

CONTRIBUTION TO THE KINEMATICS OF GROWTH  
IN CHICKENS AS INFLUENCED BY THE  
THYROID GLAND

By

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## INTRODUCTION

A fundamental corollary of the close association between genetics and the art of breeding is that contributions in the latter field are influenced, either directly or indirectly, by achievements in the former. It follows, then, that a genetic study of growth and body conformation in chickens is of a particular interest from the breeding standpoint.

In plant and animal production, the breeders' efforts are mostly concentrated on quantitative characters, yet they are among the least understood from a genetic respect. This lack of information is frequently attributed in the current texts of genetics to the complex nature of metric characters. What appears to be a single attribute is actually a combination of many traits each of which is perhaps liable to further subdivision.

Growth can be visualized as the outcome of many biochemical chains of reactions which are under the control of both internal and external factors. Genetically speaking, these factors may be divided into two main groups, the genotype and the environment. The latter can in turn be subdivided into the internal and the external environments. The concept of the internal environment as held by the physiologist, implies that in most cases the cells are not in direct contact with the blood. Instead they are bathed in what is known as the tissue fluid. It is generally accepted that free interchange of materials occurs between the blood and the tissue fluid on one hand and between the latter and the cells on the other hand. Since growth is the outcome of the continued accumulation of cells by mitotic

division, and since the latter phenomenon is one of the vital capacities of the cells, it follows then that mitosis shares the benefits of this free interchange of materials between the cells and the tissue fluids.

The biochemist maintains that the tissue fluid is composed of a variety of known and unknown substances necessary to the vital processes in the cells. Among the known components are the hormones of the endocrinal system. The nucleus, however, besides being the center of mitotic activities, is also known to be the site of the hereditary material, or more specifically, the genotype of the individual. The direct corollary of these facts is that the genes undergo a sort of interaction with the hormonal constituents of the blood, and that the outcome of the genes' effect must be governed by the kind and magnitude of this interaction. Indeed, the genetic studies of sex in general and in particular the group of traits known as sex-influenced characters are classical demonstrations for this phenomenon.

Metric characters are generally accepted to be determined by polygenes, or multiple factors. Following the argument in the previous paragraph, polygenic systems similarly undergo a sort of interaction with the hormonal constituents of the blood, or tissue fluids. In birds, the proof that there is an interaction between the polygenes for body size, for example, and the endocrine system rests on three facts. First, there is a close relationship between internal secretions and growth and metabolism. Second, there is a pronounced sexual dimorphism with respect to body size. Third, the findings that some type of recessive dwarfism in chickens is

associated with abnormal enlarged thyroid (Landauer, 1929; Upp, 1934; and Mayhew and Upp, 1932).

The present study confines itself to certain phases of this relationship, namely the interaction between the secretion of the thyroid gland and the polygenes concerned with growth. However, since polygenes are identifiable only through their effects, this interaction is considered in terms of its reflection upon certain characters pertinent to growth such as body weight and skeletal measurements. This method, though not facultative, yields results that are important from a practical breeding standpoint.

The tools used in the study are a combination of those used in applied population genetics for measuring polygenic effects in terms of genic variance and covariance, and those used in experimental endocrinology to induce hypothyroidism. From one point of view, such approach may appear discordant with the current genetic research technique, since it combines the methodology of the two main schools of genetics; the biometrical and the physiological. However, from another angle it is harmonious with the usual Mendelian procedure in which the organism is analyzed into its component traits. Indeed, modern genetics strives to go deeper into this analytic procedure even to the extent of chemical levels, and it was with this in mind that these experiments were started.

## REVIEW OF LITERATURE

The reports on the relationship between the thyroid gland and growth in chickens are very limited in number. A review of the literature reveals three main aspects of the situation. First, that a large share of these studies is on the physiological contribution of this gland to plumage characteristics and growth. The materials on this first aspect are critically reviewed by Zawadowsky (1932), Schneider (1939), and Fleischmann (1947). The second field involves the practical use of iodinated casein and goitergenic substances in commercial poultry production. This field has been recently reviewed by Blaxter, Reinke, Crampton, and Petersen (1949). Third, that a very meager part of these studies is confined to the physiogenetical aspect of the thyroid gland and growth. It is with this third group of studies that our present interest is concerned.

Landauer (1929) reported a case of "thyrogenous dwarfism" in a six month's old New Hampshire pullet which he had obtained from one of the commercial flocks in Maine. The author gave a detailed description of this single sample as to body measurements and many other anatomical features. The bird died shortly after having been under the author's careful observation for one month. The striking characteristics of this pullet were general arrest of growth, a pronounced brachycephaly, bulging eyes with swollen, surrounding tissue and with a myxoedematous swelling between eye and nostril of the left side. The skin was dry, the feathers were relatively long. He also observed that the thyroid gland was enlarged to at least twice its

normal size. Most of the gland was found to be of aplastic tissue without any colloid. The thymus had an advanced stage of involution and the parathyroids were absent. The skeletal measurements showed pronounced deviations from the normal, and Landauer maintains that this case is similar to human cretins.

Mayhew and Upm (1932) reported on eighteen cases of dwarfism in Single Comb Rhode Island Reds. They described the general appearance of the birds with emphasis on their pedigree and body weights. The newly hatched chicks did not show the character but the dwarfs were identifiable on the second week of age. These abnormal chickens resemble the bantams with relatively short legs. Their body depth was less than that of the normal chickens. A great amount of variability was noticed with respect to each one of these characteristics. Moreover, the dwarfs showed less resistance to common diseases of poultry, and most of them did not live to be very old. Accordingly, these workers classified the character as a semi-lethal one and concluded that it is due to a recessive autosomal gene. The actual count which they reported was 46 normal chicks and 11 dwarfs against the expected ratio of 42.75: 14.25. These workers likewise suggest that this condition in chickens is similar to cretinism in humans (cf. Landauer, 1929). They also added that the birds resembled very closely the specimen described by Landauer (1929).

Upm, (1934) repeated these investigations on a wider scale but on the same stock which he obtained from Mayhew. By mating 10 sires and 33 dams, all of which were proven to be heterozygous for the character, he obtained a ratio of 464 normal birds to 146 dwarfs

against the theoretical 3:1 ratio. Hence, he confirmed the previous findings that the character is due to a recessive autosomal gene since both sexes were equally represented in each group. The evidence on that was also substantiated from the data which are included in his report and which he secured from Dr. Warren of the Kansas Agricultural Experiment Station.

In neither of these two studies (Mayhew and Orr, 1932, and Orr, 1934) were the anatomical features of the thyroid gland mentioned and their findings were correlated to the specimen described by Landauer (1929) only by inference from phenotypic resemblance. By taking this inference for granted, these two reports comprise the only published material on a genetic relationship between the thyroid gland and body weight, or growth, in chickens on the level of identifiable genotypes, viz, the level of discontinuous variation. However, there is another group of reports which deal with this relationship but on the level of continuous variation. Unfortunately, these reports did not do more than demonstrate that there may be breed or strain difference in thyroidal activity which is related to growth rates.

Munro, Kossin and Macartney (1943) demonstrated differences among five breeds of chickens as to their response to the injection of an anterior pituitary extract which contained both thyrotropic and gonadotropic properties. The five breeds of this study were Single Comb White Leghorn, New Hampshire, Light Sussex, White Plymouth Rock and White Wyandotte. Seven-day old chicks were used and the study lasted for only ten days, after which the chicks were weighed, killed and the combs, thyroids, oviducts, and gonads were excised and weighed. Certain organs were observed to respond to the extract very conspicuously.

in certain breeds. The chief among those were the comb of the White Leghorn males, the thyroids of both sexes in the Wyandottes, the testes of the White Rock, particularly, and also of the White Wyandottes, and the ovaries of the New Hampshire. These workers attributed this differential response to the "genetical control over the somatic response threshold", and concluded that breed, or family, sensitivity to gonadotropins may be a criterion to reproductive superiority.

Mixner and Hon (1947) showed that "double cross" chicks produced from inbred lines of Rhode Island Reds and Single Comb White Leghorns had a higher thyroxine secretion rate than chicks produced from "single crosses" between two inbred lines. The latter hybrids, however, had a higher secretion rate than those of the cross between non-inbred New Hampshires and Barred Plymouth Rocks. The authors concluded that hybrid vigor in chickens is correlated with an increase in the thyroxine secretion rate although they did not assay the parental inbred lines involved in the study. Moreover, the characters concerned with hybrid vigor were not specified. In addition to the rate of thyroxine secretion, body weight was the only character re-reported upon and it failed to show this much effect from the claimed hybrid vigor. Thus, while thyroxine secretion rate in the "double cross" was about 35 percent higher than it was in the "single cross", the body weight of the control birds was either almost equal in both kinds of the hybrids or slightly higher in the "double cross". Their data, however, lacks information on the original inbred lines which would be necessary to demonstrate the occurrence of such claimed

correlation. Their report, however, demonstrated two facts which are interesting from our present standpoint. First, that there is an association between a higher level of thyroxine secretion and the increased heterozygosity in the "double cross" hybrid. Second, that different inbred strains of chickens when combined, yield hybrids having different levels of thyroxine secretion.

Schultze and Turner (1945) carried on an extensive study on the rate of thyroxine secretion of certain domestic animals. Their findings with respect to the fowl indicate that the thyroxine secretion rate of White Plymouth Rocks increases as the body weight increases. Sex, however, appears to have a slight differential effect on this relationship, but on body weight basis, thyroxine secretion in the female appears to be more active than in the male. They also demonstrated that the White Leghorn cockerel has a higher rate of secretion than the White Plymouth Rock cockerel. In the former breed, there is a sharp rise in both rates of growth and thyroxine secretion during the first seven weeks of age, followed by a slower rise in thyroxine rate but a decrease in growth rate. In the White Plymouth Rock cockerel this break occurs at an older age than in the White Leghorn. In general, their data show that while there is a positive correlation between the two rates up until the seventh and the twelfth weeks of age in the White Leghorn and the White Plymouth Rock, respectively, there is a negative correlation between the two rates after these periods. Castration was shown by these workers to lower thyroxine secretion about 16 percent.



Glazener, Shaffner and Jull (1949) demonstrated that a rapid-growing strain of each of the two breeds of New Hampshire and Barred Plymouth Rock had a higher level of thyroxine secretion than the corresponding slow-growing strain. The rapid-growing strain of each breed consistently showed heavier body weights throughout the period of the study. The data on the thyroxine secretion of the two strains of the New Hampshire, though confined to the period between sixth and twelfth weeks of age, showed difference between the two strains. This difference, although conspicuous during the period between the sixth and ninth weeks of age, was small during the following three weeks. The general decrease of the difference in thyroxine secretion between the two strains reveals the fact brought out by Schultze and Turner (1945) that the rate of secretion slows down as the bird approaches its mature weight. The fast-growing birds being closer to their mature weight slow down earlier than the slow-growing ones. Unfortunately, Glazener et al (1949) did not carry the study for a longer period to confirm the between strains within breed differences.

