

AN ANALYSIS OF FIVE YEARS
OF BREEDING FOR LIVABILITY
IN RHODE ISLAND REDS

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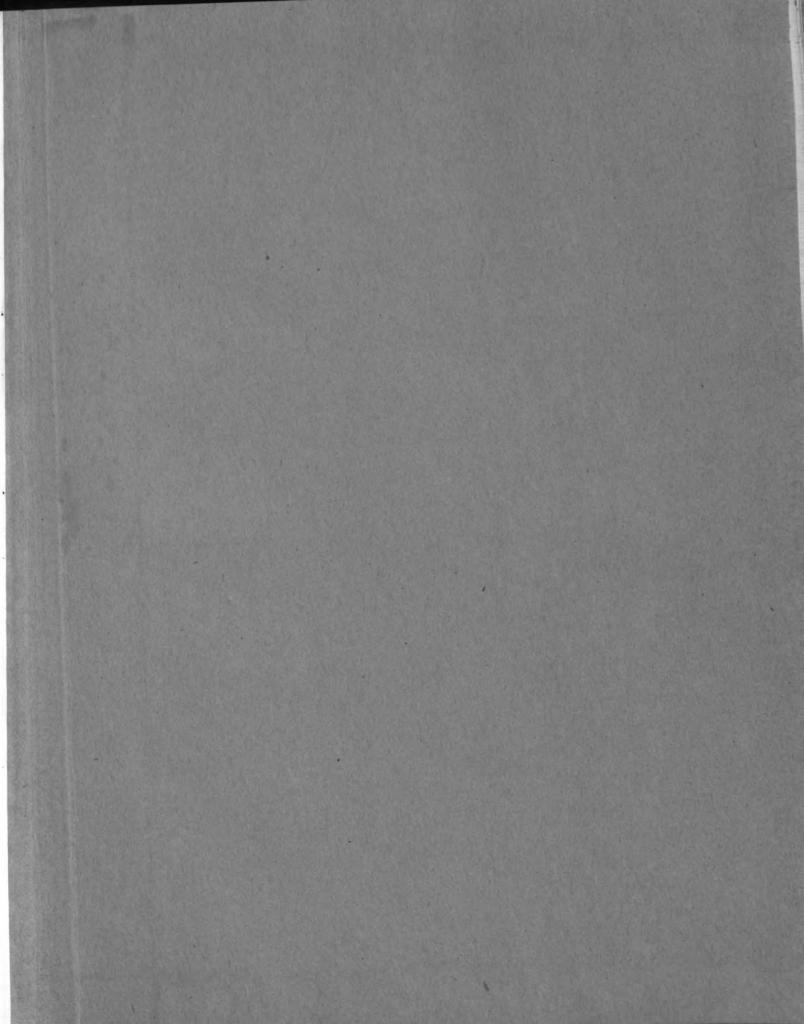
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# AN ANALYSIS OF FIVE YEARS OF BREEDING FOR LIVABILITY IN RHODE ISLAND REDS

BY

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#### INTRODUCTION

# The Mortality Problem

Heavy loss from disease is recognized as one of the limiting factors in the poultry business throughout the entire United States. The economic importance of high mortality is summarized in several books (4) (31), and in many published articles (1) (2) (5) (25) (34) (37). The greatest losses occur among pullets during their first year of laying and it is this phase of the problem that is receiving the most attention from investigators.

Darrah (16) summarized the mortality records of 1073 N. Y. State poultry farms from 1926 to 1941 and reported as follows: "The death rate of chicks has dropped considerably. In the early 1930's, about 25 chicks died out of every 100 started. In 1941, only about 15 chicks died out of each 100 started. The average death rate in the laying flocks remains as high today as it was a dozen years ago. Studies of commercial poultry farms in the early 1930's showed that about 24 layers died out of each 100 kept during the year. In a 1941 study, 25 died out of each 100 kept!

Darrah showed the effect of mortality on the income of N. Y. State poultry farms in table 1. (16).

Table 1.

Relation between Death Rate and Labor Income

(1073 N. Y. State Poultry Farm Records, 1926 to 1941)

Death Rate	Labor Income
Low (11%)	<b>\$</b> 1620
Medium (22%)	1330
High (43%)	880

# Types of Poultry Farms

The development of strains of domestic fowl resistant to disease is beginning to play a major role in the disease control program of the poultry industry. For this reason, the various kinds of poultry enterprises and their relation to the disease control program should be understood. The U. S. Agricultural Census, 1940, gives the following distribution of chickens in the United States:

Table 2.

Size of flock	Average No. per flock	Percent of Group	all Chickens Cumulative
Under 50	23	20•6	20•6
50 <b>-</b> 99	63	20.9	41.5
100-199	126	27.4	68•9
200-399	242	16.9	85.8
400-799	499	6.4	92•2
800-1599	1045	4.0	96•2
1600-3199	2136	2.3	98.5
3200% over	55 <b>73</b>	1.5	100.0

Over eighty five percent of the chickens in the United States are in flocks of less than 400. Only Three eight tenths percent are in flocks as large as 1600 chickens. Most of the farms which keep small numbers of chickens, and many which keep large numbers, do not hatch their own chicks. Chicks to maintain theseflocks are purchased from hatcheries or from poultry farms which sell chicks. Darrah (16) reported that 48 out of 100 commercial poultry farms in N. Y. State sold chicks in 1941.

Commercial egg farms keep from one thousand to many thousands of laying birds and depend on the sale of eggs as their main source of income.

Most commercial egg farms buy chicks to maintain their laying flocks and they replace from seventy five percent to one hundred percent of their birds each year. This type of poultry farm has shown a rapid increase in recent years near centers of population.

Commercial hatcheries are the main source of chicks for the small flocks and the commercial egg farms. They have shown a rapid increase in both numbers and capacity since 1930. Hartman and Vickers (29) quote figures from the U.S.D.A. Bureau of Economics Hatchery Reports which show an increase from 671 million chicks hatched in 1930 to 1,620 million in 1945. In 1930, seventy percent of all chicks hatched were hatched in commercial hatcheries, and in 1945, the percent had increases to eighty-eight & six tenths percent. Hatcheries, for the most part, own few or no chickens. Eggs for hatching are purchased from small flock owners and from commercial egg farms.

Poultry breeding farms are few in number compared to the other kinds of poultry farms, but they hold a strategic position in the industry. They depend on the sale of baby chicks, hatching eggs, and breeding stock of superior quality as their source of income, rather than market eggs and meat.

In the interrelationship of these various kinds of poultry enterprises probably lies the key which might explain the great increase in mortality of poultry laying flocks. If we could determine exactly the cause, we might have an effective aid in finding a remedy. The increase in poultry mortality since 1925 has been concurrent with the rapid increase of the hatchery business. Whether there is any relation between the two is problematical, but the increased use of hatchery chicks has certainly resulted in a wider spread of gemetic and pathological factors favorable to the development of disease. The widespread exchange of chicks between different parts of the nation has brought chicks in contact with disease producing agents of a different nature, type and virulence from those of the environment of their parents. To the extent that natural and artificial selection may have developed genetic resistance in chickens which were previously kept within a given area, the introduction of chicks from other areas may have resulted in an ever increasing number of susceptible animals. Also the rapid course of pathological agents through these susceptible animals may have resulted in an increasing virulence of the organisms (8)

The use of hatchery chicks has also been accompanied by the

practice of raising chicks in larger flocks, often partly or entirely indors, at all seasons of the year. It has also been accompanied
by the practice of keeping a larger percent of pullets in the flocks.

All of these things increase the number of susceptible animals. and
the number of months in the year when there are susceptible animals
present of the right age for disease organisms to perpetuate themselves.

Most hatcheries buy eggs from small flock owners and from commercial egg farms. Male birds are sometimes purchased from poultry breeding farms for improvement purposes, or males are swapped from one farm to another. On the whole, there is no long range breeding program for improvement of resistance to disease. The practice of constantly mixing stock from various sources has resulted in a great hodge-podge of genetic characteristics which does not lend itself to improvement by selection.

