of S^{34} illustrated in Table 2 as a result of the biological oxidation of sulfur to sulfate is significant. This may be considered as further evidence confirming results on laboratory experiments that S^{32} is not enriched, but rather, depleted, during the oxidation of sulfur to sulfate by *Thiobacilli* (*5*).

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References and Notes

1. A. P. Tudge and H. G. Thode, *Can. J. Research* 28B, 567 (1950).
2. G. E. Jones and R. L. Starkey, *Appl. Microbiol.* 5, 111 (1957).
3. C. D. Parker, *Sewage and Ind. Wastes* 23, 1477 (1951).
4. L. G. M. Baas Becking and I. R. Kaplan, *Trans. Roy. Soc. S. Australia* 79, 52 (1956).
5. We wish to acknowledge the help of Dr. C. D. Parker, who provided pure cultures of *T. concretivorus* and the sulfur and sulfate from cultures grown on H_2S.
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A New Strain of the Mouse Mammary Tumor Virus

The mammary tumor virus, transmitted from generation to generation through the mother's milk, is an important factor in the development of mammary tumors in mice. Although it has been postulated that this virus should show genetic autonomy and variability (*1*), this has not been established. The possibility that the mammary tumor viruses possessed by different inbred strains of mice may not be identical has been indicated by reports of differences in characteristics of tumor development (such as tumor incidence or mean age of tumor development) which occur when two such strains are crossed reciprocally (*2*). However, evidence of stable alterations in the activity of the virus within an inbred strain, in which the virus is detectable both before and after the change, has not been reported. To verify the assumption that the appearance of a new characteristic within a strain is the result of a change in the mammary tumor virus, it is necessary to compare the new and the original stocks in situations wherein the virus is the only variable, both in the test mice and in the females supplying virus to the test mice. In addition, the altered activity of the virus must be observed through several generations of mice to insure the stability of the change.

In our laboratory a fortunate combination of circumstances has resulted in a situation in which a change in a characteristic of an inbred strain of mice can be traced to an alteration in the activity of the mammary tumor virus. A line of Heston A mice (A/HeCRGL), inbred for over 85 generations, has been maintained in this laboratory since 1950. The mean age of mammary tumor development in the breeding females has consistently been about 12 months. In 1953 it was discovered that females in one branch of the stock were developing mammary tumors at about 8 months of age. This latter group was separated out, and it has been maintained separately as the A/viCRGL subline. It is at the present time in its 13th generation.

The experiments described below, in which all mice have been maintained as breeding females, were set up to determine whether this change in mean age of tumor development was the result of a change in the mouse or in the virus (*3*). In all experimental groups, some mice are still alive. Therefore, the final mean ages of tumor development may be slightly different from those reported here. However, the remaining mice are either so old or so few in number that their deaths (due to mammary tumors) will not affect the significance of the tumor age differences.

In the generations of stock mice concurrent with the experimental groups discussed below, 90 females of the A/He strain had a mean age of tumor development of 12.7 months; 111 females of the A/vi subline had a mean age of tumor development of 8.6 months (Table 1, experiment 1).

Reciprocal hybrids (34 A/He × A/vi hybrids, 51 A/vi × A/He hybrids) of the two stocks were collected, and their mean ages of tumor development were determined (Table 1, experiment 3). Each group of hybrid mice developed mammary tumors at a mean age similar to that of the strain to which their maternal parents belonged. These two groups of mice were identical genetically but differed in maternal influences.

Newborn mice of the A/vi subline were transferred to and nursed by females of the A/He strain and vice versa. Fourteen animals that received A/He milk either had a late age of tumor development or are alive and more than 13 months old, despite the fact that they are otherwise A/vi females; 25 females that received A/vi milk had an early mean age of tumor development despite the fact that they were otherwise A/He females (Table 1, experiment 2). Thus, the difference in mean age of tumor development is evidently mediated by factors carried in the milk.

The activity of the mammary tumor virus has been followed for two generations beyond the reciprocal hybrids by fostering females of the A/vi stock upon both groups of hybrids (Table 1, experiment 4) and by collecting the offspring of these fostered females (Table 1, experiment 5). In both of these generations, groups of 22 and 25 mice whose mammary tumor virus was originally from A/He mice are developing mammary tumors at a later age than did groups of 21 and 34 mice whose mammary tumor virus was originally from the A/vi stock. Thus, the two viruses have retained their difference in activity through these generations, despite the fact that the mice carrying each of them were similar in genotype in each generation. This eliminates the possibility that the difference in mean age of tumor development reported here is the result of a change in the genotype of the host which is expressed in the activity of the mammary tumor virus passed to the offspring.

Table 1. Summary of experiments involving the mammary tumor viruses of the A/He strain and the A/vi subline and their influence on the mean age of mammary tumor development in breeding female mice. Symbols designate genotype (in hybrids the female parent is mentioned first); numbers in parentheses indicate mean age, in months, of tumor development; arrows indicate transfer of the virus.

Expt.	Transfer of virus from A/He strain	Transfer of virus from A/vi subline
1	A/He (12.7)	A/vi (8.6)
2	↓ A/vi (> 12.8)	↓ A/He (9.6)
3	F_1* (13.1)	F_1† (9.4)
4	↓ A/vi (11.9)	↓ A/vi (9.6)
5	↓ A/vi (> 11.4)	↓ A/vi (9.4)

* A/He × A/vi. † A/vi × A/He.

The various experimental situations have shown the stability of the difference between the two viruses. Possible differences in the response of other strains of mice to these two strains of virus are being tested in a series of current experiments. In addition, the possibility of there being serological differences between the two viruses is being investigated.

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References and Notes

1. C. C. Little and P. A. Gorer, in H. Gruneberg, *The Genetics of the Mouse* (Cambridge Univ. Press, Cambridge, England, 1943), appendix I.
2. J. J. Bittner, *Cancer Research* 8, 625 (1948).
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