While the findings of Boone, Heinke, and Davidson (1950) are in agreement with those reported by Glazner et al (1949) in some respects, yet they conflict in others. The former group of workers reported a slightly higher thyroxine secretion rate for a fast-feathering strain of Rhode Island Reds, however, they concluded that there is no evidence to maintain that slow-feathering is due to an inherently low thyroid secretion rate. Moreover, they reported that their rapid-growing strain was the slow-feathering one. This is contrary to

all reports concerning this relationship (see the summaries by Jull 1940, and by Hutt, 1949). This report, however, ended with the new assumption that "the usual correlation between growth and rate of feathering may be due to coincidental factors that are not necessarily linked genetically."

Riddle (1947) established several strains of doves and pigeons differing in thyroid size and concluded that the size of this gland is genetically controlled. By crossing these strains, he noticed that the  $F_1$  tends to have metabolic levels which are intermediate to those of the parental strains. He maintains that low thyroid weight is correlated with a high level of thyroid function as indicated by higher metabolic rates. This worker noticed that "environmental goitrogenic conditions seriously interfered with the investigation of hereditary control of thyroid size in pigeon races and hybrids." He also added that his data indicated that three pigeon strains differ from two others "in a genetic factor or factors which permitted the development of goiter". This phenomenon was also observed among the dove strains.

Bates, Riddle and Lehr (1941) found that there are hereditary factors responsible for the pronounced difference between two strains of chicks with respect to their response to thyroprotein. They also noticed that during a five-year period, the thyroid weight of a strain of Carneau pigeons was increased four fold. No explanation, however, was given.

For this group of reports we should add one which though dealing with plumage color, yet has a direct bearing on our problem.

Juhn (1945) demonstrated that in females, from the cross Barred Plymouth Rocks and Brown Leghorns, the action of the genes for the color of the saddle feather is changed by lowering the metabolic rate through feeding thiouracil.

To summarize the studies reviewed, the authors attempted to demonstrate breed or strain differences in thyroidal activities. However, all these reports dealt with the problem on a phenotypic level. Even though it should be noticed that in none of these studies has an attempt been made to measure, in the usual terms of the correlation coefficient, the magnitude of the degree of the association between thyroidal activity and growth rate. The reason for this is clearly the difficulty in assembling enough data to run such statistics, viz. the correlation coefficient. Moreover, it should be noticed that in these studies the evidence of the existence of an association between thyroidal activity and growth was based primarily on the comparison of the means of two samples from the same population. One sample was used to assay for thyroidal activity and the other used to measure growth rate. It is quite evident that such procedure would be inadequate if we try to study the problem from a genetic standpoint on a family basis where the number of offspring is limited by the reproductive capacities of the parents. The family basis, however, is more adequate for genetic studies than the strain level. The reason for this is evident since by studying the families it is possible to estimate parental contributions and the effects of heredity and environment. It follows then that the conflict between such objectives and the technique used in the previous reviewed reports

is quite evident, and it was deemed necessary to find another method of approach to the problem. The method employed will be described in detail in the next section.

## EXPERIMENTAL PROCEDURE

### Historical

The birds for the experiments to be described were the current University stock of relatively rapid-growing New Hampshires

On the University Farm no selection was practiced except to satisfy the requirements of routine culling. No pedigree records were available, hence, it is impossible to state definitely whether or not the parents were related to any degree of consanguinity.

### The Parents

In July, 1949, seventy pullets and ten cockerels were picked at random from birds hatched during the spring of 1949. By the time these prospective parents were chosen, most of the pullets were in actual lay. The pullets were housed in individual wire cages in the basement of the poultry department building. In another room of the same basement, the ten cockerels were similarly housed in individual cages. This breeding stock was fed ad libitum a breeder's mash with an ample supply of fresh water. Regular pedigree operation was followed.

Since the present kind of study needs the greatest number of offspring possible, rough criteria of the reproductive capacities of the parents were used. With respect to the prospective dams, early sexual maturity and the number of eggs were the two available criteria which time could permit. Thus the pullets were subject to a short-period tests after which sixty-six birds were kept and four were discarded. As to the cockerels, sexual maturity,

as shown by a fair amount of semen, was the only criterion used. Hence, eight out of the ten cockerels were selected to be the sires.

#### Matings

All matings were done artificially according to the method described by Burrows and Quinn (1937 and 1939). The details of the method, particularly with respect to semen doses, duration of fertility, and other helpful instructions, were recently summarized by Parker (1949).

The sixty-six pullets were divided into eight groups varying in number between seven and nine each. Each of the selected sires was randomly assigned one of the mothers' groups.

Insemination began on September 12, 1949, and continued for eight weeks. Each pullet received a dose of one tenth of a cubic centimeter at least once a week. The last eggs were collected on November 9, 1949.

#### The Hatches

Four hatches were secured from these matings at two week intervals. The first hatch was on October 19, and the other three hatches were on November 2, 16, and 30, respectively. Thus the hatching season extended over a six-week period. These four hatches yielded 127, 318, 263 and 362 chicks, respectively.

#### The Treatment

The day-old chicks secured from each dam on each hatch were randomly divided into two groups equal in number. One of these

groups was assigned to the treatment and the other to the control. The assignment was purely random and no sexing of chicks was done. Ordinary wing-bands were used to identify the chick's pedigree.

This plan satisfies the following experimental requirements. First, that each dam's family and each sire's family are equally represented in both the treatment and control groups. Second, that likewise, each hatch is equally represented in both groups.

Beginning the first day of hatch, all chicks were given the Maryland Experiment Station chick starting mash.<sup>1</sup> Two-tenths percent thiouracil was added to the mash for the treated group. One tenth percent of this drug when fed to chickens of all ages is known to suffice maximum thyroïdal enlargement (Astwood, Bissell, and Hughes, 1944, Mixner, Reinke, and Turner, 1944, Andrews and Schnetzler, 1946, and Schultze and Turner, 1947). Since, according to most of these workers, a dose of two tenths percent does not cause toxicity, this dose was used in the present experiments. Both groups were fed ad libitum with an appropriate supply of fresh water. The birds were maintained as outlined until the tenth week of age.

In order to insure a uniform level of the drug-potency, the feed was mixed with thiouracil in small amounts sufficient for not more than two weeks. Mixing the feed with the drug was done in small lots of 100 pounds each in a small mechanical mixer.

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<sup>1</sup> For the formula of this mash, see Glazener, Shaffner, and Jull, 1949.

## Housing

All hatches, except the fourth, were raised in the building of the poultry department. This building was kept at a fairly satisfactory constant temperature.

The baby chicks were first raised in wire batteries containing five vertical decks. The heat of each deck was supplied by an electric hover in the middle of the battery. A partition was placed under this hover to divide each deck into equal halves. The birds were so distributed that one-half of each deck was used for birds on treatment while the remaining half contained the controls. The chicks of each group were distributed randomly over the half decks of the battery. Thus, no precautions were taken to favor or to prevent the raising of two full-sibs, or half-sibs, together.

The control birds were kept in this small battery until the end of the fourth week of age. From the fifth to the end of the tenth week they were raised in a larger wire-battery of four decks. The treated birds, being smaller in size, were kept to the end of the sixth week of age in the small battery. After the sixth week, the treated birds were transferred to a larger battery like those in which the control birds were housed. After the fourth week of age no artificial heat was supplied to either group.

On account of the large number of the fourth hatch, it had to be raised on the University poultry plant. There, these chicks were raised in two adjacent pens on the floor. They were attended by one man and received identical care except for the kind of feed.



## The Data

The following data were taken from both the treated and the control offspring.

1. Body weight in grams to the nearest gram on a Toledo scale on the first days of the second, sixth, eighth, and tenth weeks of age.

2. Three body measurements in centimeters to the nearest millimeter with a metal calliper on the first day of the tenth week of age. These body measurements were:

- a. Keel length
- b. Body depth
- c. Shank length

3. During the first two days of the eleventh week, all the birds were killed by bleeding. The thyroid gland was dissected and weighed. The glands of the treated birds were weighed on a Triple-beam balance in grams to the nearest one-hundredth of a gram. Those of the control birds were weighed in milligrams to the nearest one-fifth of milligram on a Precision balance. In both kinds of birds a single weight for the two thyroids was recorded.

In these experiments the thyroid weight is used as a criterion to its functional capacity. This criterion, though, is questionable in mammals, yet its validity in the fowl is accepted by most endocrinologists (Schultze and Turner, 1945, and Hoffman and Shaffner, 1950).

4. The testes of the males of both groups were weighed in grams to the nearest centigram on the Triple-beam balance. One single weight for both testes was likewise recorded.

## METHOD OF ANALYSIS

The analysis of the data was confined to the birds which lived until the end of the experimental period and gave a complete record with respect to the required data.

The method used in analyzing the present data is the one usually used in similar problems of quantitative inheritance. The main tools are the analysis of variance and covariance. By these analysis the total variation, or covariation, is apportioned to its sources whether genetic or environmental. The model given by Lush (1948) is followed in this report. This model fits the mating plan which has been used in these experiments. An adaptation of this model is given in Table 1 in which  $g$  is the number of sires,  $d$  is the number of dams mated to each sire, and  $n$  is the number of offspring which each dam has produced. In the last column of this table, the mean squares for the last three sources of variation are reduced to their components according to Crump (1946). The basis of this reduction is given in Table 1 in symbols, and in their corresponding figures in the Tables which contain the factual analysis. The interpretation of these components is as follows (Lush, 1948):

$B$  is the variance expected between full-sibs. It contains all the environmental variance, one half of the additive genetic variance, three-quarters of the variance due to dominance, and more than three-quarters of the epistatic variance.

$A$  is the variance expected between paternal half-sibs which have different dams but the same sire. It contains one-quarter of the genetic additive variance, one-fourth of the variance

Table 1. General plan for the analysis of variation or covariation used in the study.

Source of variation or covariation	Degree of freedom	Components of mean square or mean product
Total	$sdn-1$	
Between dams' families:	$sd-1$	
Between sires	$s-1$	$B+nA+ndS$
Between dams' families within sires' families.	$s(d-1)$	$B + nA$
Between full-sibs (i.e. within dam-family)	$sd (n-1)$	$B$

due to dominance, the variance from maternal effects, and perhaps some small fraction of epistatic variance. This component is actually a combination of the variance which is contributed by the dam and that caused by "nicking" between sires and dams.

