There are clinical correlations between low NK-cell activity and severe CMV infection[8], and there are substantial experimental data to indicate that murine CMV is controlled by NK cells[9]. By what mechanism could NK-cell-mediated regulation of CMV infection occur? The answer may lie in the attempts of CMV to escape the cytotoxic T lymphocytes by interfering with the class I pathway for antigen presentation. By removing the 'protective' class I inhibitory structures from the cell surface, the viruses may render the infected cells more sensitive to NK cells. However, both human[10] and murine[11] CMV contain a gene that resembles MHC class I genes. The protein products are mainly similar in the extracellular domains, two of which form the groove that accommodates peptide, and the human CMV class I homologue has been shown to engage and present peptides[12]. And now, Farrell *et al.*[4] and Reyburn *et al.*[5] show that both human and murine viruses use the class I homologues to escape surveillance by NK cells.

Farrell *et al.* constructed a recombinant murine CMV with a functional deletion (designated m144) in its class I homologue. The deletion did not affect replication of murine CMV in fibroblasts *in vitro*, but when mice were infected with the recombinant virus, it was severely attenuated in the spleen and liver. Depletion of NK cells in these mice led to increased synthesis of the recombinant virus, indicating that expression of the class I homologue rendered the virus (at least partially) resistant to NK cells.

The exact mechanism remains to be investigated, but the paper by Reyburn *et al.* sheds some light here. When the human CMV class I homologue (designated *UL18*) was cloned and expressed in a human cell line that lacked class I MHC, the cells became resistant to NK-cell-mediated lysis. Moreover, *UL18* conferred resistance to all NK cells, not just to certain NK clones. This is because the UL18 protein seems to interact with CD94, a component of a heterodimeric inhibitory receptor of the C-type lectin family, which is found on most NK cells and many T cells[13]. So by encoding its own class I homologue, the virus may provide itself with a mechanism to veto NK-cell attack, while using other genes to render the infected cells invisible to T-cell attack.

How does this fit in with what we know about the sensitivity of CMV to NK cells? Fibroblasts that are infected with human CMV *in vitro* are (in contrast to murine CMV-infected cells) quite sensitive to NK-cell-mediated lysis, and various NK-cell clones differ in their ability to lyse infected fibroblasts[14] — seemingly discordant with the results of Reyburn *et al.*[5]. However, expression of UL18 may be tissue specific, and replication in virally infected cells has yet to be detected. So the experiments on the lysis of human CMV-infected cells may have been examining an infection in which the inhibitory UL18 protein was not expressed[14]. It would be interesting to see whether cells expressing UL18 might be rendered sensitive to NK cells after infection with human CMV, as they could be displaying both negative and positive signalling structures.

Does murine CMV escape detection by the immune system in a similar manner? NK-cell-mediated control of infection is normal in mice with impaired MHC class I expression due to disruption of the β_2-microglobulin gene[15], indicating that host-cell class I MHC recognition may not be involved in the NK-cell response to murine CMV. But this point becomes moot if the virus expresses its own class I homologue. Expression of inhibitory receptors on the NK cells, which recognize the class I molecules, is augmented in the β_2-microglobulin-deficient mice[16]. Notably, certain mouse strains can strongly resist infection by murine CMV, with the help of a gene termed *Cmv1*, which maps within (or close to) the gene family that encodes the NK-cell inhibitory receptors for class I molecules[17].

Future studies will probably search for the expression and function of the class I homologue in infected cells *in vivo*. An intriguing question concerns its ability to bind peptides[12]. Is there no risk for presentation of viral antigens? Or is this yet another vicious trick to mislead the immune system? Moreover, could host cells evolve countermeasures to lower the expression or sensitivity of the inhibitory receptors so that NK cells cannot 'hear' the veto signal[18]? More details are needed before we can conclude whether, and how, CMV behaves like an invisible, but noisy, baby-turkey imitator, and whether the tactic is absolutely safe. Perhaps Dawkins has a clue when he sums up by citing another observation from Schliedt of "a turkey mother that savagely killed all her babies. The reason was woefully simple: she was deaf". □

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Daedalus

Water, water everywhere

All our water comes from the air, usually by way of rain. Daedalus now wants to extract water vapour from the air directly. He points out that the vapour pressure of a solution declines in strict proportion to the concentration of dissolved molecules at its surface — a fact exploited in the determination of molecular weights. If the vapour pressure of an aqueous solution falls below that of the water in the air, water vapour will condense into the liquid (which is why you should not leave the lid off a jar of syrup or condensed milk).

These products, however, are not ideal water-traps. Solutions of surfactants should be better. Their molecules cluster preferentially at the water surface, where the concentration can be hundreds of times higher than in the bulk. So quite dilute solutions should have very low vapour pressures. Indeed, one very surface-active substance, cetyl alcohol, has been added to reservoirs, ostensibly to hinder evaporation. But Daedalus reckons it works by the converse mechanism. It doesn't stop evaporation; it encourages condensation.

Aided by this new insight, DREADCO chemists are seeking better molecules for the job. They are measuring the solution vapour pressures of powerful fluorinated surfactants and non-ionic detergents, seeking the largest possible pressure drops. Their goal is an additive for reservoirs and ponds which will concentrate at the surface strongly enough to keep vapour pressure of the pond well below that of the air. The pond will then absorb humidity all the time, even without rain. Sadly, some surfactants can sink ducks and other waterfowl by wetting their feathers. A silicone duck-grooming treatment may be needed.

Water from such a pond will, of course, contain traces of surfactant. To extract and recycle it, the team is developing a large-scale froth-blower. Air is bubbled through the detergent-contaminated water; it foams up, and its surface-active molecules concentrate in the vast surface area of froth. The froth is blown off, and collapsed by heating; the resulting strong detergent solution is returned to the reservoir.

Britain's unreliable water companies will rush to install the system. But their troubles will not be over. Why bother with piped water, if you can extract your own from a small pond or tank fitted with a DREADCO detergent system? Sprawling reservoirs and vast grids of leaky pipes will no longer be needed. Only heavy industry, with its concentrated usage, will buy piped water at all.

David Jones