

Molecular dates and the mammalian radiation

In a perspectives article, Bromham *et al.*¹ raise some important concerns about the use of both molecular and paleontological data in assessing the timing of diversification of extant mammalian orders. In Box 1, they describe well the need to differentiate crown (or more generally, node-based) taxa from stem-based taxa. Unfortunately, their Fig. 2 is not clearly explained and thus confuses these kinds of taxa. As they note, the paleontologically based orders of extant placental mammals (except, perhaps, Insectivora) are recognized as appearing soon after the Cretaceous–Tertiary (K–T) boundary (thick lines in their Fig. 2). These dates are based on apomorphy- or node-based *intraordinal* diversifications for the order in question. In contrast, the extensions into the Late Cretaceous of clades shown in their Fig. 2 (the thin lines), estimated by molecular data, are *interordinal* separations. Thus, in at least this comparison, the molecular data indicate nothing about ordinal origination and diversification, but rather argue only that stem-based clades extend into the Late Cretaceous. Furthermore, the possible Cretaceous record of primates they mention in their text is a single tooth originally assigned to the primate *Purgatorius*, which was discovered at a site now regarded as Paleocene in age².

The authors' biogeographical assessment of fossil taxa requires updating. The possible placental for the early Cretaceous of Australia is now regarded by most as symmetrodont³ or early therian. Thus, although marsupials are known for the early Eocene of Australia⁴, non-chiropteran placentals do not appear until the Pliocene⁵. In South America, all definite pre-Tertiary mammals are non-therians, with both marsupials and placentals appearing only after the K–T boundary⁶. As the authors note, the Late Cretaceous of North America and Asia have a good record of mammals, but except for, perhaps, Insectivora, no modern orders of placentals are known. In fact, the latest Cretaceous record⁷ is better known than the earliest Paleocene⁸. Europe is not well known but echoes what is known in Asia for placentals⁹. As the authors also note, Africa is a cipher. Unless, however, one wishes to make the unsubstantiated argument that all 18 orders of extant placentals arose in Africa, the claim is not valid that the biogeography of placentals is too poor to help in deciphering ordinal appearances.

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Reply from L. Bromham, D. Penny and M.J. Phillips

Molecular and palaeontological dates for the radiation of modern mammals appear at odds because molecular studies propose a Cretaceous origin of many eutherian orders, but there are no uncontroversial Cretaceous fossils from modern eutherian orders, a point emphasized by David Archibald in his letter. This conflict might be partly due to different definitions of the 'origin' of an order – palaeontologists tend to focus on the appearance of members of a defined crown group, whereas molecular dates mark the split between lineages, long before they develop crown-group features¹. Both definitions are interesting and important, particularly if the timing of lineage divergence and morphological diversification are not tightly linked. We currently cannot distinguish a long Mesozoic 'phylogenetic fuse'² from a true Cretaceous radiation. Perhaps, higher phylogenetic resolution or new fossil finds could shed light on this conundrum.

To explore the apparent discrepancy between molecular and palaeontological dates, we must ask: 'If the molecular dates are true, then where are the missing fossils?' The most plausible place to hide them is Africa, or perhaps Australia or Antarctica³. We do not suggest this is necessarily true, and we certainly don't expect that 18 eutherian orders arose in Africa. Molecular evidence suggests only some eutherians 'crossed the K–T boundary'¹, which is compatible with the suggestion that a handful of basal eutherian orders form an 'African clade'^{4,5}. If the molecular dates are true, we have to hide the Cretaceous eutherians somewhere, and Africa seems the best candidate. Conversely, if the palaeontological dates are true, why are the molecular dates too old? Lineage-specific rate variation across mammals⁶ could cause consistent overestimation of the dates of divergence of mammalian orders¹. So, we are left with the conclusion that although the discrepancy between molecular and palaeontological dates seems large, at this stage neither can confidently exclude the other.

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Phase locking: another cause of synchronicity in predator–prey systems

In a recent *TREE* article, W.D. Koenig reviewed the patterns and causes of temporal synchronicity in spatially extended populations¹. Synchronicity can have different causes, one of these, spatial correlation of environmental disturbances, was extensively discussed in a news & comment in the same *TREE* issue². This mechanism, which has become known as the Moran effect, occurs when two populations are regulated by the same (linear) density dependence and are exposed to environmental disturbances. If these environmental disturbances are correlated, the fluctuations of population sizes will also be correlated. A further mechanism for synchrony is dispersal of individuals; both papers assumed that dispersal cannot counteract the desynchronizing effect of uncorrelated disturbances beyond the range of dispersal of the organisms under study. It was concluded that spatial correlations at larger spatial scales are likely to be caused by the Moran effect.

