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**Direct and indirect effects of chemical contaminants on the
behaviour, ecology and evolution of wildlife**

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1 **Direct and indirect effects of chemical contaminants on**
2 **the behaviour, ecology and evolution of wildlife**

3

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18

19

20 **Abstract**

21 Chemical contaminants (e.g. metals, pesticides, pharmaceuticals) are changing ecosystems
22 via effects on wildlife. Indeed, recent work explicitly performed under environmentally
23 realistic conditions reveals that chemical contaminants can have both direct and indirect
24 effects at multiple levels of organisation by influencing animal behaviour. Altered behaviour
25 reflects multiple physiological changes and links individual- to population-level processes,
26 thereby representing a sensitive tool for holistically assessing impacts of environmentally
27 relevant contaminant concentrations. Here, we show that even if direct effects of
28 contaminants on behavioural responses are reasonably well-documented, there are significant
29 knowledge gaps in understanding both the plasticity (i.e. individual variation) and evolution
30 of contaminant-induced behavioural changes. We explore implications of multi-level
31 processes by developing a conceptual framework that integrates direct and indirect effects on
32 behaviour under environmentally realistic contexts. Our framework illustrates how sublethal
33 behavioural effects of contaminants can be both negative and positive, varying dynamically
34 within the same individuals and populations. This is because linkages within communities
35 will act indirectly to alter and even magnify contaminant-induced effects. Given the
36 increasing pressure on wildlife and ecosystems from chemical pollution, we argue there is a
37 need to incorporate existing knowledge in ecology and evolution to improve ecological
38 hazard and risk-assessments.

39

40 **Keywords:** behavioural ecology, chemical pollution, ecotoxicology, endocrine disrupting
41 chemicals, evolution, indirect effects, sublethal

42 **1. Introduction**

43 Contamination of the environment with diverse inorganic and organic compounds, such as
44 pesticides, pharmaceuticals, and metals, represent one of the main environmental challenges
45 driven by anthropogenic activity. In 2010, the global chemical industry's value was US\$4.12
46 trillion, having risen 54% over a decade [1]. In addition, the trend towards global
47 urbanisation is concentrating chemical consumption in cities faster than environmental
48 interventions and remediation systems can be implemented, including in developing countries
49 near biodiversity hotspots [2]. The increasing production and release of chemicals means that
50 wildlife, humans and ecosystems are continuously exposed to chemical contaminants. While
51 large-scale mortality events of wildlife represent an obvious, if rare, sign of chemical
52 releases, chemical contaminants can elicit more subtle but nevertheless important and
53 harmful ecological impacts [3]. Further, chemical contamination of the environment is
54 certainly not limited to short-term, acute exposures. Effects of long-term, low-level chronic
55 exposures can be equally deleterious, though less obvious for human observers. In this
56 review, we develop a conceptual framework that integrates concepts and approaches from
57 multiple disciplines to investigate how chemical contaminants can alter animal behaviour,
58 with resultant impacts on short- (e.g. individual and community) and long-term (e.g.
59 evolutionary) responses, potentially leading to population declines.

60 Research on chemical contaminants conventionally recorded a limited range of
61 endpoints, most commonly by studying mortality following exposure in the laboratory and/or
62 by testing the impact of a single contaminant on a single species under standardised
63 laboratory conditions ([4], but see [5]). These approaches are logistically tractable and
64 repeatable but are criticised for their simplicity, particularly when such experiments neither
65 take chemical nor biological complexity into account [6]. Behaviour, on the other hand, is the
66 result of numerous complex developmental and physiological processes, and so connects

67 physiological function and ecological processes [7]. Thus, behavioural change provides a
68 comprehensive measure of both direct and indirect effects of chemical contaminants on
69 individuals, linking to population-level processes [8-10] and, importantly, is often impacted
70 at much lower contaminant concentrations than are traditional toxicological endpoints [11].
71 Here, we illustrate how behavioural responses can represent a powerful, highly quantifiable
72 and biologically relevant indicator of environmental impacts.

73 Chemical contaminants can affect animal behaviour both directly and indirectly.
74 Direct effects on behaviour in wildlife—here, we focus mostly on vertebrates—are caused by
75 contaminants acting on the physiology of an animal (e.g. impaired sensory or cognitive
76 abilities, altered endocrine/neural signalling, metabolic dysfunction). To date, research in
77 behavioural ecotoxicology has largely focussed on direct effects of contaminants on
78 individuals (e.g. activity) (see section 2). In contrast, indirect effects, when contaminant-
79 induced changes to animal behaviour in one organism or species have cascading effects on
80 other organisms and species in the exposed system, have received far less attention [12-15].
81 Indirect effects are most pronounced when a contaminant affects exposed organisms
82 differentially, such as when one species is more sensitive and another more resistant (i.e.
83 asymmetrical effects; [12,14,16]). While the importance of investigating both direct and
84 indirect effects of contaminants is evident, this multi-directional approach has rarely been
85 applied in ecotoxicology (but see [15,17]).

86 In this review, we **focus** exclusively on studies conducted under ‘natural’ conditions,
87 specifically measuring behavioural responses following contaminant exposures in the wild **or**
88 **at environmentally relevant concentrations in the laboratory**. We first critically examine
89 existing literature on the role of chemical contaminants in mediating direct effects on
90 individual behaviour (section 2). In contrast to previous reviews [14,17], our focus centres on
91 sublethal effects, particularly those induced by emerging contaminants, such as

92 pharmaceuticals. Moreover, as well as considering short-term, mean behavioural responses to
93 exposure, we discuss how chemical contaminants can alter trait variance (i.e. plasticity) and
94 act as potent evolutionary forces. Moving from effects on individuals, we investigate how
95 chemical contaminants can alter inter-specific interactions indirectly via changes in
96 behaviour of susceptible species (section 3). By integrating these collective effects, we
97 develop a conceptual framework to identify ways in which animal behaviour can be affected
98 by chemical contaminants (section 4). In doing so, we use predator-prey interactions as a case
99 study to demonstrate how our conceptual framework has real-world impact. While we
100 highlight the challenges of scale and complexity involved with predicting ecological effects
101 of chemical contaminants (section 5), we also provide directions for future research (section
102 6). Finally, the overarching aim of this review is to improve research practices by increasing
103 the ecological relevance of research approaches employed, in order to uncover global hazards
104 and risks posed by chemical contaminants.

