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Schistosoma transmission: scaling-up competence from hosts to ecosystems

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Abstract:

In a One-health context, it is urgent to establish the links between environmental degradation, biodiversity loss and the circulation of pathogens. Here we review and literally draw a general vision of aquatic environmental factors that interface with *Schistosoma* species, agents of schistosomiasis, and ultimately modulate their transmission at the ecosystem scale. From this synthesis, we introduce the concept of ecosystem competence defined as ‘the propensity of an ecosystem to amplify or mitigate an incoming quantity of a given pathogen that can be ultimately transmitted to their definitive hosts’. Ecosystem competence integrates all mechanisms at the ecosystem scale underlying the transmission risk of a given pathogen and offers a promising measure for operationalizing the One-health concept.

Scaling-up the competence concept from hosts to the ecosystem level: an illustration with *Schistosoma*

A key consequence of current global changes is a modification in the circulation of infectious diseases on a planetary scale [1,2]. In the face of rapid global change, we need a better understanding of the transmission dynamics underlying infectious disease emergence, changes in pathogen abundance, and origin and persistence of **transmission hotspots** (see glossary). This will also enable refinement of management of programs for established foci of infectious diseases, while sustainably delivering health to the other constitutive components of **social-ecological systems** [3–5]. However, this is challenging because the spread and transmission of pathogens relies on intrinsic characteristics of the pathogens, on those of their hosts and on those of the environment, all of which are potentially influenced by social-systems [4,6–8].

Reservoir hosts and vectors have received considerable attention because the dispersal, local establishment and transmission dynamics of pathogens depend on the spatial and temporal distribution of their hosts. The circulation of a given pathogen also depends on **hosts' competence** [9,10] (**Box 1**). Host competence is not a fixed property and partly depends on biotic and abiotic environmental factors [9]. Environmental changes may not only modify the distribution and abundance of hosts available for the pathogens but may also influence the **community competence** [11,12]. In multi-host communities, changes in species diversity may increase or decrease community competence and thus pathogen transmission depending on whether the most competent hosts are favoured (i.e., amplification effect) or disadvantaged (i.e., dilution effect) [11,13,14]. At the ecosystem level, several abiotic and biotic factors, increasingly influenced by social-systems, can also foster or hamper the transmission dynamics of pathogens by acting on the abundance, **infectivity** and **virulence** of the pathogen [15–18]. Together these elements converge to the idea that the transmission dynamics of pathogens are

governed by complex ecological processes that act at the ecosystem level. They call for more integrative studies that account for the interactions between pathogens, their hosts, and the environment, this strategy being at the heart of the **One-Health** concept [19,20]. In this context we propose to scale up the concept of competence initially developed at the host and community scales to the ecosystem level. We define the ‘**ecosystem competence**’ as the propensity of an ecosystem to amplify or mitigate an incoming quantity of a given pathogen that can be ultimately transmitted to their definitive hosts.

Schistosoma species are trematode parasites associated with freshwater ecosystems that are responsible for several forms of bilharziasis in humans and animals [21]. They display a complex two-host life cycle that involves a vertebrate definitive host in which the adults develop and sexually reproduce, and an intermediate freshwater snail host in which the parasite reproduces asexually (**Box 2**). The transmission ecology of *Schistosoma* is well documented and tightly linked to aquatic ecosystems whose contours are generally well delineated (e.g., pond, stream, lake), making them ideal models to draw an integrative and comprehensive mechanistic vision of the ecosystem competence concept. Here, we briefly overview how intrinsic characteristics of aquatic ecosystems can influence the transmission of *Schistosoma* and literally draw a general vision of these interactions into a central scheme. Based on this overview we next propose a more general conceptual model of ecosystem competence. We finally provide future research directions that will be necessary to assess the competence of natural ecosystems. Due to the abundance of literature on the subject, we have focused our bibliographic research on *Schistosoma mansoni* and *Schistosoma haematobium*, the two most prevailing species in human populations.

Transmission of *Schistosoma*: an ecosystem overview

In an ecosystem perspective, any factor acting on the *Schistosoma*'s free-living stages (hereafter SFLS), on the parasitic forms within their aquatic snail hosts and/or on their snail hosts, might affect the final number of cercariae produced locally and thus the infection risk for vertebrates. Ecosystem competence applied to *Schistosoma* could be defined as the propensity of a freshwater aquatic ecosystem to amplify or mitigate cercariae production, that can be ultimately transmitted to a given vertebrate host, from an incoming quantity of miracidia into that ecosystem.

Ecosystem processes influence free-living stages' abundance and infectivity.

Early experimental studies have shown that *S. mansoni* egg hatching is influenced by several abiotic factors under laboratory conditions (e.g. light, temperature) [22]. Although never validated *in natura*, some specific environmental conditions could favour or hamper egg hatching, and hence influence, at this early stage, the competence of the receiving ecosystem.

Once released, SFLS have limited intrinsic resources which force them to find a host quickly or die (i.e. 10 – 20 hours) [23]. Survival and infectivity of SFLS are influenced by water physicochemical parameters (e.g., temperature, pH) and the presence of synthesised chemicals [7,24–26]. For instance, Nguyen et al. 2020 experimentally demonstrated that increasing water temperature, as observed under current global warming, greatly improve the swimming speed and distances of *S. mansoni* miracidia and cercariae, both parameters being linked to their likelihood of encountering and infecting a host, but reduces cercariae lifespan [25].

