RÉSUMÉ

Chicken feed

WHAT, thought J. Pen *et al.*, can be done to avoid the need to supplement the diets of some domestic animals, such as pigs and chickens, with inorganic phosphorus? Feed supplementation with the enzyme phytase, which releases the nutrient from phytate (the main form of phosphorus in many plant seeds), is one way. But Pen and colleagues have tested another (*Bio/Technology* **11**, 811–814; 1993), by engineering the DNA fragment encoding phytase in the fungus *Aspergillus* into tobacco. Transgenic seeds stored the phytase stably, and were effective in releasing inorganic phosphorus in experiments *in vivo*; moreover, poultry whose diet was supplemented with the modified seeds showed significantly higher growth rates than controls. The authors now plan to try the approach with plants whose seeds are more commonly used as feedstuff.

Watch this space

TINY though they are, meteoroids in the Perseid shower could cause considerable damage if they were to strike one of the Earth's many artificial satellites. This year, with the Earth passing close behind the tail of the meteoroid parent body, the infamous comet Swift-Tuttle, the shower may rise to a storm lasting anything from 10 minutes to an hour. Martin Beech and Peter Brown (*Mon. Not. R. astr. Soc.* **262**, L35–L36; 1993) estimate from past records of exceptional Perseid and Leonid showers that an object the size of the Hubble Space Telescope has about a chance in a thousand of being hit by a hefty meteoroid — say, two or three milligrams in mass — this August. Surely no one satellite could be so unlucky?

Molecular poultice

PERHAPS the most disagreeable trick that evolution has taught the malaria parasite is how to induce the infected red blood cell to stick to endothelial surfaces and cause such complications as cerebral malaria, which tends to be fatal. The mechanism of adhesion is therefore a busy area of malaria research. A suggestion now emanating from I. W. Sherman's laboratory is that adhesion is promoted by a modified form of the abundant host cell membrane protein, band 3. From fragmentary evidence that histidine, tyrosine and lysine side chains contribute to adhesion, I. Crandall *et al.* (*Proc. natn. Acad. Sci. U.S.A.* **90**, 4703–4707; 1993) have deduced that two of the small external loops of the band 3 chain (which crosses the membrane 14 times) may be the sites in question. They have made corresponding synthetic peptides and find that these indeed inhibit adhesion *in vitro*, and when injected into infected monkeys cause release of the parasitized cells into the circulation. Therapeutic possibilities are to be explored.

is reasonable to suppose that closer to the Sun, where the terrestrial planets accreted, the planetesimals were similarly differentiated. In these small bodies, the pressures during separation of the elements would be fairly low. The Earth's metallic core would segregate at the time of accretion, with perhaps only limited opportunity for re-equilibration of metal and silicate under high pressures.

Nonetheless, if the Earth was subsequently remelted by a giant Moon-forming impact, or if the silicate mantle remained molten as a consequence of accretion, then the crystallization of the deep silicate mantle would have occurred under high pressure and the results of Keppler and Rubie will apply. With so dramatic a change to our views on the distribution of the siderophile elements in the mantle, it will be back to the drawing board for the high-pressure geochemists, as 30 years of study have not cast much light on the paradox of elemental abundances in the upper mantle.

The most popular and perhaps the most reasonable notion, that the signature represents the addition of a 'late veneer' of chondritic material is not without its own difficulties, of which the most telling is the absence of a similar veneer on the Moon. Given the present complexity of the mantle, our inadequate sampling and the possible effects of a giant impact, others must follow the lead given by Keppler and Rubie if we are to solve the paradox. □

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IMMUNOLOGY

MHC class II dimer of dimers

Hidde Ploegh and Philippe Benaroch

SIX years have passed since publication[1] of the three-dimensional structure of a class I molecule of the major histocompatibility complex (MHC). On page 33 of this issue[2] we are now treated to a similar feat, accomplished for a human class II MHC molecule, HLA-DR1 — and by the same laboratories no less.

Unlike immunoglobulins, antigen specific receptors on T lymphocytes (TCR) do not interact with intact, foreign proteins, but recognize short (8–25-residue) peptides derived from them, held tightly in the jaws of MHC molecules. MHC proteins come in two basic types, class I and class II, each of which subserves a special type of T cell. Class I products interact with CD8 (largely cytotoxic) T cells, whereas class II molecules interact with CD4 (predominantly helper) T cells. What is good for the goose is good for the gander, for it turns out that the structures of both types of MHC product are remarkably similar (a strong case for which, incidentally, was made some years ago[3]). From similarities in primary structure of class I and class II MHC products, Brown *et al.*[4] had already proposed a model for the class II MHC binding site that has stood workers in the field in good stead: experiments based on the model produced results entirely consistent with it. Its vindication now comes from the structure before us.

Buried in the fine print are references to a struggle lasting almost ten years to obtain enough material in pure enough form[5] to proceed with attempts at crystallization[6]. But that persistence has paid off: no fewer than three different crystal forms — with the added bonus of a non-crystallographic dimer — could be used to solve the structure of HLA-DR1. At first glance, a class II molecule is easily mistaken for its class I counterpart. But just as the class I structure drove a stake through the heart of any remaining dual-recognitionist demon by unveiling its peptide cargo, so the class II molecule reveals new secrets.

First is the fundamental difference between class I and class II molecules in peptide–MHC interactions. The class I peptide-binding pocket is blocked at either end and appears to impose severe restrictions on the sizes of peptides it can accommodate (8–10 residues), with longer peptides bulging out in the middle[7]. Not so for class II. The class II binding groove allows peptide to protrude from it and, consequently, longer (average 15–18-residue) peptides[8] can bind: there is no need for bulges here.

Studies of self-peptides eluted from purified HLA-DR alleles have directly established this size heterogeneity, as well as the occurrence of nested sets of peptides derived from a single protein[8,9]. Such peptides are thought to bind through a central conserved core with 'ragged' ends. Many of the contacts of DR1 with its bound peptide — in the same amino- to carboxy-terminal orientation as in class I — involve DR1 side-chains and main-chain atoms of the peptide. Only a single 'pocket', capable of accommodating a rather large peptide side chain, is observed. Other pockets are indicated by patches of polymorphic residues in the binding site. In addition, promiscuous peptides, capable of binding to many different human class II alleles, have been identified[8,10], with no obvious parallel in class I products.