnification potential, and fewer direct, adverse effects on 'non-target taxa' [18] (although further testing of this assumption is required, especially in light of endocrine disruption). From early on, some studies used population and community ecology concepts to explain 'unexpected' indirect impacts of many early pesticides. For example, Lotka–Volterra predator–prey models were integral in revealing the 'pesticide treadmill', where pesticides kill both the pest and their natural predator(s) but, owing to greater or more rapid reproduction by the pests than by their predators, pesticide use results in target pest resurgence, secondary pest outbreaks, pesticide resistance and an increased dependency on pesticides [16,17]. In addition, food-web theory was used to develop models to predict the transport and biomagnification of chemical contaminants [7].

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## Glossary

**Biomagnification**: cumulative increase in the concentration of a chemical in successively higher trophic levels generally as a result of predation.
**Community stability**: tendency of populations to persist with low temporal variability and for community composition to remain constant.
**Food-web connectance**: observed number of trophic interactions divided by the total number of possible interactions.
**Indirect effects**: effect on a species mediated by another species or factor.
**Intraguild predation**: where predators can consume each other.

Although ecotoxicologists have long been aware of the potential importance of indirect effects of contaminants, attempts to incorporate these effects thoroughly into ecological risk assessment or to forecast their direction and strength with ecological theory have been limited. For example, recent modeling-based methods for assessing effects of contaminants on natural communities assume that overall impacts represent an accumulation of independent effects estimated by conventional, single-species, laboratory dose–response tests (e.g. distribution-based extrapolation models or species-sensitivity distribution models [19]). Although these methods have enhanced ecological risk assessment, they typically ignore sublethal effects, mediating effects of other abiotic factors, and species interactions and identities, all of which can regulate community structure and processes [19]. Incorporating these neglected elements will be necessary to predict accurately community-level effects from single-species toxicity tests.

## Contaminants and the paradigms of community ecology

Chemical contaminants have traditionally been considered abiotic stressors and, in many cases, contaminants will be best incorporated into community models as abiotic disturbance. However, contaminant-induced mortality can often be similar to the effects of selective predators (albeit some significant differences; Box 1) [7,20,21]. Indeed, some ecology texts do not make strong distinctions between abiotic stress and predation (e.g. [17]), and there are cases where it might be more insightful to use community ecology models that examine contaminants as 'predators' or even 'competitors' than as an abiotic disturbance (Box 1).

Although real communities typically have networks of multi-species interactions, a useful conventional framework for organizing ideas on indirect effects in community ecology involves the use of three species interaction modules (Figure 1). These modules can be seen as the building blocks of complex species interaction webs and can reveal the dynamic consequences of interaction-web structure. In particular, our theme is that considering even broad types of contaminants as players within these interaction modules offers insights into factors that govern how indirect effects of contaminants propagate through communities. Although we recognize that contaminants do not have exactly the same role as living species do, we describe common indirect effects driven by species and how contaminants can elicit similar responses. That is, when contaminants are loosely viewed as 'species' in interaction modules, we can generate predictions for when contaminant indirect effects will be particularly strong and which communities or species will be particularly susceptible to, or even benefit from, those effects.

### Box 1. The interesting case of prescriptively applied pesticides

Similar to predators, pesticides kill organisms and induce costly changes in their traits. On the one hand, pesticides are not living organisms and so, at first, a 'pesticide-as-predator' analogy seems likely to have shortcomings. On the other hand, in situations where pesticides are prescriptively applied (i.e. in response to pest outbreaks as opposed to those that are applied proactively to prevent outbreaks), patterns of pesticide use might mirror predator responses to prey. For example, prescriptively applied pesticides and their pests might exhibit coupled cycles and coevolution that is the signature of predator–prey dynamics. This is because humans apply these pesticides when pest populations increase and often adjust application rates and/or amounts based on the size and extent of the pest outbreak. This is especially the case with the increased use of IPM. Thus, pesticide concentrations will peak and decline soon after the pest population peaks and will be proportional to the pest outbreak (but they cannot be modeled on a per capita basis). Moreover, humans are constantly tinkering with pesticide structures to subvert resistance developed by pests, similar to the coevolution of predators and prey. Although the behavior of humans is crucial to this analogy, it is the pesticide, rather than the human, that is analogous to the predator because the pesticide is what cycles and coevolves with the pest. Needless to say, this analogy only functions for the specific targeted pest, given that there would be no coupled cycles between any other organisms and the pesticide.