Biotic factors influencing abundance and infectivity of SFLS have already been thoroughly reviewed [7]. At least 17 organisms can predate *S. mansoni* miracidia (N=6) or cercariae (N=11) under laboratory conditions [7]. Although very little is known about SFLS predation in natural ecosystems, they undoubtedly constitute an important feeding resource for

several species [27]. Changes in the community of SFLS predators at the ecosystem level are hence expected to influence *Schistosoma* transmission. Additionally to predation, several organisms including plants, vertebrates and non-hosts snails can act as decoys for miracidia and cercariae hence reducing the encounter rate between SFLS and their competent hosts or possibly the infectivity of SFLS [7,28]. For instance, the presence of some non-competent molluscs causes a 25-50% reduction in the prevalence of *S. mansoni* in *Biomphalaria glabrata* under experimental conditions [29]. In the same vein, field-derived *S. mansoni* miracidia do not significantly prefer their natural host *B. pfeifferi* over the introduced species *Physa acuta* in a choice chambers experiment [30]. This suggests that the presence of *P. acuta* at natural transmission sites, can lead to a reduction in *S. mansoni* prevalence in local *B. pfeifferi* populations and ultimately to a reduction of *S. mansoni* transmission dynamics.

Together these results indicate that from an initial amount of parasite eggs hatched into a given freshwater ecosystem, the prevailing environmental conditions and biotic community can act on SFLS (**Figure 1 - Case 1**). These factors potentially influence both the number of miracidia able to infect a susceptible snail host and the final number of cercariae that can infect their vertebrate hosts, and ultimately the competence of the receiving ecosystem.

Ecosystem processes influence Schistosoma's host abundance and competence

Snail populations are highly dynamic in space and time mainly due to the fluctuations of physicochemical parameters of freshwater ecosystems such as water availability, temperature, salinity and pH [31,32] (**Figure 1 - Case 2**). The alternation of dry and wet seasons influence snail abundance with generally high abundances prevailing during the wet season and no active snails observed in some temporary ponds during the dry season which temporally stops the transmission cycle of *Schistosoma* species [31,33]. Anthropogenic modifications of ecosystems can also impact snail populations. For instance, the construction of the Diama dam

along the Senegal River, which aims at locally promoting the expansion of agriculture and food security, has shifted the saline regime from a brackish to a freshwater system upstream of the dam and has thus fostered a regional increase in snail populations hosting several human-infecting *Schistosoma* species and an increase in schistosomiasis prevalence from about 10% to >80% in local human populations [34]. Conversely, recent experiments demonstrated that the increasing salination into the Nile Delta resulting from rising sea levels could reduce the survival and reproduction of *Biomphalaria alexandrina*, the overall *S. mansoni* prevalence within snails and eventually reduce local transmission risks [35]. Cercariae production per infected snail may also increase at higher temperatures as a result of an increase in host metabolic activity and a greater energy available to the parasite [25]. However, these combined effects of high temperature can significantly reduce snails lifespan, thus reducing the time during which cercariae are released [25].

Biotic factors also influence the abundance and competence of *Schistosoma*'s intermediate hosts (**Figure 1 - Case 2**). Resource availability for snail hosts, which can be limited due to intraspecific and interspecific competition [36,37], determines their development and reproduction, and ultimately their abundance but not their immune defences [38,39]. Moreover, the development of *Schistosoma* sporocysts relies on the quantity and quality of resources acquired by their host snails. This leads to a positive relationship between resource acquisition by infected snails and their abundance and competence. For instance, an experimental 24-fold increase of resource supply rate increases up to 60-fold the cercarial production of *S. mansoni* per infected *B. glabrata* snails [38]. The diet of snail species hosting *Schistosoma* and evidence for possible competition for resource availability *in natura* is poorly documented. Subaquatic vegetation such as *Ceratophyllum sp.* seems to promote the development of periphyton and consequently an increase in abundance and competence of *Schistosoma*'s intermediate hosts [40]. The production of *S. mansoni* and *S. haematobium*

cercariae per infected snail (*i.e.*, *B. pfeifferi* and *Bulinus spp.* respectively) in the presence of *Ceratophyllum sp.* in northern Senegal is significantly higher than that of infected snails from open water or habitats dominated by emergent vegetation [40]. Similarly, the combined input of agronomic fertiliser and herbicide applied at their estimated environmental concentrations in experimental freshwater mesocosms leads to an increase in algal production, and ultimately to the increase of *B. glabrata* and *B. truncatus* densities [41]. Importantly however, neither the production nor the survival of cercariae from these snails were impacted by these treatments [41].

Predation also regulates the abundance of snail hosts [42,43]. Another consequence of the construction of the Diama dam along the Senegal River is the blocking of the annual migration of the local migratory river prawns (*Macrobrachium spp.*) that predate *Schistosoma* snail hosts. The importance of *Macrobrachium spp.* in decreasing the local abundance of *Bulinus spp.* and consequently the local prevailing human prevalence in urogenital schistosomiasis was demonstrated using semi-controlled field experiment and mathematical models [42]. Interestingly, both infected *B. glabrata* and *B. truncatus* snails are more prone to predation than uninfected ones [44]. Thus, predation potentially has synergistic negative effects on global *Schistosoma*'s transmission dynamics by limiting the encounter rate between uninfected snails and miracidia and by preferentially eliminating infected snails.

Taking all these factors into consideration, ecosystems with competitors and predators of *Schistosoma*'s snail hosts are likely to be less competent to transmit schistosomiasis to vertebrate hosts than ecosystems lacking such interactions. Conversely, environmental changes that reduce the intensity of these biotic interactions at the ecosystem scale could make them more competent to transmit schistosomiasis. For instance, laboratory and field studies in Kenyan inland areas along Lake Victoria revealed that chemicals and pesticides used in agriculture, can adversely affect biodiversity in this way and, to some extent, increase the

Schistosoma infection risks at transmission foci and hence the competence of these ecosystems [45].

Schistosoma do not stand alone within their snail hosts.

