Consider a predator–prey relationship described by the following model:

$$\frac{dv}{dt} = vr(1-v/K) - \alpha vp$$
$$\frac{dp}{dt} = \alpha\beta vp - \mu p \quad (1)$$

v and p are the densities of the prey and predator, respectively, K is the carrying capacity of the prey, μ is the death rate of the predator, r is the intrinsic growth rate of the prey and α and β are parameters controlling the capture of prey and their conversion into predator biomass. A common objection on this sort of model is that it is too simple to be of any use in the 'real world'; but in fact, the model consists mostly of irrelevant detail from the point of view of calculating an eradication threshold.

The eradication threshold of the predator is determined by the minimum prey carrying capacity, K, that can sustain a predator population (the amount of habitat that would support K prey in the absence of predation), which is straightforward to calculate. Setting the time derivatives in equations (1) equal to zero and solving for v^* and p^*, the equilibrium abundances of prey and predator,

$$v^* = \mu/\alpha\beta$$
$$p^* = \frac{r}{\alpha}(1-\mu/\alpha\beta K). \quad (2)$$

The eradication threshold is that value of K such that $p^*=0$, that is, $K=\mu/\alpha\beta=v^*$, the equilibrium abundance of the prey. So the eradication threshold is simply the unused amount of the limiting resource.

To reveal what accounts for this result, I shall rederive the estimate for the predator–prey case as derived in ref. 1 in the epidemiological context. The derivation in ref. 1 consists solely of a verbal argument which can be applied to many biological models with an obvious change of wording. Equilibrium in the system is achieved when the density of prey is reduced to that level at which a predator gives rise to a single progeny before dying. This is the equilibrium density v^*. In the absence of predation, the density of the prey would be K. So, speaking loosely, an amount $K-v^*$ prey is 'used up' in keeping the predator population alive. Biologically, this cannot be a negative number. Hence, v^* immediately presents itself as the minimum value of K required to sustain a predator population. None of the species – specific details of predator hunting efficiency, death rates, efficiency of conversion of prey biomass into predator biomass, and so on, enters into the estimate of the eradication threshold. As is demonstrably the case in epidemiological contexts, details that at first glance seem important in fact cancel each other out (for review of theory and data, see ref. 1). In addition to showing what is not important, this simple approach also makes it easier to see what features are important for estimating eradication thresholds[1,5].

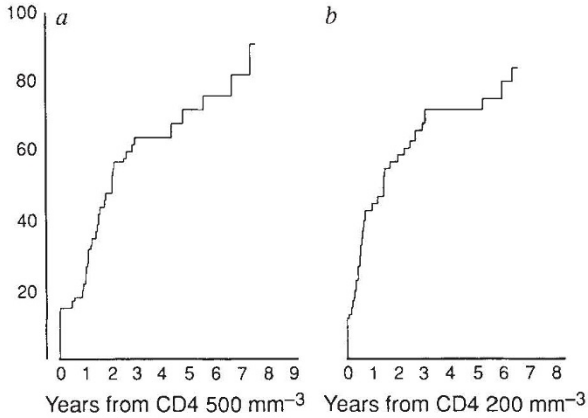
It would be trite to observe that epidemiology and conservation biology are both motivated by the same concern — the eradication of species — were it not for the fact that there are many areas of theoretical overlap between the two disciplines. Some other areas are described in ref. 6.

Sean Nee
AFRC Unit of Ecology and Behaviour
Department of Zoology,
South Parks Rd,
Oxford OX1 3PS, UK

1. Anderson, R. M. & May, R. M. *Infectious Diseases of Humans* (Oxford University Press, 1991).
2. Lande, R. *Science* **241**, 1455–1460 (1988).
3. Lande, R. *Am. Nat.* **130**, 624–635 (1987).
4. Lande, R. *Oecologia* **75**, 601–607 (1988).
5. Lawson, J. H. Nee, S., Letcher, A. & Harvey, P. H. in *Large Scale Ecology and Conservation* (eds Edwards, P. J., May, R. M. & Webb, N.) (Blackwell, Oxford, in the press).
6. Nee, S., Holmes, E. C., May, R. M. & Harvey, P. H. *Phil. Trans. R. Soc.* B (in the press).

Viral burden in HIV infection

SIR — McLean and Michie[1] and Garry and Fermin[2] argue in Scientific Correspondence that the direct cytopathic effects of the virus could well be the main cause of CD4 lymphocyte count decline during HIV infection. If such is the case, however, why does the rate of CD4 lymphocyte loss not increase concomitantly with the enormous increase in viral burden observed during the course of HIV infection? The viral burden late in HIV infection (for example when the CD4 lymphocyte count is around 200 mm^{-3}) has consistently been found to be, on average, 10–100 times greater than it is earlier (for example when the CD4 lymphocyte count is around 500 mm^{-3}). This is the case whether virus levels are measured in plasma or in cells (or, based on the little evidence available, lymphoid tissue[3]) and whether using quantitative PCR (polymerase chain reaction) or endpoint dilution culture[4,5]. In sharp contrast, the rate of CD4 lymphocyte loss tends, on average, to be no more rapid late in the infection that it is earlier (see figure). This inconsistency cannot apparently be explained by differences in viral pathogenicity. The *in vitro* properties of virus isolated late in infection suggest that it is unlikely to be less able to kill CD4 cells *in vivo* than virus present earlier in the infection[6].



Kaplan Meier estimates of the percent of haemophilic individuals having a CD4 lymphocyte count below 250 mm^{-3} (*a*) or 100 mm^{-3} (*b*) according to the number of years after the count has first fallen below 500 mm^{-3} or 200 mm^{-3} respectively. The time taken to decline from a count of 500 mm^{-3} to a count of 250 mm^{-3} (a fall of 250 mm^{-3}) is similar to the time taken to decline from a count of 200 mm^{-3} to a count of 100 mm^{-3} (a fall of 100 mm^{-3}), thus illustrating that the rate of CD4 lymphocyte count decline is, if anything, less rapid late in HIV infection than earlier. Study methods have been described previously[8].

As well as having fundamental implications for understanding HIV pathogenesis, the weakness of the association between viral load and the rate of peripheral blood CD4 lymphocyte count decline gives some indication of the potential effect of antiviral drugs. Therapies that induce substantial (about 10–100-fold) reductions in viral load might be expected to produce only small reductions in the rate of CD4 lymphocyte decline. Consistent with this prediction are data from placebo-controlled clinical trials of Zidovudine (AZT), which show an initial increase in the absolute number of CD4 cells following therapy but similar rates of decline in the Zidovudine and placebo arms thereafter[7].

Andrew N. Phillips, Caroline A. Sabin, Jonathan Elford, Margarita Bofill*, Vince Emery†, Paul D. Griffiths†, George Janossy* & Christine A. Lee‡
Departments of Public Health, Immunology, Virology† and the Haemophilia Centre‡,*
Royal Free Hospital School of Medicine, London NW3 2PF, UK

1. McLean, A. & Michie, C. *Nature* **365**, 301 (1993).
2. Garry, R. F. & Fermin, C. D. *Nature* **365**, 301–302 (1993).
3. Pantaleo, G. *et al. Nature* **362**, 355–358 (1993).
4. Piatak, M. *et al. Science* **259**, 1749–1754 (1993).
5. Bieniasz, P. D. *et al. AIDS* **7**, 803–806 (1993).
6. Asjo, B. *et al. Lancet* **ii** 660–662 (1986).
7. Cooper, D. A. *et al. N. Engl. J. Med.* **329**, 297–303 (1993).
8. Phillips, A. N. *et al. Lancet* **337**, 389–392 (1991).