The main task of selection and breeding falls on the breeding farms and the experiment stations. While these, too, have had their mortality problems, most of the work at present being done in applying genetic principles to the production of better stock is being done on these farms and the results are encouraging. In the Western

N. Y. Egg Laying test in 1945, the mortality was thirteen & five-tenths percent, compared to twenty four & seven-tenths percent the year the tests were started in 1932. (Table 3)

Table 3.

		& Mortal	ity in	Laying Tests
Year	Chicks hatched by		•	
	Commercial Hatcheries	Mich.	N. J.	West. N. Y.
1930	671,576	18	24	
1931	516,220	23	30	
1932	537 <b>,</b> 35 <b>1</b>	24	22	24.7
1933	584,547	27	26	21.9
1934	525,947	28	25	21.0
1935	649,720	30	22	20.1
1936	790 <b>•</b> 74 <b>9</b>	29	24	19.5
1937	687 <b>,</b> 595	27	<b>2</b> 5	17.7
1938	785 <b>,</b> 687	25	24	16.6
1939	916,409	28	25	14.6
1940	859,341		•	15.5
1941	1,093,300			12.4
1942	1,280,290			
1943	1,609,121			
1944	1,288,491			
1945	1,620,773			13.5

The problem of today is to get a distribution of the improved stock from the breeding farms, to the farms supplying hatching eggs, to the hatcheries, and to the farms producing eggs and meat.

Waters (47) (48) (49) (50) has proposed a "Closed flock" plan which, if universally adopted, should result in better production and lower mortality for all farmers in the country.

# Classification of Diseases.

The Ninth N. Y. State Egg Laying Tests (1940) Listed the causes of mortality of 505 birds which died in the two tests in 1940. These have been summarized in table: 4.

## Table 4.

Percent Mortality from various Causes In N. Y. State Egg Laying Tests in 1940

Cause	Percent of Total Mortality
Fowl leucosis complex	37•0
Conditions associated with egg production	18.4
Miscellaneous	16.5
Nonspecific infections	15.8
Impactions and ruptures	7.1
Nutritional	2•2
Cannibalism	2•2
Parasites	•4
Specific bacterial & virus	•4
Total	100•0

These figures are probably not representative of the average farm since they represent the best pullets from the best farms in the country, and are all pullets. Data from actual farm flocks are not available because for most deaths no positive diagnoses are made, and many birds are culled out for health or economic reasons which would have died if they had not been removed. The summary does serve to emphasize the large percent of mortality due to fowl leucosis complex and the small percent caused by specific bacterial and virus diseases. In any study of reduction of mortality by the application of gentic principles this is of considerable importance.

Beach (6) studied the autopsies of 9,526 birds which died or were culled from the University of California, poultry husbandry flock, and 1,776 autopsies of birds from California poultry farm flocks and found non-specific disease conditions responsible for sixty two & five tenths percent of the loss from the University flock and fifty nine & eight tenths percent of the California farm flocks. The distribution of the nonspecific diseases is shown in table 5.

Table 5

Percent and Distribution Nonspecific Diseases

California

#### % Mor-% of Total Distribution of nonspecific tality mortality due to non- Alimen- Urin- Repro- Misc. **s**pecific tary ary ductive Poultry Hus-22.7 62.5 28.0 15.7 39.2 16.9 bandry flock Poultry farm 46.5 59.8 32.6 flocks 19.2 31.2 16.9

The group of diseases included in the "avian leucosis complex" is classified in a report from the U. S. Regional Laboratory, East Lansing, Mich., as follows:

Lymphomatosis

Neural

Ocular

Visceral

Osteotropic

Blood forms

Erythroblastosis

Granuloblastosis

Myelocytomatosis

Sarcoma and other tumors

Methods of Disease Control

General sanitation is the method of disease control most commonly recommended to poultry raisers. A review of this subject is given by Martin (8) and by Van Es and Olney (45). As a practical means of controlling disease, the value of sanitary practices is limited to those diseases which are spread by the environment of the house and ground, and these are a relatively small percent of the total mortality of adult stock. Hutt (26) summarized the deaths of 1,922 birds which died in the N. Y. State Egg Laying tests between 1931 and 1937 and stated that thirty eight percent of the deaths were due to neoplasms, and eighty seven percent of all deaths were caused by diseases which could not be controlled by generally recommended sanitary practices.

Certain management practices are also related to disease control. The recommendation is to raise chicks in small flocks, well spread out on the land, not over 600 pullets per acre. This maintains a biological balance between the infective agents and the number of susceptible chicks. The practice of rotating ranges is an effective control of coccidiosis and worms.

Kennard (32) and Hutt (27) presented evidence that pullets survive better as layers when they are raised on the ground far enough away from mature stock to prevent infection during the first few weeks of age. Raising young stock in confinement or on wire reduces mortality during the growing period but does not increase their survival rate as layers, under ordinary laying house conditions. The practice of having all-pullet flocks reduces mortality by preventing infection from old birds, which are carriers, to young birds.

An effective means is at hand for eradicating pullorum disease from poultry of the United States by use of the agglutination test and the removal of infected birds. Tuberculosis could probably be eradicated by means of the tuberculin test.

Chickens can be successfully immunized against two specific virus diseases, fowl pox and laryngotracheitis, and recent work in New Jersey indicates success in the development of a vaccine against New Castle disease. The effectiveness of the vaccines has been well established (10) but there is still some question as to the propriety of their unrestricted sale and use.

Recent advances in the knowledge of nutrition will undoubtedly be an aid to poultry farmers in reducing mortality by establishing mutritional requirements, and aiding feed manufacturers to furnish feeds which meet the birds' needs for vitamins, amino acids, and min rals under close confinement (44).

A system of complete quarantine which would isolate individual farms from outside sources of infection is very difficult to apply, but combined with Waters "closed flock" system of breeding, which would improve genetic uniformity, it might be a practical way of reducing the high mortality rate.

There are few medicines of value in treating diseases of poultry. Round worms can be controlled by medication. Sulphur and some sulpha drugs have preventive value against coccidiosis. Sulphathiazole is useful in the treatment of coryza. Outside of these, medicine is of little value, yet farmers place their chief reliance in cures and annually spend millions of dollars for dopes and medicines (45).

The field which holds the greatest promise for the reduction of mortality in chickens is in the field of genetics. The application of genetic principles to the production of strains with livability and with resistance to disease shows considerable promise. Roberts and Card (38) in 1926, were able to produce strains with resistance to pullorum disease, and Doyle (19), in 1928, noticed differences among strains in resistance to fowl paralysis. Lambert and Knox (34) bred strains with resistance to fowl cholera. The nature of poultry diseases, which are largely neoplasms, nonspecific infections, and organic conditions such as impactions and ruptures, should be a favorable factor in efforts to increase livability by genetic selection (8).

#### PURPOSE

The object of this report is to study the results of five years of breeding which had been done.

The mortality in this flock had been reduced to a lower percent than that generally reported, even in strains of known resistance. It was thought desirable to learn what diseases were reduced or eliminated and whether the changes were due to inheritance, to changes in environment, or to chance.

Data were available to compare the mortality of young stock with the mortality of pullets raised from the survivors, as was done by Byrant (12). Changes in the age in days at which birds died from various causes would be of interest.

Opportunity was also presented to compare the causes of mortality in a flock where total mortality was exceedingly low, with the published data from other sources.

#### REVIEW OF LITERATURE

In the last few years, many reports have been made of successful experiments to reduce mortality by selective breeding. Marble (36) was able to reduce total mortality in White Leghorns from thirty nine & eight tenths percent to twenty percent infive years, and in Barred Plymouth Rocks from forty eight & seven tehths percent to twenty four & six tenths percent. Sturkie (41) reported a reduction from eighty nine percent in unselected White Leghorns to twenty seven percent after five years of breeding from families of greatest livablity. Survival age of the birds which died was increased from 229 days to

363 days. Similar results were obtained by Gildow (21) and Bostian and Dearstyne (9) and others.