Schistosoma snail host species host many other parasites and symbionts [46–48]. Considering only trematodes, 29 species were found in *B. pfeifferi* and *Biomphalaria sudanica* populations, two *S. mansoni* snail hosts in Eastern Africa [49]. Trematode diversity implies a large spectrum of potential interactions within their snail hosts including facilitation, competition or predation [46,50] (**Figure 1 - Case 3**). For instance, *Biomphalaria oligoza* and *Biomphalaria orbigny* are naturally resistant to *S. mansoni* in South America but become susceptible when previously exposed to the trematode *Zygocotyle lunata* [51]. On an epidemiological point of view, such facilitating processes might lead to the emergence –at least at short term- of schistosomiasis at sites where no apparent competent snail hosts are present, but which immune system is down-regulated by the helper, thereby enabling a state of competence to exist with a given *Schistosoma* species. In other word, the introduction of a new trematode species into an initially incompetent aquatic ecosystem can make this ecosystem competent for *Schistosoma* species as long as the facilitating trematode remains present locally.

Some trematode species established into snail hosts competent for *Schistosoma* species can also reduce the virulence of *Schistosoma* [52]. For instance, **rediae** of several echinosotomes can predate *S. mansoni* and *S. haematobium* sporocysts while feeding on the internal tissues of their snail hosts [53,54]. Some highly competitive trematode species (e.g. echinostomes or amphistomes) can also outcompete sporocysts of *Schistosoma* that are moderate competitors during coinfection [49]. Using modelling approaches, Laidemitt, Anderson, et al. 2019 predict over 50% decrease in the transmission of *S. mansoni* in the

presence of some highly competitive trematodes co-infecting *B. pfeifferi* such as *Calicophoron sukari* a widely distributed parasite across livestock.

Together, these results indicate that local trematode communities, which are primarily determined by the local aquatic and terrestrial vertebrate communities [55], might influence the competence of ecosystems with respect to *Schistosoma* species. Given that facilitation between trematodes is rarer than competition [47], and provided that *Schistosoma* species are moderate competitors [49], higher trematode diversity circulating in an ecosystem might impede the circulation of *Schistosoma* species. Thus, maintaining vertebrate host diversity to support diverse trematode communities in local snail populations could curb the transmission of *Schistosoma* species. Besides trematodes, several other organisms such as microsporidians or ichthyosporeans co-infecting or associated with *Schistosoma* intermediate hosts can also directly impact parasite virulence and the production of cercariae within snails [56,57]. This strengthens the idea that overall biodiversity associated with aquatic ecosystems influence their competence toward *Schistosoma* species.

A conceptual vision of ecosystem competence

Competence at the host scale was developed to link immunologic and metabolic phenomena at the sub-organismal level to ecological and epidemiological phenomena at the population levels to better understand the eco-evolutionary dynamics of host-parasite interactions [10] (**Figure 2A, Key Figure**). The concept of competence scaled up at the community level has helped to decipher the links between host community complexity and the transmission dynamics of pathogens [13]. Changes in community in favour of incompetent hosts at the expense of competent hosts, hamper the transmission dynamics of pathogens through a dilution effect. Conversely, an increase in the relative abundance of competent host at the expense of incompetent hosts may lead to an increase in pathogen transmission through

an amplification effect [13] (**Figure 2B**). This rather schematic link between biodiversity - defined in terms of a community composed of hosts that display varying degree of competence - and parasite transmission has been widely debated [58] and recently challenged by several studies indicating that diversity can simultaneously dilute and amplify the transmission of pathogens through complex mechanisms at the ecosystem scale [59,60]. In terms of epidemiological dynamics, it is the resulting net effect of the diluting and amplifying mechanisms at the scale of the ecosystem that ultimately drive the transmission of a given pathogen [60].

The concept of ecosystem competence aims at considering ecosystems as singular entities for which levels of competence can be assessed. Ecosystem competence is a measure of the net effect of all interacting biotic components from the sub-organismal to the community scale as influenced by physicochemical and biogeographical properties of the ecosystem within which they occur, to produce a quantity of parasite infective stages from an initial amount of parasite entering that system (**Figure 2C**). The magnitude of transmission of a given pathogen at a given focus is generally assessed based on the prevalence and intensity of that pathogen measured in the definitive host populations associated to this focus. Regarding human schistosomes, transmission hotspots are defined as freshwater aquatic sites where the surrounding human populations harbour high levels of prevalence and parasitic burden. However, in this case the magnitude of transmission combines without distinction the contribution of the amount of parasite entering into the transmission site (i.e., aquatic ecosystem) and the competence of that ecosystem.

Schistosoma **persistent hotspots** received considerable attention in the last decades [61]. Besides the effect of large and stable populations of susceptible snail hosts, additional hypotheses might explain persistent hotspots including key overtly social factors like need to access surface water leading to high frequencies of water contact, poor sanitation, and

insufficient treatment coverage, along with other factors like the presence of reservoir hosts and “super spreader” individuals [61,62]. However, many interacting factors at the ecosystem scale can amplify the number of cercariae produced locally even if the number of miracidia released into that system is low (**Figure 1, 2C**). Thus, highly competent ecosystems in which human schistosome incoming is lowered through frequent mass chemotherapy campaigns could still amplify cercarial production and remain important transmission hotspots. Conversely, poorly competent ecosystems, could represent dead ends for the parasite even with occasional high parasite incomes. In this context we argue that we should now focus on the relative contribution of the parasite pressure entering into an ecosystem and that of the competence of that ecosystem to better understand the factors driving parasites transmission dynamics.