S is the variance between unrelated birds, or it is the contribution of the sire. It does not contain any environmental variance. It contains one-quarter of the additive genetic variance and a negligible trace of epistatic variance.

It follows that the sum of S plus A equals the total of the additive genetic variance and dominance deviations among full-sib families. This total as shown above equals one-half of the additive genetic variance plus one-quarter of the variance due to dominance (Bright, 1935).

It is obvious from the genetic interpretation of these components that the separation of the variance is not rigorously accurate, since they cannot be subdivided by the present plan of analysis such as to fit their wide genetic interpretation as given above. However, since these three components are enough to provide an appropriate estimate of genetic and environmental variance, the present plan of analysis fairly satisfies the objects of these experiments. Moreover, this plan has previously been used as a tool for identical objectives in genetic studies of poultry. The reports by Hazel and Lamoreux (1947), Lerner and Cruden (1948), and Lush, Hazel and Lamoreux (1948), serve as three examples.

By using these components of variance, it is possible to make several estimates of heritability (Hetzer, Dickerson, and Zeller, 1944). In the present report, two methods suggested by Lush (1940) are used. In the first method, the heritability is estimated as the ratio between twice the sum of S plus A to B plus S plus A. In the second method, the estimate of heritability equals the ratio between four times the component S and the sum of B plus S plus A. These two estimates can be expressed in the following two equations, where  $h^2$  is the estimate of heritability:

$$(1) h^2 = \frac{2(S+A)}{B+S+A}$$

$$(2) h^2 = \frac{4S}{B+S+A}$$

The first method combines both S and A. Thus, it is more desirable since it reduces the sampling errors in these two components since sampling deviation in A may cancel those in S. It is, therefore, a reliable method if dominance and maternal effects make little contribution. Maternal effect is interpreted as those genetic or environmental factors common to full-sibs but different among paternal half-sibs. From this standpoint, the second method is preferable since A is absent in the numerator of formula (2), and since S is mostly genetic. For these reasons, the heritability of the traits reported herein are based on both methods.

The analysis of covariance followed a plan similar to that used in the analysis of variance. Hazel (1943) derived a formula to estimate the genic correlations between two characters. His

formula requires the data of the offspring and those of one of his parents. It is evident that since the present data lack the latter requirement, it is deemed necessary to use the data of the same individual. Lush (1948) discusses the different techniques for estimating genic correlation and concludes that "genetic correlations have not yet been estimated on enough different samples of data or under conditions varied enough or for enough different kinds of characteristics to permit sweeping conclusions about the best way to estimate them". The formula by Hazel (1943), however, is an example of how the covariance can be separated according to the method described above. Hence, the components of covariance are reduced in the same way used in the reduction of the variance (Table 1). These components of covariance and variance are then combined in the following formulae to estimate genetic, environmental, and phenotypic correlations (Dempster and Lerner, 1948).

- (1) The genetic correlation between the characters x and y:

$$r_{G_x G_y} = \frac{A_{xy} + S_{xy}}{\sqrt{(A_x + S_x) (A_y + S_y)}}$$

- (2) The environmental correlation between the environments of the two characters:

$$r_{E_x E_y} = \frac{B_{xy} - A_{xy} - S_{xy}}{\sqrt{(B_x - A_x - S_x) (B_y - A_y - S_y)}}$$

- (3) The phenotypic correlation between the two characters:

$$r_{xy} = \frac{B_{xy} + A_{xy} + S_{xy}}{\sqrt{(B_x + A_x + S_x) (B_y + A_y + S_y)}}$$

Where the letters with a single subscript represent the components of variance and those with double subscripts represent the components of covariance.

## RESULTS AND DISCUSSION

### Body Weight

The average body weight of the birds at the second, sixth, eighth, and tenth weeks of age are given in Table 2. To show the relative body weight of the treated and the control groups, a value is given showing the weight of the former as a percentage of the latter. By examining these data five points become evident.

First, the treated birds in both sexes are consistently smaller than the control group.

Second, the greatest loss in the weight of the treated group occurred between the second and sixth weeks of age and the magnitude of this loss decreased after this period.

Third, both groups have a considerable amount of variability. However, the variability is greater in the treated group (Mayhew and Upp, 1932), and in this group there is a progressive increase in variability accompanying the increase in age.

Fourth, the retardation of growth due to treatment is about equal for the two sexes on the second week following which the males are more adversely affected than the females. This observation is in agreement with the general situation noticed by Bird and Gutteridge (1934) who reported that adverse environmental conditions affect more drastically the growth of males than that of the females. In the present data, this sex differences becomes more conspicuous as the birds increase in age. The value representing the weight of the treated females divided by the weight of the control females is at the sixth week of age two percent greater than the corresponding

value for males. These differences increased to 2.9 and 4.1 at the eighth and tenth weeks, respectively.

Fifth, all treated birds showed a fair rate of growth in spite of the severity of the given dose of the drug. This becomes more evident when the data are plotted as shown in Figure 1, from which it is quite evident that the slopes of the two curves of the treated birds progressively increase as the age increases.

The analysis of variance for the body weight at the four periods studied is given for each sex within each group in the four tables numbered 3 - 6. In each one of these Tables the variance between dams' families is separated into: (1) the variance between sires families, and (2) the variance between dams' families within sires, viz, between the mates of a sire. The variance of these divisions when reduced to their components give a reasonable estimate of the sire's and the dam's contributions, respectively. The basis of this reduction is shown in the last column of each one of these tables. Since the average number of offspring per dam, or per sire, differs between the two sexes and between the two groups, the coefficients for the components S and A are different for each sex within a group.

The estimates of the three components of variance B, A, and S, are given in Tables 7 and 8 for the female and male progeny, respectively. From these two Tables it is quite evident that in both sexes the contribution of the sire (i.e. the component S) is always less than that of the dams (i.e. the component A). The difference between A and S, it will be recalled may be due to dominance or to any other genetic or environmental factors common to paternal half sibs. These



differences are given in the last rows of both Tables 7 and 8 as a ratio to the total variance (i.e.  $\frac{(1) \quad A - S}{A+B+S}$ )

By examining these ratios, two possibilities are to be considered. First, if the increase of A over S is entirely due to environmental factors common to maternal half-sibs, then this ratio between the treatment and the control within each sex must maintain a similar relationship from week to week. This assumption is true since both sexes within each group were raised together. Second, if dominance or genetic maternal effect or both, are to be suspected, then this ratio should not maintain this relative constancy. By examining the inequality signs between the treatment and the controls in the two last rows of Tables 8 and 9 the second possibility is the more plausible.

It will be noticed that within the males, the inequality sign changes its direction at the eighth week of age and at the tenth week in the females. These two ages are comparable to the ages on which sex differentiation as indicated by comb growth. This is also indicated from the common observation that comb growth in the females is delayed as compared with that of males, and that in both sexes comb growth is under the control of androgenic production. Hence,

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(1) The ratio  $(A-S)/(A+B+S)$  is an appropriate estimate of maternal effect if the variance between dams within a sire can be separated from the variance which is due to maternal effects, viz, the interaction between sires and dams. (See, for example Hazel and Lamoreux, 1947). Since the present plan of mating does not allow the mating of a certain dam to more than one sire, the present method of analysis does not take account of this separation of variance. Hence, the difference between A and S is interpreted in the present report as to include both these sources of variation.

it seems reasonable to conclude that androgen production is tied up with dominance or genic maternal effect or with both. The proof of this becomes more evident by comparing the differences between A and S in the two sexes. It is quite evident that (except for the treated group on the second week and the control one on the tenth week) the males have in general a greater amount of such difference than the females.

The inference that androgenic production may be responsible for the increase of A over S, however, should not entirely exclude the possibility of a common environmental factor. This is due to the fact that the size of the difference between A and S is generally decreasing in the controls (except for the control females at 10 weeks of age) after the second week, viz. from the age at which the egg size affects chick size.

One more general observation is evident from the ratios in the last rows of Tables 7 and 8. This is the fact that since these ratios differ between the treated and the control birds, the general conclusion should then be that in the absence of thyroxine the mode of the action of the genes concerned with body weight is altered (cf. Juhn, 1945). It is quite evident that any reduction in A appears as an increase in S and vice-versa. It follows then that it is possible in the absence of this hormone that some genes change their mode of action from dominance to the additive scale or vice versa. This alteration of the gene action depends primarily on the sex of the individual and the magnitude of the alteration depends upon the age and sex of the individual as indicated by the directions of the inequality signs in the last rows of Tables 7 and 8. The

effect of thyroxine on the polygenic action will become more evident in the discussion on the estimates of heritability of body weight.

Before starting this discussion, it is advisable to elaborate somewhat upon the meaning of the term heritability. The most common definition of this term is that it is the ratio of the genic variance to the total observed variance. Heritability is also defined as a measurement of the accuracy of selection. This means that in breeding problems its estimate helps to predict to what extent the observed variance of the parental generation will be repeated in the offspring of the selected parents. It should be emphasized here that by estimating the heritability of any character for the treated birds such estimate does not agree with the second definition. It is within the limits of the meaning of the first definition that the term is used in this report.

The estimates of heritability of the body weight are given in Table 9 as estimated by the two methods, which were given previously in the section on "method of analysis". These data are plotted in Figures 2 and 3.

The literature on the heritability of body weight in chickens in general, and in particular in the New Hampshire breed, is very limited. Shoffner and Sloan (1948) summarized the estimates of this heritability in different breeds as given by three different groups of authors. Among those workers, Lerner, Asmundson and Cruden (1947) estimated the heritability of body weight in the New Hampshire breed at 12 weeks of age to be 0.506 and 0.597, according to the first and the second methods, respectively. The materials used by these

workers resemble most closely that used in the present experiments. Although their data were also collected on New Hampshire birds, they raised them under quite different environmental conditions than used in these experiments and to older age. Moreover, these workers (Lerner et al, 1947), having a limited sample of 230 birds, lumped the observations on the two sexes in the estimate of heritability, but gave no information as to whether or not the sexes were equally represented in each family. It is quite evident that a character such as body weight which shows as much as 15 to 20 percent of sex difference, special care should be given to the sex ratio in the analysis of data.

There is no other adequate report in the literature with which to compare the data presented in Table 9. It is assumed that these estimates would vary considerably from flock to flock, among breeds, from time to time and according to the method of analysis used (Lush, 1948, Shoffner and Sloan, 1948 and others). This fact is also evident from the tables compiled by Phillips (1947) and Shoffner and Sloan (1948) on the heritability of different traits in livestock and poultry, respectively. Hence, it is deemed necessary to draw the attention to the fact that our present interest is to compare the estimates between the two groups, the treatment and the control, rather than to compare those of the controls with other reported figures. Keeping this in mind, we can, therefore, proceed with this comparison.

