and sublines 12 and 212 of strain dba) were observed as untreated controls, and during and following the administration of x-rays or methylcholanthrene. The methylcholanthrene was applied percutaneously twice weekly in a 0.5 per cent. solution in benzene; 720 to 880 r of x-rays were administered by fractional irradiation, 80 r daily on successive days. Young adult animals of both sexes (8 to 10 weeks of age) were used. The latent period of chemical induction of leukemia in susceptible mice averaged approximately 120 days; the latent period of x-ray induction was longer, with 120 days the shortest preleukemic period. Table 1 records the results.

TABLE 1

Mouse strain	Number of mice	Spontaneous leukemia	Methylcholanthrene induced leukemia	X-ray induced leukemia
F	421	233 (55 per cent.)		
F	122		43 (35 per cent.)*	
F	34			0†
A	80	3 (3.8 per cent.)		
A	55		0	
A	56			17 (30 per cent.)‡
dba–212 ...	14	5 (36 per cent.)		
dba–212 ...	17		11 (65 per cent.)§	
dba–212 ...	12			0†
dba–12	26	0		
dba–12	97		63 (65 per cent.)	
dba–12	12			0

* Leukemia appeared precociously; per cent. reduced because of death from induced skin tumors.
† Leukemias that appeared were not manifest precociously, but appeared at the expected time of occurrence for this strain.
‡ Thirty-one animals still living. All leukemias appeared before any spontaneous case for this strain.
§ Leukemias appeared earlier than any spontaneous case for this strain.

The F strain was high in spontaneously developed leukemia (55 per cent.), susceptible to acceleration of onset of leukemia with carcinogens (30 per cent. leukemia before 200 days of age in methylcholanthrene-treated animals as contrasted with 6 per cent. in controls), and resistant to acceleration of onset with x-rays.

Strain A was low in spontaneously developed leukemia, resistant to induction of the disease with methylcholanthrene, but has shown at least a 30 per cent. incidence following exposure to 880 r of x-rays given by fractional irradiation.

Subline 212 of strain dba was moderately susceptible to spontaneous leukemia and markedly susceptible to carcinogenic induction of the disease, but resistant to x-ray induction or acceleration. Subline 12 of the same strain proved to be resistant to spontaneous leukemia or induction with x-rays, but very susceptible to the leukemogenic action of methylcholanthrene.

The results demonstrate that susceptibility of inbred mice, strains F and dba, to either spontaneous leukemia or the carcinogenic induction of the disease did not imply susceptibility to an agent, x-rays, which was, however, leukemogenic for genetically unrelated, low leukemia, and carcinogen-resistant animals, strain A. The problem of leukemogenesis in mice is very complex—first, multiple agents can induce leukemia; second, mice of only certain genetic constitution are susceptible to only certain agents; third, genetic susceptibility to one agent, or to the spontaneous disease, can not necessarily be correlated with susceptibility to other leukemogenic agents.

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THE NEUTRALIZATION IN VITRO OF AVIAN PNEUMOENCEPHALITIS VIRUS BY NEWCASTLE DISEASE IMMUNE SERUM

AVIAN pneumoencephalitis is the name applied by Beach[1] to a disease of chicks in California from 2 to 10 weeks old, formerly called "a respiratory nervous disorder,"[2] and also to a respiratory disease of nearly or fully mature chickens which had been known in different localities as "chicken flu" and "9-day pneumonia." The former was first observed in 1940, while the latter has been prevalent since 1935. Despite the fact that the spread of the disease through a flock is very rapid, transmission by artificial means proved difficult and was not accomplished until late in 1941. The cause of pneumoencephalitis was then shown[1,3] to be a filterable virus which could be propagated in chicken embryos.

The average mortality in outbreaks of pneumoencephalitis has been small, but in some instances as many as 50 per cent. of the affected chickens have died. The disease is always of economic importance to the owners of infected flocks, however, because of the loss resulting from its temporarily depressant effect on growth or egg production. The gross lesions seen in affected chickens are mucous exudate in the trachea and, in some cases, cloudiness of the membranes which form the air sacs and mesentery. After continued propagation in embryos or rapid passage through a series of chickens, however, the

[1] J. R. Beach, *Proc. 46th Meet. U. S. Livestock Sanitary Assoc.*, 203, 1942.
[2] J. R. Beach, *Nulaid News*, 18: 13, 1940.
[3] D. E. Stover, *Amer. Jour. Vet. Res.*, 3: 207, 1942.

virus becomes so highly virulent that inoculation of chickens with minute doses of it causes death in 4 to 6 days, and hemorrhagic lesions, particularly of the proventriculus and small intestines, are present in a majority of those which succumb. An interesting, and as yet unexplained, phenomenon of the artificially induced disease is that the infected chickens do not have the respiratory symptoms which are predominant in the natural disease. This applies to chickens infected by inoculation with material from field cases as well as to those inoculated with cultured virus.

