a high-oxygen atmosphere to revert back to a low-oxygen state. This wonderful result might explain why oxygen levels stabilized permanently following the GOE.

But there are other unresolved issues in this saga. There is evidence[11–13] of low sulphur MIF values in rock formations of between 2.76 billion and 2.92 billion years old, suggestive of high atmospheric-oxygen levels preceding the accepted time of the GOE. So far, no high MIF values have been reported in that time interval. How can this be rationalized?

One possible explanation — the so-called 'yo-yo' atmosphere theory — was proposed earlier this year[11]. This theory suggests that oxygen levels first increased about 3.0 billion years ago, decreasing again about 0.2 billion years later, before their final climb to high concentrations 2.4 billion years ago (the GOE). Goldblatt and colleagues' model[5] explicitly predicts that the atmosphere was bistable for some time before the GOE, so maybe the yo-yo theory is correct.

But this would still leave some unexplained observations. For example, the Witwatersrand gold deposits in South Africa contain detrital minerals that were washed down streams between 2.8 billion and 3.0 billion years ago[14]. In the presence of oxygen, these minerals should have become oxidized and dissolved. So, either the oxygen levels were never high enough for that, or they repeatedly went up and came back down very quickly. Or perhaps oxygen concentrations did not increase at all, and the low-MIF anomaly seen in post-GOE rocks was produced by some entirely anoxic mechanism, such as the shielding of solar ultraviolet rays by an organic haze[13,15].

The jury is still out, but all these contradictory observations are stimulating a lot of creative thinking. Let us hope that this will lead to a more unified understanding of a fascinating era in Earth's history. The ancient atmosphere may have had a more complex evolution than we imagined. ■

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ECOLOGY

Moving to the ideal free home

Douglas W. Morris

Pike move between two basins of a British lake to maximize their evolutionary fitness. This adaptive behaviour suggests that habitat selection is more significant in population dynamics than was thought.

How should animals choose which habitat to live in? An evolutionary biologist is likely to answer that they should maximize their evolutionary fitness — the likelihood that their genes will be passed on to future generations — and live in the habitat that will produce the most descendants. But there is a catch. If all individuals make the same choice, the habitat will become crowded, the probabilities of survival and reproduction will suffer, and fitness will decline. Other habitats with fewer individuals might be a better option. So animals maximizing fitness through habitat selection will disperse among habitats until no individual can improve its fitness by moving.

This 'ideal free distribution'[1] assumes that an individual can estimate accurately the fitness that can be attained in different habitats, and is free to move and occupy the ideal place that maximizes fitness. Many ecologists have questioned whether the theory is hamstrung by these preconditions. Can such a model really apply to natural populations? Writing in *Proceedings of the Royal Society*, Throndt Haugen and colleagues[2] provide convincing evidence that it can. They show that, every year, pike in a northern English lake move from a habitat with low fitness (a higher mortality rate) to one with higher fitness, thus eliminating the initial difference. The research tells us that the spatial distribution of populations may often represent a dynamic equilibrium caused by habitat selection.

To understand the principle of the ideal free distribution, think of queues at airport security. As a passenger, you want to pass through as quickly as possible. If there are several queues, you choose the one that you think will move the fastest. But everyone with a boarding pass is trying to do the same. So the number of people in each queue is in dynamic equilibrium because, if one line moves more slowly than others, passengers will swap queues. Thus, at any given time, the average wait before passing through the checkpoint is similar for everyone at the end of the lines.

Now apply the same principle to animals choosing their habitat. At equilibrium, the distribution of individuals among habitats is evolutionarily stable[3]: no individual can improve its fitness by moving to another habitat. But habitats and population sizes change, disturbing that balance. Individuals will track those changes by moving from one habitat to another until their distribution regains its evolutionary stability. The density in every habitat thus depends on the density in others, just as the length

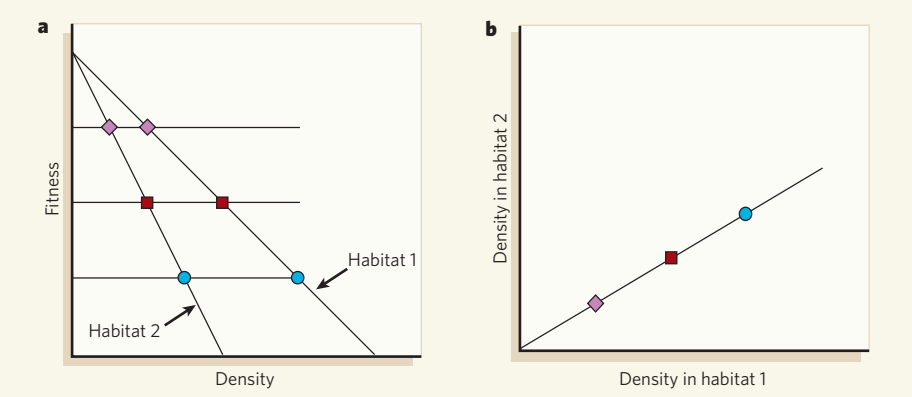

Figure 1 | Testing for an ideal free distribution. a, Evolutionary fitness declines more quickly with increasing population density in habitat 2 than it does in habitat 1. Symbols at the intersections with horizontal lines represent densities where fitness is equal in both habitats (an ideal free distribution). **b,** The equilibrium densities can be plotted against each other to yield the expected distribution of individuals in the two habitats (the habitat isodar[7]). Haugen *et al.*[2] do this for the northern and southern basins of Lake Windermere, and compare these with actual data for pike populations collected since the 1940s. The agreement between the model calculations and the data is astoundingly good.

