tempted to speculate that the chemical structure of highly polymerized RNA is perhaps different from that of DNA and contains cross links at the 2-position of the ribose. A purified sample of the $S = 24$ component should be very useful for fundamental studies of the structure of RNA. Attempts are being made to obtain homogeneous material (*11*).

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Age, Growth, and the LD_{50} of X-rays

In the case of x-rays and other ionizing radiations, the dependence of the acute LD_{50} on age has not been determined throughout the life-span of a mammal, although it is known that immature mice (*1*) and rats (*2*) are more sensitive than mature ones, and it has been indicated that mature female rats are more sensitive at 16 months than at 6 months (*3*). The present experiments contribute data for the first 75 to 80 percent of the ~900-day life-span of CAF_1 mice.

The biological, physical, and statistical methods employed are described elsewhere (*4*). The mice were weaned at about 30 days and experienced puberty at about 50 days. The radiation factors were 250-kvcp x-rays; half-value layer, 1.6 mm Cu; tissue-dose rate, ~35 r/min. Deaths were counted during the 28-day period after a single whole-body exposure. For each LD_{50} determination, there were 4 to 6 dose-groups of about 10 animals each, thus employing a total of about 50 animals.

The results (Fig. 1) showed that the LD_{50} was a linear function of log age, A, from 37 to 105 days (*5*) but was practically constant from 115 to 709 days (*6*). Thus the hypothesis that the "adult" LD_{50} is proportional to remaining life-expectancy (*7*) was not confirmed.

A change occurred in the response of the oldest animals, shown by the increased value of S, the average of the LD_{84}/LD_{50} and LD_{50}/LD_{16} ratios. For ages 600 to 709 days, three determinations of S were 1.16, 1.17, and 1.24. For ages 37 to 434 days, the mean and standard deviation of 29 determinations of S were 1.072 ± 0.024. Radiobiologically, therefore, the population became more variable at 600 days.

The diphasic dependence of the LD_{50} on log age (Fig. 1) was similar to that for log body weight (*8*), a parameter of growth. It may be said, therefore, that for the first 80 percent of the life-span the LD_{50} continued to increase until the growth rate fell to its fully mature level, after which it remained constant.

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5. $LD_{50} = -26 + 336\ A \pm 20$; $\hat{\sigma}$ (336) = 35.
6. $LD_{50} = 721 - 23\ A \pm 32$; $\hat{\sigma}$ (23) = 31.
7. H. A. Blair, *Proceedings of the International Conference on the Peaceful Uses of Atomic Energy, Geneva* (United Nations, New York, 1956), vol. 11, p. 118.
8. Let W = log weight (g). For males, W(37–103 days) = $0.43 + 0.50\ A \pm 0.02$; W(115–709 days) = $1.30 + 0.083\ A \pm 0.02$. For females, W(37–103 days) = $0.54 + 0.41\ A \pm 0.02$; W(115–600 days) = $1.08 + 0.14\ A \pm 0.02$.

27 September 1956

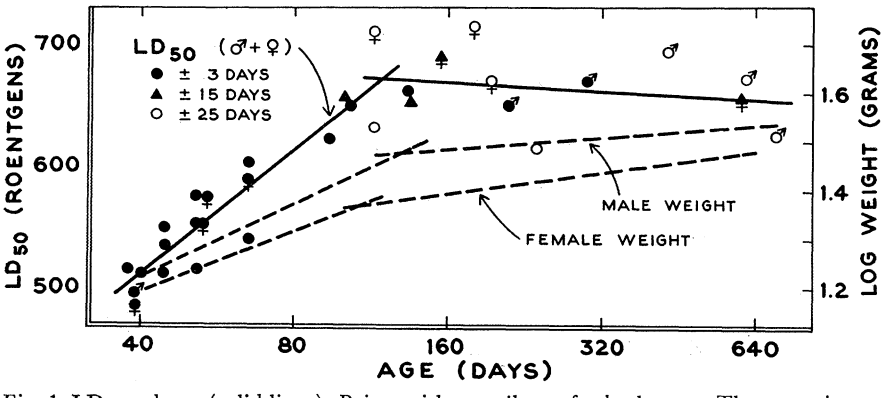

Fig. 1. LD_{50} and age (solid lines). Points without tails are for both sexes. The range in age for each point is indicated by the symbol. The dashed lines relate log body weight to age for the animals of these experiments.

