Stability and invasibility of coral communities

A recent *TREE* news and comment article[1] highlighted John Pandolfi's work on the stability of Pleistocene coral assemblages in Papua New Guinea[2]. His salient result was that coral species richness and composition in reef crest and reef slope environments at three coastline sites remained relatively constant during nine major reef-building episodes spanning 95 000 years. He found that a few common species repeatedly colonized these environments and thus suggested that coral community membership is limited and that these complex communities are highly integrated entities[3,4]. Furthermore, Pandolfi rejected a random colonization model consistent with unlimited membership of assemblages that are open to immigration from regional species pools.

Here we respond to the statement that 'surely ecology has advanced sufficiently that a study documenting 95 000 years of community similarity allows us to distinguish between a niche- and dispersal-based view of community organization'[1]. This supposes that these perspectives represent mutually exclusive alternatives and that communities composed of ecologically differentiated species should be closed to immigration. This is contrary to ecological theory, which attempts to integrate these divergent perspectives[5], and contrary to evidence from coral communities that indicates that such communities are, in fact, open to regional pools of species[6–9]. There is no reason why ecological differentiation and regional enrichment cannot jointly determine community structure. For example, coral assemblage richness and composition does change along local environmental gradients as well as among differentially rich regions[7–9]. This integrated view is consistent with the mounting evidence that most natural communities are open to regional influences through dispersal and other biogeographic phenomena[8,10–12]. It also argues for a more-inclusive conceptual basis for understanding community processes. Surely community ecology has advanced beyond the decades-old arguments that have polarized this field for so many years. We stress the need to integrate these divergent views more fully.

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Reply from M.D. Jennions

Economy of wording may have led me to write a cryptic, rather than concise, article[1]. In the context of what I wrote, however, I felt my final, admittedly pointed, sentence was intelligible. Perhaps not. Let me try again, with greater verbosity:

> Surely ecology has advanced sufficiently that a study documenting significant similarity in local species composition after nine independent events of coral reef formation (spanning a 95 000 year period)[2] is sufficient to distinguish between (1) the null hypothesis that local community composition can be explained by stochastic recruitment of species from the regional species pool without any need to invoke niche differentiation[3], and (2) alternative models in which niche differences among species are essential[4] for the explanation of local community composition?

Personally, I agree with Karlson and Cornell's statement that ecological differentiation and regional enrichment both contribute to the species composition of local communities. Indeed, their own work on corals supports this assertion[5]. The polarizing view expressed in my final sentence reflects a difference in scientific approach. The more amicable approach is to see how much importance should be attached to two or more competing hypotheses (although the difficulties this sometimes generates are exemplified by the misleading nature of statements like '60% of phenotypic variation is genetic, 40% environmental')[6]. The other approach is to test against a null hypothesis[7]. In my article, I highlighted an innovative null model by Steve Hubbell, which was presented as a plenary talk at the 8th International Coral Reef Conference[3]. To explain relative species abundance in communities, he assumes that individuals of each species can be treated as identical. My intention was to reflect the clear contrast between this null model and those alternative hypotheses that explicitly require niche differentiation among species[3].

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Direct and indirect estimates of dispersal distances

Koenig *et al.*'s recent *TREE* article on dispersal distances[1] highlights how direct (i.e. mark–recapture and telemetric) and indirect (i.e. molecular) techniques can produce markedly different estimates of gene flow. However, their choice of examples suggests that molecular estimates of dispersal rates are generally greater, and that direct methods of estimating dispersal are negatively biased.

Results of molecular research on pinnipeds also differ from those of direct marking studies, but the pattern is the opposite of that described by Koenig *et al.*[1]. The tagging of just a small proportion of grey and harbour seal pups at UK sites resulted in reports of post-weaning dispersal distances in excess of 200 km (Refs 2 and 3). The rapid spread of the phocine distemper virus to all North Sea harbour seal colonies within a few months[4] also provides indirect evidence of widespread mixing between European sites. In contrast, molecular studies of both species, using microsatellite and mitochondrial DNA techniques, indicate a high degree of differentiation between North Sea populations[5,6] (S. Goodman, PhD Thesis, University of Cambridge, UK, 1995).