Some of the examples of spatial synchronicity in the review¹ were predator–prey or host–parasite systems, which have an intrinsic propensity to oscillate. For such systems, synchronicity can be caused by phase locking. Phase locking occurs if the populations are coupled through dispersal and can act at distances exceeding the typical dispersal distance.

This can be demonstrated with a deterministic mathematical model for predator and prey populations in two connected patches, in which the local dynamics are described by a standard predator–prey model (e.g. the Lotka–Volterra or McArthur–Rosenzweig model³). If the parameters are chosen such that the populations exhibit regular oscillations when isolated, the smallest amount of dispersal results in synchronous oscillations in a system of connected patches. Even if either prey or predator does not migrate, phase locking occurs. Such results can be extended to systems with more patches: some dispersal to neighbouring patches can result in phase-locked population dynamics in large groups of patches⁴. The dispersal range in this case is small but the correlation can work at distances exceeding the dispersal range of an individual. This effect can withstand the desynchronizing effect of uncorrelated disturbances to a certain extent (Fig. 1).

The effect of phase locking will be weaker for patches that are at a larger distance. If uncorrelated noise is superimposed on such a deterministic model, it can result in a correlation that decreases with distance, resulting in a typical

spatial scale^{4,5}. It also results in population dynamics that fluctuate more strongly at a local than at a regional scale. Interestingly, for some choices of parameters, deterministic models can exhibit similar behaviour through the occurrence of diffusive instabilities^{3,4}. The decrease in correlation depends on the details of the dispersal and the density dependence.

For oscillating predator–prey systems, the Moran effect need not work because of the nonlinearity of the density dependence. Consequently, phase locking offers an alternative explanation for the synchronization of population dynamics.

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Extinction by infection

In a recent news & comment article, Cleaveland *et al.*¹ raise an interesting issue in addition to furthering the debate on pathogens as agents of biocontrol. One characteristic of candidate biocontrol pathogens is their ability to cause host extinction and Cleaveland *et al.* cite four cases where pathogens have been causally implicated in animal extinctions¹. The arguments for introduced pathogens as the proximate cause of extinction are particularly convincing for Hawaiian birds² and the thylacine (*Thylacinus cynocephalus*)³. However, these hypotheses have yet to be proven⁴, and considering the problems of investigating historical extinctions^{2,3}, definitive proof might be difficult to obtain.

Two more-recent cases cited by Cleaveland *et al.* demonstrate why wildlife diseases are currently of such concern to conservation biologists^{4,5}, although neither is known to have ultimately involved species extinctions. The black-footed ferret (*Mustela nigripes*) was rendered extinct in the wild in 1987, but this followed capture of the last remaining wild individuals as part of a captive breeding programme⁶. These animals had survived the canine distemper epizootic that had otherwise decimated the last remaining wild colony. A new fungal disease of amphibians – chytridiomycosis⁷ – appears to have caused catastrophic population declines and local extinctions of host species. At least one of the affected species (*Taudactylus acutirostris*) was thought to have become globally extinct in

