

B the odds against the possibility that the difference between reciprocal crosses is due to chance are nearly, and in excess of a million to one, respectively. In crosses C and D also the difference is obvious.

TABLE 1²

Experiment	Type of cross		No. F ₁ ♀s	Percentage mammary tumors
A	D ♀ (high tumor line)	× C 57 blk ♂ (low tumor line)	61	36.06
	C 57 blk ♀ (low tumor line)	× D ♂ (high tumor line)	307	5.53
B	D ♀ (high tumor line)	× <i>M. bactrianus</i> ♂ (low tumor line)	69	68.11
	<i>M. bactrianus</i> ♀ (low tumor line)	× D ♂ (high tumor line)	27	7.41
C	A ♀ (high tumor line)	× X ♂ (low tumor line)	22	86.3
	X ♀ (low tumor line)	× A ♂ (high tumor line)	6	0.0
D	Z ♀ (high tumor line)	× I ♂ (low tumor line)	30	90.0
	I ♀ (low tumor line)	× Z ♂ (high tumor line)	10	0.0

The greater influence of the female as compared with the male parent in determining the incidence of spontaneous mammary tumors in mice is thus clearly established. This fact makes necessary a reconsideration of any genetic conclusions drawn from data in which only one of the two types of crosses has been considered, or in which the two types have been treated as one.

The persistence of the difference in the F₂ generation has been demonstrated by an extensive study of

Derivation of F ₂ ♀s			Total F ₂ ♀s	Percentage with mammary tumors
F ₁ (D ♀ (high tumor)	× C 57 ♂ (low tumor)	<i>inter se</i>	650	31.07
F ₁ (C 57 ♀ (low tumor)	× D ♂ (high tumor)	<i>inter se</i>	489	7.39

² No detailed comparison between experiments A, B, C and D should or will be attempted until complete results are published. The important point at present is the consistent nature of the difference between the reciprocal crosses.

over 1,100 F₂ females derived from Experiment A. These show the following incidence of mammary tumors.

The F₂ females derived from the high tumor grandmothers and high tumor F₁ mothers give again a significantly greater incidence of mammary tumors. The chance of the two F₂ groups being similar is much less than one in a million.

This establishes the transmission of extra-chromosomal influence. Because of that fact the simple Mendelian genetic nature of the biological agents influencing the etiology of spontaneous mammary tumors is definitely disproved.

This should not be taken as denial of the existence of chromosomal influence. There is clear evidence that such influence also is present and effective. Further studies are already under way to attempt to determine more accurately the nature and extent of both chromosomal and extra-chromosomal influences.

STAFF OF ROSCOE B. JACKSON

MEMORIAL LABORATORY

Per C. C. LITTLE, Director

BAR HARBOR, MAINE

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