Fowl Pox Vaccine Associated with Parthenogenesis in Chicken and Turkey Eggs

Studies were initiated at the Agricultural Research Center, Beltsville, Maryland, in 1953 to determine which factor or factors were responsible for the parthenogenetic development encountered in the eggs of chickens (*1*) and turkeys (*2*). Since these studies were initiated more than 13,000 infertile chicken eggs have been incubated and examined for parthenogenesis. These eggs were produced by chickens of four different breeds—namely; New Hampshires, Barred Rocks, Rhode Island Reds, and Dark Cornish—as well as two crosses involving New Hampshires and Dark Cornish. Data obtained showed an inherited tendency on the part of certain individuals, as well as on the part of different breeds, to produce eggs that will develop parthenogenetically. The eggs of Dark Cornish and Cornish crosses were the only ones encountered showing any appreciable amount of parthenogenesis that could be detected macroscopically. The development in the case of chicken eggs consisted typically of a limited growth of membranes. These data also indicate that this tendency can be increased or decreased by selective breeding. The causal agent remains unknown. However, it was found in 1955 that a virus may be involved in the initiation of parthenogenetic development in the eggs of Dark Cornish chickens.

In 1955, the eggs laid by 42 pullets from 19 January to 4 May were incubated and examined for parthenogenesis. On 5 May, these same 42 pullets were vaccinated, 13 with pigeon pox and 29 with fowl pox vaccine. All eggs laid by these 42 females during the following 4 months were likewise examined for parthenogenesis. In each case the method of testing was the same, an initial 9- to 10-day period of incubation, following which the eggs were broken and the germinal discs were examined macroscopically for parthenogenetic development.

In 1956, the eggs of 35 Dark Cornish females were examined before and fol-

lowing vaccination with fowl pox virus on 1 April. The results of the 1955 and 1956 tests are presented in Table 1.

These data reveal that, in 1955, more than 3 times as much parthenogenesis was encountered in eggs laid after vaccination with chicken pox virus than was found in eggs laid by the same birds prior to this time ($\chi^2 = 33.0$, degrees of freedom = 1, $P < 0.001$). It is interesting to note that the milder pigeon pox virus was less effective in inducing parthenogenesis ($\chi^2 = < 1$, degrees of freedom = 1, $P < 0.35$). In the 1956 tests involving fowl pox vaccine, the difference was even greater, more than 9 times as much parthenogenesis appearing in eggs of the same birds following vaccination than was found prior to the introduction of the virus ($\chi^2 = 168$, degrees of freedom = 1, $P < 0.001$). The highest incidence of parthenogenesis was encountered in eggs laid 30 to 60 days after the birds had been vaccinated. Facilities were not available for the maintenance of nonvaccinated birds during the full term of these tests. However, data on the incidence of parthenogenesis in turkeys does not show any appreciable seasonable variation (*3*).

A further indication that fowl pox vaccine may be involved in the initiation of parthenogenesis is furnished by data obtained in 1956 with turkey eggs. In these tests a total of 3110 eggs laid by two groups of turkeys were examined for parthenogenesis over a period of 3 months.

One group was composed of 16 nonvaccinated birds, the other of 49 turkeys that had been vaccinated for fowl pox at 7 weeks and again at 30 weeks of age. Since the ancestry of each bird was known, it was possible to select birds for these tests so that in each group full sisters would be represented. The 16 nonvaccinated turkeys, representing 12 families, were housed in wire cages within a screened building where they could be kept isolated from other birds. The 49 vaccinated birds, representing the same 12 families, were kept in turkey houses where they were in direct contact with other vaccinated birds. All birds involved in these tests were virgins, having been segregated from males at 4 weeks of age, and all received the same type of feed.