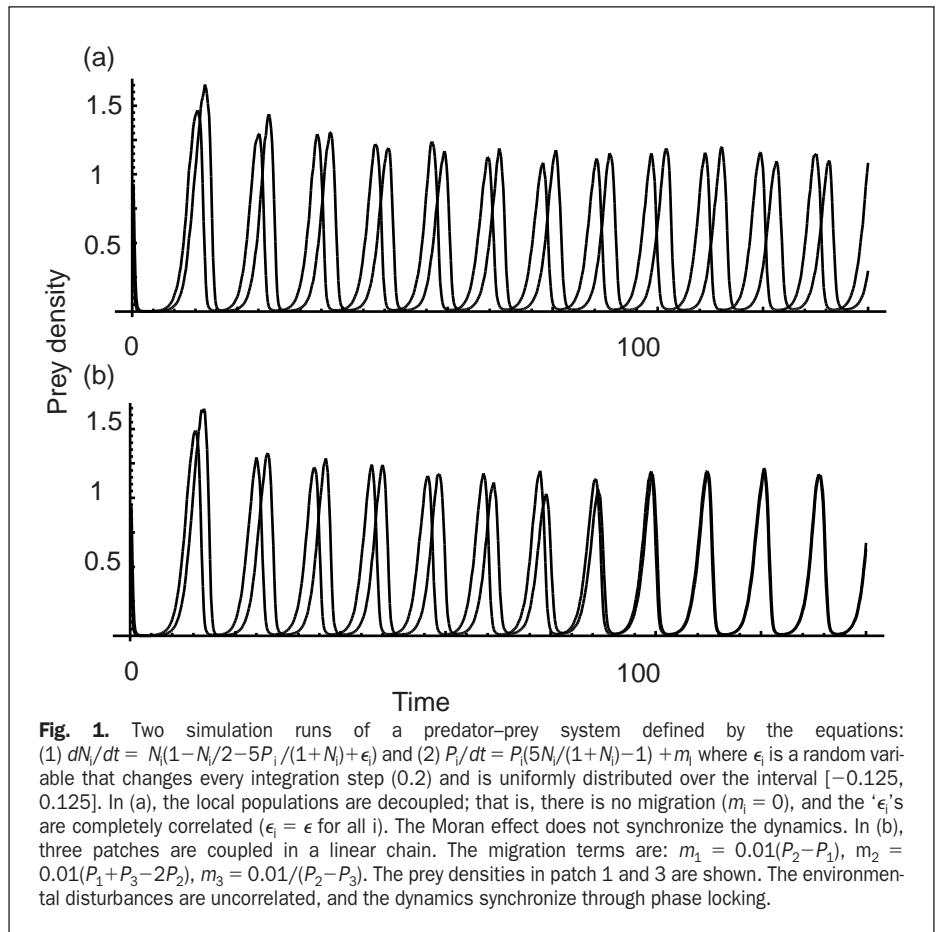


Fig. 1. Two simulation runs of a predator–prey system defined by the equations: (1) $dN_i/dt = N_i(1 - N_i/2 - 5P_i/(1 + N_i) + \epsilon_i)$ and (2) $P_i/dt = P_i(5N_i/(1 + N_i) - 1) + m_i$ where ϵ_i is a random variable that changes every integration step (0.2) and is uniformly distributed over the interval $[-0.125, 0.125]$. In (a), the local populations are decoupled; that is, there is no migration ($m_i = 0$), and the ' ϵ_i 's are completely correlated ($\epsilon_i = \epsilon$ for all i). The Moran effect does not synchronize the dynamics. In (b), three patches are coupled in a linear chain. The migration terms are: $m_1 = 0.01(P_2 - P_1)$, $m_2 = 0.01(P_1 + P_3 - 2P_2)$, $m_3 = 0.01/(P_2 - P_3)$. The prey densities in patch 1 and 3 are shown. The environmental disturbances are uncorrelated, and the dynamics synchronize through phase locking.

1994, but has since been rediscovered in the wild, albeit in very low numbers⁸.

Recent work on captive populations of *Partula* tree snails has revealed the first definitive case of an infectious agent causing the ultimate demise of a species⁹. *Partula* snails were extirpated from many of the South Pacific islands to which the genus is endemic, following introduction of a predatory snail (*Euglandina rosea*) – itself introduced as part of an *ad hoc* biocontrol measure. Currently, 12 of the 20 or so *Partula* spp. maintained in captivity are extinct in the wild. One such captive species, *P. turgida*, became extinct in 1996. Pathological investigations of the last individuals to die revealed a disseminated microsporidian (*Steinhausia* sp.) infection as the cause of death. One implication of this finding is that captive breeding programmes should no longer be considered 'safe-havens' for animals on the brink of extinction¹⁰.

Although *P. turgida* represents the only published case of extinction by infection, there is at least one example of extinction by infection by proxy. This concerns an outbreak of wasting disease in the marine eelgrass *Zostera marina*, caused by a pathogenic slime mold *Labyrinthula zosterae*¹¹, which resulted in over 90% loss of eelgrass cover in the North Atlantic Ocean between 1930 and 1933. Although some eelgrass populations survived in low-salinity refugia, a stenohaline host-specific eelgrass limpet, *Lottia alveus*, became extinct shortly thereafter¹². It is uncertain how many other cases of extinction resulting from 'knock-on' effects of disease have occurred historically, or indeed how many pathogens have become extinct with their hosts⁹.

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