The virus does not appear to be related to vesicular stomatitis or to equine encephalomyelitis, since it is non-pathogenic for guinea pigs when injected intracerebrally and because it is not neutralized when injected in mice with immune sera for equine encephalomyelitis and vesicular stomatitis.[9] The virus appears unrelated to herpes virus since rabbits show no effects following intracerebral, corneal and intradermal injections.

Sera from convalescent St. Louis[10] and Kansas City cases, in contradistinction to sera from non-contact healthy adults, appear to possess specific protective substances. Virus suspensions were mixed with undiluted human sera to give final virus dilutions of 10^{-3}, 10^{-4}, 10^{-5} and 10^{-6}. The mixtures were incubated at 37° for two hours and inoculated intracerebrally into four Swiss mice. Six sera, including one positive and one negative control, were tested at one time. The table summarizes a protocol of one of these titrations.

Thus far, sera from eight normal adults in New York tested twice have shown no protective qualities. Sera from eight St. Louis encephalitis convalescent cases, tested twice, however, all show definite protective properties.

PROTECTION TEST

Sera	Virus-Serum Dilution 10^{-3}	10^{-4}	10^{-5}	10^{-6}
J., Normal New York	5*, 5, 6, 8	7, 7, 8	8	
K., Normal New York	4, 5, 5, 8	5, 5, 5, 6	7, 7, 7	
R., Normal New York	5, 5, 5, 7	5, 5, 5, 7		
No. 10 St. Louis Convalescent	6			
No. 9, St. Louis Convalescent	7, 9, 10			
No. 33, St. Louis Convalescent	8, 10	8		

* Duration of life of mouse in days. Blanks indicate mice remained healthy.

The facts presented would indicate that the active agent is a specific filtrable virus, etiologically related to the encephalitis prevailing in St. Louis and Kansas City and that one possible mode of its transmission is by way of the upper respiratory tract. Final decision on these questions must be reserved, however, until the work in progress has been completed.

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[9] The writers are indebted to Dr. H. R. Cox for the immune sera used and assistance in these tests.

[10] These sera were obtained from cases at the St. Louis City Isolation Hospital by Dr. S. Weisman through the courtesy of Dr. J. Eschenbrenner.

THE EXISTENCE OF NON-CHROMOSOMAL INFLUENCE IN THE INCIDENCE OF MAMMARY TUMORS IN MICE[1]

THE object of this communication is to record the existence of extra-chromosomal influence, extending for more than one generation and affecting the natural incidence of spontaneous mammary tumors in mice.

The data are based on four independently conducted experiments which have continued over a period slightly in excess of three years. Seven distinct inbred strains of mice (six derived from *Mus musculus,* and one the direct descendants of wild *Mus bactrianus*) have been used. The results in all four experiments are consistent with, and confirmatory of, one another. Since in our experience females only have formed spontaneous mammary tumors, that sex alone is included in the tabulation of tumor incidence. More detailed papers on the different experiments will later be published. The present note seeks merely to record certain facts of general interest and application.

The opportunity to detect extra-chromosomal influence is offered by a comparison of the incidence of spontaneous mammary tumors among the female mice derived from reciprocal crosses between distinct "high tumor" and "low" or "non-tumor" strains. Although no mammary tumors have been recorded in certain strains designated as "low tumor," it seems better to use that term as an admission that the possibility of the future appearance of such neoplasms is recognized.

Since in such reciprocal crosses the chromosomal constitution of F_1 females is similar (both as regards sex chromosomes and autosomes) it follows that any significant difference in tumor incidence which may exist between the two types of cross is extra-chromosomal. If this difference continues beyond the F_1 generation, direct transmission through the extra-chromosomal portion of the germ-cell is clearly indicated.

In the following table the incidence of spontaneous mammary tumors in F_1 females is recorded in four experiments (A, B, C, D). The reciprocal crosses are shown in each case. In every instance the incidence of spontaneous mammary tumors in F_1 is strikingly and significantly higher when the cross is made between a female from a high tumor strain and a male from a low tumor strain, than it is when the cross is made in the reciprocal manner (low tumor line ♀ × high tumor line ♂). In experiments A and

[1] Acknowledgment of a grant from the American Academy of Arts and Sciences in partial support of this work is hereby made.

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 1[2]

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)	×	*M. bactrianus* ♂ (low tumor line)	69	68.11
	M. bactrianus ♀ (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 F_2 generation has been demonstrated by an extensive study of over 1,100 F_2 females derived from Experiment A. These show the following incidence of mammary tumors.

Derivation of F_2 ♀s	Total F_2 ♀s	Percentage with mammary tumors
F_1 (D ♀ (high tumor) × C 57 ♂ (low tumor)) *inter se*	650	31.07
F_1 (C 57 ♀ (low tumor) × D ♂ (high tumor)) *inter se*	489	7.39

The F_2 females derived from the high tumor grandmothers and high tumor F_1 mothers give again a significantly greater incidence of mammary tumors. The chance of the two F_2 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.

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[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.

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