Ecosystem competence for a given pathogen is expected to vary in time naturally or under anthropic perturbations. For instance, accumulating plastic wastes in the environment influence pathogen circulation at the ecosystem scale [63]. In this regard, schistosomes snail hosts are often found on plastic garbage that can act as physical support for them to attach and lay eggs [64,65]. When no infrastructure exists to manage these wastes, this makes areas with human activities and contact the very places where many snails are found. Moreover, macrophytes, microorganisms and invertebrates colonizing plastic wastes [66] could also influence the interactions between snail hosts and SFLS. Additionally to plastics, some commonly used agrochemicals found in waterbodies can also affect the transmission of *Schistosoma* species. In this respect, Hoover et al. 2020 conducted certainly the most integrative study to quantify the synergetic effects of agrochemicals on the final concentration of *Schistosoma* cercariae produced locally [67]. They accounted for the effect of agrochemicals on cercariae survival, on snails’ abundance, survival, and reproduction, and on the abundance of snail predators and snails’ resource availability. Combined with mathematical models, authors predicted the cercariae outcome from a system under different levels of agrochemicals

present in that system [67]. In our opinion, this study is a first step toward a measure of ecosystem competence.

Toward a measure of ecosystem competence

Measuring host competence *in natura* is difficult mainly because it is hard to assess the exposure rates of hosts to their parasites [13]. By analogy, assessing ecosystem competence is challenging because it relies on two major metrics including the parasites incoming within an ecosystem and the parasite infectious stage produced from that ecosystem. Applied to *Schistosoma* species, this means quantifying the abundance of miracidia from a given species of schistosome released into an ecosystem and the abundance of cercariae produced from that ecosystem (**Figure 2C**).

Concerning *Schistosoma* species, the use of snail and vertebrate (e.g. rodents) sentinel species could help assessing parasite incoming and exposure to cercariae in waterbodies [68] [69]. However, these approaches should be considered with great caution for obvious ethical reasons. Alternatively, the regular and precise monitoring of re-infection rates within the framework of adapted mass chemotherapy campaigns [70] should allow a measurement of the competence of ecosystems.

Emerging technologies based on environmental DNA (eDNA) are promising to meet this challenge [71,72]. Combined with quantitative approaches, eDNA allow quantifying the amount of parasites or that of their hosts present within an ecosystem [73,74]. However, these approaches capture eDNA from all free-living stages with no distinction and from dead organisms (e.g. isolated debris/cells; free DNA) [75,76]. Environmental RNAs (eRNAs) provides an interesting complementary tool for at least two main reasons. First the amount of eRNA is likely to better reflect the amount of living organisms [76,77]. Second, some RNAs are expressed specifically at different living stages [76]. In the case of *Schistosoma*, these

methods might thus allow quantifying the relative abundance of RNAs expressed by living miracidia from those expressed by living cercariae in a given ecosystem at a given time. More precisely, ecosystem competence could be quantified as a ratio between the average quantity of incoming miracidia and cercariae produced within an ecosystem. Since the emission of cercariae occur long after the incoming of miracidia within an ecosystem (due to the development time of *Schistosoma* species within their snail hosts), this would require temporal monitoring and averaging eRNAs of these two SFLS over time. While no eRNA-based tools exist for schistosomes, they were recently developed for several pathogens [78] and offer promising approaches to quantify ecosystem competence for these pathogens.

Concluding remarks

From a fundamental point of view, the ecosystem competence concept should lead to further integrative studies at the ecosystem scale to decipher the links between human impact on the environment, biodiversity and the transmission dynamics of schistosomiasis (see **Outstanding questions**).

From an applied perspective, ecosystem competence is in line with the current need of operationalizing the One Health concept [3]. It contributes to the establishment of a common vocabulary necessary to facilitate communication between actors and stakeholders of social-ecological systems for controlling infectious diseases. Beyond its conceptual value ecosystem competence also constitutes a metric of the transmission risk that results from all ecosystem processes, all of which being increasingly under the influence of the prevailing social-systems. From the perspective of maintaining or improving human and animal health, one objective might be to keep the competence of an ecosystem under an acceptable threshold for the social-system(s) while maintaining or improving the health of all constitutive components of the social-ecological system.

To eliminate schistosomiasis as a public health problem by 2030 the WHO established a roadmap based on three main strategic interventions: preventive chemotherapy, snail control with molluscicides, physical removal and environmental modification and ensuring access to safe water, sanitation and hygiene (WASH) [79]. Together these actions act on the health of people but also on the quantity of miracidia emitted in aquatic environments and on the quantity of mollusks present locally, hence contributing to the reduction, and in the best case, to the cessation of transmission. However, actions carried out from an environmental point of view are generally focused on the reduction of mollusks abundance only and, when molluscicides are used, to the detriment of the quality of the water potentially available to ensure the health of other constitutive components of the social-ecological system. We argue that accounting for ecosystem competence could lead to specific actions at transmission sites to reduce the circulation of schistosomes while preserving the health status of the social-ecological system. For example, managing inputs of anthropogenic origin (agrochemical molecules, plastic debris) at certain transmission sites associated with chemotherapy and the development of WASH, could help reducing the circulation of *Schistosoma* locally while preserving water quality. Importantly, the competence of ecosystems is defined for a given parasite/pathogen. This concept must in fact be adapted according to the life cycles and transmission modes of the pathogens considered as well as to the characteristics of the associated ecosystems.

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Declaration of Interests:

The authors declare no competing interests.

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Glossary:

Community competence: Average competence of hosts within a local community at a given time.

Ecosystem competence: propensity of an ecosystem to amplify or mitigate an incoming quantity of a given pathogen that ultimately can be transmitted to their definitive hosts.

Host competence: Propensity of a host to amplify pathogens at levels that are transmitted to other hosts.

Host resistance: Condition in which a host prevents the infection and/or the development of the pathogens and parasites through an active immunological killing process.