Experiments including a susceptible line as well as a resistant line have been conducted by several investigators. Byrant (11) in a three year experiment, developed two strains of White Leghorns which showed a significant difference (by the chi square test) in the amount of mortality. The resistant line mortality was twenty five & six tenths percent and the susceptible line was thirty six & three tenths percent. From unselected stock in which the mortality was sixty four percent, Hutt, et al, (25) (26) (27) produced a resistant strain in which mortality was lowered from eighty three percent to thirty five percent in eight years, while the susceptible line in the same period changed from seventy four percent to fifty three percent. Taylor, et al, (43) produced strains with a significant difference in mortality between two lines.

Maintenance of a susceptible strain offers some means of offsetting the error that might be included in conclusions drawn from oneline only, because of changes in the amount and type of infection.

Other means of testing the genetic basis of resistance to disease have been used. Hutt (27) compared the Cornell strains with four other unselected strains and found the Cornell resistant line superior to three out of four of the unselected strains for livability. Hutt (26) also crossed the Cornell resistant strain on males of an unrelated strain bred for resistance. The Flprogeny had less mortality than either of the parent strains. Hutt, in the same report, inoculated part of one

year's chicks with fresh lymphomatosis tissue. The results were twenty three and one tenth percent morality in the inoculated females and thirteen percent in the controls. He concluded that natural exposure was adequate to differentiate between the resistant and susceptible strains.

Taylor, et al, (43) inoculated resistant and susceptible lines. Mortality in both lines increased but there was still a significant difference between the two lines. Burmester (14) stated that chickens immunized against cell transports of lymphoid tumor strains were no more resistant to neural and visceral lymphomatosis than uninoculated chickens. Heisdorf (24) was unable to differentiate between lines of White Leghorns selected for resistance and susceptibility to neoplasms by subcutaneous inoculation with lymphoid tissue. Placing lymphoid tissue in the crops, eyes and nostrils resulted in a highly significant difference.

The possibility of a leucoagglutination test to detect the presence of lymphomatosis in chickens has been made by stafseth and by Kisslin (33). Kisslin used the rapid plate method and antigen make from normal, strained canine lymphoctyes. Such a test, if it could be made completely accurate, would be a great aid in genetic studies by identifying birds with infection below the level sufficient to cause death, and families completely free from lymphomatosis could be identified.

#### PROCEDURE

This report is based on the records of the Rhode Island Reds on the poultry farm of Michigan State College. Selection of birds was made by J. A. Davidson on the bases of progeny test records started in 1934. Egg production, egg size and hatchability were considered as well as mortality in making selections. A few birds were discarded for surface color.

All of the Rhode Island Reds that were hatched from 1934 to 1938 are included in this report and Mortality includes death from all causes. There was no culling. The eggs from this group of birds were incubated with the eggs from the Barred Plymouth Rocks and the Leghorns, the chicks were reared with the other breeds and the pullets were housed in the same house. No attempt was made to isolate the flock as a whole from outside infection.

There were from seven to nine hatches a year, between the first week in February and the last week in March. Chicks were raised in ten by twelve colony houses, three hundred chicks per house. They were hopper fed from the beginning and grain was fed after ten days to three weeks of age. Layers were fed by the "cafeteria" method, grain and a mash concentrate.

The make up of the flock is summarized in the Appendix Tables I and II. Fifty one hens were used for breeders in 1934 and twelve additional new ones in 1935. One hundred daughters and twenty seven granddaughters of the original sixty three hens were also used as breeders. During the five years, three hundred seventy eight daughters,

four hundred ninety one granddaughters, and one hundred fifty four great granddaughters were produced. These constitute the basis of this report.

In 1934, the flock consisted of fifty one families whose average size was 3.6 birds. In 1938, it consisted of forty two families with an average size of 5.7 birds. In 1934, there were twenty one families of four and over, which made up sixty five & eight tenths percent of the flock. In 1938, the average size of the families of four and over had risen to 7.6 birds and they consistituted ninety-one percent of the flock.

The conditions under which these birds were hatched, reared and housed approximate farm conditions. All birds which died were autopspied by pathologists of Michigan State College.

Data were collected and summarized from each family for the years 1934 to 1938. This includes the number of chicks hatched, percent of total mortality and mortality from leucosis complex to 190 days of age. From 190 days to 555 days of age it includes cause of death and age in days to time of death.

The data was grouped in several ways for the sake of making comparisons:

- a) By calender years
- b) By generations
- c) Related first and second generations
- d) Related second and third generations
- e) Related first, second, and third generations

Comparisons were made in all these ways using all birds in the groups, and then restricting the numbers to birds in families of four and over. It was hoped in this way to bring out evidence of genetic similarity. Families of four and over were used because four seemed the smallest number possible to use as a test of a family, and to use a larger number would reduce the number of families included to such an extent that they would not represent the generation or year.

The comparisons were measured for significance by the "t" test\* (4)

t== >2.0 significant at 5% level t== >2.6 significant at 1% level

The "t" test is adequate to measure the significance of the difference between percentages but does not prove or disprove the validity of the comparisons. Comparisons were made between years and between generations using for comparison:

Total mortality and mortality from leucosis, Appendix,
Tables III. a,b,c,d,e,

Percent Mortality from various groups of diseases,
Appendix, Tables IV. a,b,c,d,e,

Various groups of diseases as a percent of total mortality, Appendix, Tables V. a,b,c,d,e,

Survival age in days, Appendix, Tables VI.

For convenience in summarization, the causes of mortality were grouped under the following headings: Reproductive diseases, Digestive

diseases, leucosis complex, respiratory diseases, metabolic diseases, nephritis and pertonitis, and miscellaneous diseases. The diseases included under the various headings are shown in the Appendix, Table VII. Nephritis and Peritonitis were at first included in the miscellaneious but were later taken out and grouped together because they so often account for a high percent of total mortality, not because of any relationship between the two.

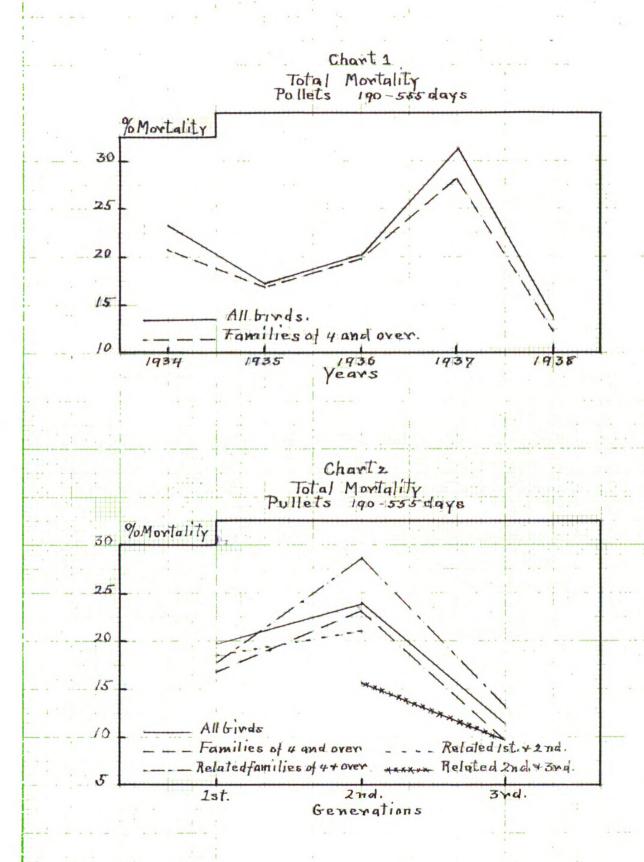
Correlations were determined between the mortality of chicks and the mortality of pullets raised from the survivours, for total mortality, and mortality from leucosis complex.