It will be recalled that two methods are used to estimate heritability. The first defines it in its broadest meaning while the

second method defines it in the narrowest sense. The first method gives the proportion of the total genic variance to the phenotypic variance, the second method gives the proportion of the additive variance to the phenotypic. Accordingly, any discrepancy between the two estimates by the two methods should be attributed to the differences between the components A and S. These differences have been sufficiently discussed in the previous section.

Figure 2 represents the estimates of heritability calculated by the first method. The general feature of these curves is that the heritability of body weight is generally high at the second and the tenth weeks of age and with a marked change in between. The treated females appear to have a higher total genic variance between the second and the seventh weeks of age. However, the difference is probably a reflection of maternal effect as represented by egg size. The difference between the two groups of females decreases until the two estimates are almost equal at about the eighth week of age. The situation is then reversed so that the genic variability of the control females is greater than that of the treated ones. Hence, it could be concluded that the absence of thyroxine decreases the total genic variability among the females.

The two male groups show quite the opposite picture. While the percentage of the total genic variance of the male control group is greater than that of the treated one before the seventh week of age the reverse is true thereafter. Thus the opposite conclusion arrived at with respect to the females applies to the male progeny.

It follows then that the treatment has different effects on the ratios of total genic variance of the two sexes. Since this situation is comparable to that pointed out by the inequality signs in Tables 7 and 8, the explanation given there applies here as well. This explanation, it will be recalled, is based on the presence of a variance factor common to paternal half-sibs. This common factor, however, cannot by the present technique of analysis be separated from dominance deviations. Accordingly, the discussion should turn to the estimates of heritability by the second method where only the percentages of the additive variance are evaluated.

For a better comparison, these estimates have been similarly plotted as shown in Figure 3. Some interesting observations can be stated here with respect to the trends of the four curves in Figure 3. The general situation can be summarized in the following two points. First, there is only a slight amount of additive variance at the second week of age. Hence, it is quite evident that most of the high corresponding estimates in Figure 2 at this age are not due to additive causes. Second, that following the second week, there is a general rise in the percentages of additive variance though this rise is interrupted by a slight negligible decrease at the eighth week of age.

Within the male progeny, the percentages of the additive variance are constantly higher in the control group than those in the treated one. The difference between these two lines indicates to what extent polygenic variance is hampered by the lack of thyroxine. It is quite evident that the great loss in the additive genic variance occurs at the sixth week of age after which it is almost

constant. The percentage of the additive genic variance of the treated males was about one-third of that of the control males.

In the female progeny the treated group has slightly higher percentage of additive variance up to the sixth week of age after which the situation is reversed. In general, the two groups of females have higher additive variance than the two male groups. Hence, there are two phases of discrepancy between the two sexes, one is between the two groups within each sex since the two groups do not behave similarly within each sex, and the second is between the two sexes regardless of the groups. These two discrepancies, however, are readily explainable on a genetic basis. The only difference between the two sexes with respect to genic material is that the female has a single X-chromosome and the "so-called" X-chromosome which is comparable to the Y-chromosome in the other organisms. The male chicken, however, has two X-chromosomes. Sex-linkage studies definitely show that the z- or the Y-chromosomes are inactive with respect to identifiable genotypes such that the sex with a single X is hemizygous with respect to the pair of genes concerned. It has also been sufficiently demonstrated that the Y-chromosome is different from the X-chromosome in the greater amount of heterochromatin which the former has. Moreover, Mather (1944) has shown that in *Drosophila melanogaster*, the Y-chromosome and the homeologous heterochromatic portion of the X, though they have no major effects, yet they are concerned with polygenic inheritance. This is also supported by the discovery in a number of plants of the heterochromatic chromosomes supernumerary to the normal complement. The findings of

Östergren (1947), that these extra heterochromatic materials have a slight effect on vigor and fertility in *Anthoxanthum* is in accordance with the relationship between heterochromatic and polygenic inheritance. To quote Mather (1949) "thus although heterochromatin contains few or no major genes, it is polygenically active."<sup>(1)</sup>

The present data accord with this point of view and it seems reasonable to conclude that the females have a higher additive variance or perhaps a greater number of polygenes for body weight.<sup>(2)</sup>

#### Body Measurements

The three body measurements observed in these experiments were taken as a sample to demonstrate the effect of thyroxins on the action of polygenes concerned with body conformation. Body conformation, however, is hard to define as a term. Hence, it was thought that by taking the measurements of the keel length, body depth and shank length, an adequate arbitrary approximation of body conformation would be obtained. Therefore, when the term, body

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(1) For a more complete discussion on heterochromatin and polygenes, see Darlington and Mather (1949) pages 150-152.

(2) The argument is based upon the formula devised by Castle (1921) a and b, and Wright (1921) for estimating the number of genetic factors in quantitative inheritance. In the derivation of this formula it has been shown that the genic variance  $\sigma^2_G = Ne^2/2$ , where  $N$  is the number of pairs of genes and  $e$  is the average effect of each allele. It follows that  $N = 2\sigma^2_G/e^2$ . Hence, it is evident that as  $\sigma^2_G$  increases, then  $N$  must also increase and that since the females have a large  $\sigma^2_G$  then they must have a greater number of polygenes. This relationship, however, holds true, assuming equal effect for each gene.



Table 2. Average body weights and standard deviation of each sex of the progeny in each group.

Sex Group	Number of birds	2nd week	6th week	8th week	10th week
Females					
Treatment	220	88.4 ± 12.2	297.1 ± 61.7	426.0 ± 101.4	598.2 ± 172.1
Control	218	93.6 ± 15.8	432.6 ± 78.8	666.5 ± 101.9	940.2 ± 127.4
Treatment:Control %		94.4	68.7	63.9	63.6
Males					
Treatment	200	89.4 ± 14.4	317.7 ± 64.8	464.7 ± 102.6	672.0 ± 171.9
Control	203	94.5 ± 19.1	476.6 ± 98.3	762.2 ± 130.2	1128.9 ± 166.3
Treatment:Control %		94.6	66.7	61.0	59.5

Table 3. Analysis of variance of body weight of the treated female progeny.

Source of variation	Degree of freedom	Sum of squares	Mean squares	6th week	10th week	Comments
Total	219	32,417.53	825,467.69			
Between dams' families	51	14,606.37	302,155.19			
Between sires' families	7	2,108.27	301.18	75,788.17	10,826.88	
Between dams within sires	44	12,498.10	284.05	226,367.02	5,144.71	
Between full sisters	168	17,811.16	106.02	521,312.50	3,114.96	
Total	219	2,234,719.71	6,427,337.73			
Between dams' families	51	811,479.02	2,427,907.71			
Between sires' families	7	194,182.68	27,740.38	617,363.63	88,194.80	B+4.23A+27.50S
Between dams within sires	44	617,296.34	14,029.46	1,810,544.08	41,148.73	B+4.23A
Between full sisters	168	1,423,240.69	8,471.67	3,999,430.02	23,806.13	B

Table 4. Analysis of variance of body weights of the control female progeny

Source of variation	Degrees of freedom	2nd week		6th week		Components of mean squares
		Sum of squares	Mean squares	Sum of squares	Mean squares	
Total	217	53,871.01		1,345,635.47		
Between dams' families	46	17,926.82		427,107.66		
Between sires' families	7	2,653.76	379.11	100,960.62	14,422.95	
Between Dams within sires	39	15,273.06	391.62	326,147.04	8,362.74	
Between full sisters	171	35,944.19	210.20	918,527.81	5,371.51	
		8th week		10th week		
Total	217	2,232,790.43		3,484,457.07		
Between dams' families	46	784,972.92		1,456,641.84		
Between sires' families	7	213,857.27	30,551.06	391,704.72	55,957.81	B+4.64A+27.25S
Between dams within sires	39	571,115.65	14,643.99	1,064,937.12	27,306.08	B+4.64A
Between full sisters	171	1,447,817.51	8,466.77	2,027,815.23	11,858.57	B

Table 5. Analysis of variance of body weights of the treated male progeny.

Source of variation	Degree of freedom	Sum of squares	Mean squares	Sum of squares	Mean squares	Sum of squares	Mean squares
				2th week	6th week	10th week	Components of
Total	199	40,932.87	834,101.50				
Between dams' families	44	14,635.30	265,210.98				
Between sires' families	7	2,620.99	374.43	50,709.68	7,244.24		
Between dams within sires	37	12,014.31	324.71	214,501.30	5,797.33		
Between full brothers	155	26,297.57	169.66	568,890.52	3,670.26		
Total	199	2,087,627.86					
Between dams' families	44	763,827.80	2,305,615.03				
Between sires' families	7	134,412.38	19,201.77	406,574.88	58,367.84		B+4.444+258
Between dams within sires	37	629,415.42	17,011.23	1,897,040.15	51,271.36		B+4.444
Between full brothers	155	1,324,000.08	8,541.94	3,542,981.57	22,857.95		B

Table 6. Analysis of variance of body weight of the control male progeny.

Source of variation	Degrees of freedom	2nd week		6th week		Components of mean squares
		Sum of squares	Mean squares	Sum of squares	Mean squares	
Total	202	73,282.74		1,939,092.75		
Between dams' families	42	27,898.04		687,282.78		
Between sires' families	7	5,403.58	771.94	161,386.82	23,055.26	
Between dams within sires	35	22,494.46	642.70	525,895.96	15,025.60	
Between full brothers	160	45,384.70	283.65	1,251,809.97	7,823.81	
		8th week		10th week		
Total	202	3,406,007.17		5,560,849.22		
Between dams' families	42	1,059,985.61		1,598,391.51		
Between sires' families	7	243,555.18	34,793.60	397,453.40	56,779.06	B+4.72A+25.37S
Between dams within sires	35	816,430.43	23,326.58	1,200,938.11	34,312.52	B+4.72A
Between full brothers	160	2,346,021.56	14,662.63	3,962,457.71	24,765.36	B

Table 7. Components of variance of body weight of the female progeny.

Components of variance	2nd week		6th week		8th week		10th week	
	treatment	control	treatment	control	treatment	control	treatment	control
S	.62	0.00	206.62	222.29	498.58	583.74	1,710.77	1,051.44
A	42.09	39.10	479.85	644.66	1313.90	1331.30	4,099.91	3,329.20
S + A	42.71	39.10	686.47	867.05	1812.48	1915.04	5,810.68	4,380.64
<hr/>								
B	106.02	210.20	3114.96	5371.51	8471.67	8966.77	22,886.13	11,858.57
B + S + A	148.73	249.30	3801.43	6238.56	10286.15	10381.81	29,616.81	16,239.21
<hr/>								
$\frac{A-S}{B+S+A}$	0.279	> 0.157	0.072	> 0.068	0.079	> 0.072	0.081	< 0.140

Table 8. Components of variance of body weights of the male progeny.

