These were shown by sera from a case of severe gastro-intestinal bleeding with many transfusions, and from three cases of secondary syphilis, two immediately after the 3-day arsphenamine drip treatment, the other immediately after 4 days' treatment with penicillin, 600,000 units in all. Malaria appeared rigorously excluded in two of the presumably false-positive cases and unlikely in the others. In three of these instances complement was also fixed strongly with *gallinaceum* antigen.

In the sera of patients with malaria, the reaction with normal stromata appeared less frequently in paretics with induced malaria than in chronically relapsing *vivax* cases. In the serological check-up of the latter group, then, the readily available normal human stromata appear to be about as sensitive an indicator as the more difficultly accessible and expensive *gallinaceum* antigens thus far available. The experimental basis for this conclusion is summarized below for 167 sera of 23 chronically relapsing *vivax* patients studied at weekly intervals for one to three months:

Antigen:	*Pl. gallinaceum*	Normal human stromata
No. of patients positive (++ fixation or more) at least once	16	14
Per cent. positive patients	70	61
No. of positive reactions in 167 tests	37	47
Per cent. positive reactions	22	28

Kligler and Yoeli[4] have noted that *knowlesi* and *gallinaceum* antigens are of roughly equivalent value in the complement fixation tests.

The immunologically non-specific malaria reaction with normal human stromata is not of the Forssman type, since all sera showing agglutination of sheep cells were absorbed with these before carrying out the complement-fixation tests. The reaction is apparently due to an auto-antibody, but occurs with the stromata of other species as well. A detailed discussion will be given in a later, more complete publication.

The normal human stromata antigen is prepared as follows: Washed human red cells, preferably from freshly drawn blood, are poured into several volumes of chilled water saturated with CO_2. The precipitated material is centrifuged in the cold, washed several times with cold 0.2 per cent. NaCl solution, taken up in a 2:1 mixture of cold 0.9 per cent. NaCl and 1.26 per cent. $NaHCO_3$ solutions, and frozen until the next day, when it is thawed and centrifuged. The insoluble material, which carries the major portion of the active antigen, is suspended smoothly in 0.9 per cent. NaCl solution and lyophilized in small quantities in ampoules which are then vacuum-sealed and stored in the cold. For use, each sample is rehydrated and diluted to about four blood volumes, or more if necessary in order not to exceed one quarter of the minimum anticomplementary dose. Different preparations varied in reactivity, but not in relation to the blood groups involved, some of the most active lots deriving from O cells.

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INDUCTION OF LEUKEMIA IN MICE[1]

MOUSE leukemia appears spontaneously in a high percentage of mice of particular inbred strains.[2,3,4,5] The disease can be induced in others by the administration of carcinogens,[5,6,7,8] exposure to x-rays,[9,10] or injection of estrogenic hormones.[11,12] Onset of the disease has been accelerated by the action of carcinogens in three of the four high leukemia strains tested;[5,6,7,13] similar studies have not been reported on x-rays and estrogens. A low leukemia strain susceptible to the induction of leukemia with estrogens proved to be resistant to induction of the disease with one of the carcinogenic hydrocarbons, methylcholanthrene.[14] X-rays and methylcholanthrene were independently and synergistically leukemogenic for F_1 hybrids of Furth's Rf and Ak stocks;[15] synergism was not demonstrable for strain dba mice which proved susceptible to induction of leukemia by methylcholanthrene but not with x-rays.[16]

The present investigation was undertaken to determine to what extent susceptibility of a strain of mice to one leukemogenic influence implies susceptibility of the same strain to other leukemia-inciting physical or chemical agents. Mice of four stocks (strains F, A,

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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

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