technological and economic fishery research, amounting to $35,000 annually for five years. The purpose of such a program was to develop research of the type outlined by this committee. Failure to secure the authorized appropriation, however, has nullified the purpose of the act. The committee strongly urges the early resumption of the authorized plan of development.

Another authorization by Congress, approved June 21, 1934, which has failed of realization provides for the construction of a marine research vessel, without which many of the fundamental problems of the marine fisheries can not be solved. The United States is the only first-class maritime nation without such a research vessel. Such facilities should be furnished as soon as possible.

Fishery Research by State Agencies

The problems best handled by state research agencies are those of more local significance that will guide the enactment and enforcement of protective regulations, or that will result in the promotion and extension of the local fishing industries and game fish supply. The program of research should be integrated with the programs of the Federal Government and coordinated with those of the neighboring states. States may meet this need for investigation by the establishment of research units in their conservation commission, or by the utilization of existing state agencies. Such units should be staffed by trained personnel to be appointed on the basis of merit, with reasonable assurance of tenure of office. Close cooperation between state-supported educational institutions and conservation officials is urged.

Stimulation of Research by Private Agencies

In contrast with conditions in Europe and Japan, where progress in aquatic biology comes chiefly from university men, few scholars in this country are devoting themselves to aquatic biology. Universities, scientific foundations and private institutions and associations should be encouraged to devote more attention to the biological problems involved in conservation of natural resources through the creation or allocation of grants in aid, fellowships and professorships in existing institutions. The Federal Government, state conservation departments, research foundations and sportsmen's organizations should cooperate in this program by allocating funds for such purposes.

Finally, the committee recommends that in order to make the results of scientific investigations available and practically applicable, adequate funds for the prompt publication of technical reports resulting from such studies should be made available by all agencies concerned.

R. V. Truitt, *Chairman*

SPECIAL ARTICLES

FURTHER DATA ON THE EXISTENCE OF EXTRA-CHROMOSOMAL INFLUENCE ON THE INCIDENCE OF MAMMARY TUMORS IN MICE

In 1933 the writers[1] included the result of two crosses among the data advanced by the staff of the Jackson Memorial Laboratory to demonstrate extra-chromosomal influence on the incidence of mammary tumors in mice.

These crosses were between an inbred strain of dilute brown mice (dba) high in incidence of mammary tumors and another inbred strain (C57 black) in which no such tumors have been recorded.

The F_1 generation produced by dilute brown females × C57 black males gave mammary tumors in 39.82 per cent. of the 113 virgin females which lived to reach "cancer age." The 664 F_2 generation animals, descended from similarly constituted F_1 females crossed with their brothers, gave 35.29 per cent. mammary tumors.

The reciprocal F_1 generation derived from C57 black females × dilute brown males gave 6.06 per cent. mammary tumors in 379 mice, while the 688 F_2 females obtained from inbreeding these F_1 animals gave 5.96 per cent. with mammary tumors. The detailed results of these completed experiments will be published in the September, 1935, number of *Genetics*.

So striking was the difference between the reciprocal crosses that it seemed desirable to produce and study certain back-cross generations. While most of the animals produced for this purpose are still alive, the preliminary results so far obtained are sufficiently interesting to justify publication.[2]

In order to understand the plan of the experiment it will be desirable to use the following symbols to designate the genetic contribution of the different strains:

C = chromosomal material derived from high mammary tumor strain—dba.

c = chromosomal material derived from non-mammary-tumor strain—C57 black.

E = extra-chromosomal influence derived from high mammary tumor strain—dba.

[1] Science, 78: 465–466.

[2] The population of virgin females from which the final data are to be derived consists of 250 mice in each of four crosses.

e = extra-chromosomal influence derived from non-mammary-tumor strain—C57 black.

The contribution of the female is shown as the numerator of a fraction; the male contribution as the denominator. Thus, a dba female is expressed by $♀\frac{CE}{C}$ and a dilute brown male as $♂\frac{C}{C}$; while a C57 black female is $♀\frac{ce}{c}$ and a male $♂\frac{c}{c}$.

Four types of back-cross were made as follows:

I. $F_1\ ♀\ \frac{ce}{C} \times ♂\ \frac{C}{C}$—containing *three* high mammary tumor chromosomal contributions and *one* non-mammary tumor. Extra-chromosomal influence non-mammary tumor.

