MARINE BIOLOGY

Spawning spot

The enigma of the eel life-cycle has become a little less enigmatic — at least as far as the Japanese eel, *Anguilla japonica*, is concerned. As described elsewhere in this issue (*Nature* **439**, 929; 2006), Katsumi Tsukamoto has filled in a missing part of the picture of the species' ocean biology.

When they are mature, eels migrate from fresh water out into the open ocean, travelling thousands of kilometres to spawn. But the when and where of the actual spawning event, although suspected in general terms, have been hard to pin down. This is partly because of the technical challenges of isolating and identifying the tiny, transparent larvae just after they hatch. Tsukamoto has tackled those

challenges to pinpoint the timing — which aptly coincided with a new moon in June — and strategic location of *A. japonica* spawning.

This fishing feat also meant that the newly hatched larvae could be photographed. As shown in this picture, teeth, jaws and pigmented eyes are evident within five days of hatching, when the larvae are still less than 5 millimetres long.

Thanks to the efforts in the 1920s of a Danish oceanographer, Johannes Schmidt, the spawning

area of the European (*A. anguilla*) and American (*A. rostrata*) eels was identified as the Sargasso Sea in the western North Atlantic, spreading over some 5 million square kilometres. But it seems that the nursery of the Japanese eel, as identified by Tsukamoto, is much more compact — spawning is confined to a seamount northwest of Guam in the Philippine Sea, a location that enables the larvae to ride the northbound Kuroshio Current

to their freshwater destinations in eastern Asia.

This discovery will be applauded by researchers specializing in eel biology, as well as by those intrigued by a life history that constitutes one of nature's wonders. But there is a commercial aspect too: eels are of great economic value, not least in Asia, and there is a pressing need for management and conservation of natural populations.

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PARASITOLOGY

Switching like for like

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To remain hidden from its host's immune system, the malaria parasite must vary the proteins on the surface of the infected cell. The genes encoding these proteins are very similar, so how does the parasite express just one at a time?

Selecting one gene for activation out of many closely similar ones is tricky. Mammals address this problem when they switch on just one odorant receptor gene in each olfactory neuron, choosing between a thousand receptor genes. Bacteria and protozoa address it when they vary the proteins displayed on their surface. How an organism can activate just one gene and keep all the other closely related ones silent — a process known as allelic exclusion — has long remained elusive. In this issue, Voss *et al.* (page 1004)¹ analyse how this selection process works for the *var* gene family of the malaria parasite *Plasmodium falciparum*.

The *var* genes are expressed when the parasite multiplies in the red blood cells (erythrocytes) of its host. The genes encode proteins known as *P. falciparum* erythrocyte membrane protein 1 (PfEMP1), so-called because they are exported from the parasite and end up in the membrane of the red blood cell (Fig. 1). PfEMP1 is key to the lethality of *P. falciparum* infection, because the PfEMP1 molecules on the erythrocyte surface cause the cell to stick to capillary walls. In this way, the infected cells are prevented from circulating through the spleen where they would be recognized by immune cells and removed from the circulation. So PfEMP1 provides the parasite with more time

to multiply. The unfortunate side effect is that capillaries become clogged, resulting in the severe symptoms of tropical malaria.

Putting 'alien' proteins, or antigens, on the erythrocyte surface inevitably advertises the parasite's presence to the host immune system, and eventually results in an immune response against the infected cell. To escape this threat, the parasite uses antigenic variation of its PfEMP1. The *var* family consists of 60 genes, each encoding a different PfEMP1.

Figure 1 | A red blood cell infected with *Plasmodium falciparum*. The surface of the infected cell is studded with proteins of the PfEMP1 family, encoded by *var* genes. The various PfEMP1 molecules can bind to one of a series of receptors on the endothelial cells that line the capillary walls. (Modified from ref. 14.)

By switching the *var* gene expressed, the parasite changes the PfEMP1 on the host-cell surface before the host immune system can mount a full response to it. This allows the parasite to survive and multiply for another week.

Antigenic variation of surface PfEMP1 works in practice only if the parasite expresses just one *var* gene at a time. If all 60 genes were simultaneously expressed, the host would make antibodies against all 60 at once and eliminate the infected cells. The parasite must also be able to switch the *var* gene expressed, otherwise it would continue to make the same surface antigen and be doomed by the host immune response. Key elements in the strategy of antigenic variation are therefore a large gene family, the ability to activate only one gene and keep the others silent (allelic exclusion), and the ability to switch the gene that is active².

The *var* genes encode large proteins (relative molecular mass 250,000–300,000) and have