TABLE 2

Preserved CA fluid PR8 strain	Methanol, %	Phosphate buffer pH 7, *M*	CCA titer	Yield, %
Batch #1	20	0.1	68	54
	25	0.1	34	27
	30	0.1	0	0
	Original CA fluid		126	
Batch #2	14	0.1	39	41
	17	0.1	40	42
	20	0.1	48	50
	Original CA fluid		96	
Batch #3	23	0.1	96	44
	23	0.2	172	78
	20	0.3	164	74
	23	0.3	178	81
	26	0.3	219	100
	Original CA fluid		220	

or previous treatment of the CA fluids with such preservatives as merthiolate and formalin in concentrations of 1: 5,000 and 1: 10,000, respectively.

References

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2. NIH directions for titration of chicken red cell agglutination (CCA) activity of influenza virus and vaccines according to Hirst's method, as modified for use in the laboratories of the Rockefeller Institute at Princeton, N. J., Sept. 16, 1946.

A Sex-linked Lethal Gene in the Fowl[1]

K. Goodwin, F. B. Hutt, and R. K. Cole

Department of Poultry Husbandry, Cornell University, Ithaca, New York

This report deals with a sex-linked recessive lethal gene that was found in White Leghorns. The gene, for which the symbol *xl* is suggested, exerts its lethal action during the growing period, with affected females having been observed as early as 23 days of age and as late as 123 days of age. The abnormal physiological mechanism resulting in death has not as yet been determined; nevertheless, the gene induces a set of symptoms which identify it as a specific character.

In the affected chick the symptoms are hyperacute, to the extent that after only a few hours' illness the bird either succumbs, or else it undergoes a remarkable recovery. Sick chicks are invariably discovered early in the morning; the onset of symptoms apparently does not occur during daylight hours. Affected birds display an extreme listlessness, resting quietly on their keels with the tip of the beak touching the floor. In the later stages they may pass into a complete coma. Occasionally dyspnea, or tetanic spasms, or both, are seen. The first attack is not always fatal, some birds having been observed to survive as many as 3 attacks before succumbing to a subsequent one. Among the 64 affected females observed in 1947 and 1948, 2 apparently made a complete recovery (in one instance after 1 attack, and in the other instance after 2), and the birds lived to reproduce. Deaths appear to be concentrated on certain days, suggesting that some environmental factor, as yet unrecognized, precipitates the condition. The dead birds are well-fleshed, and findings at autopsy are essentially negative.

The condition came to the attention of this department when a breeder of White Leghorns experienced a number of losses for which no specific cause could be found. Since these chicks were all from one sire, the influence of heredity was suspected by the owner and by Grayson B. Mitchell, who made the autopsies. The suspected sire (K-1) and 4 dams from the mating that had produced affected chicks on the breeder's farm were then given to us for study.

During a 2-year period, sire K-1 was mated to these 4 females and to unrelated females from the college breeding flock. The results of these matings, given in Table 1, show that: (*a*) approximately one-half the daughters succumbed to the *xl* lethal, (*b*) no sons were affected, and (*c*) the frequency of the lethal condition in the progeny was independent of any relation of the dams to the sire, K-1.

TABLE 1

GENETIC TESTS WITH SIRE K-1 AND WITH 13 OF HIS SONS

Matings	Male progeny			Female progeny		
	No.	Died, other causes	Died, *xl* lethal	No.	Died, other causes	Died, *xl* lethal
K-1 × 4 related ♀♀	23	2	0	13	1	7
K-1 × 14 unrelated ♀♀	32	5	0	42	4	21*
Totals for Sire K-1	55	7	0	55	5	28
Three carrier sons of K-1†	89	4	0	77	5	36
Ten noncarrier sons of K-1†	244	8	0	287	5	0

* Includes 2 birds that showed symptoms but did not die.
† Mated with unrelated females.

These preliminary matings provided evidence that the lethal effect was caused by a single sex-linked recessive gene, and that sire K-1 was heterozygous. One-half the daughters received the lethal gene from sire K-1 and died, whereas the one-half receiving the normal allele survived. No affected sons could be produced in this type of mating, since each received the dominant normal allele from his dam.

By chance segregation, one-half the sons of sire K-1 should have received the recessive allele from him and should therefore have been heterozygous (*Xl xl*) for the character. When 13 of these were tested, 3 were found to be carriers, as was proved by the fact that approxi-

[1] Number 23 in the series by F.B.H. entitled "Genetics of the Fowl."

mately one-half their daughters were affected (Table 1).

The 10 sire-families not showing any affected offspring by 10 weeks of age were discarded; the 3 families containing affected chicks were kept intact for about 5 months to secure data on the frequency of the character. Mortality from causes other than the lethal gene was very low, and no males were affected by the character.