II. $♀\ \frac{CE}{C} \times F_1\ ♂\ \frac{c}{C}$—similar to I except extra-chromosomal influence is from high mammary tumor strain.

III. $F_1\ ♀\ \frac{ce}{C} \times \frac{c}{c}$—containing *three* non-mammary tumor chromosomal contributions and *one* high mammary tumor. Extra-chromosomal influence non-mammary tumor.

IV. $F_1\ ♀\ \frac{CE}{c} \times \frac{c}{c}$—similar to III except extra-chromosomal influence is from high mammary tumor strain.

The total number of virgin female mice which have died in each cross, together with the incidence of mammary tumors, is as shown in Table 1.

TABLE 1

	Total mice dead	Non-mammary tumor	Mammary tumor	Per cent. mammary tumor
I	10	10	0	0.0
II	47	16	31	65.9
III	13	13	0	0.0
IV	23	7	16	69.5
Total...	93			

In crosses I and II the chromosomal contributions to female back-cross mice by their F_1 parents should be equal, three being from the high tumor strain and one from the non-tumor strain. The extra-chromosomal influence was, however, obviously very different, being from the high tumor strain in II and from the non-tumor strain in I.

The high incidence of mammary tumors does not follow the high tumor chromosomes, but is found in the cross where the extra-chromosomal influence is derived from a high tumor strain. The percentages are strikingly distinct, being 0 for I and 65.9 for II.

Further evidence of the relative unimportance of the chromosomes is to be found in a comparison of I and III. Here the extra-chromosomal contribution is similar in the two crosses, both being from the non-tumor strain. A C57 black male with no "high tumor" chromosomes was used in III, producing animals with three non-tumor and one high tumor chromosomal contributions. In I an F_1 with "high tumor" chromosomes derived from a dilute brown mother was used. The total chromosome contribution was the reverse of III, being three "high tumor" to one non-tumor. Although this great chromosomal difference exists, neither cross has as yet formed a mammary tumor. Later figures may, however, modify this result somewhat.

Cross I compared with Cross IV is interesting in that I introduces $\frac{3}{4}$ high tumor chromosome contribution, $\frac{1}{4}$ non-tumor, with non-tumor extra-chromosomal influence and gives 0 per cent. mammary tumors while IV introduces $\frac{3}{4}$ non-tumor chromosomes and $\frac{1}{4}$ high tumor and high tumor extra-chromosomal influence, giving 69.5 per cent. mammary tumors.

Cross II compared with Cross III presents a slightly different situation. Cross II combines $\frac{3}{4}$ high tumor and $\frac{1}{4}$ non-tumor chromosomes with a high tumor extra-chromosomal influence. Its incidence of mammary tumors is 65.9 per cent. Cross III is the opposite, having only $\frac{1}{4}$ high tumor chromosomes and a non-tumor extra-chromosomal influence. Its tumor incidence is 0.

Cross II may also be compared with Cross IV. In this case, although the chromosomal contributions are diametrically opposite, being $\frac{3}{4}$ high tumor in II and $\frac{3}{4}$ non-tumor in IV, *both* crosses have high tumor extra-chromosomal influence. The percentages of tumor incidence are 65.9 and 69.5, respectively. It is interesting to note that while the incidence is as nearly identical as could be expected, the slight excess that does exist occurs in the cross with *more* "non-tumor" chromosomes. The significance of this fact is, however, doubtful.

In a comparison between Crosses III and IV the chromosome situation is identical in the two crosses, but the extra-chromosomal contribution of IV is directly transmitted from a high tumor dilute brown grandmother, while III has no such derivation. In this case the incidence of tumors is strikingly different, being 0 per cent. in III and 69.5 per cent. in IV.

It will be noted that the incidence of mammary tumors in back-crosses II and IV (65.9 and 69.5 per cent.) is distinctly higher than in the F_1 and F_2 generations which were derived from high tumor female ancestry (39.82 and 35.29 per cent.). The significance of this difference is at present doubtful. It is quite possible that as larger numbers of non-tumor

animals begin to die in the older age groups of the back-cross it will tend to lower the final percentage of tumors in these generations. If the present high tumor incidence of mammary tumors in Cross II persists it might be considered the result of chromosomal influences from the dilute brown chromosomes. Cross IV, however, should be actually no greater than on the F_1 and F_2 generations above mentioned. If, therefore, chromosomal influences are operative to any measurable degree Cross IV should eventually show no higher incidence of mammary tumors than 40 per cent., while Cross II should remain appreciably higher than that figure. Further data are needed to settle this point.

