pattern, with t and t_1 again representing apostatic relations (Case 2). In this case there is a non trivial stable equilibrium at $\hat{q} = \sqrt{[(t-w)/(t-w+t_1)]}$ so that when $t > w$ the tendency of the conspicuous warning pattern to increase will be held in check by apostatic selection. Though this example probably underestimates the complexity of the predator–prey relations in question it does demonstrate that there is no *a priori* reason to reject the hypothesis that *marginella* is a warning pattern maintained in polymorphic condition with more cryptic forms by apostatic selection.

Dr Hebert's counter proposal is difficult to accept. The *marginella* pattern of black on white is too sharp and the black on most specimens too unbroken to reasonably suggest a resemblance to bird excreta. But, as Hutchinson[2] has noted, the colour form *trilineata* may vaguely resemble grass seeds and I do not rule out protective resemblance as a factor in *Philaenus spumarius* polymorphism.

I thank Dr L. Van Valen for comments.

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[1] Clarke, B., and O'Donald, P., *Heredity*, **19**, 201 (1964).
[2] Hutchinson, G. E., *Entomologist's mon. Mag.*, **99**, 175 (1963).

Genetic control of natural resistance to *Leishmania donovani*

SIR,—In studies of infections in mice with *Leishmania donovani*, an intracellular parasite, (in preparation) several observations are relevant to the recent letter[1] on mouse susceptibility to *Salmonella* showing that seven mouse strains fell into two resistance categories.

Colleagues and I have found that the growth rate of *L. donovani* (Ethiopian strain L82) populations in mice varied greatly and examined the acute phase, the first 2 weeks after infection, in detail. Amastigotes from hamster spleen, cleaned by differential centrifugation, were injected intravenously into three mice from each of 25 inbred strains and five F_1 hybrids. Parasite numbers at day 15 in the liver were estimated by Stauber's method[2,3]. The ratio of parasites to liver cells was determined on liver imprints and multiplied by the liver weight in mg to give a reproducible index of parasite load in LDU (Leishman-Donovan units).

The mean liver parasite load on day 1 of 30.5 LDU did not differ between strains but at day 15 inbred mice fell into two distinct categories which did not overlap. Twelve highly susceptible strains showed about a hundred-fold multiplication whereas the remaining 13 strains were resistant with less than eight-fold increase. No intermediates were seen except for an F_1 cross between a susceptible and a resistant strain which was of intermediate susceptibility. The observation suggested a rather simple genetic control of resistance. Our series included the seven strains studied by Plant and Glynn[1] and the results are in Table 1. There is precise correspondence between resistance to *S. typhimurium* and to *L. donovani*.

In breeding experiments resistant C3H mice were crossed with susceptible NMRI. The F_1 progeny were both selfed and back-crossed to each parental strain. Mice were infected as before. Liver counts from mice killed after 2 weeks are summarised in Fig. 1. Susceptible mice reappear as a distinct group clearly separable from a wider group corresponding to resistant and F_1 categories. The proportion of susceptible mice does not differ significantly from one quarter in the F_2 and one half in the back cross to susceptible parents. Also, the observed distribution of other counts fits closely to the predicted distribution on Mendelian expectations using independent estimates of the means and variances of counts in F_1 and resistant mice.

This is strong evidence for control of *L. donovani* growth in the mouse liver by a single gene or tight linkage group. The coincidence of response pattern to *L. donovani* and *S. typhimurium* has a 1:64 probability of having arisen by chance as there are concordant results for six separate mouse strains (C57BL and B10.D2 are genetically very similar). It is therefore possible that we may be dealing with the same genetic mechanism

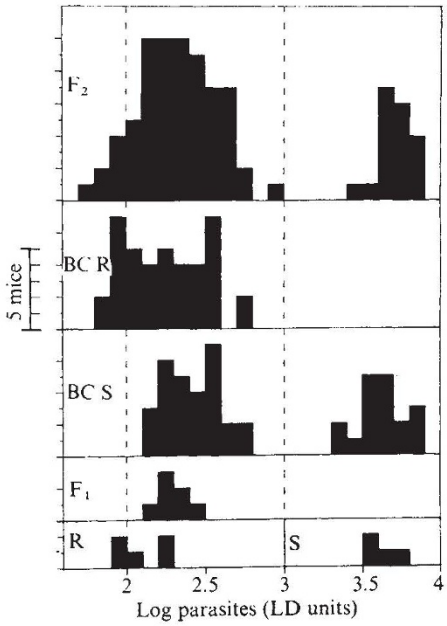

Fig. 1 Histogram of liver parasite burdens, 14–16 d after infection with 10^7 *L. donovani*, from 16th passage, October 1972. Mice derived from crossing susceptible (S) NMRI and resistant (R) C3H strains. Results from F_2 backcrosses (BC) to S and to R, F_1, and parental strains are shown. Parasite counts have been logarithmically transformed.

There is no evidence from our 25 strains that any *H*—2 alleles correspond with acute phase resistance or susceptibility, nor have attempts to map the gene (provisionally name *Lsh*, leishmaniasis resistance) given support for any linkage to *Ir* or *H*—2 loci (D. J. B. and B. A. Taylor, in preparation). Results so far do not suggest any direct relation of acute leishmaniasis susceptibility to *Ir*1, *H*—2 or the ability to mount an acquired immunological response.

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Yours faithfully,

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Table 1 Parasite numbers in mouse livers

Mouse strain	Mean count (LDU)	Mean log count* (± s.e.)	Parasite increase (day 15 count / day 1 count)	Susceptibility *Leishmania*	Susceptibility *Salmonella*†
BALB/c	3,438	3.534 ± 0.030	115.1	S	S
C57BL	3,697	3.565 ± 0.035	123.3	S	S
BIO D2 new	6,410	3.793 ± 0.078	208.4	S	S
DBA/2	94	1.967 ± 0.044	3.1	R	R
C3H/He	165	2.216 ± 0.030	5.5	R	R
A	149	2.170 ± 0.030	5.0	R	R
CBA/Ce	125	2.095 ± 0.040	4.2	R	R

* Here the counts in LDU have been logarithmically transformed which normalises the distribution and renders the variance independent of the mean.
† *Salmonella typhimurium* susceptibility from ref. 1 for comparison.
Parasite numbers were measured 15 d after intravenous infection with 10^7 amastigotes of *Leishmania donovani* derived from hamster spleen, 14th passage, May 1972. Three mice of each strain used.