105

106 **2. Direct effects on individual behaviour**

107

108 Here, we discuss why, in a rapidly changing world, we need to expand our concept of direct
109 effects—perhaps more accurately ‘mean behavioural responses’—to incorporate the potential
110 for chemical contaminants to affect both plasticity in, and evolution of, behavioural
111 responses.

112

113 **2.1. Direct effects**

114 Exposure to chemical contaminants can result in direct effects on a range of both ‘general’
115 behaviours (e.g. activity levels)—changes in which can have knock-on effects on multiple
116 fitness-related traits—and specific mechanisms underpinning specific behaviours. Given that

117 behaviour is the product of inter-connected physiological, **anatomical**, and neurological
118 processes, and, in the wild, organisms are usually exposed to chemical cocktails rather than
119 single contaminants, pinpointing mechanistic pathways between exposure to a contaminant
120 and a behavioural change can be challenging. For example, round gobies (*Neogobius*
121 *melanostomus*) collected from heavily contaminated industrial sites (e.g. PCBs, PAHs,
122 metals) [18] or exposed to municipal wastewater effluent [19] both showed reduced
123 aggression, even though the contaminant mixtures were very different.

124 Disruption of reproductive behaviours resulting from exposure to chemical
125 contaminants has been increasingly studied in both laboratory and field settings because of
126 the obvious population-level consequences [8]. Mechanisms underlying such behavioural
127 changes include contaminant actions on endocrine and neural signalling, via changes to
128 receptors, enzymes and/or transporters [20-22]. For instance, environmental exposures to
129 organochlorine pesticides reduces parental care behaviour in predatory birds [23]. Studies on
130 fish have demonstrated that exposure to municipal wastewater treatment plant effluent (e.g.
131 [19]), and the active ingredients in (and metabolites of) the oral contraceptive pill, reduce
132 nest building and courtship behaviours (reviewed in [20]). Furthermore, exposure to the
133 insecticide endosulfan disrupts pheromonal communication between the sexes in red-spotted
134 newts (*Notophthalmus viridescens*), leading to disrupted mate choice and depressed mating
135 success [24]. Apparently subtle changes in reproductive behaviour could potentially be as
136 devastating for fitness as major malformations of reproductive morphology, because an
137 animal that fails to attract a mate or care for offspring appropriately will accrue zero fitness.

138 Changes in animal movement (e.g. frequency and speed) following contaminant
139 exposure are common behavioural endpoints in ecotoxicological studies [25, 26]. For
140 example, small-scale activity, which is often measured in the laboratory, has high ecological
141 importance because it increases encounter rates with both resources (e.g. food, potential

142 mates) and risks (e.g. predators, diseases). Activity also underlies individual dispersal and
143 migration tendencies [27,28], although smaller scale movements measured in the laboratory
144 do not automatically reflect larger scale movements in the field. Chemical contaminants can
145 alter these movement behaviours by disrupting either sensory capabilities used to locate
146 suitable environments and resources (e.g. inability to detect chemical cues; [29-31]) or
147 physiological pathways governing and supporting movement (e.g. neural/endocrine
148 disruption, metabolic dysfunction; [32,33]). Contaminants can, for instance, directly impair
149 movement, making animals less adept at capturing prey and/or escaping predators, as has
150 been noted in vertebrates exposed to acetylcholinesterase-inhibiting pesticides [34]. So far,
151 only a handful of studies have connected these measures to dispersal or migration in the wild.
152 One such study showed that Atlantic salmon (*Salmo salar*) smolts exposed to the anxiolytic
153 pharmaceutical oxazepam migrate faster both in laboratory migration pools and down a river
154 [35]. In contrast, while round gobies collected from heavily contaminated environments
155 dispersed more slowly in a laboratory maze, there was no evidence that dispersal was
156 affected in the wild [36]. Recent work has also demonstrated that exposure of European
157 starlings (*Sturnus vulgaris*) to a polychlorinated biphenyl (PCB) mixture in the laboratory
158 resulted in reduced activity and incorrect orientation for migration [37], indicating that
159 exposed birds might migrate later and less accurately in the wild. Overall, activity seems to
160 be a sensitive and relatively easily measured endpoint but its potential to indicate individual
161 fitness or population-level processes is assumed rather than proven, in most cases.

162 Chemical contaminants can also interfere with complex behaviours, such as predator-
163 avoidance, grouping and aggression, which have direct implications for fitness and
164 population dynamics. By acting on the sensory system, contaminants can affect an
165 organism's responses to conspecifics or predators by, for example, reducing their ability to
166 detect stimuli, but also rendering them less active or motivated to respond [29]. **If receivers**

167 are unable to detect prey, predators or signals from conspecifics, or alternatively if signallers
168 emit altered signals, this could lead to ineffective communication [38]. The resulting
169 disruption of group interactions and coordination could potentially reduce the anti-predator
170 and food-location benefits of grouping [39]. By impacting conspecific detection pathways,
171 chemical contaminants can also alter aggression and dominance hierarchies among
172 individuals. For example, captive rainbow trout (*Oncorhynchus mykiss*) exposed to cadmium,
173 which damages the olfactory epithelium, were less aggressive towards an unexposed rival
174 and therefore, formed dominance hierarchies faster [40].

175 Interestingly, some chemicals, such as psychoactive pharmaceuticals, have actually
176 been designed to modulate adaptive stress or fear responses. Thus, they have great potential
177 to impact foraging and anti-predator responses of wild animals (e.g. [41-44]). Indeed, recent
178 studies have shown that exposure of fish to environmentally relevant concentrations of the
179 antidepressant fluoxetine can extend the duration of 'freezing' behaviour [44] after predatory
180 attack and increase activity levels regardless of the presence of a predator [43]. Because
181 natural selection favours individuals that can quickly and accurately detect and assess risk,
182 any disruption of this fine-tuned system is likely to have important implications for individual
183 fitness [45] (see electronic supplementary material for more on predator-prey effects).

