normal littermate was born as well. But, the main source of mature T cells or thymic factor during embryonic life should be the mother¹. Thus these findings support the concept that the weak θ -positive lymphocytes may be prethymic T-cell precursors^{2,3} as the normal thymus or its products are not essential for their appearance in nude mice. They also suggest that nude mice may not be totally devoid of T-cell function.

FRANCIS LOOR
LENA-BRITT HAGG
KATHLEEN S. MAYOR
GEORGES E. ROELANTS

Basel Institute for Immunology,
Grenzacherstrasse 487,
CH-4058 Basel,
Switzerland

- ¹ Raff, M. C., *Nature*, **246**, 350 (1973).
² Loor, F., and Roelants, G. E., *Nature*, **251**, 229 (1974).
³ Roelants, G. E., et al., *Eur. J. Immunol.*, **5**, 127 (1975).
⁴ Raff, M. C., *Nature*, **251**, 184 (1974).
⁵ Humber, D. P., Pinder, M., and Hetherington, C. M., *Transplantation*, **19**, 91 (1975).

Y chromosome and aggression in mice

GENETIC differences in the aggression of mice have been attributed to the Y chromosome¹. As such a claim has obvious implications in the current controversy over the behavioural correlates of the XYY karyotype in man, alternative explanations must be considered.

The evidence depends on reciprocal differences in F_1 hybrids, where male hybrids scored closer to the male than the female parent. Although this rules out X chromosome inheritance, it does not rule out maternal effects. The authors state that "Because the most aggressive hybrids developed in the uterine and postnatal environment of the less aggressive C57BL/10/Bg strain, these maternal conditions seem to make no major contribution to the aggressive behaviour of these strains and hybrids". This assumes that maternal effects always make the offspring more like the mother, that is, that any maternal and additive genetic effects are always in the same direction. But there are no *a priori* reasons why this should be so, especially when one is considering a trait such as aggression, that they measured only in males. Clearly, if maternal effects are acting in an opposite direction to additive genetic effects, we have a situation of

apparent paternal inheritance and Y chromosome effects.

Data do exist² that demonstrate such maternal effects on the steroid responses of C57BL/10J and DBA/2 mouse strains, which presumably are fairly closely related to the C57BL/10/Bg and DBA/1/Bg used here. In the non-handled females there was a large positive additive genetic effect, where the DBA genotype acted to raise the corticosteroid response, and an equally significant negative maternal effect, where the DBA maternal environment lowered the response. Such a complex form of interaction is not merely a statistical artefact, but has definite evolutionary advantages in situations where natural selection favours an intermediate level of performance.

Fulker³ gives further examples of such "maternal buffering" and in Table 1 I have reanalysed the data under discussion by his method to give the four parameters, m = mean $[d]$ = additive genetic effect, $[d_m]$ = maternal effect and $[h]$ = dominance.

Using *t*-tests, m and $[d]$ turn out to be highly significant ($P < 0.001$) in both experiments, the large $[d]$ values contradicting the comment of Selmanoff, Jumonville, Maxson and Ginsburg¹ that no autosomal contribution was found for aggression. Admittedly the present model does separate additive and maternal effects more clearly than their scaling approach. $[d_m]$ is more significant ($P = 0.02$) in the larger experiment, though it should be noted that in both experiments it is only one-third the size of $[d]$, the additive genetic component. $[h]$ is of borderline significance in both experiments, though its fluctuations in sign between experiments are puzzling and should have been mentioned.

Although such maternal and genetic influences in opposite directions could explain the data and, in view of the previous evidence of others^{2,3} are possibly more likely than Y chromosome inheritance, the latter cannot yet be ruled out completely with these data, as $[d_m]$ and $[d_Y]$ (for the effect of a Y chromosome) have identical coefficients^{4,5} in these four generations and cannot be separated. Further experiments are the only way of deciding the issue. No examples exist in mice, but, working on *Drosophila* behaviour, I have described an extension of the m , $[d]$, $[d_m]$ and $[h]$ model to the situation where F_2 s and backcrosses are tested⁵. I have discussed several examples

of complex interactions between maternal and autosomal effects that could be detected unequivocally with these extra generations. Until such further generations are tested in these mice, the hypothesis of Y-linked inheritance of aggression in mice remains unproven.

