

at this current level was limited to a few minutes by the attainable temperature rise. Although this output is too small for most applications, the authors outline plans to increase the yield to a million neutrons per second, comparable to that of some commercial portable neutron generators. Nevertheless, even at the level already attained, there are laboratory uses, such as measuring neutron detector response or for student practical demonstrations, for which a simple,

inexpensive, monoenergetic neutron source would be most valuable. ■

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HIV

Viral blitzkrieg

R. Paul Johnson and Amitinder Kaur

It takes years for AIDS to develop from the damage inflicted on the immune system by HIV or its simian counterpart. Surprisingly, as many as half of the body's memory T cells may die at a very early stage of infection.

HIV and the related simian immunodeficiency virus (SIV) cause AIDS by infecting the master regulatory cells of the immune system — T helper cells, better known as CD4⁺ T cells. It is generally years before enough damage is done to this cellular army for full-blown AIDS to develop. Nevertheless, two reports in this issue^{1,2} (pages 1093 and 1148) suggest that the outcome of the battle between SIV and its host may be determined by a dramatic opening salvo, in which the virus eliminates around half of the host's memory CD4⁺ T cells within four days — thus setting the stage for a lengthy war of attrition.

CD4⁺ T lymphocytes are so called because they express a receptor protein termed CD4; this is necessary for T-cell function, but has also been co-opted by HIV and SIV to gain entry to the cells. Previous results suggested that HIV/SIV replication is restricted

to a relatively small fraction (0.01–1%) of CD4⁺ T cells in the chronic stages of infection³. This low frequency of infection seemed to reflect the fact that the viruses require the presence of a co-receptor in addition to CD4 to gain entry, and also that they replicate best in activated memory CD4⁺ T cells (memory cells being those previously stimulated by foreign antigen). The preferred co-receptor for most HIV and SIV strains is CCR5, which is expressed only in a subset of memory CD4⁺ T cells.

Despite the apparent low frequency of T-cell infection in chronic infection, seminal studies in SIV-infected monkeys⁴ — subsequently confirmed in HIV-infected humans^{5,6} — revealed a rapid and widespread depletion of CD4⁺ T cells in the gut (mucosal T cells) during the first few weeks of infection. T cells in the blood or lymph nodes did not show the same degree of

depletion. This predilection of HIV and SIV for replicating in gut lymphocytes was felt to be a consequence of the relatively large populations of activated CD4⁺ T cells at this site that express CCR5. However, the proportion of cells actually infected with SIV was not known; nor was it clear whether T-cell activation was in fact required for infection, or how T cells were killed.

Mattapallil *et al.*¹ have now examined the role of SIV in the depletion of CD4⁺ T cells during early (acute) stages of infection. As previously reported⁴, SIV rapidly depleted CD4⁺ T cells in the gut. Remarkably, however, 60–80% of memory CD4⁺ T cells were concurrently depleted at all sites. Using a technique that can detect a single copy of SIV DNA, the authors determined that 30–60% of all memory CD4⁺ T lymphocytes were infected with SIV within 10 days of infection, regardless of their location, and that most of these cells had disappeared 4 days later. These percentages far exceed the number of CD4⁺ T cells that express CCR5, but the authors propose that this apparent contradiction may be resolved by their finding of low levels of CCR5-encoding messenger RNA in memory cells in which CCR5 protein could not be detected by flow cytometry. This implies that such cells may in fact express sufficient levels of CCR5 protein to render them permissive for SIV infection. Alternatively, SIV may be entering the cells using other co-receptors.

Li and colleagues' paper² provides a complementary perspective on this viral blitzkrieg. By identifying cells expressing SIV RNA in tissue sections, these investigators characterized the activation state of virus-producing cells (viral production requires SIV DNA to be transcribed into RNA). Consistent with their earlier work⁷, they found that most infected cells did not express markers of activation (CD25 or CD69), nor did they express Ki67 — a molecule found in

Behavioural ecology

Cue for kin

If you yourself can't breed, you can at least help your relatives with their offspring. Such altruistic behaviour occurs in long-tailed tits (*Aegithalos caudatus*, pictured), which Stuart Sharp and colleagues have studied to find out what cues enable a 'helper' to recognize kin. Their report appears elsewhere in this issue (*Nature* **434**, 1127–1130; 2005).

Adult long-tailed tits pair off and attempt to breed each year, but many don't succeed because of high rates of predation on the eggs or nestlings. The childless parents may then turn to assisting kin in feeding their brood — which makes sense in evolutionary terms but requires some

form of recognition system. Long-tailed tits are not the greatest of vocalists. They sing infrequently but do have an individually characteristic contact call, the 'churr', which develops even before fledging and is retained in the adult bird.

The first part of Sharp and colleagues' research involved the playback to individuals of churr calls belonging to a close relative and a non-relative, and a further two trials in which the frequency of these calls had been tweaked. The responses of birds to the untweaked calls of relatives differed significantly from their responses to the other three calls. From this, the authors

conclude that the churr call provides cues involved in kin recognition.

The most innovative part of the study, however, was an investigation into how much churr acquisition owes to nurture (learning) and nature (genetics). This took the form of swapping young birds between nests, so that adult birds were raising foster nestlings along with their true offspring. The churr calls of the fostered birds were the same as those of their nestmates, and unlike those of their biological siblings raised elsewhere — so it seems that the churr is in large part learned.

The pattern of helping observed in long-tailed tits is consistent with



the use of this learned cue in the great majority of cases. But this kin-recognition system evidently isn't flawless: in 6% of cases, an adult helped unrelated nestlings. As the authors point out, recognition systems are rarely perfect. **Tim Lincoln**

ANDREW MACCOLL