Figure 2 | Divided lake. Lake Windermere is separated by a shallow sill into two habitats for pike.

of one security queue depends on the others.

There are two ways to test the theory. First, you can do an experiment: you measure the fitness, density and dispersal of a model species living in adjacent habitats. Then you vary the population size; the densities and fitness of individuals in the two habitats should also vary. If, despite changes in density, the mean fitness actually remains equal in each habitat, the animals are following an ideal free distribution.

But what if you can't do the experiment? Then you can stand the theory on its head. Instead of testing whether fitness is equal, you measure fitness at different population sizes in different habitats. You assume that fitness is equalized, and then calculate how many animals should live in each habitat (Fig. 1).

Haugen *et al.*[2] use an incredible data set to perform this second type of test. For more than 40 years, biologists captured, measured, marked and recaptured pike (*Esox lucius*) in Lake Windermere in northwest England. This lake has two basins (Fig. 2), which are separate pike habitats. Pike reproduce only once each year, so it is appropriate to census their densities and assess habitat selection annually.

The authors use capture histories of individually marked pike to estimate survival and dispersal[4] of the fish. The probability of observing a given fish depends on the probability of it staying alive between census periods, the probability of it being captured when alive, and the probability of it moving between the two basins. Each of these probabilities depends on such things as the size and sex of the fish, and the basin in which it currently lives. Haugen *et al.* searched for the most likely models[5] describing the pattern of pike captures in each basin. They merged these models with data on egg production to calculate density-dependent population growth rates in the two habitats. By assuming that these growth rates were equalized through habitat selection, they could back-calculate the number of pike expected in each basin each year. There was a nearly perfect fit between predicted and actual densities.

Thus, pike choosing between the two basins of Lake Windermere behaved as though capable of ideal free habitat selection. Critics will demand more proof. Do pike disperse to the basin where higher fitness was assured? And, when placed in an experimental environment, do pike adjust their habitat choice to equalize fitness?

Haugen *et al.*[2] answer both questions. The population density in both basins increases during spawning. Although the southern basin is more productive, pike survival in the south decreases more quickly with population density than it does in the north. Thus, the fitness curves in the two basins diverge (Fig. 1). At an ideal free equilibrium, every fish in each basin will, on average, produce the same number of descendants. But even if the system is in equilibrium after spawning, the different survival rates in each basin cause fitness to rebound more rapidly in the south. The system will move away from its former equilibrium. So with each passing year — if pike are ideal and free — their net annual dispersal should be biased towards the southern basin.

And it was, most of the time. There was a three-year anomaly when pike reversed their dispersal and headed north. This coincided with a remarkable, serendipitous experiment. Fisheries officials, during only those three years, had reduced the population of pike in the northern basin by approximately 20%.

Most previous tests of ideal free theory have been at a small scale where foragers choose between patches differing in food supply[6]. Pike in Lake Windermere provide compelling evidence that the ideal free distribution occurs at much larger scales where populations are regulated. Dispersal between basins equalizes fitness and homogenizes population growth. Although we need additional tests and experiments at these large scales, the message seems clear: if you study populations, then you must include the potential for habitat selection. ■

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CELL BIOLOGY

Mitochondria shape up

Barbara Conradt

Mitochondria are central to the process of programmed cell death that kills damaged or superfluous cells. Surprisingly, components of the death machinery turn out to be essential for keeping these organelles in shape.

Proteins of the Bcl-2 family are evolutionarily conserved regulators of programmed cell death (apoptosis). However, their mechanisms of action are still not fully resolved[1,2]. On page 658 of this issue, Youle and colleagues[3] report that the mammalian Bcl-2 family members Bax and Bak have additional functions to their apoptotic ones: they control the morphology of mitochondria, the cellular organelles responsible for energy generation. Other components of the apoptotic machinery also perform tasks outside apoptosis[4,5]. These tasks are unrelated to the proteins' apoptotic roles, however, whereas the non-apoptotic and apoptotic functions of Bax and Bak are linked. This latest study therefore improves our understanding not only of the regulation of mitochondrial morphogenesis, but also of the apoptotic functions of Bcl-2 proteins.

Bax and Bak are prototypical 'killer' proteins: their pro-apoptotic activities are low in healthy cells but high in cells instructed to die; their overexpression in cultured mammalian cells accelerates apoptosis; and, in mice lacking both Bax and Bak (Bax/Bak DKO mice), most apoptosis is blocked[1,2]. The ability of Bax and Bak to kill cells is intimately associated with the mitochondria. Whereas these two proteins are found singly in healthy cells, in cells earmarked for apoptosis they tend to group together into oligomers in the outer mitochondrial membrane (OMM). This oligomerization and association with mitochondria has two consequences: it causes the organelles to change