Host susceptibility: Condition in which the host immune system fails to inactivate or kill a pathogen/parasite, resulting in an infection that progresses to patency and establish viable infections in individuals within a host population.

Host suitability/unsuitability: Condition in which the physiological/physicochemical needs of the parasite are adequately met or not adequately met in the host internal environment, respectively. The host internal environment is physiologically suitable or not suitable to support pathogens/parasites development and growth.

Host tolerance: Tolerance prevents hypersensitivity of the immune system to exogenous pathogens/parasites and to limit host damage during infection. Tolerance is a defensive approach that relies on tissue damage control systems to prevent infections from causing harm to the host. Tolerance protects the host by promoting host health while having a neutral to sometimes positive effect on pathogen fitness. Tolerance allows for some pathogenic organisms to successfully infect a host and avoid elimination.

One-Health: Integrated and unifying approach that aims to sustainably balance and optimize the health of people, animals, and ecosystems. This concept recognizes the health of humans, animals, plants, and the environment are closely linked and interdependent [19].

Pathogen infectivity: Propensity of a pathogen to penetrate its host and escape the first defences of the host immune system.

Pathogen virulence: Propensity of the pathogen to develop and multiply in a host (generally conceived to have important impacts on host fitness).

Persistent hotspots: Sites where prevalence and intensities of a given *Schistosoma* species in human populations remain high despite frequent rounds of mass drug administration (*MDA*) programs.

Rediae: Intra-mollusc larval stage of some trematode species. Like sporocysts of schistosomes, rediae develop in the body of a snail and reproduce asexually. From rediae, cercariae differentiate during successive maturing waves before leaving the molluscan host.

Social-ecological system: system that includes an ecosystem (composed of independent biological units interacting with each other and with their physicochemical environment) and one or more social subsystems (composed of societal elements such as economies, public policies, institutions, culture of local societies). These two subsystems are intricately linked and in constant interaction.

Transmission hotspots: Sites of elevated transmission efficiency or a higher risk of disease acquisition [80].

Box 1. Host competence

Host competence is the propensity of a host to amplify an incoming amount of pathogen at levels that are transmitted to other hosts/vectors [9,10]. It is a complex phenomenon composed of several traits including **tolerance**, **susceptibility** (or **resistance**) and **suitability** (**Figure I**) [81]. These traits mainly rely on two intrinsic physiological characteristics of the host including metabolism and the immune system. Metabolism and immune system are under the control of both molecular and environmental determinants. At the molecular level, genetic and epigenetic factors interact to shape host competence [82]. Recently it has been also demonstrated that successive exposures of snail to schistosome infection can modify the host competence. This modification of host competence may occur due to direct competition between pathogens installed in snail tissue (see **Figure 2C**) or due to the enhancement of snail immune capacities, designated as ‘immune priming’ for invertebrates. Past experiences and successive exposures with pathogens improve the efficiency of innate immune system and alters the susceptibility of the host following secondary exposures [83–85]. By influencing host susceptibility or compatibility parameters, immune priming may therefore influence the dynamics of the Schistosome transmission in the field. As expected *in natura*, repeated non-infectious exposures of *B. glabrata* snails to miracidia of *Schistosoma sp.* would result in closing the compatibility filter and to a reduction of the quantity of cercariae produced. Moreover, the influence of immune priming on the evolution of pathogen virulence has to be also considered. It is hypothesized that host populations with high rates of primed individuals have the potential to maintain more virulent pathogens than can susceptible host populations [86]. In such ecosystems, the impact of immune priming would thus lead to a reduction in host competence and pathogen transmission but a spreading of higher virulent pathogen phenotypes in the environment.

The host competence concept might evolve together with our changing notion of what comprises a host. Hosts are increasingly recognized as holobionts [87]. Each partner intrinsic to the hosts might influence host competence. In *Schistosoma* snail hosts, bacterial communities and their effect on competence are poorly documented [88–90]. However, several organisms associated with hosts influence schistosome development within their hosts, hence modifying their competence (e.g., other trematodes, microsporidia; see full text).

Box 2. Classical version of *Schistosoma* life cycle

Pairs of adult worms live and reproduce sexually within their vertebrate definitive hosts (i.e., relatively stable environment), producing around 20–3500 eggs a day [91]. Nearly half of produced eggs are expelled through urine or faeces [92]. When they are released in freshwater (i.e., relatively unstable environment), eggs hatch, releasing miracidia (first schistosome free-living stage). Miracidia actively search for their freshwater gastropod intermediate hosts by chemotaxis [93].

After the parasite reaches the intermediate host and escapes the early defences of the host's immune system [94], miracidia transform into primary or mother sporocysts which reproduce asexually giving rise successive stages of sporocysts. From sporocysts, cercariae (the second schistosome free-living stage) differentiate during successive maturing waves before leaving the molluscan host [95]. Once fully-developed, tens to thousands of cercariae are released discontinuously within the aquatic environment per patent mollusc per day, for the remaining lifespan of the molluscs. Cercariae then actively seek their definitive hosts, whose spectrum depends on *Schistosoma* species [96] and develop into adults after skin penetration.

Figure 1. Ecosystem overview of the life cycle of *Schistosoma* species.

The life cycle of *Schistosoma* species is represented by the solid black circle. The life stages miracidium, sporocyst, cercariae of the *Schistosoma* species are represented in grey. This ecosystem perspective accounts for biotic communities, and physicochemical property (in green) of aquatic ecosystems that influence parasite transmission and ultimately their ability to infect their definitive hosts. Case 1: Ecosystem processes influence the abundance and infectivity of free-living stages of *Schistosoma*. Case 2: Ecosystem processes shape schistosome intermediate hosts abundance and competence. Case 3: 3 Ecosystem processes influence schistosomes virulence toward their intermediate hosts.