#### RESULTS

Total mortality in this flock was reduced to thirteen & five tenths percent after five years of selection. The first year of the study it was twenty three & two tenths percent. This twenty three & two tenths percent is the mortality of the 1934 progeny, not the mortality of the parents. Mortality records of the parent stock before 1934 were not available.

The reduction was not consistent from year to year (Chart 1). In the families of four and over, total mortality followed the same general pattern but was about two percent less than when all birds were considered.

When the flock is considered by generations instead of calender years, the results are shown in table 6.



Generation

Table 6.

Total Mortality in Percent, by Generations

		Generati	011
Group	First	Second	Third
All Birds	19.8	23.1	11.0
All birds in families of 4 & over	16.8	21.0	9•5
Related 1st. & 2nd. generations in families of l&over	18.3	21.0	
Related 2nd. & 3rd. Generations in families of 4 & over		15.6	9•5
Related 1st., 2nd., & 3rd,. generations in families of 4 & over	17.1	28.7	12.9

In all groups, second generation mortality was higher than first generation, and third generation was lower than either first or second.

Changes in mortality from leucosis complex are greater and show more consistent pattern than changes in total mortality (Chart 3).

This is especially evident when the data is arranged by generations (chart 4). In all but the related 1st., 2nd., and 3rd., generations group; the second generation is lower than the first and the third is lower than the second. This one group which is the exception contains the smallest number of birds. Here the second generation was a little higher than the first but the third generation was the lowest of all thegroups.

Chick mortality, on the whole, increased over the five year period (Charts 5 & 6).

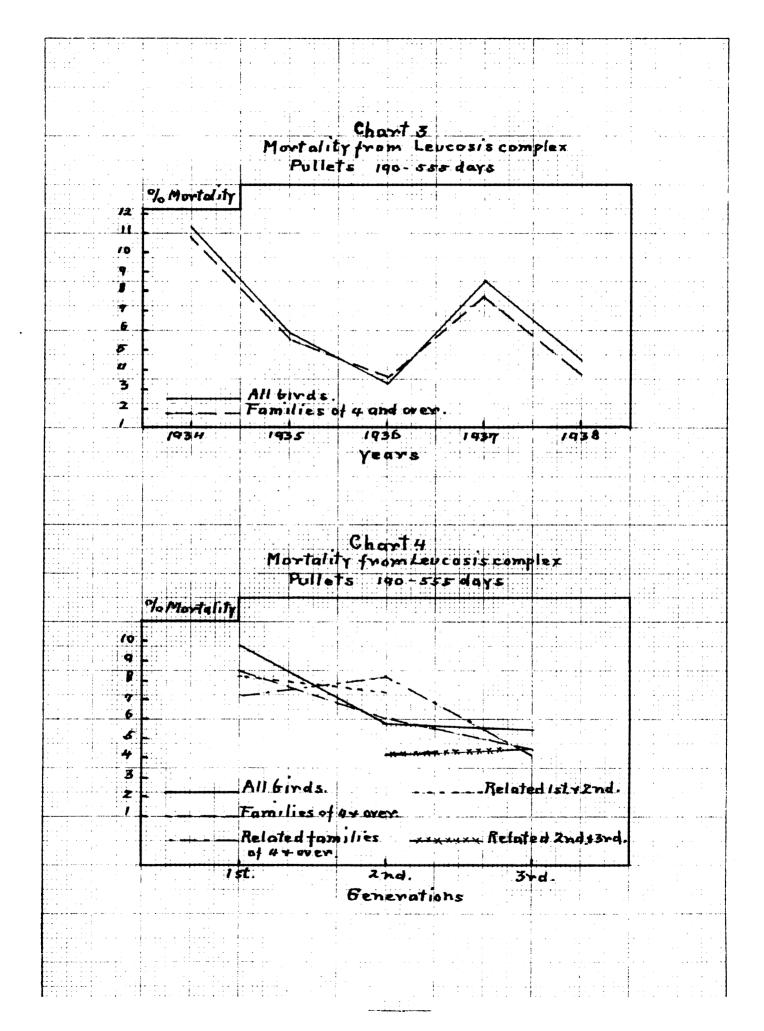
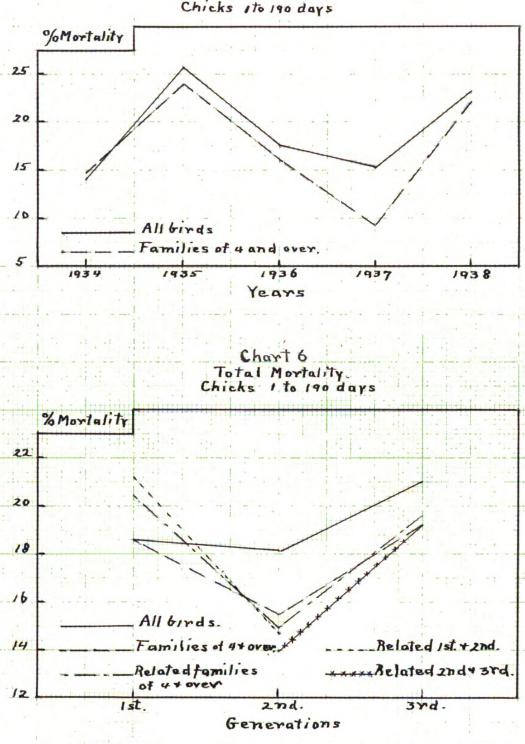


Chart 5 Total Mortality Chicks 1 to 190 days



There was no relation between chick mortality and pullet mortality when total mortality is considered. In fact, changes in pullet mortality seem to be just opposite to changes in chick mortality.

Changes in chick mortality from leucosis complex are quite consistent and downward and more related to changes in pullet mortality than when total mortality is considered. (Charts 7&8).

There seems to be a relationship between mortality from leucosis complex and mortality from other causes but the results are not always consistent (Appendix, Table IIIa). From 1934 to 1936, mortality from leucosis complex decreased from eleven & three tenths percent to three and three tenths percent and most of the other diseases decreased, yet mortality from the metabolic diseases increased.

From 1936 to 1937, leucosis complex and most of the other diseases increased, while the metabolic diseases decreased.

When the birds are summarized by generations (Appendix, Table IVc), the loss from leucosis complex decreases each year, while losses from other diseases show a decided increase in the second generation.

In 1938, in the group which includes all birds in families of four and over (Appendix, Table IVb), total mortality was twelve & five percent, and in the third generation birds in families of four and over (Appendix, TableIVd) it was nine & five tenths percent. The complete list of causes of mortality in these two groups is given in Table 7.