184

185 **2.2 Plasticity**

186 Individuals can adjust their behaviour in response to chemical contaminants, i.e. they show
187 phenotypic plasticity [7]. This 'plasticity' in behaviours has been the subject of much interest
188 in behavioural ecology, because of its role in enabling species to cope with rapid
189 environmental change [46, 47]. However, most studies so far have focused primarily on the
190 mean behavioural responses of the contaminated population, with little to no mention of the
191 variance in the trait. To date, we are unaware of any research explicitly investigating how

192 contaminants can modulate behavioural plasticity or flexibility (i.e. how responsive
193 individuals are to environmental variation) (but see [41]; section 5). Predictions as to how
194 plasticity will be modulated by chemical contaminants are not straightforward. **If a behaviour**
195 **is attenuated by a contaminant by, for example, all individuals becoming inactive regardless**
196 **of environmental conditions, this could erode plasticity. Thus, there would be no benefit to**
197 **individuals having variable responses to environmental changes, because they would never**
198 **be expressed. Consequently, over time this could decrease the intensity of selection for**
199 **plasticity.** In turn, this could reduce population variation in responsiveness to environmental
200 change, reflecting a decrease in variance in behavioural responsiveness of all individuals.
201 Conversely, one study found that exposure of jumping spiders (*Eris militaris*) to pesticides
202 led to an increase in within-individual behavioural variability, whilst not changing the
203 population's average level of predatory behaviour [48]. There is a clear need to integrate new
204 experimental designs, technologies and statistical approaches (e.g. [35,47-50]) from
205 behavioural ecology to measure individual behavioural responses under varying
206 environmental conditions, such as, for example, multi-stressor studies, to better understand
207 the consequences of contaminant exposure.

208

209 **2.3 Chemical contamination drives evolution**

210 There is growing interest in the long-term, multi-generational consequences of chemical
211 contamination and how contaminants might modulate population persistence and
212 evolutionary trajectories. Our current focus is on how selection can act directly on exposed
213 organisms, although it is important to acknowledge that selection may also operate indirectly
214 via impacts of chemical contaminants on, for example, a species' prey, or competitors (see
215 section 4).

216 It is established that exposure to chemical contaminants can result in the evolution of
217 physiological resistance, with perhaps the best-studied example being the micro-evolution of
218 resistance in populations exposed to metal pollution (see [51,52]). By contrast, far less is
219 known about how this resistance might affect the subsequent behavioural responses of
220 exposed organisms. Adaptive physiological adjustments could reduce the likelihood that
221 downstream behaviours are maladaptive. **On the other hand, changes in physiology can also**
222 **have negative effects on behaviour and life histories via the reallocation of resources required**
223 **for growth and reproduction.** For example, laboratory selection for cadmium resistance in
224 least killifish (*Heterandria formosa*) resulted in decreased fecundity, female life expectancy,
225 and brood size [53]. Whether such trade-offs also impinge on behaviour remains to be tested.

226 Even in the absence of physiological resistance, organisms can simply change their
227 behaviour, for example altering their diet, to avoid contaminants. However, it is often unclear
228 whether these behavioural changes reflect plasticity or evolved responses [54,55]. Studies
229 have shown spatial avoidance of contaminated sediments and water by aquatic invertebrates
230 [55] and vertebrates [54,55], as well as adjustment of migration routes by salmon in response
231 to metal pollution [56]. Other species show temporal avoidance of potential contaminant
232 exposure by employing a faster life history or changing reproductive timing [52]. An
233 interesting hypothesis is that the adaptive potential of an organism to respond rapidly to
234 strong selection favouring earlier maturation and reproduction could, in turn, facilitate
235 adaptations to novel stressors, such as chemical contaminants [57].

236 If organisms have neither evolved physiological tolerance nor behavioural
237 compensation, exposure to chemical contaminants can result in drastic population declines
238 [58]. **This potentially creates a destructive feedback loop where a reduction in population size**
239 **leads to further loss of genetic diversity, thus restricting the adaptive potential of populations**
240 **[59, 60], including adaptive behavioural responses.** Chemical contaminants (e.g. persistent

241 organic pollutants) can also affect mutation rate (e.g. [61]), which may either compensate for
242 the loss of genetic diversity during population bottlenecks (e.g. marsh frogs, *Rana ridibunda*;
243 [62]) or otherwise alter population responses to contaminants [63]. However, most
244 contaminant-induced mutations are likely to be deleterious [64]. Thus, adaptive behaviour
245 that shields genotypes from otherwise harsh selection imposed by chemical contaminants
246 could allow for population persistence and the maintenance of adequate levels of standing
247 genetic variation crucial for further adaptation [65].

248 Chemical contaminants can also impact the strength and targets of selection via their
249 direct effects on behaviour. For example, since sexually selected behaviours can affect the
250 rate and trajectory of evolution (e.g. [66]), contaminants that interfere with sexual selection
251 (e.g. endocrine-disrupting chemicals, EDCs; [67]) have considerable potential to affect
252 subsequent evolution. For example, in European starlings, treatment with an EDC mixture
253 resulted in males producing longer and more complex songs that are preferred by females,
254 despite exposed males also having suppressed immune responses [68]. Whereas, in guppies
255 (*Poecilia reticulata*), exposure to the agricultural contaminant 17 β -trenbolone increased the
256 occurrence of coercive copulatory behaviour in males, thus circumventing female mate
257 choice [69]. While such changes that weaken sexual selection could further contribute to
258 population decline [70], some studies find the opposite effect, whereby sexual selection
259 enhances the evolution of mechanisms to cope with contaminants, presumably resulting in
260 population growth. For example, flour beetles (*Tribolium castaneum*) evolved resistance to a
261 pyrethroid pesticide faster when sexual selection was allowed to occur compared to when it
262 was experimentally precluded [71].