Figure 2. Key Figure. The competence concept applied at different scales.

(A) Host scale [10], (B) community scale [11] and (C) ecosystem scale (this study). White squares represent intermediate host snails of *Schistosoma* species. Light blue squares represent the intermediate host communities. Blue squares represent aquatic ecosystems. Vertical lines above hosts, host communities and aquatic ecosystems represent the incoming miracidia released by definitive hosts. Line width reflects the quantity and infectivity of miracidia. The quantity and infectivity of miracidia is initially the same for each system (host, host community, aquatic ecosystem). Vertical arrows represent cercariae produced by intermediate host snails. Vertical arrows width reflects the quantity and infectivity of cercariae. The quantity and infectivity of cercariae produced is different depending on the competence of each system (host, host community, aquatic ecosystem). Horizontal arrows in (C) represent the biotic and physicochemical factors of ecosystems that affect negatively (in red) or positively (in green), (a) the quantity and infectivity of *Schistosoma* free living stages, (b) the intermediate host community abundance and competence and/or directly the *Schistosoma* virulence within their intermediate host. The dotted square represents an infected intermediate host eliminated by biotic or physicochemical factors. The red cross represents a developing schistosome in its

intermediate host eliminated by biotic or physicochemical factors. The green circle represents the development of a schistosome within an initially incompetent intermediate host, but whose competence was primed by biotic or physicochemical factors.

Figure I (Box 1): Schematic vision of host competence.

The large rectangle represents a host. The vertical line above the host represents the incoming pathogen. The vertical arrows represent the pathogen produced by the host. Vertical arrows widths from light grey to dark grey reflect the quantity and infectivity of pathogen from low production with a low infectivity to high production with high infectivity. The quantity and infectivity of pathogen produced is different depending on the host competence, which varies according to several hosts traits under the influence of genetic, epigenetic and environmental determinants.

Highlights :

- Health authorities call for more integrative studies that account for the complex interactions between pathogens, their hosts, and the social-ecological systems to better understand pathogens transmission dynamics.
- Accumulating empirical and theoretical evidence have highlighted several scale-dependent mechanisms by which biodiversity and the prevailing environmental conditions can dilute or amplify pathogens circulation.
- Anthropogenic activities, that affect the aquatic environments and the associated biodiversity, influence the virulence and infectivity of *Schistosoma* species, the abundance and competence of their snail hosts, and ultimately the parasites transmission at the ecosystem scale.
- The concept of competence that was first developed as a property of host individuals and then extended to host communities can also be scaled-up to ecosystems.

Outstanding questions

- What is the relative importance of locally diverse species assemblages versus the abundance of competent snail intermediate hosts, independent of other environmental factors (biotic and abiotic), on the risk of schistosomiasis transmission to humans?
- Through which ecological mechanisms do social systems influence ecosystem competence with respect to *Schistosoma* species?
- Can an ecosystem poorly competent for *Schistosoma*, conversely, be highly competent for another pathogen?
- How the concept of ecosystem competence applies to other pathogens?
- How can we easily and effectively measure the competence of an ecosystem with respect to a given pathogen?
- What are the levers to act on to limit the competence of ecosystems in a context of disease control while maintaining or improving the health of other constitutive components of socio-ecological systems?
- How to consider the competence in a meta-ecosystem context?

Physico-chemical water conditions
(Agro)chemicals
Heavy metals

Free-living stage (cercaria)

- Decoy / Dilution effect
- Predation

1

Physico-chemical water conditions
(Agro)chemicals
Heavy metals

Free-living stage (miracidia)

- Decoy / Dilution effect
- Predation

1

**Schistosome - trematode
(intramolluscal interactions)**

- | | |
|-------------------|----------------|
| Direct: | Indirect: |
| - Competition | - Priming |
| - Hyperparasitism | - Facilitation |
| - Predation | |