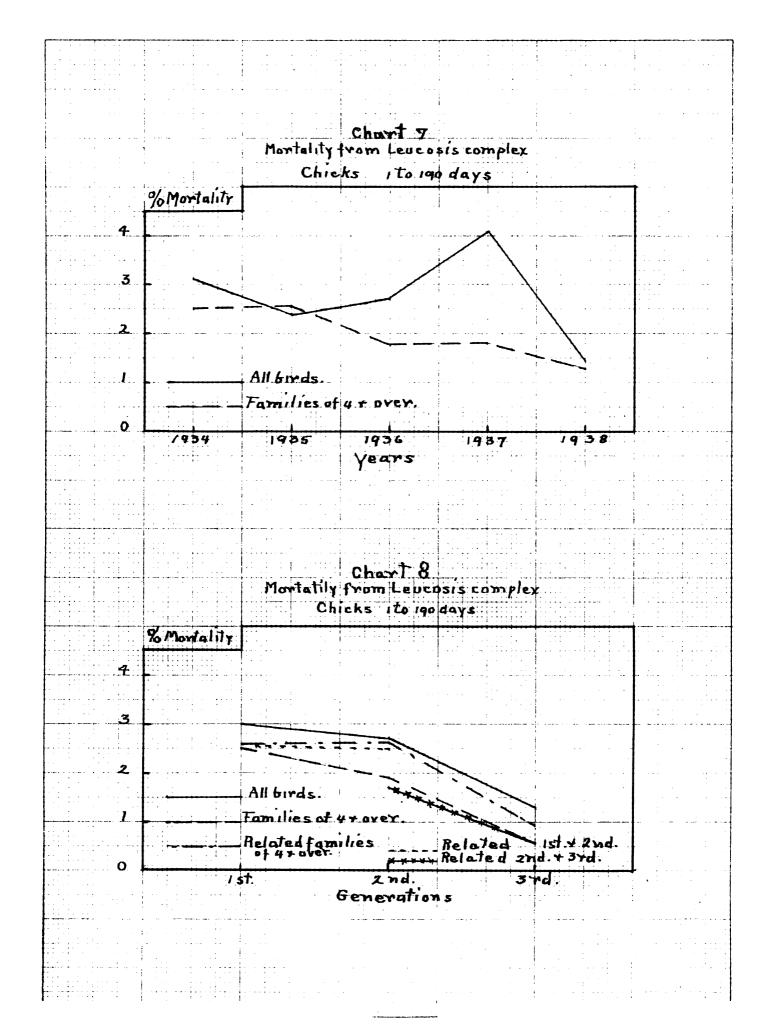


Chart 9
Correlation Chick Mortality + Pullet Mortality
Total Mortality All birds.

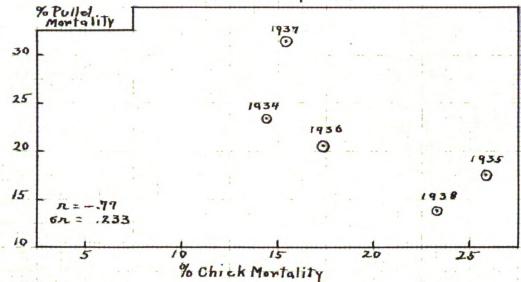


Chart 10 Correlation Chick Mortality + Pullet Mortality Leucosis Complex Allbirds

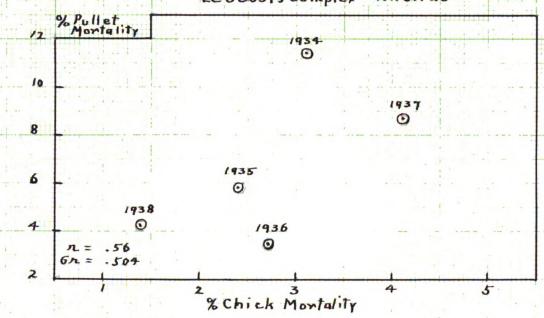


Table 7

Causes of Mortality in Two Groups of Birds with Low Total Mortality

Causes of Mortality	1938, All Birds in families of 4 and over	3rd. Generation of Related 1st,. 2nd,. & 3rd. Generations in families of 4 & over
Pickout	2	
Salpingitis	4	3
Impacted oviduct	1	
Enteritis	2	_
Liver diseases	2 1 3 5 2	1
Colibacillosis	Ţ	•
Lymphomatosis	2	1
Leucosis	2	4 1 1
Laryngotracheitis	2	÷ .
Impacted trachea	1	<b>T</b>
Obesity	1	_
Visceral gout	3 1	1
Peritonitis		1
Hemorrhage of heart	1	1
Unknown	1	
Total died	30	14
% died	12.2	9•5

When mortality is reduced to near ten percent leucosis complex still accounts for about one third of total mortality, reproductive, digestive and metabolic diseases account for most of the balance (Appendix, Table Vb and Vc).

There was a small decrease in the survival age of the birds
which died (Appendix, Table VI a to g) which is contrary to expectations.

It is true for all groups of diseases, even the leucosis complex. Generally, a reduction in the rate of mortality from leucosis complex is accompanied by an increase in survival age.

The five year average survival age for diseases in the leucosis complex group was:

Tumors	50 <b>1</b> days
Lymphomatosis	426 days
Neurolymphomatosis	<b>336</b> days
Leucosis	380 days

## DISCUSSION

For inheritance studies, comparisons of characteristics by consecutive years is not very accurate because pullets are trap-nested a year before they are used for breeding and therefore have no progeny until their second year. There should be more relation between alternate years than between consecutive years. And even alternate years is not a completely accurate comparison because some birds are used more than once and may have progeny of more than one generation in the same year (Appendix, Table 1).

Summaries by generations are also subject to error because some birds of a particular generation may be produced in more than one year, and there were no means at hand to measure variations in the amount and virulence of plant infection from year to year.

The "t" tests to determine the significance of the comparisons are shown in the following summaries:

Table 8

All Birds, by years					
Consecutive years	Difference in %	t value	Alternate years	Difference in %	t value
1934 <b>-</b> 35 1935 <b>-</b> 36 1936 <b>-</b> 37 <b>1937-3</b> 8	-6.1 3.2 11.4 -18.2	1.37 .79 **2.67 **4.55	1936-38	-2.9 14.6 -6.8 -9.7	•73 **•13 *2•05 *2•58
<b>A</b> :	ll Birds in Fa	milies of f	Cour and over,	by Tears	
1934-35 1935-36 1936-37 1937-38	-3.7 2.8 8.4 -15.9	•73 •64 1.85 **3.82	1937-38	-0.9 11.2 -7.5 -8.4	•19 *2•28 *2•17 1•99
	All	Birds, by	Generations		

Generations	Difference in %	t value
1st & 2nd	4.1	1.45
2nd & 3rd	<b>-1</b> 2 <b>.</b> 9	**4.08
lst & 3rd	<b>-</b> 8 <b>.</b> 8	**2.72

All Birds in Families of four and over, by Generations

Generations	Difference in %	t value
lst & 2nd	6.3	* 2.1
2nd & 3rd	<b>-</b> 13 <b>.</b> 6	** 4.3
lst & 3rd	<del>-</del> 7•3	<b>*2.23</b>

Related Generations, in Families of four and over

1st & 2nd	11.6	1.72
2nd & 3rd	-15.8	*2.44
1st & 3rd	- 4.2	•63

Related 1st and 2nd Generations, in Families of 4 & over

lst & 2nd 2.7 .69
Related 2nd & 3rd Generations, in Families of 4 & over
2nd & 3rd \* 6.1 1.52

In general, the variations in total mortality are no greater than would be expected for the number of birds involved. The comparisons of alternate years are a little more consistent and a little more significant than consecutive years but not enough so to justify and conclusions. The value of the figures as a basis of evidence of genetic changes is offset by a high percent of mortality from cannibalism in the year 1937 (Appendix, Table IVa). Cannibalism is included in the reproductive diseases, and in 1937, was eight & four tenths percent higher than the average of the other four years. This also accounts for some, but not all, of the high mortalityin the second generation birds.

The birds in the related 1st., and 2nd., and 3rd. generations, in families of four and over, ought to be the best measure of genetic changes. The changes are significant, but the change from the 1st. to the 2nd. generation is an increase of eleven & six tenths percent while the change from the 2nd. to the 3rd. generation is a decrease of fifteen & eight tenths percent ( Table 8). Some of this increase and decrease is due to cannibalism in 1937, which year was largely second generation birds. The value of the figures for this group is further offset, however, by the small number of birds in the group. This group represents birds in families of four and over which are descended from hens in families of four and over, which are themselves descended from first generation families of four and over. Only three third generation families with only thirty one progeny are in this group (Appendix, Table IIg).

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There is no explanation for the increase in chick mortality and the decrease in mortality of pullets raised from the survivors of the same flock. The correlation is, r = -.77 - .233 The "t" value is 2.266, which is not significant for the five comparisons. Bryant (12), found a positive correlation between chick mortality and pullet mortality when hisdata was grouped by percent mortality in families. Just the opposite results are obtained here when the data is arranged by years, including all chicks and all pullets.