Components of variance	2nd week		6th week		8th week		10th week	
	treat-ment	control	treat-ment	control	treat-ment	control	treat-ment	control
S	1.99	5.09	57.88	316.50	87.62	451.99	292.36	885.56
A	24.92	76.07	479.07	1525.80	1907.50	1835.58	6395.42	2022.70
S + A	36.91	81.16	536.95	1842.30	1995.12	2287.57	6687.78	2908.26
B	169.66	283.65	3670.26	7823.81	8541.94	14,662.63	2857.95	24765.36
B + S + A	206.56	364.81	4207.21	9666.11	10537.06	16950.20	29541.23	27673.62
$\frac{A-S}{B+S+A}$	0.159 <	0.195 <	0.100 <	0.125 <	0.173 >	0.082 >	0.267 >	0.041

Table 9. Estimates of heritability of body weight of each sex of the progeny in each group.

Sex	Group	Number of birds	2nd week		6th week		8th week		10th week	
			(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Females	Treatment	220	0.574	0.017	0.361	0.217	0.352	0.154	0.392	0.231
	Control	218	0.314	0.000	0.278	0.143	0.369	0.225	0.540	0.259
Males	Treatment	200	0.357	0.039	0.255	0.055	0.379	0.033	0.452	0.038
	Control	203	0.445	0.056	0.381	0.131	0.270	0.107	0.210	0.128

$$(1) h^2 = \frac{2(S + A)}{S + A + B}$$

$$(2) h^2 = \frac{4S}{S + A + B}$$



Figure 1. Body weights on 2nd, 6th, 8th, and 10th weeks of age.

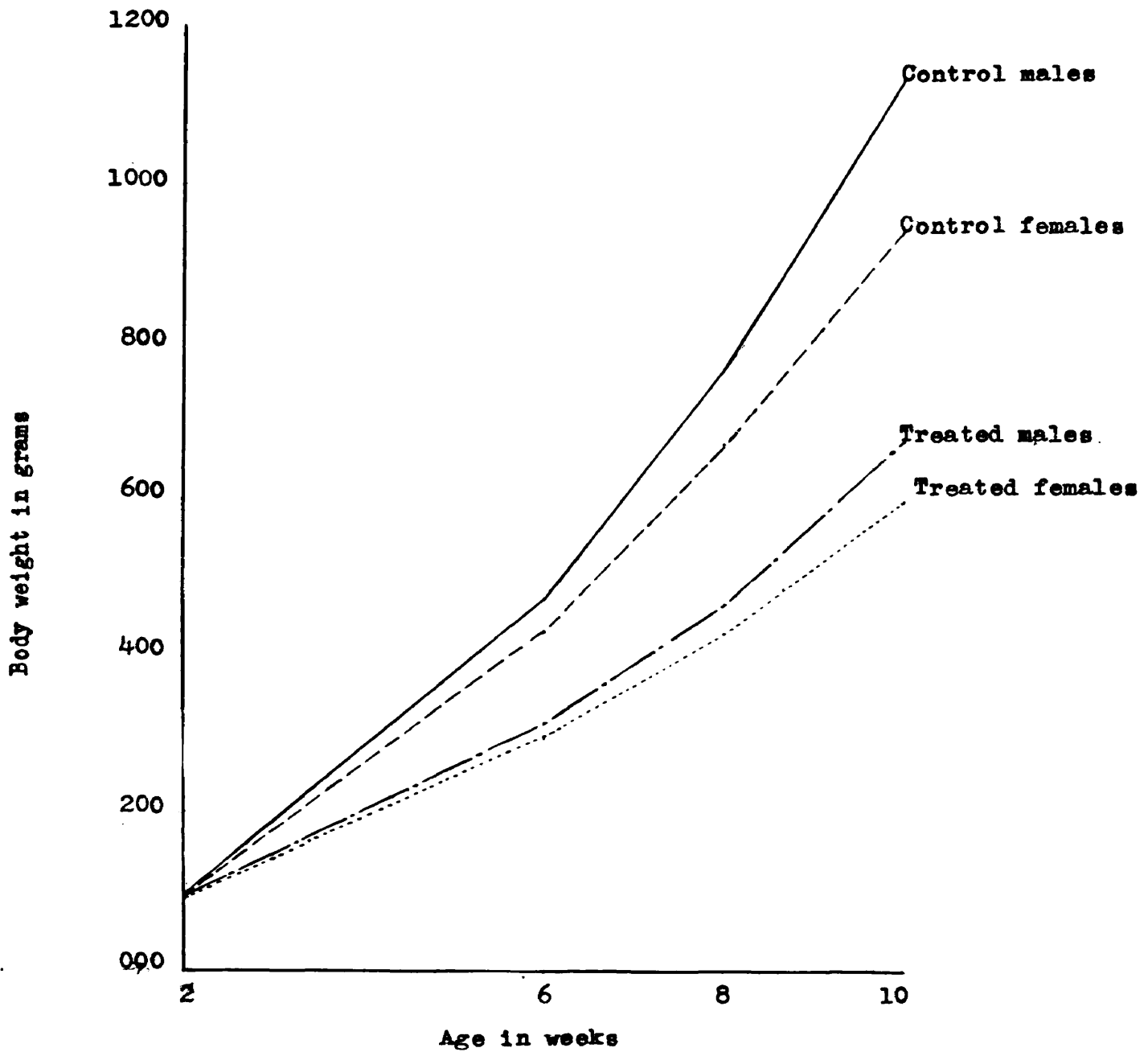


Figure 2. Estimates of heritability of body weight of each sex of the progeny in each group --  $h^2 = 2(S + A)/(S + A + B)$

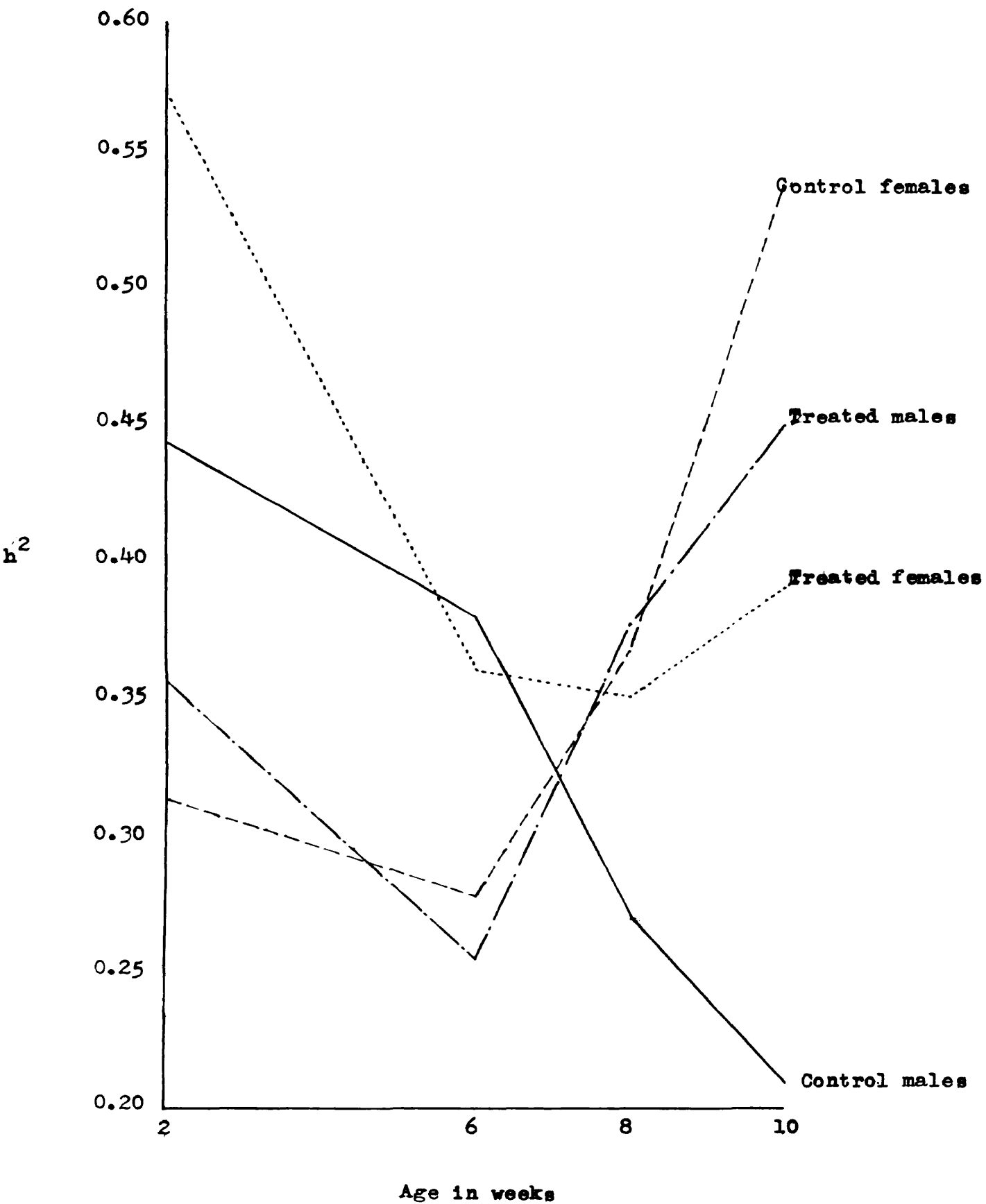
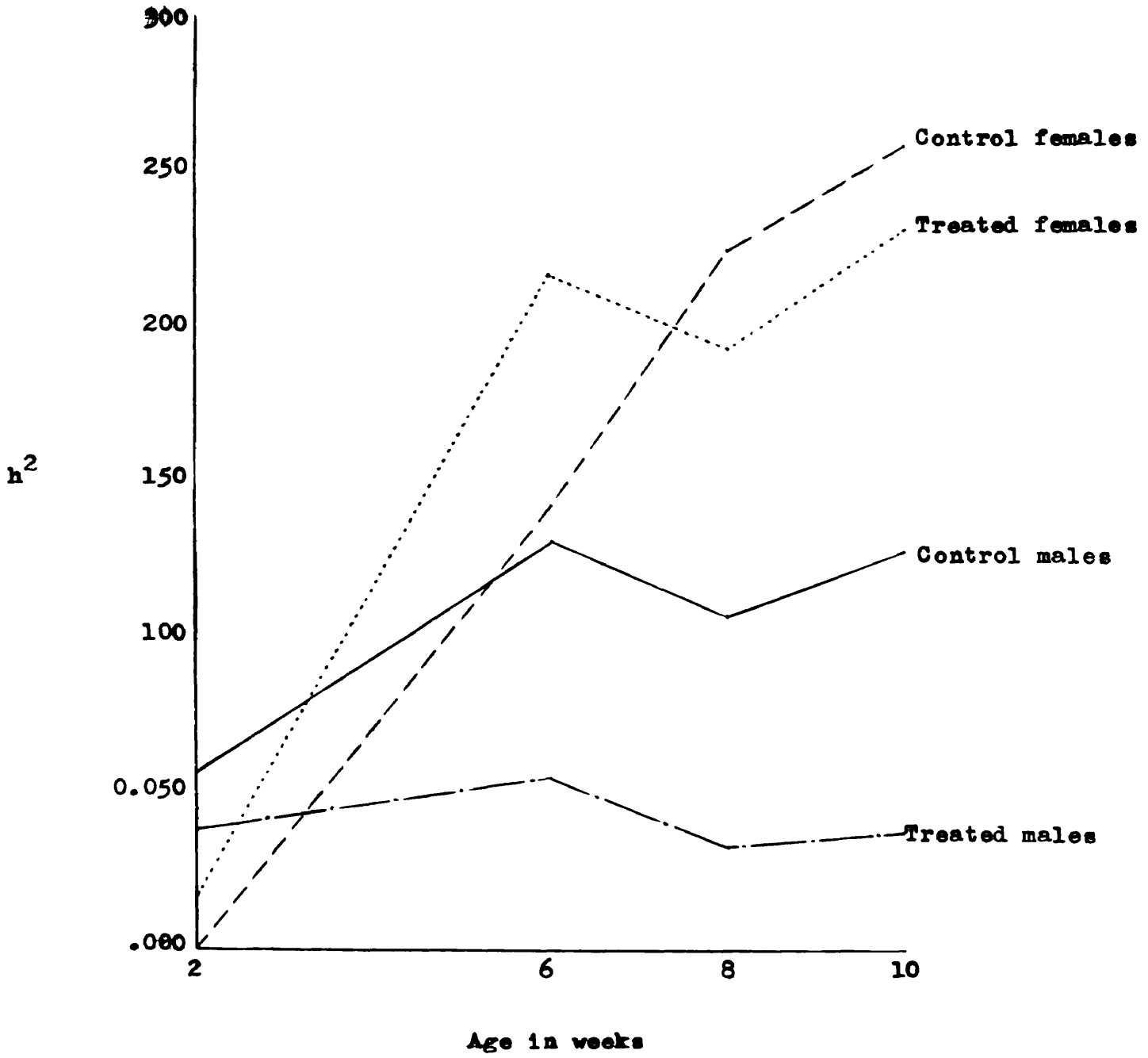


