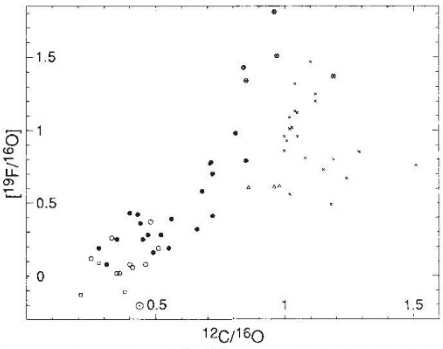

Logarithm of the $^{19}F/^{16}O$ ratio relative to the mean value in K and M stars plotted as a function of the $^{12}C/^{16}O$ ratio. The symbols are for stars of different spectral types. Open squares are K stars, closed squares are Ba stars, open circles are M stars, closed circles are S stars, circled crosses are SC stars, crosses are N stars, and open triangles are J stars. The trend of increased fluorine abundance with increasing carbon abundance and spectral type is strong evidence that fluorine is produced in helium-shell flashes within these stars.

consistent with other considerations[4–6].

These data also suggest a problem for competing schemes for the formation of fluorine in stars. For example, it has been proposed by Woosley *et al.*[7] that fluorine could be produced by neutrino-induced nucleon emission from ^{20}Ne during a type II supernova explosion. If that were true then the fluorine abundance should be correlated with other elements produced in supernovae (like oxygen)[5,6]. One star in the sample was depleted in oxygen by roughly a factor of two. If fluorine were produced in supernovae, the fluorine to oxygen ratio should have been about the same as the Solar System value. Instead, the ratio is less, suggesting[5,6] that fluorine was produced more slowly than oxygen. This points to the slowly evolving low-mass AGB stars as the source for fluorine rather than the rapidly evolving massive stars responsible for type II supernovae.

So, if anyone ever asks where the fluorine for your fluoride toothpaste comes from, you can answer that fluorine is probably formed in the frequent furious fusion flashes of an inflated florid star, and on the new evidence you would probably be right. □

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1. Jorissen, A., Smith, V. V. & Lambert, D. L. *Astr. Astrophys.* **261**, 164–187 (1992).
2. Iben, I. & Renzini, A. *A. Rev. Astr. Astrophys.* **21**, 271–342 (1983).
3. Schwarzschild, M. & Härm, R. *Astrophys. J.* **142**, 855–867 (1965).
4. Käppler, F., Beer, H. & Wisshak, K. *Rep. Prog. Phys.* **52**, 945–1013 (1989).
5. Mathews, G. J. & Cowan, J. J. *Nature* **345**, 491–494 (1990).
6. Mathews, G. J., Bazan, G. & Cowan, J. J. *Astrophys. J.* **391**, 719–735 (1990).
7. Woosley, S. E., Hartmann, D. H., Hoffman, R. D. & Haxton, W. C. *Astrophys. J.* **356**, 272–301 (1990).

SUPERANTIGENS

Playing upon both sides

Ursula Esser and Peter Parham

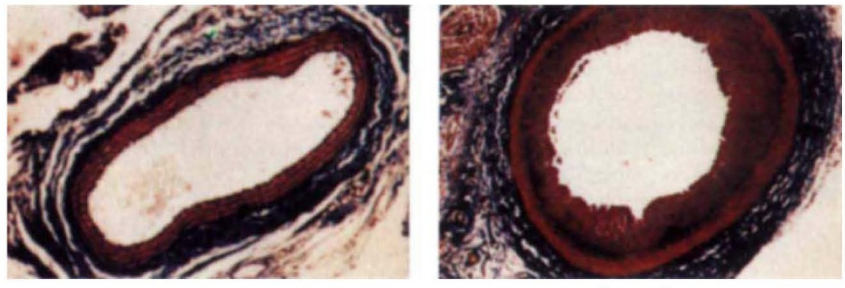
In the wars between host and pathogen, whose side are superantigens on? In the case of the superantigens of mouse mammary tumour retroviruses, the answer seems to be 'both'. The picture emerging from recent reports[1,2] is not only of how superantigens facilitate virus infectivity, replication and survival, but also of how they may be kidnapped and exploited by the host. More broadly, we can see a meeting of minds on superantigens, for the topic excited considerable debate at a conference dealing with the interplay of microbes and their hosts, held a month ago in Montana. After many years of going their own ways, microbiologists and immunologists are taking an interest in each other's wares.

Superantigens bind to class II major histocompatibility complex (MHC) molecules stimulating families of T cells bearing particular Vβ chains. These potent proteins can derive from bacteria or viruses, and they differ from ordinary antigens by the greater numbers of T cells they trigger, their lack of processing into short peptides and the different sites of interaction with T-cell receptors and MHC molecules[3]. In humans, staphylococcal enterotoxins are bacterial superantigens active in food poisoning and toxic shock syndrome, while on the viral front, rabies[4] and HIV[5,6] are getting in on the act. But, at the moment, it is on mouse mammary tumour viruses (MMTVs)[7,8] that the spotlight has fallen.

The MMTVs and their superantigens come in different guises; the exogenous viruses are inherited as infectious particles transmitted through mother's milk, whereas the endogenous viruses exist as proviruses integrated into the host genome and are passed on through its replication[7,8]. T cells with antigen receptors using complementary Vβ chains are stimulated by a superantigen, and the consequence of such encounters depends upon the developmental stage at which they occur. 'Natural' exposure to bacterial superantigens often involves mature T cells and results in a strong polyclonal immune response[3]. In contrast, the superantigens of endogenous and exogenous MMTVs are encountered early in life and produce deletion or inactivation of the complementary T-cell families. In this manner the roster of MMTV carried by strains of mice — and there are many differences between them — shapes their T-cell repertoires, each virus determining the fate of part of the total T-cell pool.

Patterns of Vβ deletion can be manipulated experimentally by making mice transgenic for MMTVs. An extension of this approach has shown that the pre-

Anti-gene therapy?



Antisense Control

ANTISENSE DNA has been used successfully to study the function of genes in intact cells, but its use as a therapy has been stymied by difficulties in delivery to the appropriate cells or organs — until now. On page 67 of this issue, M. Simons *et al.* describe how they have delivered antisense oligonucleotides to injured carotid arteries in rats. Injury such as that caused by balloon angioplasty, a therapy commonly used to 'widen' arteries in people, causes proliferation of the cells of the inner vessel wall, as is apparent in the right panel, with dangerous consequences. By painting an antisense oligodeoxynucleotide directed against the c-*myb* proto-oncogene onto the stripped and exposed artery wall in the injured region in a hardening, non-toxic gel, Simons *et al.* prevented the proliferation of cells in the vessel wall (left panel). After entering the cells, the antisense oligonucleotide is presumed to hybridize specifically to the messenger RNA encoding c-*myb*, thereby preventing its translation into protein. This approach therefore provides both a new route to cardiovascular therapy and a means to studying gene function in intact animals. T. L. B.