Because of the highly virulent nature of the cultured pneumoencephalitis virus, it seemed of interest to determine if it might be related to the viruses of Newcastle disease or fowl plague, two highly fatal diseases of chickens which were not present in the United States. Through the cooperation of the Bureau of Animal Industry of the U. S. Department of Agriculture, a small quantity of anti-serum for each virus was received from England in February, 1943, and was used for *in vitro* neutralization tests with pneumoencephalitis virus. In these tests, mixtures of equal parts of serum, undiluted or diluted with saline, and of embryo-cultured virus were prepared and used for the intramuscular inoculation of chickens, 61 days old, in doses of 0.1 cc.

Chickens were not infected by inoculation with 1,000 infective doses of pneumoencephalitis virus when it was mixed with an equal quantity of undiluted or 1:10 or 1:100 dilutions of Newcastle disease immune serum. The virus was not affected, however, by mixing it with the fowl plague immune serum. These results indicate that the virus of pneumoencephalitis is immunologically identical with the virus of Newcastle disease.

It is hoped that the further studies of avian pneumoencephalitis which are in progress may yield an explanation of marked difference between the characteristics of the natural and artificially induced disease.

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THE ISOLATION OF THE ST. LOUIS ENCEPHALITIS VIRUS FROM CHICKEN MITES (DERMANYSSUS GALLINAE) IN NATURE*

EPIDEMICS of St. Louis encephalitis have occurred in St. Louis and in St. Louis County in 1933 and 1937. Since the epidemic of 1937 sporadic cases of the disease have been identified[1, 2] in St. Louis County. Although few in number the occurrence of these sporadic cases indicates that an endemic focus exists. The St. Louis area seemed to offer the opportunity for investigating the problem of inapparent viral infection in a community during a non-epidemic period. Therefore, a survey for the presence of type specific antibody to the St. Louis encephalitis virus in the human and animal population of the St. Louis area was undertaken by one of us to determine to what extent the population is being immunized to the virus of St. Louis encephalitis. In this study the data[3] obtained up to this time indicate that few individuals who have come into St. Louis County since 1937 show specific antibody to the St. Louis virus. On the other hand, a significant number of chickens, approximately one year of age, has shown a low titer of neutralizing antibody for the virus.

The work of Hammon *et al.*[4] has shown the natural occurrence of the St. Louis encephalitis virus in the mosquito *Culex tarsalis Coquillet* during epidemic periods. The experimental transmission of the St. Louis virus in chickens and pigeons by 9 species of mosquitoes from 3 genera has been reported.[5] These findings, together with other epidemiological studies by Hammon and his colleagues,[6] appear to indicate beyond reasonable doubt that the mosquito is a vector concerned in human epidemics of St. Louis encephalitis. However, the demonstration of neutralizing antibody in a significant number of one-year-old chickens in certain flocks in an area where the human population does not appear to be developing antibody suggested the possibility that some blood-sucking vector which does not bite man was transmitting the disease to fowl.

The common chicken mite, *Dermanyssus gallinae*, frequently infests fowl in this area. The chicken mite belongs to the same order of Arachnida as does the tick and the life cycles of the two are comparable in so far as the mite requires a blood meal before the moulting of the nymphs and before the oviposition by the adult females.[7] Therefore, the chicken mite seemed a possible vector in light of the experiments which have shown that ticks, under experimental conditions, are capable of becoming infected with two neurotropic viruses, the *Dermacentor andersoni* with the western equine encephalomyelitis virus[8] and the *Dermacentor variabilis* with the St.

* From the Department of Pathology and the Department of Pediatrics, Washington University School of Medicine. Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

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[2] R. J. Blattner and F. M. Heys, to be published.

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[5] W. McD. Hammon and W. C. Reeves, *Jour. Exp. Med.*, 78: 241, 1943.

[6] W. McD. Hammon, W. C. Reeves, B. Brookman and C. M. Gjullin, *Jour. Inf. Dis.*, 70: 278, 1942.

[7] H. P. Wood, *U. S. Department of Agriculture Bull.* No. 553, 1917.