263 Given the importance of evolution in facilitating population persistence, a key
264 question is: what might limit the ability of organisms to evolve adaptive physiological or
265 behavioural responses to contaminants? One possibility is that it may be difficult to

266 adaptively respond simultaneously to multiple contaminants, or, more broadly, multiple
267 stressors that exert conflicting selection pressures [72]. Resistance to a single class of
268 contaminants, such as pesticides, can evolve very fast, but evolving resistance to cocktails of
269 contaminants with different modes of action is likely to be much slower. Here, the ability to
270 cope with a particular contaminant could make it more difficult to deal with another [63]. A
271 complementary idea emphasises the role of evolutionary history—i.e., the notion that
272 organisms often have greater difficulty coping with stressors that are truly ‘novel’, as
273 opposed to those that are mechanistically similar to those that are familiar [73]. Clearly there
274 is a need is for a deeper mechanistic understanding of when and why plastic or evolutionary
275 responses to one contaminant should facilitate or conflict with responses to another.

276

277 **3. Indirect effects of chemical contaminants on behaviour via interspecies interactions**

278 Contaminants can, as outlined above, exert direct effects on the behaviour of species, which
279 often results in decreases in organism abundance. However, species and their behaviours can
280 also be altered *indirectly* because changes in behaviour (or abundance) of susceptible species
281 will lead to cascading indirect effects—even on resistant species—at all trophic levels within
282 a community. One of the most commonly documented indirect effects of contamination is
283 predator responses to reduced prey abundance caused by contaminant-induced direct lethality
284 or reproductive failure in their prey species. A population crash of fathead minnows
285 (*Pimephales promelas*), caused by experimental EE2-exposure of a whole lake, led to
286 cascading indirect effects: zooplankton populations in the exposed lake increased without
287 minnow predation, while the biomass of larger lake trout (*Salvelinus namaycush*) decreased
288 without minnows as a prey item [14]. Indirect effects can also reduce the efficacy of
289 ecosystem services provided by wildlife. For instance, population crashes of *Gyps* vultures in
290 India due to diclofenac toxicity resulted in an increase in feral dogs scavenging on decaying

291 carcasses and a consequent increase in human rabies infections from dog bites [74]. **In**
292 **contrast**, examples of indirect effects caused specifically by changes to animal behaviour are
293 rare in the literature [16]. For example, mummichog (*Fundulus heteroclitus*) from industrial
294 sites were less active and less adept at capturing prey grass shrimp (*Palaemonetes paludosus*)
295 than were fish at pristine sites, allowing these prey to grow larger and become more abundant
296 [75]. We predict that contaminant-induced increases in boldness or aggression in one species,
297 for example, will change the competition and predation pressures on, and thus alter the
298 behaviour of, other species within a community (Figure 1). **Contaminant-disrupted courtship**
299 **leading to declines in abundance**, are predicted to have cascading effects on the interspecies
300 interactions across a community. **Here, we use cascading effects as a tool to illustrate the**
301 **importance of indirect effects in ecological risk-assessment, although other indirect effects**
302 **such as keystone predator effects and exploitative competition can also be locally important**
303 **[76]. The key point, here, is the need to understand the mechanism, i.e. the contaminant**
304 **induced change in behaviour(s), initiating the cascade.**

305 Given the complexity of studying multi-species responses to contaminants [12], it is
306 not surprising that indirect community effects, particularly those acting via changed
307 behaviours, have not yet been broadly studied and quantified. First, multiple organisms must
308 be studied simultaneously in real time using environmentally realistic mesocosms or field-
309 based studies. Second, the system often must be studied for longer durations than are typical
310 of laboratory exposures (i.e. several months to years). One might argue that studying indirect
311 effects is redundant since the net effect on the community is the ultimate endpoint. However,
312 since species compositions differ between most environments and reactions to contaminants
313 can be highly species-specific, the net effect on a mesocosm community will only provide the
314 outcome for that particular community. Without a mechanistic understanding of which
315 behaviours in which species are affected and how, the generality, and, as such, the predictive

316 power of mesocosm studies for risk-assessment of particular contaminants is limited at best.
317 Knowledge of indirect effects is also crucial for modelling ecological risk, a promising and
318 cost-effective tool that will help to reduce the number of animals required for
319 ecotoxicological testing.

320

321 **4. Conceptual framework for understanding the ecological and evolutionary impacts of** 322 **chemical contaminants**

323

324 Here, we have developed a conceptual framework that can be used by researchers aiming to
325 design experiments or research programmes that move away from the ‘one chemical – one
326 species – one (usually lethal) endpoint’ style of ecotoxicology (but see [71]) towards a more
327 holistic approach. Specifically, our framework demonstrates the direct and indirect effects of
328 chemical contaminants on the behaviour of individuals within a population, and of species
329 within communities. We draw upon knowledge and literature from ecology and lay out
330 potential scenarios of community-level effects caused by chemical contaminants (Figure 1).
331 Since communities are composed of interconnected populations overlapping in time and
332 space, the effects of chemical contaminants on communities necessarily manifest in the
333 interactions within and among populations [72]. For example, some of the most
334 salient interactions shaping ecological communities worldwide are between prey and their
335 predators [72,73]. All animals are either prey or predators at some point in their lives and this
336 interaction often has considerable consequences on individual fitness and population size
337 [74].

338 Imagine that a chemical contaminant is introduced into an ecosystem. This chemical
339 does not change the behaviour of top predator ‘species B’, but does increase the boldness of a
340 second top predator ‘species A’, resulting in ‘species A’ taking more risks, spending longer

341 foraging and less time avoiding predators. ‘Species C’, the prey of species A, which is
342 resistant to the contaminant, is indirectly affected because increased **time and energy spent to**
343 **anti-predator behaviours** but it is still consumed at a higher rate than when the ecosystem was
344 uncontaminated. Thus, prey species C decreases in numbers, which, in turn, causes its own
345 plant prey ‘species D’ to proliferate, thereby shifting the nutrient cycling and changing the
346 ecosystem for all species (Figure 1a). Notably, if the contaminant’s action was conserved
347 across taxa, such that species C also became bolder, its population would rapidly decline by
348 predation-induced mortality from species A. Further, the decreased numbers of prey species
349 C could potentially result in predator species B changing its foraging preference to alternative
350 prey. The risky behaviour of species A will increase its own probability of being preyed
351 upon, attacked by competitor species B and/or eating novel but toxic or infected foods. This
352 would, in turn, decrease the predation pressure from predator species A on species C, and
353 could potentially decrease competition between species A and B (Figure 1b) [72]. We have
354 included dynamic feedback loops to magnify the actions of the chemical contaminant on both
355 directly and indirectly affected species, which, in turn, have community-level consequences
356 and can alter ecosystem functioning (Figure 1b).