The value of population theory on predator–prey dynamics for providing insights into the relationship between pests and prescriptively applied pesticides will depend on understanding the limits of this pesticide-as-predator analogy. There are also several questions that still need to be addressed. For instance, are the coupled oscillations between prescriptively applied pesticides and pests indefinite and offset by a quarter phase, as predicted by Lotka–Volterra models, or do they exhibit delayed density dependence, damped oscillations and stable-point equilibria [63]? Can predator–prey theory offer insights into how to disrupt any stability in these cycles to drive pests to long-term extirpation? Is pesticide resistance developing at a rate that requires evolution to be incorporated into any ecological models (e.g. [66])?

### *Keystone effects*

The simple view of contaminants is that they have negative effects on organisms. However, community ecology has long emphasized that both abiotic factors and keystone species can increase or maintain species diversity when competitive exclusion characterizes interactions on a focal trophic level [22,23]. A keystone species is defined as a predator or consumer that enhances the abundance of one or more inferior competitors by reducing the abundance of a superior competitor (Figure 1a) [24]. Disturbance can function similarly if it too reduces competitive exclusion [22].

Predation or disturbance that maintains or increases diversity is thought to be due to a fundamental tradeoff between competitive ability and resistance to the predation or disturbance. By analogy, a keystone contaminant should hinge on a tradeoff between tolerance to toxins and competitive ability, one that has been documented repeatedly (e.g. [25–27]). For example, Hanazato [28] showed that short-lived insecticides increase zooplankton diversity by selectively killing competitive dominants. Sheffield and Lochmiller [29] and Risch and Carroll [30] showed that