3

**Host
Abundance/competence**



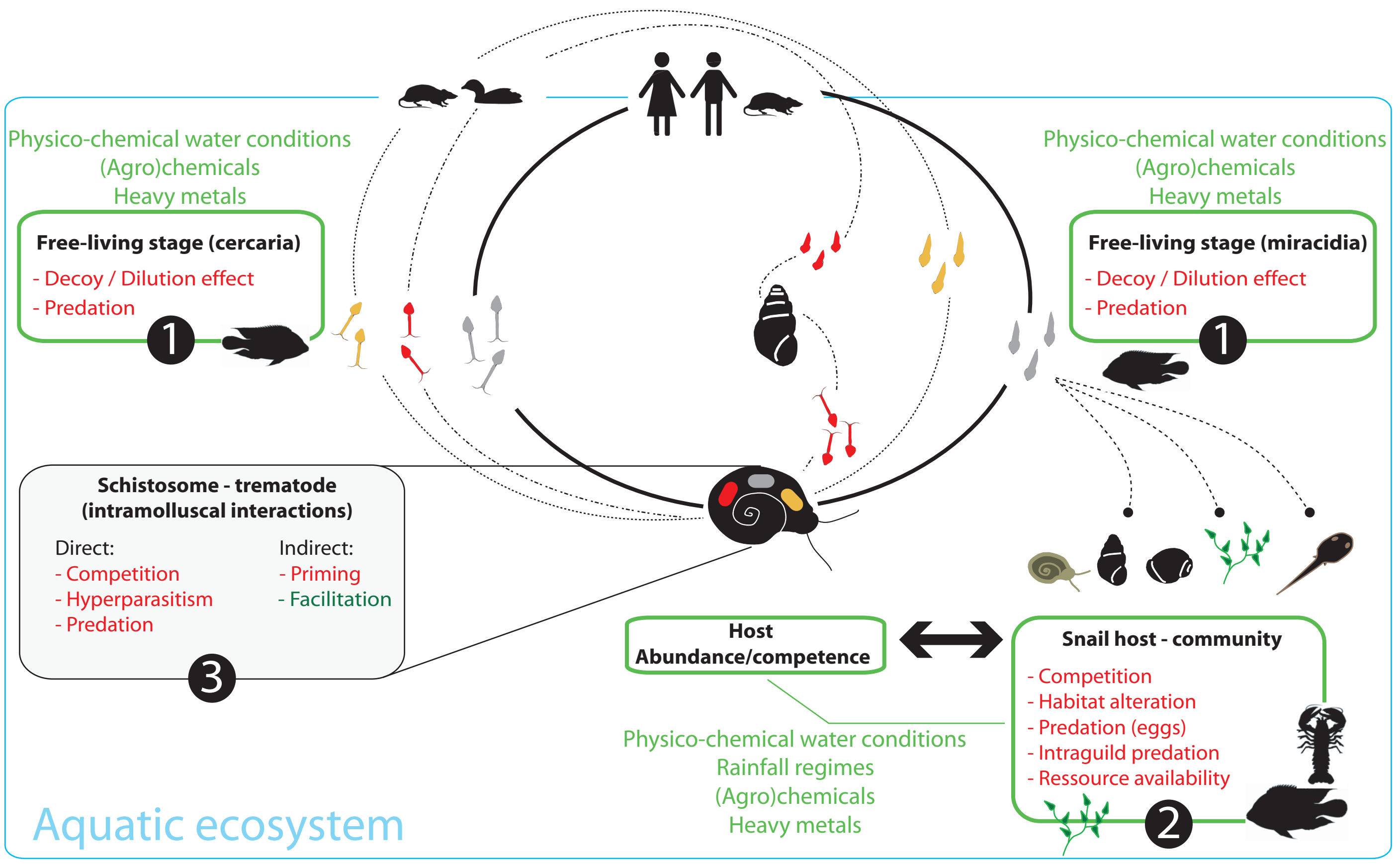
Snail host - community

- Competition
- Habitat alteration
- Predation (eggs)
- Intraguild predation
- Ressource availability

