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# The terminology of metacommunity ecology

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One of the most promising theoretical frameworks for studying responses to ecological change is the metacommunity concept. Recent work by Logue *et al*. [1] provided the first comprehensive synthesis of empirical metacommunity research since the seminal work by Leibold *et al*. published in 2004 [2]. In their original work [2], Leibold *et al*. outlined the theoretical framework for the study of a metacommunity, a set of local communities connected by dispersing species. Logue *et al*. [1] point out some of the limitations of the original concept as well as more recent research. More importantly, they make three recommendations for future research in metacommunity ecology to continue to advance this field: (i) the extension of empirical approaches to different and varied metacommunity systems; (ii) the integration of established metacommunity paradigms; and (iii) work incorporating meta-ecosystem and evolutionary mechanisms. These are important recommendations, but we think that their second recommendation is a necessary condition for the other two. We propose that a revision of the terminology used by Leibold *et al*. [2] can already accomplish part of this integration.

Leibold *et al*. [2] proposed four paradigms of metacommunities: (i) neutral model; (ii) patch dynamics; (iii) mass effects; and (iv) species sorting, synthesizing concepts of intra- and intercommunity dynamics [2]. These four paradigms have formed the basis for the modern metacommunity concept, and much literature has characterized metacommunities based on the degree to which the systems conform to these paradigms (e.g. [2]). What the terminology of Leibold *et al*. [2] does not explicitly recognize is that two of these metacommunity paradigms (mass effects and patch dynamics) are actually special cases of the species-sorting paradigm. In the patch dynamics paradigm, the interacting species differ from each other, but in a very specific way. They specialize in their abilities as either competitors or colonizers within a uniform environment. Patches of varying species composition develop as species effectively colonize a site or outcompete other species. A second, less described, type of patch dynamics starts from a species-sorting setting, with environmental heterogeneity and associated species differences. Strong priority effects in such a system caused by dispersal limitation can lead to different and stable communities [3]. For the mass effects paradigm, source–sink dynamics enable species to exist at sites normally considered marginal or outside of their environmental range because high dispersal ensures a constant supply of new colonizers to these sites [2]. Therefore, these species have very different niche requirements, essentially a species-sorting framework, but dispersal maintains species in sites with negative growth rates. The only way to detect mass effects truly is to model or measure population growth rates of a species in an environment with and without dispersal. Thus, the distinguishing feature of a patch dynamic versus a species-sorting versus a mass effect framework is the amount of dispersal present between communities within a metacommunity: dispersal is limiting for some of the species in the patch dynamic framework, efficient for (the majority of) the species in a species-sorting framework, and high for some of the species in the mass effect framework.

The terminology used by Leibold *et al*. [2] ensures consistency with the previous concepts in the literature. For example, terms such as 'patch dynamics' or 'mass effects' were already well documented in the literature before this work (e.g. [3–7]). Although we doubt that it was the intention of Leibold *et al*. [2] to suggest that these four paradigms were mutually exclusive or that each metacommunity system was uniquely associated with a single paradigm, the summary of research provided by Logue *et al*. [1] shows that work since 2004 has largely focused on delineating between the four paradigms (e.g. [8]). Indeed, Logue *et al*. [1] showed that the majority of observational metacommunity studies were aimed at testing for patch dynamics or mass effects. Thus, the terminology of the four paradigms ensures consistency over time, but continuing to use these terms while not explicitly recognizing, for instance, that patch dynamics and mass effects are special cases of the species-sorting paradigm does not address the actual relationships between the different paradigms. More importantly, these relationships illustrate the fundamental mechanistic processes responsible for metacommunities. We suggest that ecologists could move on from characterizing metacommunities as 'species sorting', 'patch dynamics', 'mass effects' or 'neutral model'. Rather, they should break these paradigms down and go back to the basics of thinking about what these paradigms mean for dispersal and environmental signals in metacommunities. This avoids the issue of having four discrete paradigms, and rather puts the focus on the relationships at play in metacommunities and how the mechanisms behind the patterns that describe each of the paradigms interact in a metacommunity. We thus propose to think about metacommunities as neutral, and species sorting with limiting (patch dynamics, sensu [2]),

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efficient (species sorting, sensu [2]), and high dispersal (mass effects, sensu [2]) as these terms capture the causal mechanisms and are, thus, more explicit.

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# Peto's paradox and cancer: a response to Caulin and Maley

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Although cancer is assumed to originate from a single cell and to engage mainly old individuals, it occurs at similar rates in species differing widely in total cell number ($10^9$–$10^{17}$ among mammals) and life span; this has been called Peto's paradox. In a recent TREE review on attempts to resolve this paradox, Caulin and Maley suggested that evolution can help to find ways to prevent cancer [1]. However, there is evidence that an evolutionary perspective on cancer can be further extended. Many tumour-suppressive mechanisms are well-conserved, but there is reason to believe that evolution has also fine-tuned for each species a multi-faceted network of tumour-influencing factors, including procancer mutagenic enzymes and natural anticancer weapons, producing the observed cancer frequency of approximately 0.3 per individual's lifetime [2,3]. Most likely, a significantly higher rate of cancer would increase the fraction of affected young individuals and thus reduce reproduction. Conversely, the reason why the rate is not significantly lower is that this would require more investment in anticancer defence without bringing any selective advantage. A concept of an evolutionary defined cancer rate can provide experimental results presented by both sides in the on-going academic conflict in determining whether the cause of cancer is cellular mutagenesis or a rare failure of healthy cells combating cancer [4]. It may take at least two partners for a clinically relevant cancer to be formed: on the one hand a single cell with errors in molecules or signalling pathways and on the other a tissue, organ or organism with deficient homeostasis; the erroneous cell then takes advantage of a moment of host weakness to embark on a selfish adventure.

Some examples can illustrate the need for evolution to place tumour-influencing factors in regulatory homeostatic networks. T lymphocytes are then often prevented by other network components from attacking cancer cells, for the reason that they can, if permitted to use all of their armoury, produce serious autoimmune disease (as observed in melanoma patients treated with the ipilimumab monoclonal antibody, functioning to activate these very T lymphocytes) [5]. Enzymes with DNA-modifying activity such as V(D)J recombinase and topoisomerases act as two-edged swords: on the one hand facilitating T lymphocyte defence against microbes and tumours, and on the other mediating chromosome translocations driving lymphomas and solid cancers [6]. Thus, evolution tinkers with its networks to weigh benefits against costs [7]. Furthermore, there is evidence that a mutation causing loss of production of the sialic acid Neu5Gc variant has been selected for despite its contribution to colon cancer, because it brings a net benefit by reducing malaria [8]. Thus, evolution is playing all possible cards; including both cellular procancer molecules and cancer resistance functions, along with taking into consideration homeostatic effects unrelated to cancer.

If evolution is shaping our cancer repertoire, then it follows as a logical consequence that an array of cancer-regulating factors does exist. What can keep evolution from tinkering with all types of such factors? Why do we put theories about cancer against each other? Is it not apparent that there will always be mutated cells willing to start up a tumour (explaining cancer monoclonality) and that evolution has done its task to ensure that such antisocial behaviour is kept at an acceptable level? This can also explain why cancer strikes mainly in old age, although there is little fitness to be gained from reducing cancer death in 70-year-old people, evolution will tinker to make cancer and other ageing manifestations develop after reproduction (and child-rearing) has been completed.

Are there any practical consequences of a concept of evolutionary defined homeostatic networks regulating cancer? If it was established how to release host factors with anticancer potential from an inhibitory influence

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