Palaeontology

Triassic 'scenarios'

from M. Howgate

THE tetrapod land faunas of the early part of the Triassic period were dominated by mammal-like reptiles. Herds of herbivorous dicynodonts were being preyed upon by top carnivores such as *Cynognathus*. However by the end of the period this entire fauna had all but vanished to be replaced in all the large-body terrestrial ecological niches by the early dinosaurs and their relatives — the archosaurs. The problems posed by this major faunal replacement and the disparate assemblage of theories it has thrown up were addressed by Alan Charig, from the British Museum (Natural History), at a recent symposium*.

Charig started with a defence of the 'scenario' as a valid method of interpreting past events — a method dismissed by some as mere evolutionary storytelling. By 'scenario' Charig means "a sequential narrative of hypothesized prehistorical events based on conjectural interpretations of palaeontological data". Such a scenario could be deemed to be scientific provided that it were based on a firm foundation of empirical evidence, the interpretations made being the most parsimonious possible and the scenario as formulated open to subsequent modification.

It was on these latter points that Charig himself opposed some of the existing scenarios constructed to explain the faunal transition, on the grounds that they relied too much on "the conjectured effects of conjectured environmental conditions upon conjectured physiological characteristics". The theories he particularly had in mind were those involving hot-blooded dinosaurs and their ilk, that seek to explain the dominance of the archosaurian fauna in terms of their hypothetical ectothermic constitution; or conversely for cold-blooded dinosaurs, theories that depend on a hypothetical deterioration in climate. Similar objections apply to those theories which produce a non-competitive scenario by relying solely on extrinsic factors, such as changes in climate and flora to eliminate the mammal-like reptiles.

Charig's own position is that the replacement was dominated by a competition for food resources (actively or passively — it makes no difference) in which the primary selective factor, and the only one for which we have direct evidence in the fossil record, was the improvement in locomotion in the archosaurian lineage. Thus while the archosaurs attained first a 'semi-improved' and then a 'fully improved' dinosaurian gait, the mammal-like therapsids, despite alleged improvements in thermal regulation, never achieved a comparable stance and gait.

Throughout the Early Triassic then, a gradual replacement took place in which archosaurian carnivores out-competed their cynodont adversaries, the latter becoming smaller, fewer and less diverse as the replacement 'snowballed' during Middle Triassic times. By mid-Ischigualasto times (Carnian of Argentina) the terrestrial fauna was one of carnivorous pseudosuchian archosaurs preying on a herbivorous assemblage composed of dicynodonts, those previously carnivorous cynodonts which had succeeded in making the transition to herbivory — the traversodontids — and the aberrant rhynchosaurs [now separated from the rhynchocephalians and placed among the archosauromorphs by Mike Benton (Nature Conservancy Council, Newbury) in another paper at the same symposium].

The post-Ischigualasto formations reveal a second wave of replacement, this time among the herbivores, and Charig sees his scenario developing as follows. The archosaurs, some now with a fully improved gait and hence dinosaurs, over-exploit the therapsid herbivores and rhynchosaurs and drive them to extinction. Then faced with a drastically reduced food supply but an abundance of vegetation, selection pressure drove some of the omnivorous archosaurs to switch to a vegetable diet. So by the beginning of the Jurassic period a new balance had been attained in which both carnivores and herbivores had the same fully improved gait. □

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*A symposium on 'The Structure, Development and Evolution of Reptiles' was held in honour of Professor Angus Bellairs by the Zoological Society of London on 26–28 May 1983.

Immunology

The importance of T3 in the activation of T lymphocytes

from Peter Beverley

MONOCLONAL antibodies against leukocyte surface antigens were first used for identifying and separating subsets of cells, but more recently attention has been focused on the molecules identified by the antibodies. Clearly, if an antibody modifies a cellular function this implies that the molecule to which the antibody binds may be involved in that function. Monoclonal antibodies thus provide probes for investigation of function at the molecular level.

The anti-T3 monoclonal antibody OKT3 was first described as an antibody recognizing all T cells[1], and it has since been used, along with others of the same specificity, to identify and enumerate human T cells in health and disease. Early biochemical data identified T3 as a 20,000-molecular weight glycoprotein with some heterogeneity of charge[2], but T3 might have remained for some time a molecule looking for a function had it not been shown that anti-T3 antibody had profound effects on T-cell function.

Anti-T3 antibody in very low concentrations is a powerful mitogen for human T cells[3] and at the least this observation identifies T3 as a molecule (or molecules) likely to play a part in the activation of the cells. That binding of a ligand to T3 is not sufficient in itself to induce division in resting T cells is shown by the requirement for accessory cells, demonstrated previously by cell depletion and reconstitution experiments[3], and confirmed in this issue of *Nature* by the identification of a genetically determined defect in the ability of some human non-T cells to provide accessory function for T-cell responses to mouse IgG1 anti-T3 antibodies[4].

Anti-T3 has since been shown to block a variety of T-cell functions including the proliferative response to soluble antigens and both the generation in mixed leukocyte culture and expression of T-cell cytolysis[5]. While the mitogenic effect of the antibody made interpretation of the proliferative data difficult, the blocking of the effector phase of cytolysis was not open to this criticism and, taken with the mitogenic effect of anti-T3, strongly implied that the T3 molecule was involved in T-cell recognition or triggering by antigen. Recent experiments reported in *Nature* and elsewhere have amply confirmed this view.

The results can be summarized as follows. If interleukin-2-dependent T-cell clones are exposed to anti-T3 antibody, T3 is rapidly lost from the cell surface by a process known as modulation. Cells in which modulation has occurred are no longer capable of responding by proliferation to alloantigen[6] or soluble antigen[7] nor, in the case of cytotoxic clones, can they kill appropriate target cells. That loss of T3 is linked to loss of antigen-specific receptors is implied by the parallel loss of a putative T-cell receptor detected by a clone-specific monoclonal antibody[7]. Similarly, T3 is modulated if cells are exposed to the anti-clonotype antibody or to soluble antigen, again implying that the antigen-specific receptor and T3 are linked. Modulated

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