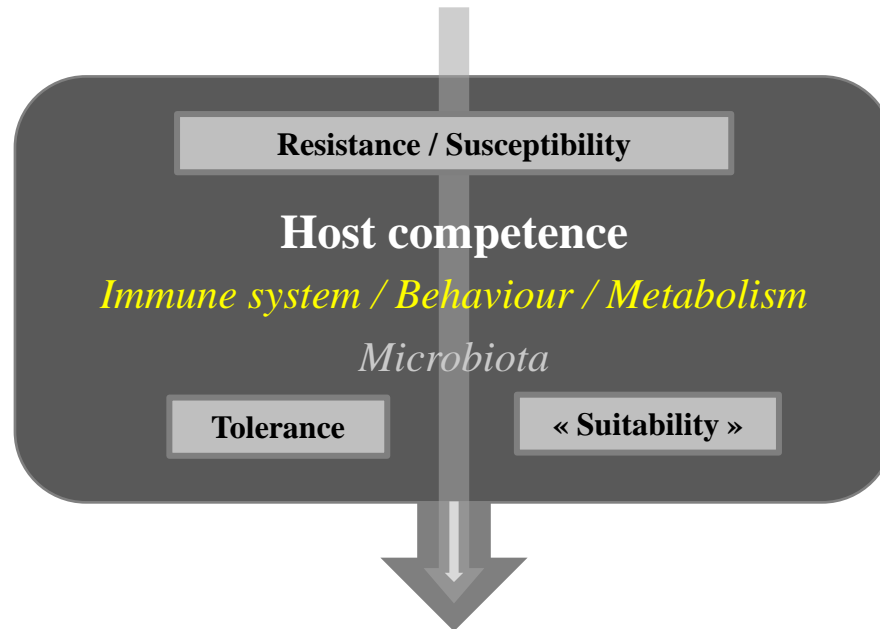
2

Physico-chemical water conditions
Rainfall regimes
(Agro)chemicals
Heavy metals

Aquatic ecosystem



Pathogen input



Resistance / Susceptibility

Host competence

Immune system / Behaviour / Metabolism

Microbiota

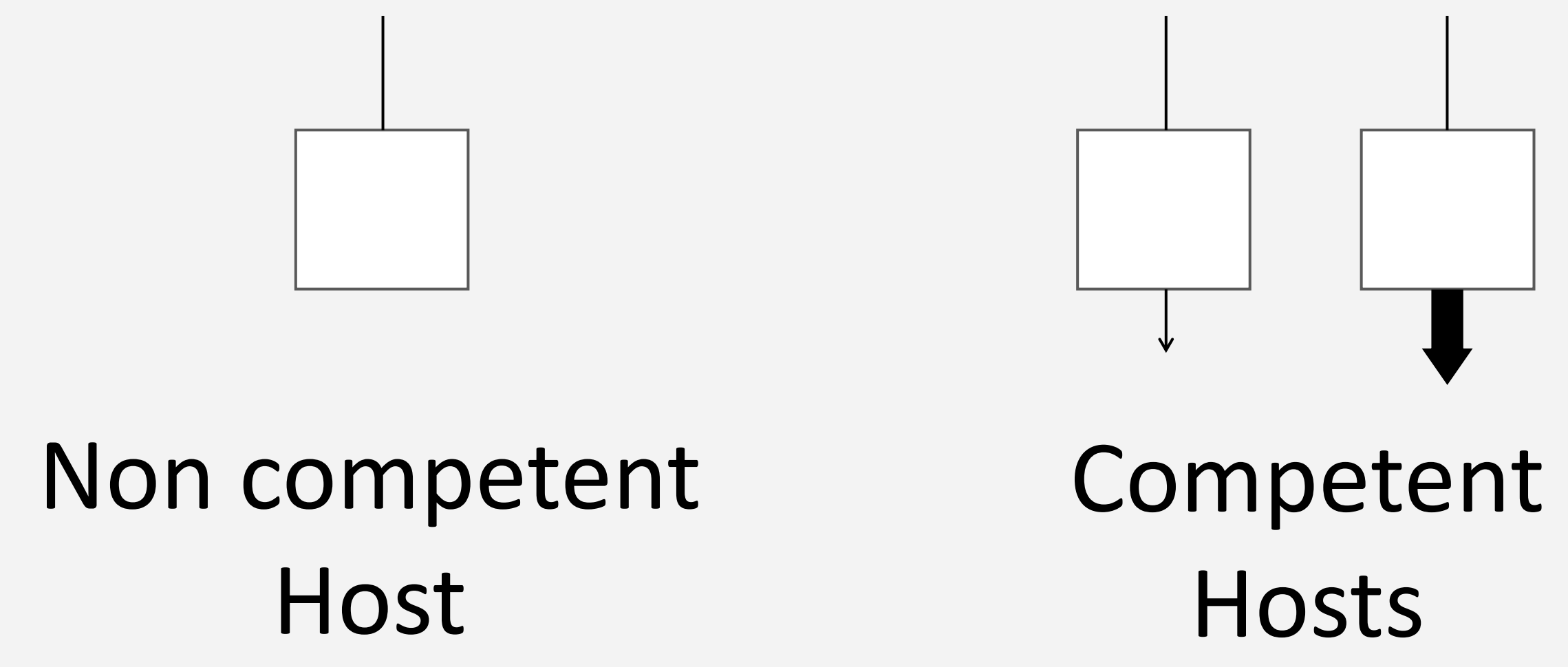
Tolerance

« Suitability »

Pathogen output

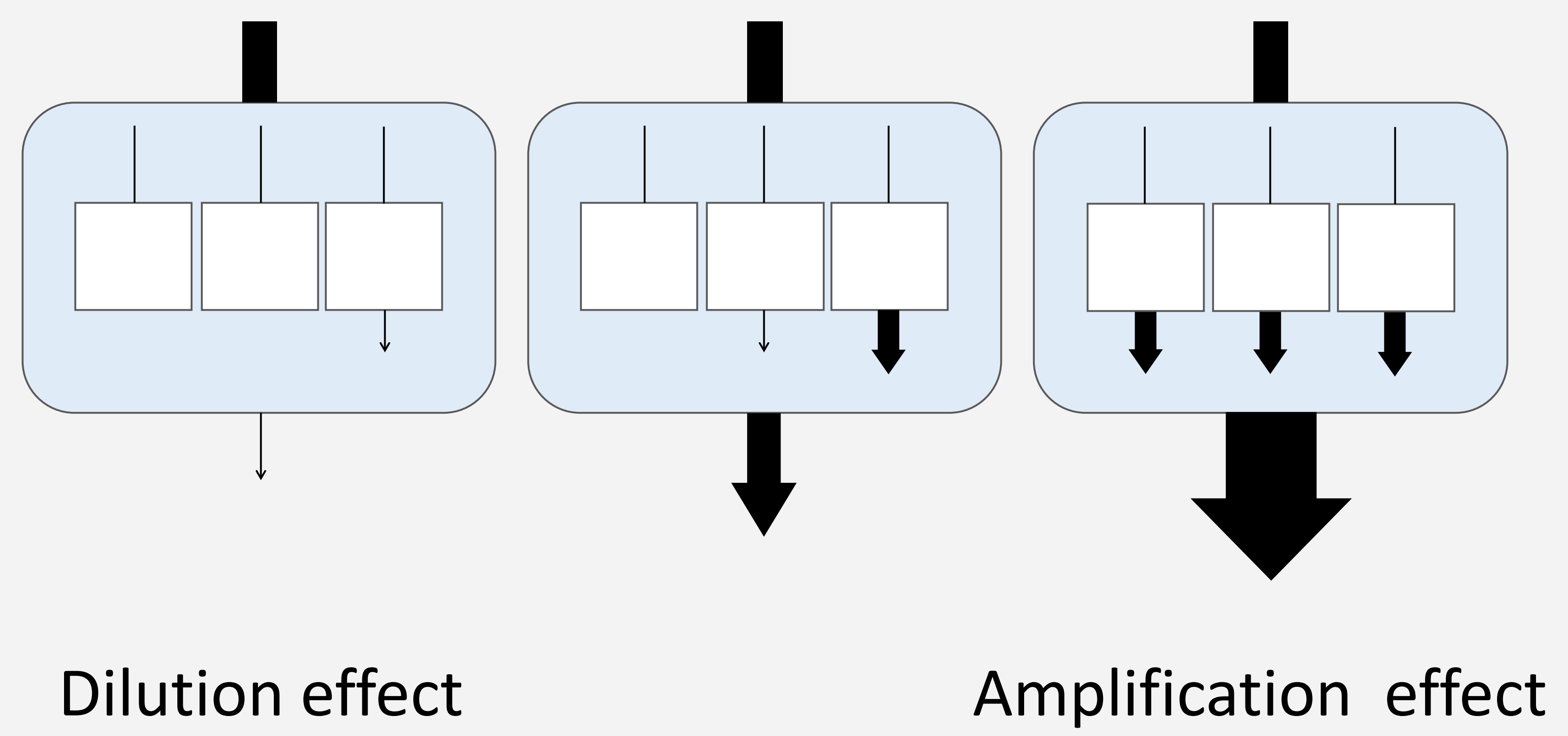
A

Host
Competence



B

Hosts community
competence



C

Ecosystem competence