357 Importantly, indirect effects due to contaminant-induced behavioural shifts could
358 cause systems to respond far more strongly and quickly than an assessment of direct effects
359 alone, or simply monitoring changes in the abundance of key predators, would predict [73].
360 Moreover, contaminant-mediated effects could yield novel forms of ecological interactions
361 by, for example, inducing prey-switching due to changes in predatory behaviour and/or
362 changes in prey abundance or quality, or by differentially altering the vulnerability of
363 individuals or species to parasites [75]. Also, we have focused on the top-down effects, but
364 some contaminants will affect primary productivity and so will have bottom-up impacts.
365 These can be difficult to predict but, again, could have indirect, sublethal effects by

366 increasing competition for food and/or necessitating greater foraging distances. Such a
367 framework allows us to integrate and go beyond individual experiments and encourages
368 researchers to assess behavioural change within its environmental context. By understanding
369 the behavioural mechanism underpinning multi-level changes, modelling, for example, can
370 be used to predict the impacts of contaminants with similar modes of action for enhanced
371 environmental risk assessments [77]. As an implementation plan, we provide Figure 2,
372 which directs researchers to consider which experimental design (laboratory, mesocosm or
373 whole ecosystem manipulations) and level (individual, species or community), or modelling
374 approaches are required, and which endpoints should or could be tested. Our basic framework
375 can, therefore, be applied to specific behaviours and/or interspecific interactions, as well as to
376 different levels of organisation, as required.

377

378 **5. Problems of scale and complexity: predicting effects in the wild from effects in the** 379 **laboratory**

380

381 Predicting the ecological effects and behavioural perturbations caused by chemical
382 contaminants is valuable for guiding legislation and policy to protect wildlife but it is also
383 challenging for many reasons. Behaviour is inherently variable—although so are many of the
384 physiological endpoints currently measured—and how organisms respond to any given
385 contaminant may vary across an individual's lifetime, between sexes, among individuals of
386 the same species, and across species with different life-histories, habitat use, trophic position,
387 and/or physiology [7,10,33,75,78].

388 Most earlier standardised ecotoxicological tests used model species that are easily
389 cultured with simple, measurable endpoints [4], which allowed direct comparisons of toxicity
390 among different compounds. This long-used approach has efficiently generated hazard and

391 risk-assessments for many chemical contaminants under the premise that similar species are
392 equally affected by the contaminant. Of course, the ‘all species are the same’ argument does
393 not hold for the effects of many contaminants (e.g. pharmaceuticals [79]). Inter- and intra-
394 species differences in physiology, behaviour and life history, when coupled with differential
395 metabolism, generate substantial differences among species and individuals in susceptibility
396 and responses to chemical contaminants. Unfortunately, our understanding of comparative
397 mechanistic responses to contaminants still remains quite limited, even for model laboratory
398 organisms.

399 Susceptibility differences between species are one of the key challenges in
400 ecotoxicology. For example, studies have shown that small wild-caught prey fish are more
401 sensitive to the anxiolytic effects of the pharmaceutical oxazepam than larger predatory fish
402 or laboratory-reared fish [5,80,81]. This could be due to species differences in the rate and
403 extent of pharmaceuticals being taken up, metabolised and concentrated. Indeed,
404 bioconcentration of pharmaceuticals in fish tissues can differ by several orders of magnitude
405 between species [82], and even across life-history stages [83]. Therefore, two species
406 inhabiting the same polluted system can be exposed to very different internal concentrations
407 of contaminants [81]. Moreover, tests including a less vulnerable life-stage might
408 underestimate ecological risk [83]. Such differential exposures, and the associated effects,
409 make it very difficult to predict the ecological effects of chemical contaminants in the
410 environment [16].

411 Differential behavioural responses to chemical contaminants in laboratory-reared
412 versus wild species have also been explained by the lack of predation risk or high
413 competition in laboratory environments, which selects for inherited behavioural phenotypes
414 that are often bolder, more aggressive and less responsive to predators than wild-type
415 individuals [84]. For example, in assessing the risk of chemicals that potentially modify anti-

416 predator behaviour, using a laboratory fish model that may exhibit a suppressed basal
417 behavioural response to predators may greatly underestimate actual risk in the field (Figure
418 3). Also, the distribution of behavioural traits studied should be characterised within each test
419 group [83]. This consideration is critically important because a contaminant that acts to
420 increase activity and/or boldness will more likely generate behavioural change in individuals
421 originating from a (wild-type) population of low competition/high predation, compared with
422 a (lab-reared) high-competition/low-predation population that contains many active and bold
423 individuals (Figure 3). Even in the wild, populations of the same species under different
424 predation pressures are known to have evolved different physiology, morphology and
425 behaviours [84]. In terms of our conceptual framework, such population-level differences in
426 behavioural responses will alter both the state of a community prior to contamination, and the
427 magnitude of feedback loops triggered by a contaminant. Such differences between
428 populations, generated by differing selection regimes, have received very little attention
429 despite clearly being important considerations when assessing contaminant vulnerability.

430

431 **6. Future directions**

432 The use of behavioural studies enables us to link the effects of contaminants at multiple
433 levels of organisation, from individual to ecosystem. This is an invaluable asset, because
434 chemical contaminants have a wide range of actions and effects. At the individual-level, the
435 fields of behavioural ecology and so-called ‘personalised medicine’ are increasingly realising
436 the need to analyse inter-individual variation in responses, not just population means [46].
437 Far from being ‘noise’, plasticity in responses in itself represents a trait that can shape the
438 capacity of individuals and populations to cope with environmental change in the short term.
439 In this review, we illustrate that chemical contaminants can impact the capacity of
440 populations to persist into the future by altering the strength and targets of evolutionary

441 selection, for example via direct effects of behaviour. To date, a mechanistic understanding
442 of how evolutionary and plastic responses interact to facilitate population persistence is
443 lacking. This also limits our ability to predict how populations respond if legislation succeeds
444 in reducing concentrations of specific chemical contaminants. Consequently, we have
445 identified avenues to fill the knowledge gaps and challenge the often simplistic assessment of
446 direct effects of contaminants, specifically in terms of how behaviour and other endpoints
447 should be measured, analysed and interpreted.