Figure 3. Estimates of heritability of body weight of each sex of the progeny in each group --  $h^2 = 4S/(S + A + B)$



conformation is mentioned in this report, its meaning should not exceed what these body measurements are able to illustrate.

The means and the standard deviations of these three characters are given in Table 10 from which it is evident that the measurements of the treated birds are on the average about 25 percent smaller than those of the controls. <sup>(1)</sup> The reduction in body depth, however, was slightly less than that in the other two characters.

Tables 11, 12, 13 and 14 give the analysis of variance of these three measurements for each group within each sex separately. The components of variance are separated and given in Tables 15 and 16. In the last row of each one of these two Tables the total phenotypic variance is shown (i.e. the sum  $B+S+A$ ). It will be noticed that the phenotypic variance of these three characters in the treated group is about three times as great as that for the control groups. The total variance of body weight was reported in Tables 7 and 8 from which it is evident that at ten weeks of age the phenotypic variance of the treated group is less than twice that of the control group. The direct corollary of this comparison is that the absence of thyroxine increases the phenotypic variability of body conformation more than that of body weight.

The estimates of the heritability of each one of these

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(1) cf. the description given by Landauer (1929), Mayhew and Upton (1932), and Upton (1934).

three body measurements are given in Table 17. It is evident from these data that in both sexes the estimates for the treated group by either method are in general greater than those of the control group. The only exception to this general observation is the heritability of the body depth of the females as estimated by the first method. This general increase in the genic variance through depriving the animal from thyroxine is in accord with the observation on the phenotypic level.

Once more we notice that in each one of these three body measurements, the control females have higher percentages of additive variance than those of the males. This is evident from comparing the estimates of heritability by the second method between the two sexes.

#### Thyroid Weight and Testes Weight

The means of the thyroid weights and testes weights are given in Table 18. It is evident from these data that while the average thyroid weight of the treated birds increases to as much as seven-fold times its normal average weight, the average testes weight of the treated males loses 35 percent of its average normal weight. Since both the thyroid weight and the testes weights may be correlated with body weight and since the latter was seriously depressed by the treatment, it is preferable to postpone the discussion on the relationship among these three weights until the correlations among them are discussed.

The hypertrophy of the thyroid gland as caused by feeding thiouracil is known to be a function of two factors. First, the

Table 10. Measurements of keel length, body depth, and shank length at 10 weeks of age.

Sex	Group	Number of birds	Means in Millimeters		
			Keel length	Body depth	Shank length
Females	Treatment	220	65.8 ± 11.0	73.0 ± 9.9	69.2 ± 10.2
	Control	218	86.7 ± 5.7	92.5 ± 5.4	91.3 ± 5.4
	Treatment: control %		75.9	78.9	75.8
	Treatment		200	68.6 ± 11.3	75.7 ± 9.6
Males	Control	203	92.6 ± 5.9	97.3 ± 6.1	99.2 ± 6.3
	Treatment: Control %		74.1	77.8	74.1

Table 11. Analysis of variance of keel length, body depth, and shank length of the treated female progeny at 10 weeks of age.

Source of Variation	D.F.	Keel length		Body depth		shank length		Components of Variance
		S.S. (1)	M.S. (2)	S.S.	M.S.	S.S.	M.S.	
Total	219	26,200.38	21,627.93	22,560.23				
Between Dams' Families	51	12,344.59	8,764.78	10,264.48				
Between Sires' Families	7	3,617.09	2,432.22	247.46	424.06	2,968.44	424.06	34.23 A + 27.50 B
Between Dams Within Sires	44	8,727.50	198.35	6,332.56	143.92	7,296.04	165.82	B + 4.23 A
Between Full-Sisters	100	13,855.79	82.47	12,863.15	76.57	12,295.75	73.19	B

(1) Sum of squares

(2) Mean square

Table 12. Analysis of variance of keel length, body depth, and shank length of the control female progeny at 10 weeks of age.

Source of Variation	D/F	Keel length		Body depth		Shank length		Components of Variance
		S.S.	M.S.	S.S.	M.S.	S.S.	M.S.	
Total	217	6,828.37		6,218.43		6,355.61		
Between Dams' Families	46	2,161.61		2,819.77		2,598.98		
Between Sires' Families	7	678.24	96.89	594.42	84.92	504.73	72.10	B+4.64A+27.25S
Between Dams' Within Sires	39	1,483.37	38.04	2,225.35	57.06	2,094.25	53.70	D+4.64A
Between Full-Sisters	171	4,656.76	27.29	3,398.66	19.88	3,756.63	21.97	B



Table 13. Analysis of variance of keel length, body depth, and shank length of the treated male progeny at 10 weeks of age.

Source of Variation	D/F	Keel length		Body depth		Shank length		Components of Variance
		S.S.	M.S.	S.S.	M.S.	S.S.	M.S.	
Total	199	24,868.38		18,156.88		20,015.75		
Between dams' families	44	10,953.75		7,054.87		7,659.35		
Between sires' families	7	3,182.08	454.58	1,915.19	273.60	1,416.46	202.35	B+4.44A+25S
Between dams within sires	37	7,771.67	210.05	5,139.68	138.91	6,242.89	168.73	B+4.44A
Between full brothers	155	13,914.63	89.77	11,102.01	71.63	12,356.40	79.72	B

Table 14. Analysis of variance of keel length, body depth and shank length of the control male progeny at 10 weeks of age.

Source of variation	D/F	Keel length		Body depth		Shank length		Components of variance
		S.S.	M.S.	S.S.	M.S.	S.S.	M.S.	
Total	202	6,866.21		7,446.67		8,012.27		
Between dams' families	42	2,614.96		2,222.74		2,162.71		
Between sires' families	7	719.32	102.76	497.79	71.11	398.64	56.95	344.72A+25.37S
Between dams within sires	35	1,895.64	54.16	1,724.95	49.28	1,764.07	50.40	344.72A
Between full brothers	160	4,251.25	26.57	5,223.93	32.65	5,849.56	36.56	2

Table 15. Components of variance for keel length, body depth, and shank length of the female progeny at 10 weeks of age.

Components of variance	Keel length		Body depth		Shank length	
	Treatment	Control	Treatment	Control	Treatment	Control
S	11.58	2.56	7.40	1.62	9.39	0.68
A	27.39	2.32	15.92	8.01	21.90	6.84
S + A	38.97	4.88	23.32	9.63	31.29	7.52
B	82.47	27.29	76.57	19.88	73.19	21.97
B + S + A	121.44	32.17	99.89	28.91	104.48	29.49

Table 16. Components of variance for keel length, body depth and shank length of the male progeny at 10 weeks of age.

Components of variance	Keel length		Body depth		Shank length	
	Treat-ment	[control]	Treat-ment	[control]	Treat-ment	[control]
S	9.78	1.92	5.39	0.86	1.34	0.26
A	27.09	5.85	15.15	3.52	20.05	2.93
S + A	36.87	7.77	20.54	4.38	21.39	3.19
B	89.77	26.57	71.63	32.65	79.72	36.56
B + S + A	126.64	34.34	92.17	37.09	101.11	39.75

Table 17. Estimates of heritability of keel length, body depth, and shank length for each sex of the progeny in each group.

Sex	Group	Number of birds	$h^2$					
			Keel length (1)	Keel length (2)	Body depth (1)	Body depth (2)	Shank length (1)	Shank length (2)
Females	Treatment	220	0.642	0.381	0.467	0.296	0.599	0.359
	Control	218	0.303	0.318	0.625	0.141	0.510	0.092
Males	Treatment	200	0.582	0.309	0.446	0.234	0.423	0.053
	Control	203	0.453	0.224	0.237	0.093	0.161	0.026

$$(1) h^2 = \frac{2(S+A)}{B+S+A}$$

$$(2) h^2 = \frac{4S}{B+S+A}$$

ability of the anterior pituitary to produce thyrotropic hormones, and second, the ability of the thyroid gland itself to respond to the thyretropic hormones. Since these two factors are inseparable in the present study, the term "goiterogenic capacity" of the thyroid is to be used here.