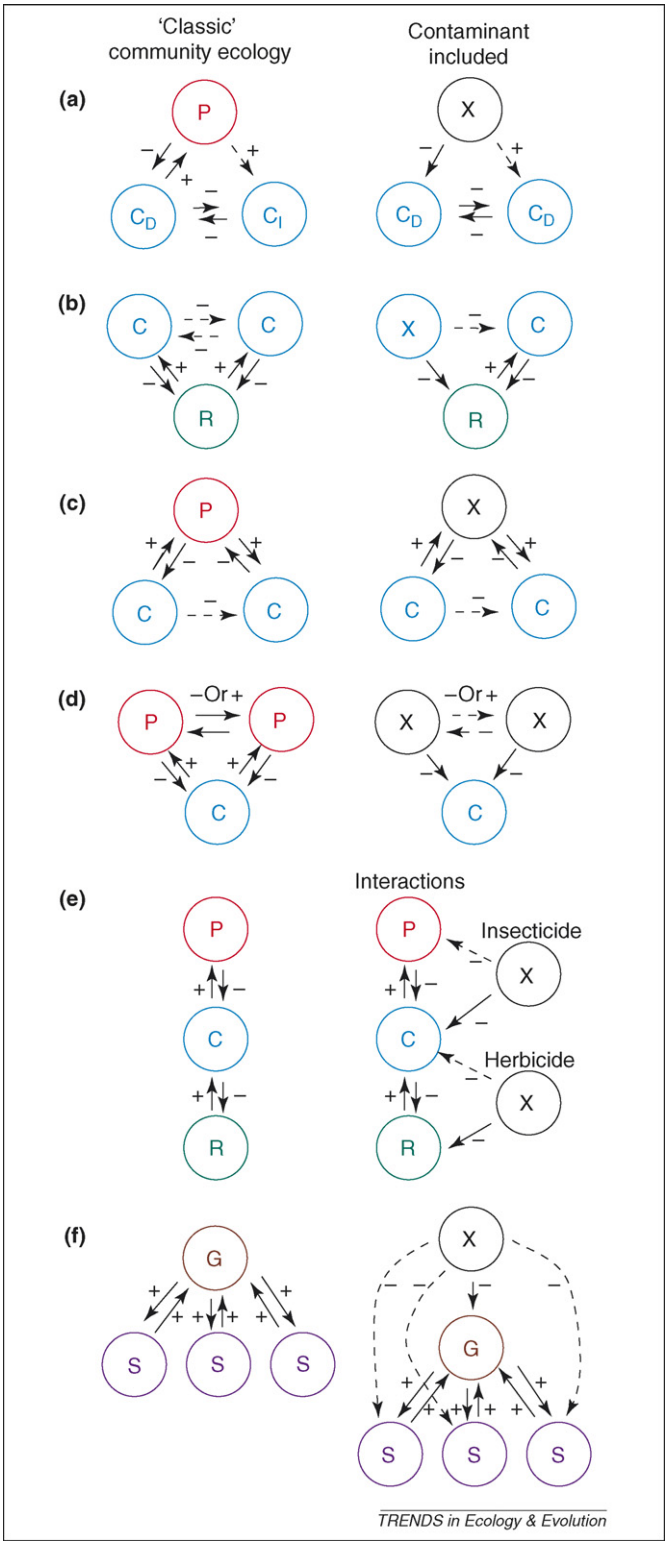


**Figure 1**. Species interaction modules depicting the hypothesized effects of contaminants relative to the 'classic' community ecology scenario without contaminants. In these interactions modules, density-mediated interactions can be replaced by trait-mediated interactions to obtain the analogous trait-mediated indirect effects. **(a)** Keystone predation: contaminant impacts on competitive dominant species ($C_D$) enable competitive inferior species ($C_I$) to persist. **(b)** Consumptive competition: contaminant impacts on prey negatively impact predators of that prey. **(c)** Apparent competition: as a result of shared sensitivity to a pesticide, pesticide use to control a pest species negatively impacts other species that do not compete with the pest. **(d)** Multiple predators: contaminants can interact additively, synergistically, or antagonistically with one another, similar to intraguild predators. **(e)** Tri-trophic interactions: contaminants can have direct and indirect effects at three, and sometimes more, trophic levels. **(f)** Direct mutualism: contaminants can affect the central hub of generalists by having indirect effects on the specialists that rely heavily on these generalists. Key: C, consumer; G, generalist; P, predator; R, resource; S, specialist; X, contaminant. Dashed lines represent indirect effects, solid lines are direct effects. Positive and negative effects are signified by + and −, respectively.

insecticides altered competitive interactions in mammal and insect communities, respectively, in favor of competitively inferior species. A common driver of the tradeoff between competitive ability and predation resistance is the frequent positive correlation between prey foraging activity and predator encounter rates. Interestingly, increased foraging activity might also increase encounters with heterogeneously distributed pollutants and should elevate oxygen demands, thus increasing the intake of contaminated air or water.

Despite the generality of tradeoffs between competitive ability and predation resistance, predation elevating or maintaining diversity is not as common as initially expected [31]. This is presumably because predation, and disturbance in general, tend only to increase diversity at low to intermediate intensities. In the face of frequent and severe stress, diversity is reduced because only a few species can tolerate it [22,23]. The insight for ecotoxicology is that, whereas we generally expect high concentrations of contaminants to reduce diversity, low to intermediate concentrations (or perhaps exposure durations) might maintain or increase it, but only if the contaminant has disproportionately large impacts on competitive dominants. This dependency on the concentration of the contaminant and its relative impact on competitive dominants might be partly why it appears rare for contaminants to increase or maintain diversity.

### *Consumptive competition*

Consumptive competition occurs when predator or consumer species inhibit one another by consuming a shared resource (Figure 1b). Some contaminants can have similar effects. For example, herbicides often have negative effects on herbivores by reducing plant availability [8,9,13,14]. Although this relationship is unidirectional and thus does not strictly fit the definition of competition, it does involve a highly asymmetric negative relationship driven by a 'shared resource', as does most competition [17]. This suggests that the application of competition theory could provide insight into understanding community dynamics in contaminated systems. For instance, classic niche theory predicts that competition will be stronger if species have greater niche overlap, and that species with narrow niches might thus often escape competition. By analogy, a contaminant should have a large indirect effect on an organism if the range of taxa negatively impacted by a contaminant overlaps greatly with the dietary range of that organism. Because they forage on fewer resource types, specialists might be affected less frequently than generalists by indirect effects of contaminants (e.g. [32]). However, if the resources of the specialist are affected (directly or indirectly) by the contaminant, the magnitude of the effect might be greater for specialists, particularly if they cannot easily switch their diets to consume species that are tolerant to the contaminant.

