# In the news

## GLOWING GNATS MALARIA'S DOWNFALL

A mosquito has been engineered that is resistant to malaria infection. "It is an audacious scientific project that could offer hope to 300 m malaria sufferers worldwide," (*The Guardian*, 21 March 2007). "Large numbers of [these] GM mosquitoes would be released in areas where malaria is common, where they would interbreed with wild ones," (*The Times*, 20 March 2007).

Every year, malaria infects 300–500 million people worldwide, and kills 2.7 million. The idea of genetically modifying mosquitoes to break the life cycle of the malaria parasite is not new. "Though the first GM mosquitoes were created seven years ago, they proved to be less fit than their wild counterparts," (*The Times*, 20 March 2007). This time, it is different. The new, fluorescent-eyed strain created by a team from John Hopkins University in Maryland, USA, "…had a higher survival rate and laid more eggs. After nine generations, 70% of the insects belonged to the malaria-resistant strain," (*The Daily Mail*, 21 March 2007).

In a parallel effort, a team at Imperial College London, UK, created a strain in which males have fluorescent testicles, "…allowing scientists to easily separate them from females," sterilize them and release them "…into the wild so they mate with wild females but have no offspring," (*The Daily Telegraph*, 21 March 2007).

Despite the promising news, the results will need to be repeated using the actual parasite that causes the most dangerous form of malaria in humans. The Maryland team used a parasite that does not infect humans and the mosquito that is the main Asian, not African, vector.

Moreover, environmental groups have concerns about "…supplanting a naturally occurring species with a genetically engineered variant. […] Even scientists involved accept that further research is needed before any GM insects could be introduced into the wild," (*The Times*, 20 March 2007).

*Magdalena Skipper*

EVOLUTION

# Colour vision for mice

Primates have trichromatic rather than dichromatic vision, because their genomes encode three rather than two types of cone photopigment. But was the acquisition of a new receptor type in a dichromatic ancestor sufficient for trichromacy to evolve, or was more complex neural rewiring required? A new study shows that mice can acquire new colour vision simply by expressing an additional human photoreceptor.

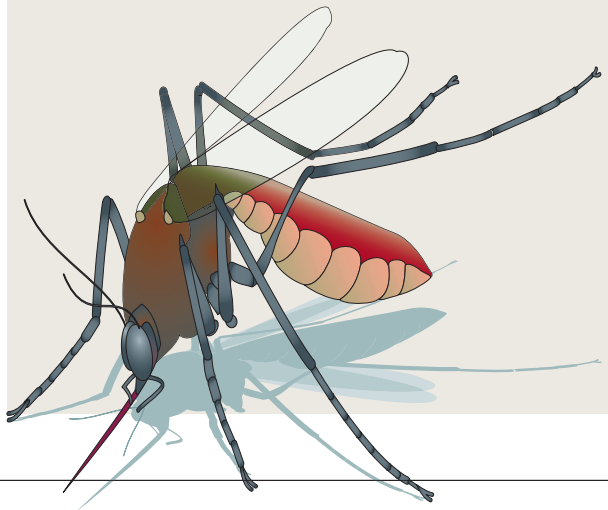
Most mammals have just two types of photoreceptor, sensitive to short (S) or medium (M) wavelengths of light. However, in most New World monkeys, the X-chromosomal gene that encodes the M receptor has a second allele that is sensitive to long (L) wavelengths; therefore, heterozygous females have S, M and L receptors and are consequently trichromatic. In Old World primates, the X-chromosomal locus has duplicated and diverged, giving all individuals trichromatic vision; however, the New World monkey system is thought to be ancestral.

Gerald Jacobs and colleagues attempted to recreate the New World monkey situation in mice by replacing the M locus with the human L locus and breeding heterozygous females. The process of random X-chromosomal inactivation in clones of cells means that there are separate populations of M- and L-expressing cones in the eyes of such individuals, but the proportions of the two are variable. Initial physiological tests showed that the L cones were functional, so the authors carried out behavioural tests to assess new colour vision.

TECHNOLOGY

# Driving out insect-borne pathogens

A potential way to tackle pathogens such as malaria and dengue is to use transgenic insect vectors that are refractory to parasite transmission. However, there is a question mark over whether these modified vectors could successfully infiltrate the wild population. Using the fruitfly as a model, a recent study shows how this could be achieved: the authors have created a transgene that drives its own spread throughout an insect population.



To have a chance of taking over a population, transgenic vectors must have a fitness advantage over unmodified insects. Chen and colleagues showed how such an advantage can be generated in a *Drosophila melanogaster* population. They made a transgene carrying a 'toxin' that is expressed specifically in the female germ line, consisting of two microRNAs (miRNAs) that silence *Myd88*, a gene that is essential for embryonic development. The transgene also includes an 'antidote' — a version of *Myd88* that is expressed in embryos and is immune to the effects of the miRNAs. The authors confirmed that when a transgenic female lays eggs, offspring that lack the transgene die as embryos — owing to a lack of *Myd88* expression — whereas transgenic offspring survive.