The analysis of variance of the thyroid weight and the testes weights are given in Tables 19, 20, 21, and 22. The components of this variance are separated as shown in Table 23 from which the estimates of the heritability of the thyroid weight and testes weights are calculated by the two methods (Table 24).

The most striking observation on the data of Table 24 is the fact that the heritability of the thyroid weight as estimated by the second method is equal to zero in both sexes while it is equal to 0.613 and 0.711 for the females and the males, respectively, when estimated by the first method. The direct conclusion from this observation is the fact that none of the additive variance is concerned with the goiterogenic capacity of the gland and that dominance is to be suspected (cf. Middle, 1947).

Further evidence for this hypothesis is found when the frequency distributions of the thyroid weight of both the treated and the control groups are plotted. (Figure 4). Since there is a slight sexual dimorphism in the thyroid weight of either the treated or the control group (Table 18), the data of both sexes were combined. Hence, the upper curve of Figure 4 is based on a total of 420 treated birds and the lower one represents 421 control birds.

The curve of the control birds shows a typical normal dis-

tribution with a slight positive skewness, while that for the treated birds shows a very sharp negative skewness which is evidence that the goiterogenic capacity of the thyroid may be influenced by non-additive genes.

A selection program is now under way to study the mode of inheritance of the goiterogenic capacity through the establishment of two lines representing the extremes of this character.

Several interesting questions may arise if the existence of one or more genes for goiter is definitely established in chickens. Speculating on this problem, it could be asked: what is the frequency of such gene in the different flocks of the different breeds? The estimate of the frequency of such proposed gene would be relatively easy since the feeding of thiouracil reveals its presence. It also could be asked: what is the relationship, if any, between this gene, or genes, and that of "thyrogenous dwarfism"? (Mayhew and Hoar, 1932, and Top, 1934) Are they identical? If so, then why these genes appear sporadically under normal conditions as shown by the reports of these workers? Is this sudden appearance due to chance recombination of the recessive alleles or is it also conditioned by certain physiological or genetical factors? Moreover, what is the relationship between this gene or genes and the hypophysis?

It is clear that these observations open up a broad field of investigation which deserves the attention of the research workers.

Returning to the data of Table 24, it will be noticed that the females have a higher percentages of additive variance of the

thyroid weight than the males (0.139 versus 0.081, respectively). This is in accord with the hypothesis previously mentioned that the heterochromatic z-chromosome of the female is polygenically active.

With respect to the testes weight, it is evident from Table 24 that the heritability of this character as estimated by either method, is considerably higher in the treated birds than in the control. It is quite evident that the lack of thyroxine favors genic variability of testes weight and more particularly the additive portion of the variance of this character.

The fact that the treatment causes a loss in the additive variance of the thyroid weight and an increase in the additive variance of testes weight may indicate the presence of some sort of a compensatory mechanism between the two glands. It is evident that such a relationship is better understood when the correlations between these two glands are evaluated on both phenotypic and genotypic levels as shown below.

#### Estimates of correlation

The correlation studies reported herein are concerned with two general phases. First, the analysis of the relationship, if any, between body weight and each of the thyroid weight and testes weight. Second, the relationship between the weights of the latter two glands.

Analysis of covariance has been determined for each possible combination of the three characters of body weight at ten weeks of age, thyroid weight and testes weight. These analysis are present in Tables 25-32. Following the general plan of Table 1, the mean covariance has been



Table 18. Average thyroid weight and testes weight of each sex of the progeny in each group.

Sex	Thyroid Weight			Testes Weight		
	Treatment Number of Birds	Mean Weight in Centigrams	Control Number of Birds	Control Mean Weight in Milligrams	Treatment Mean Weight in Centigrams	Control Mean Weight in Centigrams
Females	220	183.0	218	105.7		
Males	200	182.2	203	107.0	86.9	135.6

Table 19. Analysis of variance of thyroid weight of the treated female progeny.

Source of Variation	D/F	Sum of Squares	Mean squares	Components of Variance
Total	219	5,453,923.98		
Between dams' families	51	2,472,205.35		
Between sires' families	7	231,548.54	3 3078.36	B + 4.23A + 27.508
Between dams within sires	44	2,240,656.81	50,924.02	B + 4.23 A
Between full-sisters	168	2,981,718.63	17,748.33	B

Table 20. Analysis of variance of thyroid weight of the control female progeny.

Source of variation	D/F	Sum of squares	Mean square	Components of variance
Total	217	267,919.19		
Between dams' families	46	120,062.42		
Between sires' families	7	46,633.36	6,661.91	B + 4.64 A + 27.25S
Between dams within sires	39	73,429.06	1,882.80	B + 4.64 A
Between full sisters	171	147,856.87	864.66	B

Table 21. Analysis of variance of thyroid weight and testis weight of the treated male progeny.

Source of Variation	D/F	Thyroid Weight		Testes Weight		Components of Variance
		S.S.	M.S.	S.S.	M.S.	
Total	199	5,101,038.16		1,294,442.39		
Between dams' families	44	2,418,334.80		552,671.17		
Between sires' families	7	209,716.36	29,959.48	279,010.25	39,858.61	B+4.44A+258
Between dams within sires	37	2,208,618.44	59,692.40	273,660.92	7,396.24	B+4.44A
Between full brothers	155	2,682,703.36	17,307.76	741,771.22	4,785.62	B

Table 22. Analysis of variance of thyroid weight and Testes weight of the control male progeny.

Source of Variation	D/Y	Thyroid Weight		Testes Weight		Components of variance
		S.S.	M.S.	S.S.	M.S.	
Total	202	258,599.12		5,382,492.26		
Between dams' families	42	96,602.62		1,713,203.39		
Between sires' families	7	31,641.13	4,520.16	442,014.20	63,144.89	B+4.72A+25.37S
Between dams within sires	35	64,961.49	1,856.04	1,271,189.19	36,319.69	B+4.72A
Between full brothers	160	161,996.50	1,012.48	3,669,288.87	22,933.86	B

Table 23. Components of variance of thyroid weight and testes weight.

Components of Variance	Females		Males		Testes weight	
	Treatment	Control	Treatment	Control	Treatment	Control
S	0.000	175.38	0.00	105.01	1,298.49	1,057.36
A	7,842.95	219.42	9,546.09	178.72	587.98	2,836.15
S + A	7,842.95	394.80	9,546.09	283.73	1,886.47	3,893.51
B	17,748.33	864.66	17,307.76	1,012.48	4,785.62	22,933.06
B + S + A	25,591.28	1,259.46	26,853.85	1,296.21	6,672.09	26,826.57

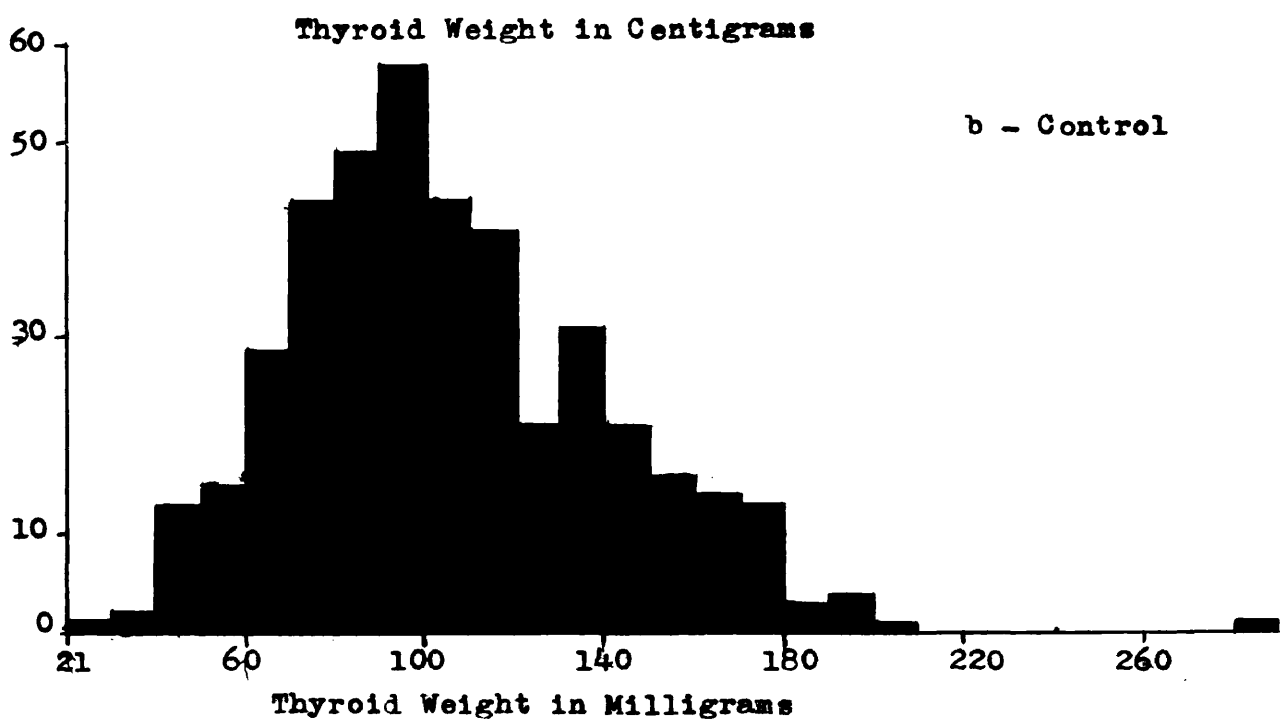
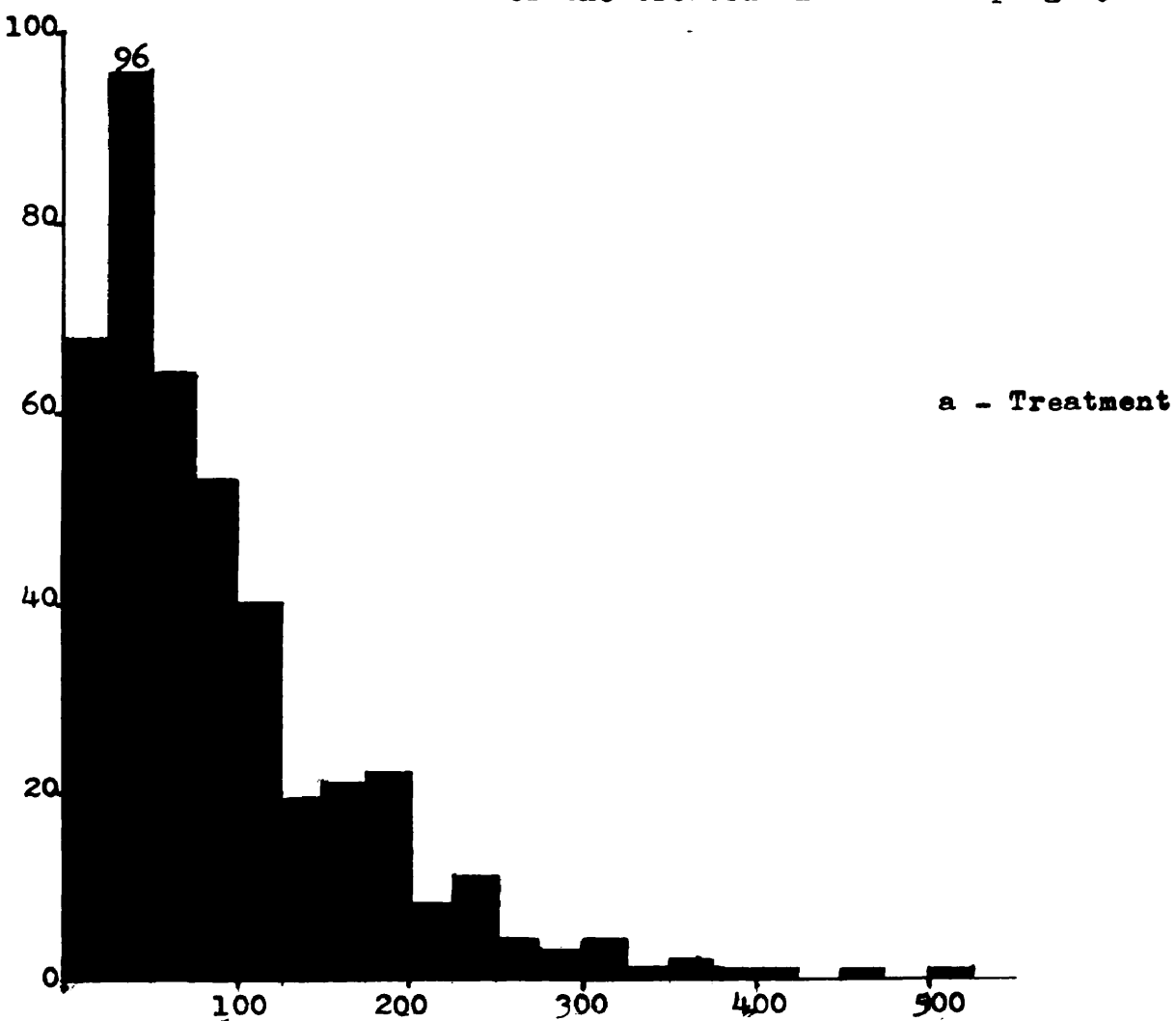
Table 24. Estimates of heritability of thyroid weight and testis weight.

