Control of feline leukaemia virus

HARDY *et al.*[1] report the success of a programme for preventing spread of feline leukaemia virus (FeLV) among pet cats by screening for FeLV viraemic individuals, removal of carriers and quarantine of contacts. Although evidence that the programme was of some success seems strong, the study's methodology deserves comment. Major problems concern the comparisons between 'treated' and 'untreated' groups.

(1) The groups were 'self-selected' by owners. Though it is probable that pet owners' responses to such a programme reflect different approaches to the care of pets, we are given no information on the cluster-size distribution, location, management, sex or age structures of the groups—though each such variable has major implications for the spread of an infectious agent.

(2) The only information given as to the previous status of the groups is that the treated group had a lower initial prevalence rate of FeLV carriers than did the untreated control (0.224–0.312). This indicates that the treated group was at a lower risk of infection even before the programme and thus it is improper to attribute subsequent differences in incidence rates to the programme alone.

(3) The report of a 0.193 incidence rate in the untreated group over three months is surprising, as it implies an annual incidence rate of $1.0-(1.0-0.193)^4$ or 0.576, which is far higher than the prevalence rate in either group—though the authors claim that most viraemic cats remain viraemic for life, they report a low natural mortality rate for viraemic individuals and they make no reference to reversions to virus negative among the untreated group cats.

(4) The programme entailed both removal of viraemic individuals and also quarantine of contacts. As neither action was imposed upon the untreated group it is improper to attribute the lowered incidence rate to removal of FeLV carriers alone.

(5) Mortality 'rates' are given without reference to time period or to background mortality among 'uninfected' cats. Without such references they are uninterpretable.

(6) The authors' conclusion that infection in most cats occurred between 18 and 24 months of age does not follow logically from their data on median ages—and, if it did, it would emphasise the necessity to adjust for age when comparing incidence and mortality rates. No such adjustments have been made.

The implications of Hardy *et al.*'s study are important. Though some of the problems mentioned here may have arisen due to the demands of brevity in publication, it should be stressed that such studies deserve a careful methodology. This is especially true in the context of current interest in the epidemiological implications of oncornaviruses.

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1 Hardy, W.D. *et al.* Nature **263**, 326–328 (1976).

HARDY AND MCCLELLAND REPLY—We agree with Dr Fine that a 42-fold decrease in feline leukaemia virus infection (FeLV) in the households which implemented our FeLV test and removal programme is evidence that our programme was successful[1]. Our study was not a strict epidemiological survey, indeed, an epidemiological study of lymphosarcoma in cats failed to detect the contagious nature of the disease[2]. But, we would like to answer Dr Fine point by point.

Matters Arising

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(1) All the owners were given the same advice regardless of whether or not they chose to have their infected cats removed. The management of the households was the same for both groups and the average number of cats per household (Fine definition of cluster) in those households in which the cats were removed was 16.6 cats per household while in the other group it was 16.5 cats per household (Table 2, ref. 1). Households in both groups were located throughout the USA. The two groups are thus quite comparable.

(2) Although it is true that the initial FeLV infection rate of the two groups was different, the difference in the rates of FeLV infection between the two household groups after our programme had been implemented was much greater and was statistically highly significant ($P<0.001$).

(3) Dr Fine should not assume that the FeLV infection rate of 19.3% in the first 3 months will be perpetuated. The infection rate decreases because, as we reported, approximately 41% of the exposed cats in a household develop protective FeLV neutralising antibody titres though they never test positive for FeLV. We reported previously that 33% of healthy exposed cats became FeLV infected[3]. This figure was the result of a single sampling of many households in which the cats had been exposed to FeLV for varying periods of time. In some households, 100% of the cats were infected, while in others only 10% were infected, which shows that the FeLV infection rate can vary greatly. In contrast to Dr Fine we do not think that our observed mortality rate of 52% for secondarily infected cats and 8.5% for first test FeLV uninfected cats is low. With regard to his other points in this area; most cats do remain viraemic for life and reversions happened only rarely among the cats in this study.

(4) Both groups of households were quarantined as referred to in refs 13 and 16.

(5) The observation period for the study was 2 y, as mentioned in the paper[1].

(6) The median age of cats which became infected with FeLV was simply our observation on 49 cats—we did not make any conclusions about age susceptibility of pet cats in the general population.

In conclusion, we would like to add that removal of the carrier host is a common method for controlling and preventing the spread of many infectious veterinary diseases when vaccination is not available. For instance, bovine tuberculosis, brucellosis, foot and mouth disease, hog cholera, African swine fever, vesicular exanthema, rinderpest and glanders are controlled by test and removal programmes. Thus, the concept of preventing the spread of the contagious feline leukaemia virus by a similar programme should not, we think, be difficult to accept.

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1 Hardy, W. D. *et al. Nature* **263**, 326–328 (1976).
2 Schneider, R. *Int. J. Cancer* **10**, 338–344 (1972).
3 Hardy, W. D., Jr, Old, L. J., Hess, P. W., Essex, M. & Cotter, S. M. *Nature* **244**, 266–269 (1973).