The correlation between chick mortality and pullet mortality from leucosis complex is positive,  $r = .56 - .50\mu$ . The correlation is not significant for the five comparisons. When the data is grouped by generations and includes all chicks and all pullets (Appendix, Table IIIc), The correlation is, r = .9 - .09. This is a high degree of correlation.

The consistent reduction in mortality from leucosis complex in both chicks and pullets is the most important and satisfactory result of this study.

Bearse, et al, (7) reported that leucosis complexcaused 30 percent of the mortality in a resistant line during the first six years of breeding and forty nine percent in a susceptible line. The thirty percent is close to the results of this study after five years of selection. Other investigators have noted that a reduction in leucosis complex is accompanied by a reduction in other diseases (11) (26) (41). Waters (52) suggests that deaths from these two groups may have some association. Is the reduction in other diseases due to the reduction in neoplasms, or vice versa? It may be that the presence of leucosis infection in a degree less than sufficient to cause death may predispose birds to other

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infections. This view is supported by the fact that nonspecific reproductive and digestive disorders still cause most deaths when mortality from meoplasms is reduced to allow percent by selective breeding.

The decrease in the survival age of the birds which died, when an increase was expected, is difficult to explain. It should be pointed out, however, that in the first year of the study (1934), the survival age was 400 days. This is much higher than in most report of resistant strains after several years of selection, and the variations from 400 days are relatively small.

The number of birds which die in a flock depends on the genetic resistance of the birds and the amount of plant infection. That there was sufficient infection in the environment of this flock is indicated by the mortality of the flocks of White Leghorns and Barred Plymouth Rocks which were reared and housed with them. Both the Leghorns and the Rocks had higher mortality, the Rocks being considerably higher. The method of selecting breeding pens used for the Rhode Island Reds and described in this paper, was not used for the Leghorns and Barred Rocks.

The mortality in this flock was reduced to a rate considerably below the average for commercial flocks. It seems evident that part of this reduction is due to genetic resistance developed by breeding from families which had low mortality. Especially is this true for the reduction in losses from leucosis complex. Part of the reduction may be due to reduced infection, and the reduction in mortality form nonspecific infections may be coincidental with reduction in leucosis complex, but there is enough

evidence o mhether th poultrymen housed are could also be greatly The m wting the hers are to and ten to Tear the p

each male arentested! to build a The practi à Tale Woul trat time ful life l for three

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evidence of increased genetic resistance to make it worth while to examine whether the same method of selection used here could be used by commercial poultrymen. The methods by which these birds were hatched, reared and housed are similar to commercial farm methods. If the method of selection could also be applied with the same results, the financial returns could be greatly improved. The evidence is that it could be.

The method of selecting breeders which is the most accurate in esti mating their genetic make-up is the "progeny test" method. By this method, hens are tentatively mated to a male in individual pens, that is, one male and ten to fifteen hens in each pen. The chicks are raised and the next year the pullets are trapnested and records of the progeny of each hen and each male are compiled. On the basis of this records, the hens and males are"tested". The assumption is that the poultry man sould then proceed to build a flock using these tested birds for breeders year after year. The practical difficultyin the way of such a procedure is that a hen or a male would be three years old before it could be fully tested, and by that time many would be dead and the rest would have relatively short useful life left. It would also be necessary to carry many extra male bird s for three years until their families had been tested. It should also be realized that the pullets which are being trapnested to prove certain hens, males and families are an important part of the flock. A farmer could never have an entire flock made up of tested or proven birds. Most of the flock each year is being used to test or prove a few of the others.

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A short cut to this method is the use of cockerels which are tested on the basis of their sisters rather thn their progeny. This reduces the number of males it is necessary to carry and permits their use at an earlier age. Such a method was used in selecting males in this flock. of the 26 different male birds used, only 3 were used twice. Most of them were sib-tested cockerels used for the first time. Of the 190 hens used for breeders during the five years, 143 were used only once, 34 were used twice, and 13 were used three times.

The data offers an opportunity to examine another question which has interested the writer for several years. The question is whether the resistance which birds seem to develop against disease is a biological entity which gives resistance against all diseases and can be transmitted as a unit. Or is resistance developed against diseases one at a time and only this limited resistance transmitted? Waters (52) noted the relation between mortality from leucosis complex and mortality from other diseases, and Taylor (43) states that "individual families seem to vary greatly in their ability to withstand particular diseases".

There is evidence here to support both sides. There are families as large as 20 birds which had little or no mortality, sometimes fro more than one year and when mated to different males. Hen No. N934 had 12 daughters in 1936 and no mortality; 14 daughters in 1937 and 3 died, one each form impacted oviduct, cropbound and pickout; 8 daughters in 1938 and one died from laryngotracheitis; a record of 34 daughters in three years and 4 deaths, none from leucosis complex. Hen No. N907 had 13

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daughters in 1937 and 7 died; 2 from leucosis, 1 from lymphomatosis, 1 from pickout, 3 undnown. In 1938, she had 6 daughters by a different male and none died. The best family in the five years was hen No. N583 In 1936, she had 24 daughters of which 2 died; in 1937, 7 of these daughters produced 60 pullets of which 5 died, 2 from leucosis, 2 from salpingitis, and 1 from peritonitis.

There are instances of families which had high mortality from leucosis complex and also other diseases, and others that had no neoplasms but high mortality from other causes.

The term "vigor", is often used by poultry breeders and occurs constantly in poultry literature yet it is a term that is hard to describe and harder yet to measure. The cause of the present high mortality in poultry flocks is ofted blamed on the practice of selection for high production without accompanying selection for Vigor. Used in this way, vigor, is synonymous with resistance to disease. This question is not answered definitely by the data at hand but it is the considered opinion of the writer that there is no such thing as vigor, in the sense generally used. It is not a biological entity. Vigor is a vague something which is the result of good health, rather than the cause of good health.

## CONCLUSIONS

A method of selection, using progeny tested hens and their families mated to sib-tested cockerels, was effective in reducing the mortality ina flock of Rhode Island Reds from twenty-three & two tenth percent

to thirteen & five tenths percent and mortality from leucosis complex from eleven & three tenths percent to four & one tenth percent. The same method could be used by commercial farms.

When mortality is reduced to thirteen percent, leucosis complex causes about one-third of the total loss. Nonspecific reproductive, digestive, and metabolic diseases cause most of the balance.

Reduction in mortality from leucosis complex diseases is accompanied by a reduction in other diseases.

There is a relationship between the percent mortality from leucosis complex in chicks and the percent mortality from leucosis complex in the pullets raised from the survivors. The correlation for total mortality between chicks and pullets is negative.

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APPENDIX

Table I Analysis of the Flock Dams and Progency

	Great grand- daughters							•	154		154
Progeny	Grand- daughters				201		87	06	715	17	164
	Daughters	181	69	71	Ħ	34	н	ဆ			378
	Grand- daughters								27		27
Dams	Daughters				38		17	20	п	16	100
	Original	17	16 (134)	12 (new)	(134)	6 (135)	(ηε.) τ	2 (135)			63
\$ 0 2	Teg.	1934	) (	1,4,3,5	,	1930	ני כי רי	193(	C C	1930	Totals

Table IIa
Number and Size of Families
By Years, All Birds

Year	No. of Families	Total Progeny	Ave. size of Families
1934	51	184	3.6
1935	26	140	5.4
1936	42	246	5.9
1937	36	186	5.1
1938	42	267	6.4

Table IIb

Number and Size of Families

By Years, All Birds in Families of Four and Over

ïear	No. of Families	Total Progeny	Ave. size of Families	% of the Flock
1934	21	121	<b>5.</b> 8	65.8
1935	17	123	7.2	88.0
1936	26	208	8.3	81.4
1937	22	156	7.0	84.0
1938	32	242	7.6	91.0

Table IIc Number and size of Families By Generations, All Birds

Gener- ation	No. of Families	Total Progeny	Ave. size of Families
lst.	92	378	4.1
2nd.	87	490	5.6
3rd•	21	154	7•3

