and, in particular, of the thymus. This phenomenon may explain in part the protective effect of homologous marrow cell suspensions and activated ectopic marrow against lethal doses of irradiation in mice and rats (*4, 5*). In view of the inhibitory effect of thigh shielding on the development of radiation-induced lymphoid tumors, it seems reasonable to predict that a similar, though perhaps less pronounced, inhibition of this neoplastic process will be observed after injection of exogenous marrow cells into irradiated mice.

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The Interaction of Genetic and Environmental Influences Affecting the Incidence of Avian Leucosis

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There is some doubt whether the various forms of avian leucosis, such as neural, visceral, and ocular lymphomatosis, erythroblastosis and granuloblastosis (the last two are uncommon), can be induced by a single virus, or whether several different viruses are involved, but there is agreement among pathologists and poultrymen that the disease is the most serious one afflicting domestic fowls today.

Most mortality from leucosis occurs between 2 and 16 months after hatching. In that period deaths from this disease may reach 50% or more. Recent tests of about 3300 severely exposed female chicks representing 36 different, improved strains showed that about 20% died of neoplasms (nearly all leucosis) before reaching 500 days of age (*1*).

In the light of present knowledge, the most important factors determining the mortality from leucosis in any exposed flock seem to be (*a*) the genetic constitution of the birds, (*b*) the age at which they are exposed, and (*c*) the severity of that exposure. This last apparently depends in part upon environmental conditions still unknown. It is the purpose of this note to show how these 3 factors interact and how knowledge of that interaction can be used to attain a satisfactory measure of control.

(*a*) Genetic resistance to leucosis has been studied continuously by the writers for the past 18 years. The feasibility has been demonstrated of breeding strains of White Leghorns capable of high egg production and so resistant that, when severely exposed to leucosis, mortality from that disease is almost negligible. Previous reports (*2, 3*) showed that deaths from neoplasms among these birds up to 500 days of age in 3 successive years, 1944–46, varied only from 5–8% in unculled flocks. In the birds hatched in 1951, mortality from leucosis alone (excluding other neoplasms) in that same period among 2177 females of the C- and K-resistant lines was only 2.7%, but it was 61% for birds of the susceptible strain. These had been mixed with the others since hatching and even during that process.

(*b*) The relation of age to susceptibility became clear when different investigators showed independently that chicks not genetically resistant to leucosis, when reared in complete isolation until about 5 months of age, are then highly resistant when brought to infected premises (*4, 5*). Subsequent experiments in which chickens reared in isolation, and hence presumably uninfected, were taken at various ages to infected quarters showed that most of this resistance had been developed by 10–16 weeks (*6, 7*) and in some cases even at 6 weeks (*8*) or 30 days (*9*). The utilization of this knowledge for control of the disease has been difficult, and in most cases impracticable, because few poultrymen can afford to maintain a separate, isolated establishment for rearing their birds.

(*c*) The severity of natural exposure varies greatly from flock to flock within any one area, from year to year in any one flock, and even from one weekly hatch to the next at any one farm. Among environmental factors that might contribute to this variability, only one has been clearly incriminated, namely, the proximity of the chicks to adult fowls during the critical first few weeks after hatching. During 3 successive years, deaths from neoplasms (mostly lymphomatosis) up to 500 days of age among birds that had been brooded only 40 feet from adult stock were 1.6–4 times as numerous as in full sisters that had been brooded over 100 feet from older birds (*10*). Although that difference in environment, with its accompanying difference in severity of exposure, was maintained only during the critical first 13 days from hatching, after which all chicks were kept on the same rearing range until 5 months old, it proved to be the deciding factor (apart from genetic resistance) in determining the amount of mortality from leucosis in these birds at later ages.

The relation of these three interacting factors—inheritance, age at exposure, and severity of exposure—to mortality from leucosis is clearly revealed in Fig. 1, which shows the mortality from that disease in the 4 annual generations of our White Leghorns raised in the years 1948–51. All females alive at 6 weeks of age are included, and, for simplicity, those of the 2 resistant strains are shown as a single group.

In 1948, 1949, and 1951, chicks exposed right after hatching apparently underwent the usual severe exposure desired to facilitate selection, and, as a result, mortality from leucosis in the susceptible strain ranged from 51–64.5%. Among birds of the resistant strains, which had been brooded, raised, and maintained with the others, corresponding mortality was only 2.7–7.8%.