A total of 738 eggs was produced by the 16 nonvaccinated turkeys during the 3-month test period. Of these, 180, or 24.4 percent, showed parthenogenetic development when they were examined following a 9- to 10-day period of incubation. Membranes only were found in 144; 47 showed blood formation in addition to membranes; and 19 eggs contained well-formed embryos.

The 49 vaccinated females produced a total of 2362 eggs during the same period. Parthenogenetic development occurred in 750, or 31.8 percent, of these eggs.

Table 1. Incidence of parthenogenetic development found in eggs of Dark Cornish chickens before vaccination and following vaccination with pigeon pox and fowl pox vaccine.

Item	1955		1956
Before vaccination			
Number of birds on test	13	29	35
Number of eggs tested	497	1190	1294
Number of eggs showing parthenogenetic development	9	12	21
Percentage of eggs showing parthenogenetic development	1.8	1.0	1.62
After vaccination with	*Pigeon pox*	*Fowl pox*	*Fowl pox*
Number of birds on test	13	29	35
Number of eggs tested	808	1653	1675
Number of eggs showing parthenogenetic development	23	53	266
Percentage of eggs showing parthenogenetic development	2.9	3.2	15.9

The parthenogenetic development was classified as follows: 548 eggs contained membranes only; 100 contained blood in addition to membranes; and 102 contained well-formed embryos.

The records of these two groups of full sisters reveal that a significantly higher percentage of parthenogenesis occurred in the eggs laid by the vaccinated group of turkeys, 31.8 percent as compared with 24.4 percent of total eggs tested ($\chi^2 = 12.3$, degrees of freedom = 1, $P < 0.001$) (*4*). This increased incidence of parthenogenesis in the eggs of the vaccinated group was evident in each of the three categories listed—that is, membranes, blood formation, and embryos.

The results secured in the foregoing tests indicate that some agent, possibly of a physiological nature, had some part in initiating parthenogenetic development. When this agent was present at, or near, the optimum level in the blood stream of genetically susceptible hens, the development initiated tended to proceed further and show a higher degree of organization. The agent, whatever its nature, possibly may be transmitted from parent to offspring through the egg, since, in eggs of both nonvaccinated groups of chickens and turkeys, a considerable percentage of eggs was encountered showing parthenogenetic development. This was true, even though these nonvaccinated females were isolated from other birds, and every attempt had been made from time of hatching to keep them disease-free.

It would appear, therefore, that at least two conditions are necessary before an advanced type of parthenogenesis occurs. First, a susceptible strain of birds is necessary. This implies not only that birds produce readily activated eggs but also that the parthenogenetic cells possess a sufficiently high inherent viability to survive until such time as the eggs are placed in an incubator. In this sense the eggs produced by most of our domestic breeds of chickens cannot be considered as susceptible, since in nearly every instance the parthenogenetic cells have died by the time the eggs are laid. The second condition is that an activating agent must be present in the blood stream. When this agent is present at, or near, an optimum level, it has the effect of inducing parthenogenetic development.

It is not known, at present, whether the fowl pox virus as such is the sole agent initiating parthenogenesis or whether some contaminant which may be present in the vaccine is also involved. Neither is it known just how this virus may exert its effect, whether it is a direct or an indirect one.

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Spontaneous Activity in Denervated Insect Muscle

Following section and peripheral degeneration of the motor nerve supply, insect muscle generally has been thought to become inactive, even in response to direct electric stimulation (*1*). Recent physiological investigations of the thoracic spiracle muscles of cockroaches, *Periplaneta americana* (L.), have demonstrated a strikingly different mode of behavior, which is deemed of sufficient interest to the general question of the irritability of insect muscle to warrant preliminary description.

For each thoracic spiracle of the American roach there is a single occlusor