### Apparent competition and indirect mutualism

Apparent competition involves negative interactions between prey species through a shared predator rather than shared resources. An increase in either prey species increases the abundance of a shared predator, resulting in increased predation on the other species (Figure 1c) [33]. Here, we view pesticides as being analogous to the shared predator. One might think that, because contaminants are not living organisms, they would not respond positively to changes in the abundance of vulnerable taxa; however, some pesticides, as controlled by humans, respond to pest numbers (Box 1), suggesting that pesticide-driven apparent competition is possible. That is, an increase in either of two pests can induce an increase in pesticide use, which negatively impacts both pests. For example, pesticide-driven apparent competition is likely to occur with the control of black fly and mosquito populations, taxa that rarely compete but can have similar sensitivities to pesticides (e.g. [34]).

Ecological theory also predicts indirect, positive, bidirectional (mutualism) and unidirectional (commensalism) effects between species. For example, prey species might share a predator but, unlike apparent competition, an increase in either prey species might divert the attention of the predator away from the other species, resulting in no increase in total predation and a positive, bidirectional, indirect effect between the prey species [35]. Similarly, given a scenario where there are limits on total pesticide use (e.g. owing to costs or regulations) and where pests are controlled with different pesticides, an increase in one pest could divert overall pesticide use away from the other, with the side effect of releasing this pest from control (and vice versa). Clearly, an understanding of pesticide use by humans is crucial for predicting even the qualitative direction of indirect impacts on communities. As with apparent competition, and consistent with analogous predator–prey theory [35], the strength of pesticide-driven indirect mutualism or commensalism should depend upon the encounter rates and degree of shared sensitivity of the pests with the applied pesticides.

### Multiple predator effects

Most organisms are exposed to more than one type of predator [36]. Similarly, organisms are often exposed to multiple contaminants [37]. In some cases, the combined presence of two predator taxa results in greater than expected prey mortality (relative to a null multiplicative model), whereas in other cases, two predator species result in less than expected prey mortality [36]. In multiple predator theory, these are referred to as risk enhancement and risk reduction, respectively. Risk enhancement tends to occur when the foraging strategies of predators complement one another, so that prey responses to one predator can increase prey susceptibility to the other predator. Risk reduction commonly occurs when predators engage in either strong interference competition or intraguild predation (Figure 1d) [36].

Despite the lack of coupled cycles, or feedbacks, between many contaminants and their vulnerable taxa (Box 1), much of multiple predator theory does not explicitly rely on these feedbacks; hence, the emergent impacts of multiple predators should have parallels to the effects of contaminant mixtures. For example, mixtures of contaminants can have additive or synergistic direct effects on species (Figure 1d) [37]. These effects tend to occur when the contaminants differ in their modes of action [37], similar to how complementary foraging strategies might enhance the foraging success of intraguild predators. There are many examples, such as the combination of the herbicide atrazine and organophosphate insecticides synergistically increasing their toxicity to invertebrates [38]. Antagonistic interactions between contaminants often occur when contaminants have similar chemical structures competing for binding sites [37], akin to similar predators exhibiting interference and exploitative competition. These parallels between multiple contaminants and multiple predators might enable multiple predator theory to serve as a framework for predicting the direct and indirect effects of multiple contaminants.

### Tri-trophic interactions

Changes in the abundance of species mediated through consumer–resource interactions can also have indirect effects that proliferate across three or more trophic levels. Tri-trophic interactions can be generated by top-down or bottom-up effects (Figure 1e) [39–41]. Tri-trophic indirect effects have been repeatedly documented with pesticides and hydrocarbon pollutants [8,13,14], providing further evidence that indirect community pathways can have an important role in understanding contaminant effects. For instance, herbicides and fungicides can have bottom-up effects because they reduce primary productivity and fungi, major food sources for higher trophic levels [8,13,14], but the relative contributions of other contaminant types to top-down and bottom-up processes seem less predictable.