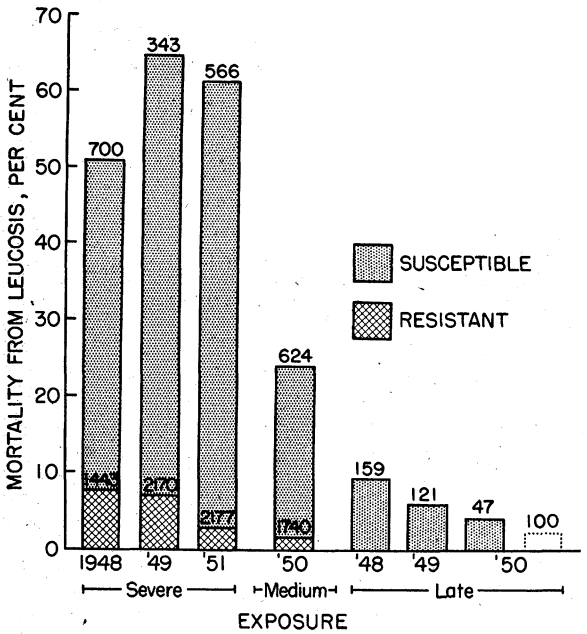

FIG. 1. Mortality from leucosis from 42–500 days of age in 4 annual generations of White Leghorns. Figures above the columns give the numbers of females in the different groups. The protective value of genetic resistance is shown by comparisons between the resistant and susceptible strains, when exposure was severe, and also in 1950, when it was of medium intensity. The protective value to a genetically susceptible stock of isolation during the critical early ages is shown by comparisons in 1948 and 1949 between such birds exposed severely (right after hatching) and their full sisters not exposed until 160 days later. The short dotted column at the right represents genetically resistant birds exposed late, among which not one died of leucosis.

The differences between these groups in all 3 years show clearly the extent to which resistance and susceptibility depend upon the genetic constitution.

However, in both 1948 and 1949, two of the 8 or 9 weekly hatches of the susceptible stock were sent right from the incubators to a distant farm that carried no other fowls. From this isolation the birds were not returned to infected premises until about 160 days old. With that comparatively late exposure, mortality from leucosis was reduced to 9.4% in one year and 5.8% in the next. Comparison of these low figures with the corresponding high ones for full sisters severely exposed in the same 2 years shows how protection by isolation during the critical early stages of life will reduce subsequent mortality to a comparatively negligible minimum, even in a stock that is genetically highly susceptible. Details of these experiments were given elsewhere (*11*).

In 1950 mortality from leucosis was only 23.9% in the susceptible strain and 1.5% in the resistant ones. That the exposure of these chicks to the causative agents had been of only medium intensity was obvious, but the reasons for it were not. Conditions under which the birds were brooded and reared (*i.e.*, the exposure) did not differ in any recognizable way from those of other years. Any possibility that the strains had undergone radical changes genetically seems unlikely because (*a*) mortality was simultaneously reduced in all 3 strains below the levels of previous years, and (*b*) in 1951 it was restored to a more normal 61% in the susceptible line.

Such inexplicable variations from year to year in the severity of exposure, along with similar differences between chicks hatched early and those hatched late in the same season, constitute some of the difficulties confronting those who study the etiology of avian leucosis. While it is desirable in genetic studies to eliminate such variation by inoculating all birds with a standard dose of the pathogen, attempts to do so have thus far failed because response to artificial inoculation apparently gives little or no measure of familial differences in susceptibility to infection through normal channels (*12, 13*).

With the medium exposure in 1950, the difference in mortality between the different stocks was greatly reduced, and the protective value of genetic resistance was correspondingly less evident. As in the 2 previous years, some birds of the susceptible stock were reared in isolation, but a change was made by sending with them chicks of the resistant strains. These were all returned to infected premises at about 160 days, as before. Mortality from leucosis to 500 days of age was only 4.3% in the susceptible stock, but not one of the 100 females of the resistant strains died of that disease. The contrast between these figures and those for the birds exposed early in the same year shows the protection afforded by isolation. It is also evident that the dams of these isolated chicks, whether genetically resistant or genetically susceptible, did not transmit, through the egg, to their offspring any pathogen that could destroy that protection.

Altogether, these results over a 4-year period show that satisfactory control of leucosis can be attained (1) in spite of severe exposure, by using a highly resistant stock, or (2) even with a genetically susceptible stock, by rearing the birds in isolation.