Differences between results from direct and indirect studies clearly demand a closer examination of the potential biases involved in each approach. For pinnipeds, where direct methods produce greater estimates of dispersal, the paradox is more difficult to resolve. Certainly, tagging studies could over-estimate dispersal. Most recoveries are of animals that fail to survive their first year, potentially underestimating philopatry. Additionally, data on post-weaning movements may not represent actual recruitment

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patterns, and contemporary rates of dispersal may not reflect historical population structure. However, indirect molecular studies are also subject to bias and may underestimate gene flow. Genetic estimates of differentiation [e.g. F_{ST} (Ref. 7) and its analogues] and migration rate make several assumptions that are commonly inappropriate or, at best, have unclear validity for natural populations. Deviations from mutation–drift equilibrium, nonrandom mating, the presence of selection (through linkage of neutral loci to other loci experiencing selection) and complex geographic structuring of populations all have the potential to bias estimates of gene flow[8]. Some measures also assume that sample sizes are balanced, and that large sample sizes (hundreds of individuals) are required to achieve acceptable confidence intervals. Finally, allozyme studies frequently conclude that there is no genetic subdivision between mammalian populations (as has been the case for pinnipeds[9]), whereas subsequent analyses using mitochondrial or microsatellite DNA reveal significant differentiation[5,6]. Thus, the power of different genetic systems to detect variation and resolve population subdivision must be considered[10].

Molecular approaches clearly offer important new tools for understanding gene flow, but we cannot assume that they provide the unbiased results that traditional direct methods have failed to obtain. The technological advances in telemetric devices to which Koenig *et al.* [1] look forward may, in future, improve our understanding of vertebrate dispersal. In the meantime, comparisons of direct and indirect estimates could have the potential to yield information about demographic histories of populations, but such comparisons would benefit from a more-detailed assessment of the consequences of violating the assumptions underlying these molecular analyses.

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Genetics of host–parasite interactions

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The evolution of host susceptibility or resistance to parasites has important consequences for the evolution of parasite virulence, host sexual selection, population dynamics of both host and parasite populations, and programs of biological control. The general observation of a fraction of individuals within a population that is not parasitized, and/or the variability in parasite intensity among hosts, may reflect several phenomena acting at different levels of ecological organization. Yet, host–parasite coevolution requires host susceptibility and parasite virulence to be genetically variable. In spite of evolutionary and epidemiological implications of genetic heterogeneities in host–parasite systems, evidence concerning natural populations is still scarce. Here, we wish to emphasize why we need a better knowledge of the genetics of host–parasite interaction in natural populations and to review the evidence concerning the heritability of host susceptibility or resistance to parasites in natural populations of animals.

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Models of host–parasite coevolution usually assume that variation in host resistance to parasite infection is, at least partially, genetically determined[1–3]. However, despite evolutionary (e.g. the evolution of parasite pathogenicity, genetic adaptation to local environment) and epidemiological (e.g. population dynamics of both hosts and parasites) consequences of genetic variation for parasite resistance, the presence of genetic heterogeneity in host populations with respect to probability of infection has been neglected[4], at least in natural populations of animals.

Most experimental evidence concerning genetic variability in susceptibility to parasites in animals comes from studies dealing with variation in disease resistance in domesticated and laboratory populations[5,6]. The emphasis of these studies is understandable given the potential implication arising from the importance of the evolution of mechanisms of pathogen resistance for pest control and veterinary medicine. However, we have several reasons to expect a discrepancy between patterns observed from laboratory studies and natural situations.

Before addressing this particular issue, we review the evolutionary, ecological and epidemiological implications of genetic variation of susceptibility to parasite infections.

Host–parasite genetics and the evolution of virulence

In the past decade, a burst of work dealing with host–parasite interactions has provided a theoretical framework that has improved our understanding of the evolution of parasite virulence[2,3,7–10]. The underlying idea is that parasite reproduction is traded against the negative effects of parasite proliferation on host fitness. In other words, the parasite faces a dilemma: should it speed up its own reproduction, with the potential damage to host survival, or manage host survival to increase its transmission? When the opportunity for transmission to a new host is high, the optimum value of this trade-off tends toward an increase in parasite virulence, whereas when the opportunity of transmission is low, the optimum tends toward a decrease in virulence[8]. Linked to the effect of the opportunity of transmission for the evolution of virulence is the mode of transmission from one host to another. Vertical transmission, that is, transmission between closely related hosts (usually between mother and offspring) should select for benign parasites, whereas horizontal transmission (between unrelated hosts) should select for virulent parasites[10]. Experimental and comparative evidence in agreement with this theoretical prediction is now accumulating[11,12].

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