The factors that determine the strength of top-down and bottom-up effects remain contentious, but some generalities have emerged. These effects are generally weaker with greater diversity, greater omnivory, weaker species interactions, invertebrate relative to vertebrate carnivores, detritus-based food webs, and in closed experimental and terrestrial (relative to aquatic) systems ([39–43] but see [44]). Similar factors might dictate the strength of pesticide-induced cascades. If so, this should facilitate identifying contaminant-sensitive communities, species that transmit toxicant effects, and contaminants that are especially harmful to ecosystems.

### Trait-mediated indirect effects

Here we have described indirect effects in community ecology that are driven primarily by changes in the density of species, termed density-mediated indirect interactions (DMIIs). We now know that indirect effects do not require changes in species abundances but can occur merely with the modification of species traits [45]. For example, carnivores can have strong positive effects on plants not by killing herbivores, but by reducing herbivore feeding rates or by promoting herbivore dispersal [46].

A recent meta-analysis revealed that these trait-mediated indirect interactions (TMIIs) can have equal to or greater impacts on communities than can DMIIs [46].

The same might be true for contaminants because, at ecologically relevant concentrations, they probably cause more trait changes than direct mortality (e.g. [47]; Figure 1). For instance, a common effect of contaminants is reduced growth because of the metabolic costs of detoxification. For prey that can grow to exceed the gape of their predators or those that need to grow large enough to metamorphose before their environment dries, reduced growth could mean longer exposure to potentially more dangerous aquatic predators and an increased risk of desiccation [48,49]. Contaminant-induced changes in behavior also increase the predation and desiccation risk of non-targeted species [8,50]. Physiological trait changes associated with chemical exposure, such as the effects of endocrine disruptors on reproduction [4] and the impacts of immunotoxicity on disease risk [5], are now of great concern to toxicologists and the public.

Despite pervasive sublethal effects of modern pesticides, TMIIs have been understudied even more so than DMIIs in ecotoxicology. This might be because TMIIs are more difficult to predict. A useful approach to help guide searches for contaminant-induced sublethal effects and their associated TMIIs is to compare the observed effects of a contaminant on a community to a null model that includes only known, direct, lethal effects of the contaminant and the predicted accompanying DMIIs. Substantial deviations from the null model would suggest the location of unaccounted for contaminant effects.

### *Direct mutualism*

Although we have focused on antagonistic interactions between species, such as those between predators and prey or between herbivores and plants, many communities can have substantial numbers of direct facilitative and mutualistic interactions [51], such as those between plant species and their pollinators and seed dispersers. Bascompte *et al.* [52] recently used network theory to examine communities of mutualists and discovered that networks of mutualists tend to be highly complex, with specialists that tend not to interact with other specialists but with a central and dense core of generalists that all interact with each other (Figure 1f). This architecture is consistent across many mutualistic webs and appears to be shaped by coevolution selecting for convergence and complementarity of traits among interacting species that facilitate the incorporation of new species into mutualistic interactions [53]. By contrast, antagonistic interactions between predators and prey and herbivores and plants tend to generate smaller clusters within the larger interaction web (i.e. more compartmentalized) and fewer species interactions because prey and plants are selected to escape these negative interactions [53].

Bascompte *et al.* emphasize that the tendency for specialists to associate with generalists should enhance community stability essentially because generalist hubs should be reliable; they should be buffered against stochastic fluctuations in any one of their mutualists. However, this network structure could backfire when contaminants are involved. If contaminants have substantial direct or indirect deleterious effects on the species of the central hub of mutualistic networks, it could be catastrophic to the many specialists that rely on this hub [54] and could lead to system collapse (Figure 1f). By contrast, webs dominated by antagonistic interactions are more compartmentalized than those dominated by mutualistic interactions and, thus, adverse indirect effects of contaminants might be less likely to influence substantial portions of the broader interaction web. Consequently, antagonistic webs might be more 'tolerant' to contaminants than are mutualistic webs. Network theory, which was crucial in revealing the architecture of mutualistic webs, should facilitate identifying the types of communities that are susceptible to contaminants and the core species in mutualistic webs that hold the network together.