448 With the rise in emerging contaminants, many of which are designed to exert
449 sublethal effects on evolutionarily conserved physiological systems at ecologically realistic
450 concentrations, it is important to update existing frameworks for studying their short- and
451 long- term consequences. Sublethal behavioural effects can be both ‘positive’ and ‘negative’
452 for individuals, populations and communities. As illustrated by our conceptual framework
453 (Figure 1) effects can vary dynamically within the same individuals and populations. Indeed,
454 this could be described as a key feature of emerging or dilute contaminants. Importantly,
455 behavioural effects can lead to top-down and/or bottom-up effects. For example, changes at a
456 lower trophic level could have sublethal effects by increasing competition for food and/or
457 necessitating greater foraging distances. This is because linkages within communities will act
458 indirectly to alter and even magnify contaminant-induced effects. Future work, integrating
459 modelling, remote sensors and tracking technologies and statistical analyses should focus on
460 quantifying changes on the individual level and how the linkages within these networks are
461 affected by contaminants. We argue that understanding the behavioural and ecological
462 mechanisms underpinning contaminant-induced population changes will greatly increase the
463 accuracy and power of Environmental Risk Assessment to protect wildlife and ecosystems
464 from disturbance by chemical contaminants.

465

466 **Authors' contributions**

467 MS, TB and KEA organised the symposia on which this paper is based, developed the
468 conceptual framework, edited the manuscript and created figures. All authors contributed to
469 publication writing. All authors gave final approval for publication.

470

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486

487

488 **Figure legends**

489 Figure 1. Outline of our conceptual framework modelling the direct and indirect effects of a
490 chemical contaminant using predator-prey dynamics as a case study. Two predatory species
491 (A and B) are exposed to a chemical contaminant. a) State 1 shows initial changes to species
492 in the food web at the individual and community levels; b) State 2 includes feedback loops,
493 which show dynamic interactions between species in time and space. Increases and decreases
494 in population size for each species are indicated by arrows. The solid arrows indicate direct
495 effects, dashed arrows indirect effects, dotted arrows nutrient cycling, and blue arrows
496 species interactions.

497

498 Figure 2. Implementation plan suggesting methodological approaches for utilising our
499 conceptual framework to identify the routes by which animal behaviour is affected
500 by chemical contaminants. For each level of biological organisation (individual, species,
501 community and ecosystem), we highlight some of the factors that should or could be
502 quantified or experimentally manipulated.

503

504 Figure 3. The distribution of expressions of a trait (here, activity) in two populations from
505 environments with different levels of predation risk. a) Population collected from the field
506 (high predation); b) Laboratory-bred population (low predation). Black arrows illustrate the
507 potential for contaminant-induced increases in activity in the populations (the longer the
508 arrow, the greater the potential change).

509

510

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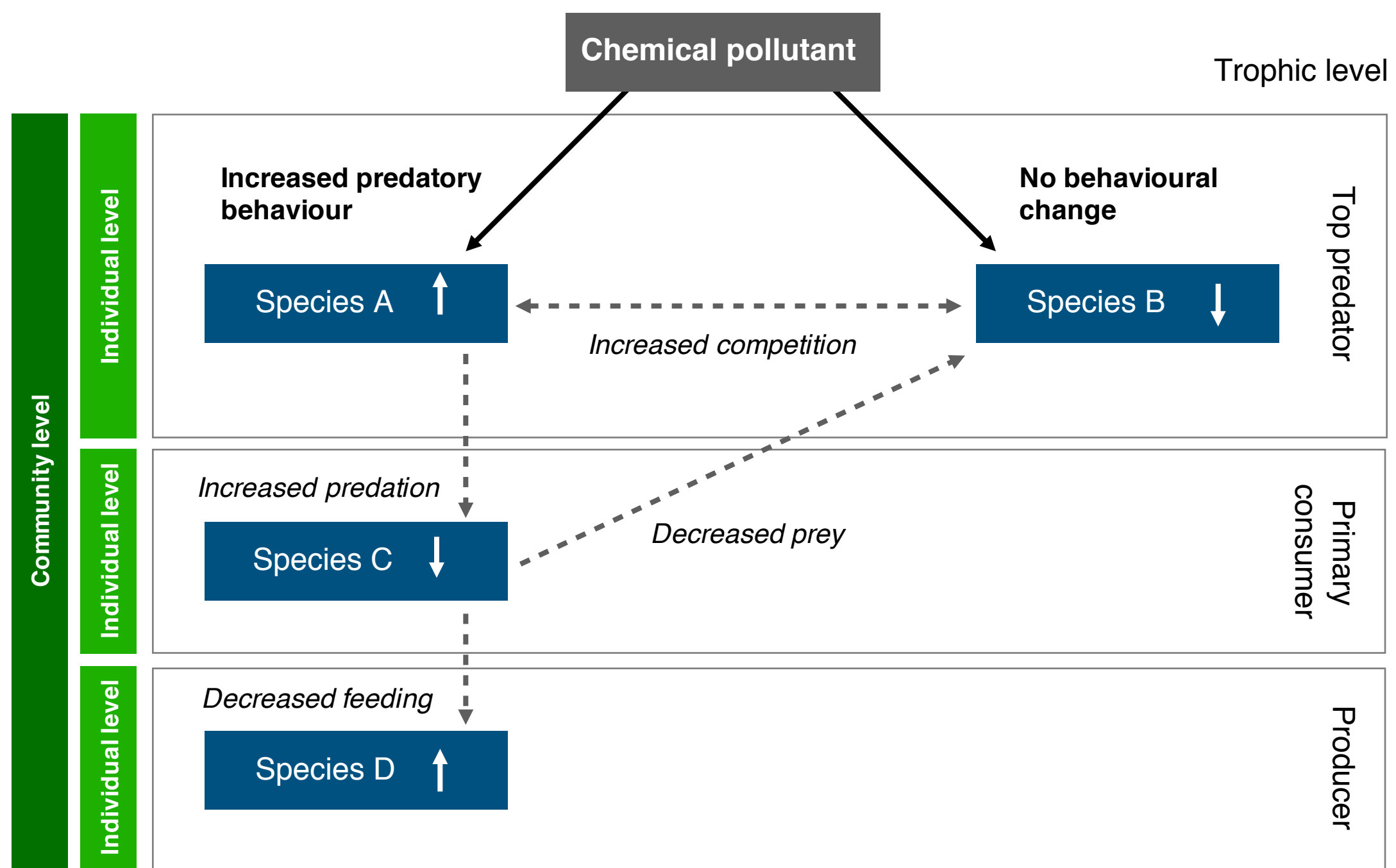
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- 730

