PHOTOTAXIS

Jumping to Conclusions

ALL frogs may look before they leap, but it is now clear that all anuran species are not alike in their phototactic preferences. Although since the turn of the century it has been widely believed that frogs preferentially face and move towards blue light, Robert Jaeger of the University of Maryland and Jack Hailman of the University of Wisconsin report on page 189 of this issue of *Nature* a new kind of anuran phototactic behaviour; the tropical frog *Eleutherodactylus rhodopsis* does not seem to be sensitive to any particular colour of light, but responds only to the apparent brightness of the light source.

The study of colour vision in frogs has fascinated zoologists chiefly for two reasons. First, the mechanism by which anurans recognize and respond to blue light is an example of very primitive colour vision; it serves as a model which may help to explain the more highly developed systems found in other animals. Second, it is hard to see what benefit this limited colour sense confers on the species —and similarly in what way the lack of colour response may penalize *E. rhodopsis*, if at all.

The principal problem in devising experiments to investigate colour preference in a phototactic response is to ensure that what is measured is really a response to the colour of the light stimulus, that is, true colour vision, and not merely to the intensity of the light used. The test used by Jaeger and Hailman was similar to that designed originally by W. R. A. Muntz, a pioneer in the study of colour vision in frogs. The amphibians were placed singly in a black box facing two screens towards which the animal could jump. The screens were illuminated with different colours and intensities of light and the frogs were allowed to choose which of the screens to jump towards (being helped on their way by a friendly prod with a blunt instrument).

The use of appropriate combinations of filters and light intensities made it statistically possible to show that the leopard frog *Rana pipiens* shows a true colour preference in leaping predominantly towards blue light, whereas the tropical frog *E. rhodopsis* shows no such discrimination, but responds only to the apparent brightness of the source.

Neither the physiological nor the functional significance of this difference in the phototactic behaviour of the two species seems clear at present, but Jaeger and Hailman hope that a more extensive comparative survey of phototaxis in anuran amphibians will clarify the picture. The early work of Muntz (*J. Neurophysiol.*, **25**, 712; 1962, for example) clearly established that in ranid frogs, there are specific optic nerve fibres which run from the retina to that part of the brain called the dorsal thalamus which are responsible wholly for the detection of the onset of illumination and for distinguishing between blue and other spectral colours. Other optic fibres, concerned with movement of small and large objects, run to the optic tectum. In man, where colour vision is highly developed, the tectal network is comparatively unimportant, whereas most of the optic fibres run to the dorsal thalamus, and thence to the cerebral cortex. It will be interesting to see whether there are significant differences in the structure of the optic nerve system in the brain of *E. rhodopsis* compared with *R. pipiens*.

On the other hand, Muntz has suggested that the blue light response in frogs is a defence mechanism whereby a badly frightened frog can instantly recognize and leap towards the sanctuary of water; it will be equally interesting to see whether similar ecological considerations underlie the lack of response to colour in the tropical frog.

No Polyoma Repressor

DRAWING analogies between the lysogenization of *Escherichia coli* by temperate phages such as lambda and the transformation of cultivated animal cells by the small DNA tumour viruses SV40 and polyoma virus is a fashionable exercise. And it is useful so long as it is not taken too seriously, for in many respects the two phenomena are not closely analogous. If anybody doubts that, he should read the account by Basilico and Wang, in next Wednesday's *Nature New Biology*, of experiments which rule out the idea that a dominant and diffusible immunity factor, which specifically represses the polyoma virus genome, exists in polyoma transformed cells. By contrast, of course, the lysogeny of *E. coli* by lambda phage involves the synthesis of just such an immunity factor; it not only prevents the complete transcription of the infecting phage genome responsible for lysogenization but also renders the lysogenic cell resistant or immune to superinfection.

Lysogeny occurs when bacterial cells are infected by a phage which has the potential to replicate completely in those bacteria and as a result kill them; it is not known precisely which factors determine whether a particular infection will result in lysogeny rather than replication and lysis. Transformation, on the other hand, can occur either when wild type virus infects a type of cell which cannot support the complete replication of the virus, or when a defective virus, inherently incapable of complete replication, infects a permissive cell which can support the complete replication of wild type virus.

A priori it might be anticipated that lysogeny should, as it does, depend on some repressor factor, which somehow prevents the complete expression of the phage genome. If that were not the case, every infection should result in the replication of the virus and death of its host. This argument does not, however, apply to transformation, an event which involves either a defective viral genome in a permissive cellular environment or a functionally complete viral genome in a non-permissive environment, in which the genome is incapable of full expression.

It has been shown repeatedly that permissive mouse cells transformed by polyoma virus are not resistant to superinfection with wild type virus. This observation is precisely that anticipated. Two French groups have, however, recently reported experiments which, they believe, show that cells transformed by polyoma virus or simian virus 40 contain a "repressor" which specifically inhibits viral multiplication. But not every, indeed very few, tumour virologists were convinced by this claim and Basilico and Wang's results add another nail to its coffin.

These workers made hybrids by fusing non-permissive hamster (BHK) cells, transformed by polyoma virus with uninfected 3T3 mouse cells which support the multiplication of polyoma virus. So long as the hybrid cells retain the hamster chromosomes carrying the integrated transforming polyoma genomes they should also contain any putative repressor or immunity factor and as a result resist superinfection. In fact, however, the hybrid cells, with a slight excess of hamster cell chromosomes compared with the normal complement, were susceptible to superinfection. In superinfected populations of hybrid cells, the frequency of virus-producing cells and cells dying as a result of polyoma multiplication was the same as that in populations of normal 3T3 cells infected with the virus.

The only way to reconcile these observations with the notion that the transformed cells contain a diffusible "repressor" is to postulate further that 3T3 cells contain a dominant and diffusible "antirepressor"; such flights of fancy are completely unwarranted in the face of the much simpler hypothesis that a repressor does not exist. In short, transformation and lysogeny are analogous in so far as both phenomena involve the integration of a viral genome into a host cell chromosome. But the ways in which the expression of the viral genome is blocked in the two situations are by no means analogous apparently.