Unfortunately few poultrymen can provide the complete isolation best afforded by a separate farm for rearing, and the number of strains genetically resistant to leucosis, though constantly increasing, is still far too few. However the roles of heredity and environment in the causation of this disease are not all-or-none reactions. By using the most resistant stock available, and by brooding and rearing the chicks as remotely from adult stock as is practicable, mortality from leucosis should be much less than when either of these precautions is ignored. Methods of attaining such control, and of breeding resistant stock with a minimum of risk, have been discussed elsewhere (*14, 15*).

Leucosis is a serious problem in many countries. In the United States, it has been estimated to cause an annual loss of 65 million dollars (*16*). It is not likely to be eradicated, nor can it be controlled at present by any means other than those discussed above. Animal breeders and animal pathologists have lagged notoriously behind plant breeders and plant patholo-

gists in combating disease by the development of resistant strains, but it seems highly desirable that this useful weapon be utilized in the fight against leucosis. Numerous poultry breeders are now attempting to develop their own resistant strains. Records in the first 2 New York Random Sample Laying Tests, in which chicks from leading poultry breeders are deliberately exposed to leucosis, show that some of them have already succeeded to remarkable degrees (*1*). It is to be hoped that such tests will be duplicated elsewhere, and that the encouragement thus given to the production of genetically resistant strains will eventually lead to a wider distribution of such desirable stock.

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Comments and Communications

"True" Scientists

I AGREE with Dr. Hammett (SCIENCE, **117**, 64, [1953]) that a degree of detachment or objectivity is desirable in the pursuit of science. However, his letter makes four points with which I cannot agree, and which I think deserve comment in times such as these. These points are: (1) that scientists practice science for diversion; (2) that "true" scientists are judged by standards different from those applied to other people; (3) that a "true" scientist should not be interested in ". . . wages, fame, or fortune . . ." or in the conditions under which he lives; (4) that "true" scientists are disappearing. For a scientist, detachment in his work is fine, but detachment from his environment may be fatal.

The *American Collegiate Dictionary* gives two definitions for "diversion," either of which, if accepted, makes me take a cloudy view of those scientists whom Dr. Hammett uses as bases for his statement: "Diversion, *n.* 1. act of diverting or turning aside, as from a course . . . 2. distraction from business, care, etc.; recreation; entertainment; amusement; a pastime." Neither of these represents the motivation of the scientists I know; are they representative of Dr. Hammett's "true" scientists?

I cannot deny the statement that a true scientist is concerned with following his vocation to the best of his ability within his capacities. So is a true sausage-stuffer or a true bank-robber. Certainly this spirit does not differentiate the scientist from his fellow humans. I object violently to the idea that the scientist is set apart from the rest of mankind by virtue of what he does. The idea of the scientist as a funny man in an ivory tower, who doesn't care of what form of government he is a part, was dispelled from the minds of scientists, press, and public by the last war. In addition to being untrue, such a belief is prejudicial to the interests of science, since in these days to be different is to be suspect.

Dr. Hammett's "true" scientist would be much too busy in his ill-equipped laboratory ever to sit with a plebeian bottle of beer (domestic, of course) and watch his small-screen table-model TV set, or to take his undoubtedly large family for a ride in the Austin. The few hours per day at home would be spent in deep thought, punctuated intermittently by trips to the outside plumbing. Who can set the scientist apart from his neighbor by stating that he cares little about the conditions under which he lives? Why should possession of a certain standard of living divert a scientist from practicing science any more than it diverts a carpenter from practicing carpentry? The professional scientist is not different from others in needing a satisfactory standard of living, in desiring rewards commensurate with his training and productivity, and in wanting to play a part in his own future and that of his family, if a "true" scientist is allowed one. Any scientist, as any other mature person who works for a living, is very properly concerned with ". . . wages, fame, or fortune . . .", since they help shape the society in which he lives.

Dr. Hammett is worried because "true" scientists are becoming extinct. I cannot dispute this, because I have no notion of what "true" may mean applied to scientists. However the race of practicing scientists is certainly increasing. I can cite, for example, the extent of support of "fundamental" research, numbers of research papers in various fields, etc. Research today is a major industry, dependent upon the output of the serious scientist.

Whatever the motivation of Dr. Hammett's letter, at least one practicing scientist disagrees with it. If