a) State 1 – Initial changes



b) State 2 – Feedback loops

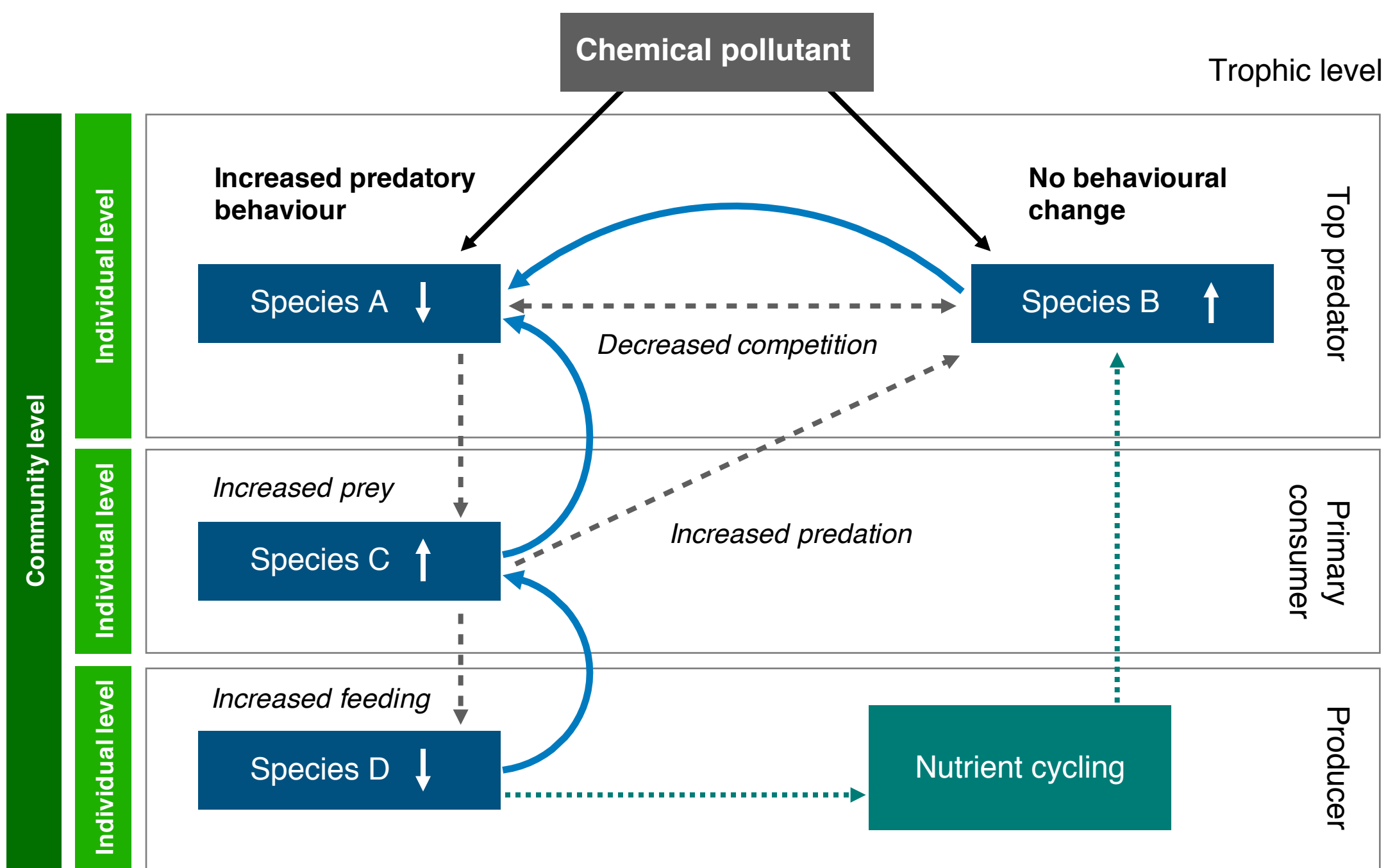


FIGURE 1

