new work of Maryanski *et al.*[3] shows that when mice are immunized against syngeneic tumour cells that have been transfected with the fully functional gene for a human class I protein, cytotoxic T lymphocytes are generated that recognize a fragment of the human antigen in association with self class I molecules. The site on the human protein recognized by some class I-restricted T cells is defined by a stretch of 16 amino acids. The complete human class I antigen is simultaneously expressed at the surface, but this native form seems irrelevant for restricted recognition. The work of Townsend *et al.*[2], although not defining a peptide sequence of influenza haemagglutinin, shows that whether leader-sequence intact haemagglutinin is produced and expressed as an integral cell membrane component or whether leader-sequence deleted haemagglutinin is made in the cytosol and rapidly degraded, the same class I/ haemagglutinin determinant is displayed at the surface for recognition. This means that the form of haemagglutinin recognized is denatured and implies strongly that it is a degraded form. The important message from this and other work is that irrespective of whether proteins are normally made in the cytosol or on the endoplasmic reticulum, they all provide degradation products that can associate on the cell surface with class I MHC molecules for the perusal of cytotoxic T lymphocytes.

This pathway of degradation of endogenously synthesized proteins leading to class I presentation seems to be different from that for the degradation of soluble exogenous proteins taken into the cell by endosomes and leading to class II presentation. For example, the latter pathway is sensitive to the drug chloroquine, whereas the former is resistant, and peptides derived from endogenously synthesized haemagglutinin might not interact with the class II pathway[6].

Now that we are confronted with the fact that degradation products of endogenously produced proteins (whether originally destined for the nucleus, cytosol or membranes) can be presented on the cell surface with native class I molecules, we can ask whether class I presentation ever involves protein antigens that come from the exterior of the cell. This is central to understanding the induction phase of a class I-restricted cytotoxic T-lymphocyte response to certain types of viral infections. If the only cell capable of presenting antigen to class I-restricted T cells is the infected cell itself, then sensitization would have to occur peripherally in the case of a virus that did not productively infect cells in the lymphoid organs. This is at odds with an old dogma that immune sensitization occurs centrally, in draining lymph nodes or in the spleen. There is, however, evidence in the transplantation literature suggesting that antigens can be picked up from the periphery and presented in the central lymph nodes. For example, female mice of a given H-2 type can be primed to reject a syngeneic male graft by cells from a male of a different H-2 type — presumably because the male-specific antigen is picked up and presented on host cells. In most cases, however, one does not know if the rejection mechanism is dependent on class I or class II MHC presentation. But from *in vivo* systems, there is clear evidence for such class I antigen presentation. Class I cytotoxic T lymphocytes can be primed to foreign minor antigens or to simian virus 40 T antigen that have been introduced on MHC-different cells[7,8]. Furthermore, minor antigens may be picked up and presented by cells to tolerize class I-restricted T cells[9]. In these cases, the foreign antigens must have been taken up by host cells and presented in association with their self class I molecules.

How can one explain the class I presentation of these exogenously derived cellular antigens? One way is to propose that the cell synthesizing the foreign antigen is also producing and leaking processed peptides that may bind class I molecules on cells of the lymphoid system. That this is feasible is demonstrated by the finding that synthetic peptides can be added externally to cells *in vitro*, thereby creating the cytotoxic T-lymphocyte target antigen[3,4]. But it does not seem to be a likely occurrence *in vivo* where peptides are being flushed from the system, nor does it offer a basis for selecting for class I over class II presentation. Another more plausible way to take cellular antigens that are exogenous and to present them as endogenous, class I-associated antigens is via specialized antigen-presenting cells that phagocytose large cellular debris and shuttle the resulting peptide degradation products to their endogenous class I presenting system. Such a phagocytic cell may or may not express class II molecules.

The important distinction from the class II presentation systems here is that the exogenous antigens come as cell debris and not as soluble material or small particles which can be pinocytosed. Such a

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Wrinkle ridges on the Moon and the Earth

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STUDY of the surfaces of Mars, Mercury and the Moon has revealed the existence of wrinkle ridges, complex geomorphic structures variously proposed to have volcanic, tectonic or combined volcanic/tectonic origins. Generally they consist of long linear ridges, with heights measured in metres, that may stretch for hundreds of kilometres. Such a lunar wrinkle ridge is shown (left) in a photograph of the southwestern part of the Mare Imbrium taken from Apollo 15; the field of view (about 370 km across) gives some idea of the scale of these features. Analysis of the ridges to determine their origins has been hampered by the absence of terrestrial data from which to extrapolate. J.B. Plescia and M.P. Golombek have recently investigated structures of Earth that they believe are the analogues of the planetary ridges; their report can be found in *Bulletin of the Geological Society of America* 97, 1289–1299 (1986). A typical terrestrial ridge of total length 37 km is shown (right), which formed after an earthquake at Meckering, Western Australia; the road and vehicles allow scaling. From this and other examples from many widespread localities, the authors argue that the ridges on Earth are the terrestrial equivalents of planetary wrinkle ridges. The major discrepancy is one of scale, in that the examples on Earth are considerably shorter; this is explained in terms of the relatively heterogenous terrestrial stress systems, which effectively limit fault development. Planetary wrinkle ridges, it is suggested, developed following deformation associated with thrust faulting close to the planet surface. □