Table IId

Number and Size of Families

By Generations, All Birds in Families of Four and Over

Gener- ation	No. of Families	Total Progeny	Ave. size of Families	% of the Flock
1st.	կկ	277	6•3	73•4
2nd.	56	426	7•6	87•0
3rd.	<b>1</b> 8	147	8•2	95•6

Table IIe

Number and Size of Families

Related 1st. and 2nd. Generations, Families of Four and Over

Gener-	No. of	Total	Ave. size of Families
ation	Families	Progeny	
1st.	ስተ	153	6.9
2nd.	55	289	7.2

Table IIf

Number and Size of Families
Related 2nd. and 3rd. Generations, Families of Four and Over

Gener-	No. of	Total	Ave. size of Families
ation	Families	Progeny	
2nd.	13	126	9•7
3rd.	18	147	8•2

Table IIg
Number and size of Families
Related 1st., 2nd., and 3rd. Generations

Gener- ation	No. of Families	Total progeny	Ave. size of Families
lst.	16	129	8.1
2nd.	<b>3</b> 3	254	<b>7.8</b>
3rd•	3	31	10.3

Total Mortality and Mortality from Neoplasms
By Years, All Birds

				Chicks	cks				Pullets	င် န	
No. Hens No. (Breeders) Hatched	No.	Ę.	Total	tal %	Neop]	Neoplasms	No. Housed	Tol	Total	Neop	Neoplasms
		<u> </u>	died	died	died	died		died	died	died	died
930	930		133	133 14•3	28	3.1	184	43 23.2	23.2	21	11.3
28 593	593		154	25.8	큐	7.7	οήτ	24	17.1	ω	5.7
101 87	1047		181	17.3	28	28 2•7	545	50	20•3	80	3.3
41C 04	777		109		59	4.1	186	59	186 59 31.7	16	16 8•6
1034	1034		240		77		267	36	13.5	7	4.1

Total Mortality and Mortality from Neoplasms By Years, All Birds in Families of  $^{\rm F}$ our and Over Table IIIb

	Sms	ied ied	7.0.	5.6	3.!4	7.7	3.7
ά	Neopla	No. % died died	13 1	2	7	12	6
Pullets	٦, ,	. NO• % died died	20•6	16.9	19.7	28•1	12.2
	Tota	No. died	25	21	다	7/1	30
	No	Housed	121	124	208	156	246
	asms	No• % died died	2.57	2.5	1.8	1.8	1.3
_	Neopl	No. died	7	11	15	σ,	12
Chicks	tal	No• % died died	9•أل	24.0	15.8	9•2	22.0
	Tot	No. died	65	105	128	917	182
	No.	natched	5171	7738	811	500	888
	No. Hens	(breeders)	21	17	56	23	32
		Year	1934	1935	1936	1937	1938

Total Mortality and Mortality from Neoplasms By Generations, All Birds

	asms	died died	8.5	6•1	4.5
ø	Neopl	died	32	77	7
Pullets	Total	died	19.8	23.9	0.11
	Tot	died	75	711	17
	No.	Topono!!	378	760	154
	lasms q	died	3.0	2.7	1.3
Gnicks	Neoplasms	died	52	29	ω
	Total	died	18.6	18.1	22.5
	Tot:	died	323	358	136
	No.	<b>J</b> 01100 B11	1732	1969	909
	No. Hens	(grapa rg)	92	87	21
	Gener-		lst.	2nd.	3rd.

Table IIId Total Mortality from Neoplasms

By Generations, All Birds in Families of Four and Over

	sms	% died	7.5	6•17	3.4
ts	Neopla	No. % died died	21	77	<b>7</b> V
Pullets	rị.	% died	16.8	23.1	9 5•5
	Tota	No. % died died	87	100	77
	No.	Housed	285	430	747
	Sms	No• % died died	2.5	1.9	•56
Chicks	Neoplas	No. died	56	30	m
	Total	No. % died died	18•5	15.5	19.1
		No. died	191	232	103
	No.	Hatched	1030	1512	240
	No. Hens	(Breeders)	7-1-1	26	21
	Gener-	ation	lst.	2nd•	3rd.

Table IIIf

Total Mortality and Mortality from Neoplasms

By Generations, Related 2nd, and 3rd. Generations in Families of Four and Over

	Sms %	died	3.1	3•1
m			4 3.1	
Pullets	al %	died	20 15.6	9•5
	Tot No.	died	50	<b>זו</b> ר
	No• Housed		128	
m	Neoplasms No•	died	1.7	<b>3</b> 5.
				٣
	년 %	died	58 14.0	19•1
	Tota No.	died	58	103
	No. Hatched		415	240
	No. Hens (Breeders)	•	ဆ	18
	Gener- ation		2nd.	3rd.

Table IVa
Percent Mortality from Various Causes
By Years, All Birds

Cause	1934	1935	1936	1937	1938
Reproductive Digestive Leucosis Complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	3.8 1.1 11.3 1.1 2.2 1.1 2.7	2.8 2.8 5.7 0.7 1.4 0.7 2.8	2.4 1.6 3.3 1.2 6.9 1.2 2.7	11.3 3.2 8.6 2.1 2.1 1.6 2.7	2.6 2.6 4.1 1.1 1.5 0.8 0.8
Totals	23.3	17.1	20.3	31.7	13.5

Table IVb
Percent Mortality from Varous Causes
By Years, All Birds in Families of Four and Over

Cause	<b>1</b> 934	1935	1936	1937	1938
Reproductive Digestive Leucosis Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	5.8 0.0 10.7 0.0 1.6 0.0 2.4	3.2 3.2 5.7 0.0 1.6 0.8 2.5	2.9 1.4 3.4 1.4 7.4 1.0 1.9	10.3 1.0 7.7 1.1 1.9 1.3 3.2	2.8 2.2 3.7 1.2 1.6 0.0 0.8
Totals	20.6	16.9	19•7	28.1	12.2

Table IVc
Percent Mortality from Various Causes
By Generations, All Birds

Cause	First Generation	Second Generation	Third Generation
Reproductive	2.9	6.3	1.9
Digestive	1.6	3.1	1.3
Leucosis Compled	8.5	4.9	4.5
Respiratory	0.8	1.4	1.3
Metabolic	1.6	4.9	0.7
Peritonitis & Nephritis	1.1	1.0	0.9
Miscellaneous	3•4	2.2	0.7
Totals	19.8	23•9	11.0

Table IVd
Percent Mortality from Various Causes
By Generations
All Birds in Families of Four or Over

Causes	First Generation	Second Generation	Third Generation
Reproductive	3.9	6.0	2.4
Digestive	1.4	2.3	0.7
Leucosis Complex	7.4	4.9	3.4
Respiratory	0.0	1.6	1.4
Metabolic	1.7	5.1	0.7
Peritonitis & Nephritis	3 0.4	0.9	0.7
Miscellaneous	2.1	2.3	0.7
Total	16.8	23.1	9•5

Table IVe
Percent Mortality from Various Causes
By Generations
Related 1st. and 2nd. Generations, Families of Four and Over

Cause	First Generation	Second Generation
Reproductive	5 <b>.</b> 2	6.9
Digestive	2.6	2.8
Leucosis complex	7.2	6.2
Respiratory	0.0	2.1
Metabolic	0.7	5.9
Peritonis & Nephritis	0•7	0.7
Miscellaneous	2.0	3.8
Total	18•3	28.և

Table IVf
Percent Mortality from Various Causes
Related 2nd. and 3rd. Generations, Families of Four and Over

Cause	Second Generation	Th <b>ird</b> Generation
Reproductive	3.1	2.4
Digestive	0.8	0.7
Leucosis Complex	3 <b>.</b> 1	3•4
Respiratory	2•3	1.4
Metabolic	<b>3.</b> 9	0.7
Peritonitis & Nephritis	1.6	0.7
Miscellaneous	0.8	0.7
Total	15.6	9•5

Table IVg
Percent Mortality from Varous Causes
Related 1st., 2nd., and 3rd. Generations, Families of
Four and Over