## Possible extensions

Some researchers have quantified the impacts of contaminants on ecosystem processes but, in general, our knowledge remains scant [1,21,55]. Increasing evidence links community composition to ecosystem functions [56], suggesting that a promising extension of the community ecology approach is to predict the indirect effects of contaminants on ecosystem processes. Both Odum and Rapport *et al.* [57,58] formulated similar expectations for ecosystem responses to stress. Efforts that merge community regulation models, such as that of Menge and Sutherland [23], with ecosystem stress models should be particularly fruitful because their synthesis would enable predicting ecosystem responses to stress, as well as identifying ecosystems with functions or services that are vulnerable to certain stressors and the species responsible for this fragility. However, functional endpoints are typically less sensitive to stress than are structural endpoints because of functional redundancies among species [13,14,21]. Thus, an improved understanding of species interactions and functions will be needed to predict accurately species-mediated effects of contaminants on ecosystem processes.

## Benefits of the approach

We propose that there are three crucial questions that need to be addressed in ecotoxicology: (i) which community types or structures are most sensitive to pollution? (ii) Which species are most threatened by contaminants? and (iii) on which of the tens of thousands of registered chemicals should we focus our attention? Community ecology can facilitate addressing each question because it generates testable predictions regarding which communities will be sensitive to contaminants, which contaminants will be particularly insidious to communities, and which species are crucial for transmitting adverse contaminant effects. These predictions are based on our knowledge of factors that alter community stability by contributing to the transmission of indirect effects.

Community viability analysis (CVA), the analog to population viability analysis [42], and network theory [52–54] are promising tools for predicting the indirect effects of contaminants. They use knowledge of species richness, species interaction strengths, trophic links and their distributions to identify: (i) 'key species' whose loss (and possibly decline) triggers secondary extinctions; and (ii) fragile communities where the loss of a species, on

average, causes a considerable reduction in system stability. These key species might represent valuable model species for chemical testing, especially given that some governments are reluctant to incorporate community-level testing requirements for chemical registration decisions [11]. These approaches might also assist the chemical registration process by predicting responses to present and untested contaminants (e.g. [59]). Likewise, identifying fragile communities that receive contaminants will help to target remediation and management measures appropriately and prioritize systems in dire need of IPM strategies and biological pest control.

A community ecology context should also help focus our research efforts appropriately on a more manageable number of contaminants. To help prioritize research on contaminants, Pimentel and Edwards [60] suggested that we consider five chemical attributes: persistence, water solubility, lipid–water coefficient, toxicity characteristics (how broadly toxic they are) and adsorption. We suggest that the ecological attributes of chemicals should also be emphasized, such as the expectation that a chemical will reduce community stability and trigger secondary extinctions, and the effect a chemical has on fragile communities and threatened, endangered, at-risk and invasive species. For example, from a pest control perspective, the indirect negative impact of a given pesticide, via its negative effect on the natural enemies of the pest, is reduced if the pesticide has greater adverse effects on intraguild natural enemies than on strict natural enemies. This is because empirical and theoretical work on food-web connectance suggests that predators that specialize on prey species (low connectance) are more effective at controlling prey populations than are intraguild predators that consume both the prey and its natural enemies (high connectance) [61]. Ultimately, we need a greater emphasis on simultaneously comparing the effects of multiple contaminants at standardized and relevant concentrations [47]. This will facilitate evaluating which contaminants are most insidious to our ecosystems and assessing whether dealing with general categories of contaminants can simplify the complexity of ecotoxicology (Box 2).