DAVID A. HAY

Department of Genetics and Human
Variation,
La Trobe University,
Bundoora, Victoria, 3083, Australia

- ¹ Selmanoff, M. K., Jumonville, J. E., Maxson, S. C., and Ginsburg, B. E., *Nature*, **253**, 529 (1975).
² Treiman, D. M., Fulker, D. W., and Levine, S., *Dev. Psychobiol.*, **3**, 131 (1970).
³ Fulker, D. W., *Behav. Genet.*, **1**, 119 (1970).
⁴ Mather, K., and Jinks, J. L., *Biometrical Genetics* (second ed.) (Chapman and Hall, London, 1971).
⁵ Hay, D. A., *Heredity*, **28**, 311 (1972).

SELMANOFF *et al.* REPLY—HAY¹ suggests that there are autosomal effects, that a reversal of dominance occurs, and that our differences in reciprocal F_1 s are attributable to factors other than the source of the Y chromosome.

We took these possibilities into account, and suggested that autosomal factors and maternal factors may be involved (see our fifth paragraph, ref. 2). Similarly, what Hay interprets as reversals in dominance are described in our fourth paragraph, and explanations for these changes are proposed.

Hay's explanation of the reciprocal differences in aggression of F_1 hybrids depends on the assumptions made in applying the diallel model³ and on Fulker's⁴ findings of genetic and maternal influences acting in opposite directions. In using a Mendelian analysis, we take a more parsimonious approach to the assumptions that must be made. There are, in addition, very few examples where maternal and genetic influences act in opposite directions; whereas, there is now a growing body of evidence for the involvement of the Y chromosome in the inheritance of particular characteristics⁵. Aggressive behaviour is complex and undoubtedly has multiple genetic determination. As mentioned in our paper, there is no evidence for Y chromosome inheritance with respect to differences in aggressive behaviour where the DBA/2/Bg strain is involved⁵. We have, therefore, suggested that the most parsimonious explanation for all of our findings is that there is genetic variation in the Y chromosome of the mouse, such that some Ys, but not others, make substantial contributions to aggressive behaviour, as do autosomal genes and early experience. Further research is needed to substantiate this hypothesis.

Behavioral Genetics Laboratory,
The University of Connecticut

- ¹ Hay, D. A., *Nature*, **255**, 658 (1975).
² Selmanoff, M. K., Jumonville, J. E., Maxson, S. C., and Ginsburg, B. E., *Nature*, **253**, 529 (1975).
³ Mather, K., and Jinks, J. L., *Biometrical Genetics* (second ed.) (Chapman and Hall, London, 1971).
⁴ Fulker, D. W., *Behav. Genet.*, **1**, 119 (1970).
⁵ Selmanoff, M. K., Maxson, S. C., Ginsburg, B. E., *Behav. Genet.* (in the press).

Table 1 Estimates for the four-parameter genetic model of mouse aggression

	Initial experiment	d.f.	Replication	d.f.
m	19.65 ± 2.76*	36	21.35 ± 2.57	123
$[d]$	20.90 ± 4.17	36	18.60 ± 3.17	123
$[d_m]$	-6.25 ± 3.13	18	-4.55 ± 1.86	83
$[h]$	8.10 ± 4.17	36	-6.10 ± 3.17	123

* ± s.e.

Details of these parameters and how to calculate them and their s.e. are given by Fulker³ (except that his formulae for s.e. of $[d_m]$ and s.e. of $[d]$ on page 121 are accidentally transposed) and more fully by Mather and Jinks⁴.