Cause	First Generation	Second Gener <b>ati</b> on	Third Generation
Reproductive	6.2	7•5	0.0
Digestive	0.8	2.0	3.2
Leucosis complex	6.2	7.1	3.2
Respiratory	0.0	2.0	3.2
Metabolic	2.3	6.3	0.0
Peritonitis & Nephritis	0.0	1.4	0.0
Miscellaneous	2.3	3.5	3•2
Total	17.8	28.7	12.0

Table Va
Causes of Mortality as a Percent of Total Mortality
By Years, All birds

Cause	1934	1935	1936	1937	1938
Reproduction Digestive Leucosis complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	16.3 4.7 48.7 4.7 9.3 4.7	16.7 16.7 38.2 4.2 0.3 4.2 16.7	12.0 8.0 16.0 6.0 34.0 6.0	25.0 10.2 27.1 6.8 .6.8 5.1 8.5	19.4 19.4 30.6 8.3 11.1 5.6 5.6
Total	100.0	100.0	100.0	100.1	100.0

Table Vb
Causes of Mortality as a Percent of Tetal Mortality
By Years, All birds in families of four and over

Cause	1934	1935	1936	1937-	<b>19</b> 38
Reproductive Digestive Leucosis complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	28.0 0.0 52.0 0.0 8.0 0.0 12.0	19.0 19.0 33.0 0.0 9.5 4.7 14.5	14.6 7.3 17.1 7.3 39.0 4.9 9.8	36.4 6.8 27.3 6.8 6.8 4.5	23.2 16.6 30.0 10.0 13.0 0.0 6.7
Total	100.0	100.0	100.0	100.0	100.1

Table Vc
Causes of Mortality as a Percent of Total Mortality

By Generations, All birds

Cause

	First	Second	Third
	Generation	Generation	Generation
Reproductive Digestive Leucosis complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	14.6	26.5	17.6
	8.0	12.8	11.7
	42.6	20.5	11.0
	4.0	6.0	11.7
	8.0	20.5	6.0
	5.8	4.3	5.9
	17.3	9.4	5.9
Total	100.3	100.0	99•8

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Table Vd Causes of Mortality as a Percent of Total Mortality By Generations

		,	
All birds	in families of First	Second	Third
Cause	Generation	Generation	Generation
	•		
Reproductive	23.0	26.0	21.4
Digestive	8•3	10.0	7•3
Leucosis complem	43.7	21.0	35•7
Respiratory	0.0	7.0	14.3
Metabolic	10.4	22.0	7•3
Peritonitis & Nephritis	2.1	4.0	7•3
Miscellaneous	12.5	10.0	7•3
Total	100.0	100.0	100.6

Table Ve
Causes of Mortality as a cause of Total Mortality
By Generations
Related 1st. and 2nd. Generations, Families of four and over

Caus <b>e</b>	First Generat <b>ion</b>	Second Generation
Reproductive Bigestive Leucosis Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	28.6 14.3 39.4 0.0 3.6 3.6 10.7	24.0 9.8 22.0 7.3 20.8 2.4 13.4
Total	99,9	100.1

Table Vf
Causes of Mortality as a percent of Total Mortality
By Generations
Related 2nd. and 3rd. Generations, Families of four and over

Cause

	First Generation	Second Generation
Reproductive	20.0	21.4
Digestive	5•0	7.1
Leucosis compled	20.0	35.6
Respiratory	15.0	14.3
Metabolic	25.0	7.1
Peritonis & Nephritis	10.0	7.1
Miscellaneous	5.0	7.1
Total	100.0	99•7

Table Vg
Causes of Mortality as a Percent of Total Mortality
Related 1st., 2nd., and 3rd. Generations
Families of four and over

Cause	First Generation	Second Generation	Third Generation
Reproductive Digestive Leucosis complex Respiratory Metabolic Peritonis & Nephritis Miscellaneous	34.8 4.3 34.8 0.0 13.0 0.0	26.0 6.8 24.7 6.8 21.9 1.4 12.3	0.0 25.0 25.0 25.0 0.0 0.0
Total	100.1	99 <b>•9</b>	100.0

Table VIa Survival Age in Days By Years, All birds

Cause	1934	1935	1936	1937	1938
Reproductive Digestive Leucosis complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	406.1 386 394.7 511 394.5 355.5 465.6	429 430 424•1 446 393•5 498 451	293.6 410.7 342.7 228 267.9 321.6 407.4	393.1 385.8 374.4 341.5 443.5 420.6 386.3	385.1 318.7 388.1 250 369 435.5 530
Average	400.1	431.8	320.3	388.0	358.1

Table VIb
Survival Age in Days
By Years, All birds in Families of four and over

Cause	<b>1</b> 934	1935	1936	1937	1938
Reproductive Digestive	406	կ29 կ30	293.6 439.3	384•2 379	371.4 295.2
Leucosis complex Respiratory	387.4	441.1	349•7 228	383•5 344	388 <b>.</b> 1 260 <b>.</b> 3
Metabolic	457.5	388•5 498	271 327	427•3 469	432.5
Peritonitis & Nephritis Miscellaneous	422.3	490	433 <b>•</b> 5	386 <b>•</b> 2	430
Average	453.1	436.7	332.5	387.7	364.7

Table VIc Survival Age in Days By Generations, All birds

Cause	First	Second	Third
	Generation	Generation	Generation
Reproductive Digesting Leucosis Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	414.4	364.6	438
	415.3	360.6	390•5
	402.3	359.1	376•9
	489.3	286.0	232•0
	380.8	305.9	463•0
	367.5	383.2	532
	477.7	401.7	531
Average	417.8	350•6	395•4

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## Table VId Survival Age in Days By Generations All birds in Families of four and over

Cause	First Generation	Second Generation	Third Generation
Reproductive Digestive Leucosis Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	1,11,.5 280 1,31.1 302.7 1,98 1,140	353.6 355.5 368.5 286 300.6 398 419.5	438 368 412•6 232 463 532
Average	418	348.9	409

Table VIe
Survival Age in Days
By Generations
Related 1st. and 2nd. Generations, Families of four and over

Cause	First Generation	Second Generation
Reproductive	405	357•1
Digestive	461.7	345.8
Leucosis complex	417.5	374
Respiratory		307.5
Metabolic	344	303.1
Peritonitis & Nephritis	429	469
Miscellaneous	394	429.9
Average	415	<b>3</b> 58•5

Table VIf
Survival Age in Days
By Generations
Related 2nd. and 3rd. Generations, Families of four and over

Cause	Second Generation	Third Generation
Reproductive Digestive Leucosis complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	418.5 512 273 258 252.4 327 461	438 368 410•6 232 463
Average	321.4	409

Table VIg
Survival Age in Days
Related 1st., 2nd., and 3rd. Generations
Families of four and over

Cause	First Generation	Second Generation	Third Generation
Reproductive Digestive Leucosis complex Respiratory Metabolic Peritonitis & Nephritis Miscellaneous	406.7 392 423.7 402	363.4 388.2 369.2 307.2 306 426	368 371 216
Average	419	360•3	371.5

## Table VII Diseases classified under various headings

Reproductive diseases

Respiratory diseases

Laryngotracheitis

Impacted trachea

Coryza

Roup

Pickout

Salpingitis Internal eggs Ruptured yolk

Hemorrhage of ovary

Cystic ovary Impacted oviduct Ruptured oviduct

Metabolic diseases

Visceral gout

Obesity

Digestive diseases

Proventriculitis

Enteritis Impacted crop Impacted intestines Inflammation of vent Liver disease

Colibacillosis Chronic coccidiosis Peritonitis and Nephritis

Peritonitis Nephritis

Leucosis complex

Tumors Lymphomatosis Neurolymphomatosis Leucosis

Miscellaneous diseases

Parisites Injury Heat Crippled

Hemorrhage of heart

Abscess Unknown ROOM USE ONLY