The relative age of the populations in the four crosses is a factor to be considered. The mean age in days of the four is as follows: I, 499; II, 444; III, 439; IV, 431. It will be noted that such difference as exists has resulted in as good or better chance for the crosses showing no mammary tumors as for those showing them.

While the incidence of mammary tumors has shown a clearly "extra-chromosomal" type of distribution, the two pairs of mendelian color factors B black—b non-black (brown), and D intense—d, dilute have segregated normally. The actual count in the two crosses where such segregation was discernible is given in Table 2.

TABLE 2

	Black	Dilute black	Brown	Dilute brown	Total
I					
Observed	170	162	134	172	636
Expected	159	159	159	159	638
II					
Observed	163	133	128	152	576
Expected	144	144	144	144	576

The experiments are being continued and will be fully discussed on their completion. It should be clearly understood that larger numbers of animals may modify somewhat the quantitative values of the various groups. On the other hand, there is little doubt that the major difference between Groups II and IV, on one hand, and I and III, on the other, will persist.

Although the numbers of mice so far autopsied in Crosses I and III are small, the absence of mammary tumors is interesting and may, if it persists, have considerable significance. The fact that the completed series of F_1 and F_2 animals, to which reference has been made, showed in all crosses some mammary tumors remains to be explained by further experimentation. Using the symbols already employed, the F_1 and F_2 generations may be tabulated as follows:

dBF_1 ♀ $\frac{CE}{C} \times$ ♂ $\frac{c}{c}$ = 39.82 per cent. mammary tumors in 113 mice

BdF_1 ♀ $\frac{ce}{c} \times$ ♂ $\frac{C}{C}$ = 6.06 per cent. mammary tumors in 379 mice

dBF_2 ♀ $\frac{CE}{c} \times$ ♂ $\frac{C}{c}$ = 35.29 per cent. mammary tumors in 664 mice

BdF_2 ♀ $\frac{ce}{C} \times$ ♂ $\frac{C}{c}$ = 5.96 per cent. mammary tumors in 688 mice

We may thus conclude that in the four types of back-cross cited, the incidence of mammary tumors in mice depends primarily upon the direct transmission of extra-chromosomal influences. This confirms completely the earlier experiments with F_1 and F_2 mice.

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ADRENAL INSUFFICIENCY IN THE MARMOT AND OPOSSUM AND THEORIES OF CORTICO-ADRENAL FUNCTION

COMMON laboratory animals (cat, dog, rat, rabbit, guinea-pig) which have been adrenalectomized show diminutions in serum sodium and chloride content, and Addisonian patients in crisis are apparently similarly affected.[1, 2, 3, 4] It has also been demonstrated that an increased renal excretion of sodium and chloride is either the result or the cause of the subnormal values found in blood serum and tissues after adrenal removal. If compensation for excessive loss of sodium chloride is made by the injection or oral administration of saline, adrenal insufficiency is said to be relieved[2, 3, 5]; again, if the sodium content of the body is reduced experimentally, a condition resembling that of adrenal insufficiency is said to result.[7]

On the basis of these observations several experimenters have expressed the belief that the adrenal cortex is intimately concerned with sodium chloride

[1] H. Silvette and S. W. Britton, *Amer. Jour. Physiol.*, 104, 399, 1933; *ibid.*, 108, 535, 1934; *ibid.*, in press; also unpublished results.

[2] R. F. Loeb *et al.*, SCIENCE, 76: 420, 1932; *Proc. Soc. Exp. Biol. Med.*, 30: 808, 1933; *Jour. Exp. Med.*, 57: 775, 1933.

[3] G. A. Harrop *et al.*, *Jour. Amer. Med. Assoc.*, 100: 1850, 1933; *Jour. Exp. Med.*, 57: 305; *ibid.*, 58: 1, 17.

[4] W. W. Swingle *et al.*, *Amer. Jour. Physiol.*, 107: 259, 1934.

[5] J. M. Rogoff, *Jour. Amer. Med. Assoc.*, 103: 1764, 1934.

[7] A. Gilman, *Amer. Jour. Physiol.*, 108: 662, 1934.