Journal Club

Phanerozoic biodiversity: rocks or real?

The marine biodiversity curve is an icon of paleobiology. The familiar curve shows increasing global diversity in the early Paleozoic, then a plateau, followed by increasing diversity through the Mesozoic and Cenozoic. The curve also gives us often quoted magnitudes of mass extinctions and the relay race of Evolutionary Faunas. When Sepkoski initially published his compilation, Raup, his colleague at the University of Chicago, questioned whether the curve represented a biological signal or was an artifact of the rock record. Raup's argument was compelling but his data modest, so the objection was tabled in the flush of new insight from Sepkoski's database.

Today, a new generation of Chicago paleontologists has reopened the question. In a new paper, Peters and Foote [1] use the number of geological formations (distinct rock units) as a proxy for the amount of marine rock available for paleontological sampling. They found a strong, positive correlation between diversity during a given interval and the number of geological formations. Similarly, global diversity was positively correlated with the proportion of continents flooded during an interval

and with the actual area of exposed marine rock. Peters and Foote speculate that, during times of high sea levels, more rock would be deposited and diversity might be higher owing to an increase in habitable area. To test this possibility, they compared the degree of continental flooding to diversity while keeping the amount of rock sampled constant. Only a small amount of the variation in diversity could be explained by continental flooding alone. Thus, apparent variation in diversity might be a function of the amount of rock sampled, rather than an evolutionary phenomenon.

Although the iconic diversity curve might be specious, the pattern of Evolutionary Faunas and their different average turnover rates stand firm. However, attempts to correlate variation in global diversity with Earth system events might be a geological snipe hunt.

1 Peters, S.E. and Foote, M. (2001) Biodiversity in the Phanerozoic: a reinterpretation. *Paleobiology* 27, 583–601

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Phage λ takes the first exit

Once upon a time, biologists were accused of physics envy, of wanting universal laws and neat equations. The opposite could now be true, with physicists and mathematicians looking to biology for challenging problems and complex systems. Their contribution is often to recognize that a biological system resembles a mathematical framework for which a set of solutions already exists: the same equations have the same solutions, irrespective of their origin. This approach is illustrated by a recent model developed by Aurell and Sneppen [1] that frames the transition between lytic and lysogenic phases of bacteriophage replication as a first exit problem, which is a way of characterizing the expected time at which a random process will end.

For example, the end point of a game of poker occurs when all but one player runs out of money (assuming no one has the sense to stop before his or her purse is empty). The time it takes to reach this endpoint will depend, to some extent, on chance events (the deal of the cards). When the temperate phage λ infects *Escherichia coli*, it enters one of two pathways: lytic (replicates and kills the host) or lysogenic (enters the genome of the host and is inherited passively by the progeny of the host). The quiescent state of a lysogenic phage is maintained by specific proteins that bind to sites on the phage genome.

This protein–DNA interaction acts as a switch: if the concentration of the regulatory proteins drops below a crucial level, repression of transcription ceases, and the phage enters the lytic phase (and it's curtains for the *E. coli*). This system has been modelled previously by deterministic equations, for which the system has a stable equilibrium – the lysogenic state. This deterministic description might be valid if the regulating proteins were in very high concentrations in the cell. But, in reality, these molecules exist in small enough numbers that there is an element of randomness to the rate at which they bind and leave the receptor. The Brownian motion of the regulatory proteins results in the expected number of hits to the target sites growing with time only as its square root. This randomness, analogous to the effect of genetic drift on allele frequencies in small populations, introduces noise into the regulation of lysogeny. The beauty of this model is that it predicts that λ can exit the lysogenic phase and go into active replication simply through noise in the signalling system. This random model is somewhat at odds with the apparent robustness of the lysogenic state to experimental manipulation, suggesting that there could be additional factors at work. But in any case, this paper demonstrates that, rather than biologists with physics envy, we now have physicists modelling the messiness of biological systems.

1 Aurell, E. and Sneppen, K. (2002) Epigenetics as a first exit problem. *Phys. Rev. Lett.* 88, 048101

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