Community ecology theory also engenders predictions of stressor combinations that might enhance or diminish deleterious indirect effects (Figure 1d). For example, top-down control of primary productivity by arthropods should be reduced by insecticides, exacerbating the eutrophication effects of nutrients alone. Herbicides should reduce the impacts of nutrient inputs by reducing primary productivity. And, because both insect grazers and photosynthetic organisms directly or indirectly rely on organic material, nutrient inputs might accelerate the recovery of systems impacted by insecticides or herbicides. There is increasing evidence supporting all of these predictions [62–64]. This suggests that the timing of agrochemical applications could be slightly altered to reduce negative impacts on ecosystems and that community ecology offers a foundation for stressor mixture models that could substantially reduce the complexity of ecotoxicology and be a tool for improving IPM, ecosystem management and ecological risk assessment.

Finally, because contaminants modify species abundances, they have a history of use for elucidating important ecological relationships and processes (reviewed in [7,8]); thus, a more thorough integration of ecotoxicology and community ecology should mutually benefit both disciplines. It is time to use ecological theory more

**Box 2. Future research directions**

**General support for the benefit of ecological theory**
How common are keystone and intraguild contaminant effects? What are the mechanisms underlying tradeoffs between susceptibility to contaminants and competitive ability? Do conclusions about the strengths of trophic cascades apply to contaminants?

**Fundamental community ecology**
The community ecology framework depends upon our level of understanding of indirect effects and the dynamics of species interactions; thus, advances in these areas are crucial (particularly with regards to TMIIs, CVA and network theory).

**Spatiotemporal scales**
Effects of contaminants can be influenced by historical factors [67] and recovery processes, such as density-mediated compensation and dispersal [64,68]. However, these processes that occur before and after stressor exposure are typically neglected [34,50,67–69]. Similar to temporal dynamics, spatial aspects can be important and difficult to interpret. For instance, are species rare in contaminated habitats because they were killed by the contaminant or because they avoided it? A challenge will be to integrate individual- to ecosystem-level responses [70] and spatiotemporal dynamics more thoroughly into ecotoxicology.

**Managing mixtures and multiple stressors**
How common are antagonistic, additive and synergistic interactions between contaminants and are they predictable from community ecology theory? Can we predict the composition of communities exposed to contaminant mixtures by using interaction-web models to adjust simultaneously the densities of taxa (at a range of levels) that are directly vulnerable to the components of the mixtures? What biotic and abiotic factors are most likely to interact synergistically with contaminants and why? Two of the greatest hurdles to overcome are predicting responses to contaminant mixtures and the cumulative effects of contaminants with other stressors [48,50,71].

**Mathematical modeling**
The few attempts so far to develop interaction–web models to predict contaminant effects have been successful [8,59,64,65]. Because many contaminants biomagnify, linking these interaction–web models with contaminant transport models is an important challenge [62].

**Contaminants and the concept of the guild**
Many species show consistent sensitivity to contaminant types [8,14,25], suggesting that grouping contaminants with similar modes of action, analogous to guilds in community ecology, could reduce the complexity of ecotoxicology. However, how much variation in community-level responses is there within and between contaminant 'guilds', and what is the appropriate level of grouping, type (e.g. herbicide versus insecticide), class (e.g. organophosphate insecticide versus triazine herbicide), sub-class, and so on?

**Resistance and population dynamics**
What are the consequences of resistance to contaminants and the associated genetic bottlenecks? How rapidly is resistance occurring and should this evolutionary process be incorporated into ecological models [66]?

**Inactive ingredients**
Some inactive ingredients in chemical formulations can be more toxic and less predictable than active ingredients (e.g. [72]). How do we manage this when trade formulations are often confidential?

**Ecosystem functions**
Can we predict contaminant-induced changes in ecosystem processes by knowing species sensitivities, interactions and functions?

extensively to understand contaminant effects, and for ecologists to examine their own systems more thoroughly in light of chemical contamination.

The species interaction-web approach to ecotoxicology is being adopted in the Netherlands and, in the few cases where it has been used, has successfully predicted contaminant effects [59,64,65]. (See also US Environmental Protection Agency. AQUATOX model validation reports, http://www.epa.gov/ost/models/aquatox/download.html). This framework is still in its infancy and will require substantial advances in our understanding of higher-level contaminant effects and other complexities (Box 2). However, the important upshot of simplifying and unifying ecotoxicology under the predictive and mechanistic framework of community ecology is that it should help improve remediation and management strategies, IPM, ecological risk assessment and overall environmental health.

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