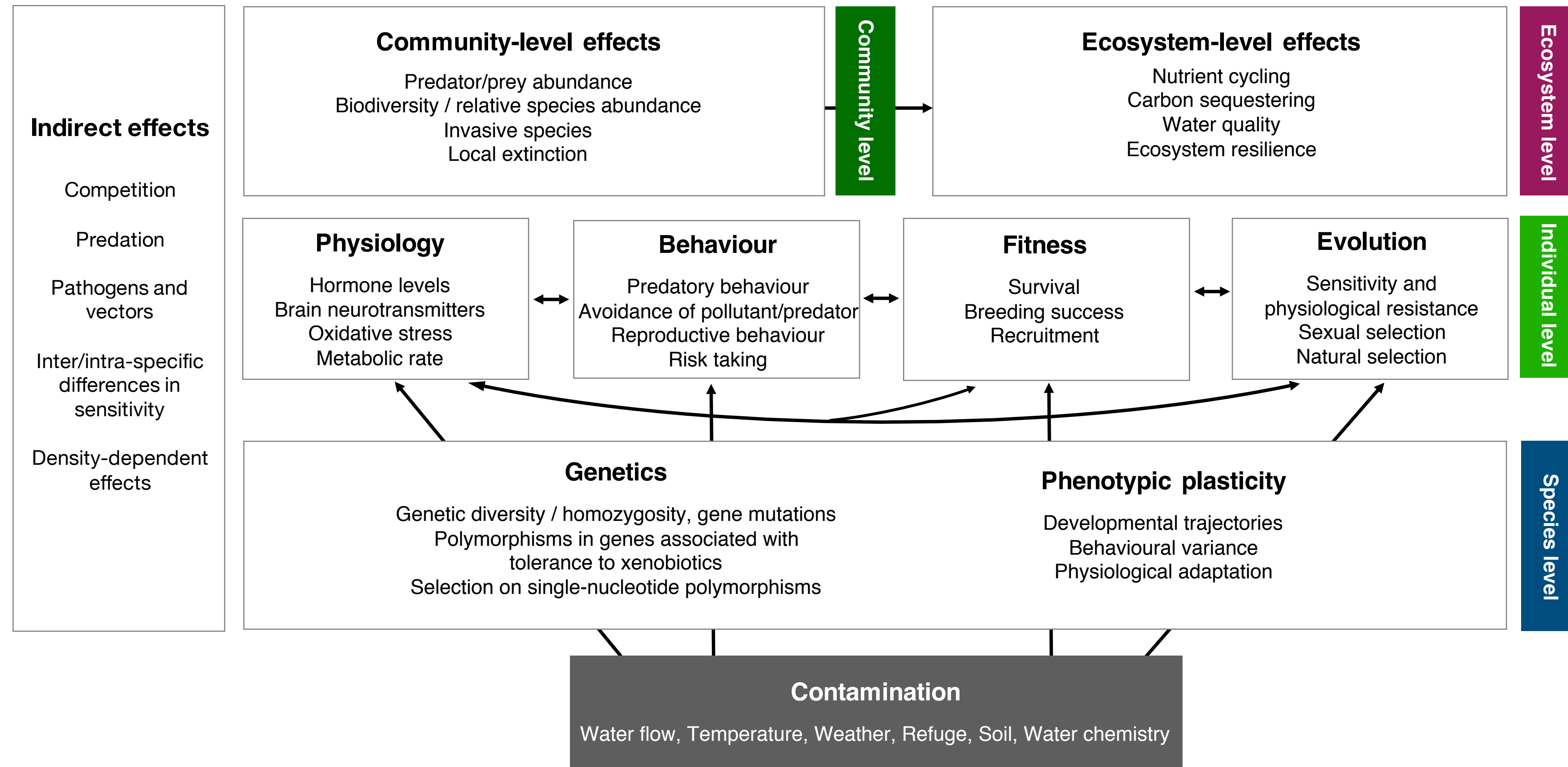
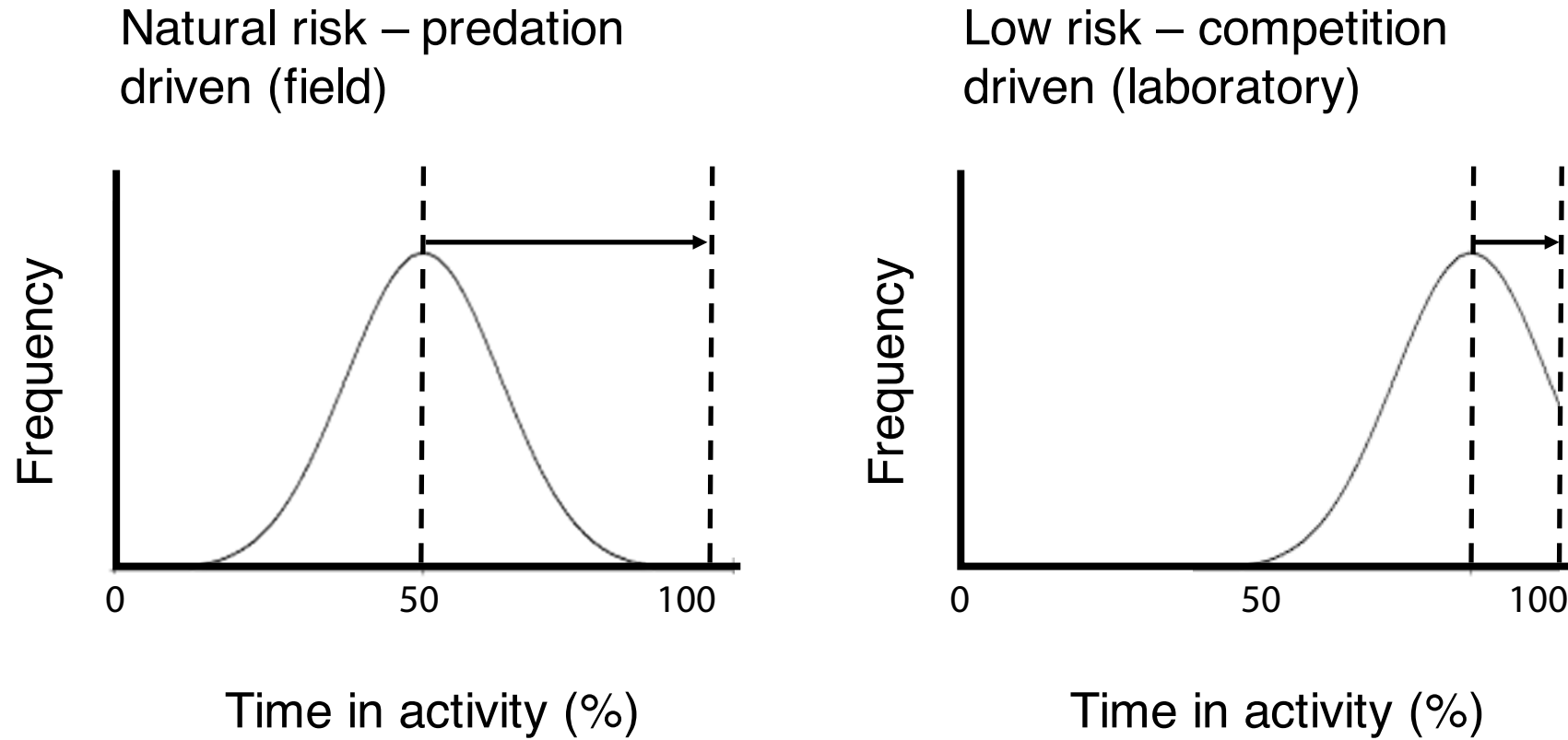


FIGURE 2

**FIGURE 3**