Subsequent tests of 10 other sons of heterozygous sires revealed that 4 were carriers, thus making a total of 7 carriers among 23 cockerels tested. These results do not differ significantly from the expected number of 11.5 carriers ($P = .05–.10$).

As was pointed out previously, 2 daughters of sire K-1 were able to survive all attacks of the condition and became, to all outward appearances at least, perfectly normal adults. As a further test of the hypothesis of sex-linkage, these 2 females (presumably *xl*/—) were mated to a known carrier male. In such a mating one-half the pullet chicks should have been affected as before, and, in addition, one-half the cockerel chicks should have received a sex chromosome bearing *xl* from each of their parents, and should therefore express the character. Only a limited number of progeny (35) was obtained from these matings. Among these chicks, 40% of each sex died. Of the 8 males that died, 5 did so suddenly, at ages ranging from 11 to 17 days. At autopsy no abnormal changes were seen. The 3 other sons died at later ages, and these all showed symptoms identical with those which distinguish pullets affected with the *xl* syndrome. We interpret these facts as evidence that at least the sons which showed the syndrome were homozygous for the sex-linked gene *xl*.

Preliminary physiological tests have yielded no substantial information as to the mode of action of the lethal gene. The attacks almost invariably develop during, or just following, a 10- to 14-hour period of darkness and quiet. Attempts to revive comatose birds by intramuscular injection of adrenalin, or by intravenous injection of glucose, calcium gluconate, or parathormone, were all unsuccessful.

One other sex-linked gene with lethal properties is known for the fowl. The sex-linked gene *n* for naked (*3*) is lethal to about half the naked chicks during incubation, and to most of those that hatch, unless these are brooded at high temperatures. In the case of the lethal gene *xl*, chicks destined to show the lethal syndrome cannot be distinguished from their normal siblings. In this respect it is similar to the delayed lethal studied in the rat by Crew and Kon (*1*). The presence of the gene *xl* in the hemizygous state is apparently not inevitably lethal since about 3% of such birds escape death.

When added to the lists of mutations previously recognized in the fowl (*2*), the *xl* lethal raises the number of known sex-linked genes to 12, and that of lethal genes to 22.

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A Precision Method of Counting Radioactive Liquid Samples[1]

Arthur J. Freedman[2] and David N. Hume

Laboratory for Nuclear Science and Engineering and *Department of Chemistry, Massachusetts Institute of Technology, Cambridge*

The lack of an accurately reproducible method of preparing radioactive samples for counting has often prevented the use of radioactive tracers in precise analytical work. Most of the common methods for handling solid samples will generally give reproducible results with carrier-free tracers. However, the end result of an analytical procedure is usually a sample containing a weighable amount of inert solid material, and in such cases self-scattering and self-absorption effects make the observed activity of the sample critically sensitive to small variations in the form and dimensions of the sample.

Good reviews of techniques currently in use for handling radioactive samples have recently been published (*2*, *3*). While attempting to minimize self-scattering and self-absorption errors, the authors tested many of the common techniques, and in no case could results consistently precise to better than 1% be obtained with samples containing more than a milligram or so of solid material. Curve *A* in Fig. 1 shows a typical set of counting data obtained during the course of this work.

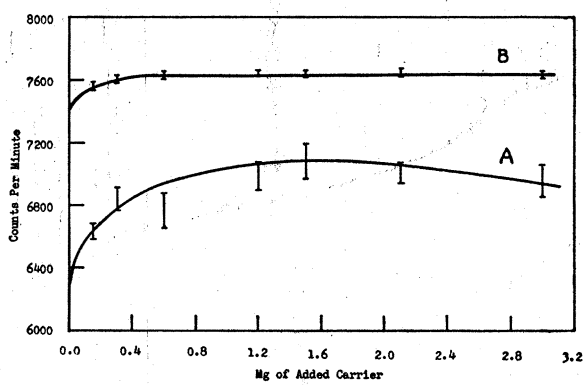

Fig. 1. Observed radiostrontium activity as a function of added inactive carrier. *A*, evaporated solid samples; *B*, lacquer-covered liquid samples.

The isotope used was Sr^{90} (25 y, $\beta = 0.61$ mev) which decays to Y^{90} (65 h, $\beta = 2.35$ mev). Yttrium sulfate in dilute sulfuric acid was used as carrier, and the samples were evaporated on 1-mil polystyrene film in order to minimize back-scattering effects. The samples were counted in the third shelf of the standard arrangement for use with an end-window Geiger tube. Each point in the figure represents an average of 3 counts on each

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[2] Present address: Los Alamos Scientific Laboratory, Los Alamos, N. M.