Sex	Group	Number of Birds	Thyroid Weight (1)	Thyroid Weight (2)	Testes Weight (1)	Testes Weight (2)
Females	Treatment	220	0.613	0.600		
	Control	218	0.627	0.139		
Males	Treatment	200	0.711	0.600	0.565	0.778
	Control	203	0.438	0.081	0.290	0.158

$$(1) h^2 = \frac{2(S+A)}{S+A+B}$$

$$(2) h^2 = \frac{4B}{S+A+B}$$

**Figure 4. Frequency distribution of the thyroid weights of the treated and control progeny.**





reduced to its components as shown in the last column of each one of these Tables. Hence, these components are calculated as given in Tables 33 and 34. From these components the genetic, environmental and phenotypic correlations are calculated according to the three formulae previously given under the "method of analysis". These estimates are given in Table 35.

In the control group, comparing the genetic correlation between body weight and thyroid weight, shows that there is a high genetic relationship between these weights. This fact is readily explainable by the close relationship between metabolic rate and growth and the thyroid gland.

The smallest reported correlation in the control group is that between body weight and testes weight, viz. 0.130. This weak relationship is evident from the fact that the function of the testis is primarily reproductive. Although this correlation is low, yet it indicates some relationship between the testes weight and body weight.

In the control group, the genetic correlation between the thyroid weight and the testes weight is a medium sized one (0.400); this fact may be due to the endocrinal relationship between the two glands through the hypophysis. However, since both glands are correlated with the body weight, it seems advisable to resort to a partial correlation between them as will be shown later.

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(1) The term genetic correlation as used in this section refers to the correlation for the estimate of which the total genetic covariance and variance have been used.

If this comparison is extended to the treated groups, we have quite a different situation. The genetic correlation between body weight and thyroid weight are still the highest among the observed estimates for this group. However, the situation with respect to sex is reversed so that while in the controls the females have a higher genetic correlation than that of the males, the reverse is true under the treatment. Moreover, the genetic correlation between body weight and testes weight is higher in the treated birds than it is in the control. Meanwhile, the genetic correlation between thyroid gland and testes weight is almost the same under the treatment and the control.

The data of Table 35 could be divided into two distinct groups as to the relative magnitude of the genetic correlation to the corresponding phenotypic one. The first group comprises the five pairs of characters of the control and the treated females and the treated males. The second group comprises the three pairs of characters of the control males. The distinction between these two groups lies in the fact that while the genetic correlation is greater than the phenotypic ones in the first group, the opposite situation is true with respect to the second group. It is conceivable that if the two environments of a pair of characters have something in common, then the phenotypic correlation becomes greater than the genetic one. Accordingly there must be something common to the environment of each pair of characters reported in the second group. However, this common factor could not be thyroxine, since the control females have not a higher phenotypic correlation

than their genetic one. Hence, we have to look for another factor and in order to explain it the technique of partial correlation is resorted to below.

Returning to the correlation between the weights of the thyroid and testes, it has been previously stated that since each one of these glands is correlated with the body weight, then it seems more reasonable to evaluate the relation between the two glands in terms of partial correlations. These statistics between the two glands were calculated according to the formula given by Snedecor (1946). For the control group the partial correlation between thyroidal weight and testes weight (i.e. the body weight being held constant) are 0.391 and 0.305 on the genotypic and phenotypic levels, respectively (Table 36). It is evident that the relative situation between the correlations on the two levels is reversed so that the partial genotypic correlations is actually higher than the phenotypic one when the body weight is kept constant. The direct corollary from this inference is the fact that the body weight is the common factor which is responsible for the increase in resemblance between the weights of the two glands.

The partial correlations between the two glands in the treated group are 0.061 and 0.034 on the genotypic and the phenotypic levels, respectively. Therefore, it is evident that the variability of body weight comprises almost all the genetic and phenotypic correlated variability between the two glands in the treated birds.

If the thyroid is kept constant (Table 36), the partial correlation between body weight and testes weight in the control

group becomes negligible. This fact reveals that the variance of the thyroid weight accounts almost entirely for the correlated variability between body and testes weights. In the treated birds, though the partial correlations are less than the simple ones, yet the former still indicate a fair relationship between body weight and testes weight even when the thyroid weight is kept constant. Hence, it seems reasonable to infer that by depriving the animal of thyroxine the testes weight is more highly correlated with the body weight regardless of the goiterogenic capacity of the thyroid gland.

Keeping testes weight constant, it is evident that the situation does not alter the relationship between body weight and thyroid weight as previously estimated by the simple correlation. Therefore, it could be concluded that the variance of testes weight has negligible effect on the correlated variability between the thyroid weight and body weight.

#### General Discussion

In this part of the discussion an attempt is made to account in general for the results as reported and discussed separately for each character in the previous pages.

The results show that lack of thyroxine has one general effect on the phenotypic variability of all the characters studied. This effect is to increase such variability. This increase in variability, however, is limited to the phenotypic level, the effects

Table 25. Analysis of covariance of body weight and thyroid weight - treated female progeny at 10 weeks of age.

Source of Covariation	D/F	Sum of Products	Mean Product	Components of Covariance
Total	219	3,135,601.64		
Between dams' families	51	1,432,161.81		
Between sires' families	7	302,878.02	43,268.29	B+4.23A+27.50S
Between dams within sires	44	1,129,283.79	25,665.54	B+4.23A
Between full-sisters	168	1,703,439.83	10,139.52	B

Table 26. Analysis of covariance of body weight and thyroid weight - control female progeny at 10 weeks of age.

Source of covariation	D/F	Sum of products	Mean products	Components of covariance
Total	217	550,782.1		
Between dams' families	46	279,389.3		
Between sires' families	7	124,916.6	17,845.2	B <sub>4</sub> 4.64A <sub>27.25</sub> S
Between dams within sires	39	154,472.7	3,960.8	B <sub>4</sub> 4.64A
Between full sisters	171	271,392.8	1,587.1	B

Table 27. Analysis of covariance of body weight and thyroid weight - treated male progeny at 10 weeks of age.

Source of covariation	D/F	Sum of products	Mean products	Components of covariance
Total	199	3,324,477.34		
Between dams' families	44	1,702,826.43		
Between sires' families	7	141,845.25	20,263.61	<del>B44.44A+25S</del>
Between dams' within sires	37	1,560,981.18	42,188.68	<del>B44.44A</del>
Between full brothers	155	1,621,650.91	10,462.26	B

Table 28. Analysis of covariance of body weight and thyroid weight - control male progeny at 10 weeks of age.

Source of covariation	D/F	Sum of products	Mean products	Components of covariance
Total	202	746,475.67		
Between dams' families	42	222,513.27		
Between sires' families	7	94,527.77	13,503.97	B+4.72A+25.37S
Between dams within sires	35	127,985.50	3,656.73	B+4.72A
Between full brothers	160	523,962.40	3,274.77	B



Table 29. Analysis of covariance of body weight and testes weight - treated progeny at 10 weeks of age.

Source of covariance	D/F	Sum of products	Mean products	Components of covariance
Total	199	1,290,692.50		
Between dams' families	44	553,829.30		
Between sires' families	7	273,760.38	39,108.63	$B+4.44A+25S$
Between dams within sires	37	280,068.92	7,569.43	$B+4.44A$
Between full brothers	155	736,863.20	4,753.96	B

**Table 30. Analysis of covariance of body weight and testes weight - control progeny at 10 weeks of age.**

Source of covariance	D/F	Sum of products	Mean products	Components of covariance
Total	202	1,802,536.61		
Between dams' families	42	494,329.91		
Between sires' families	7	156,890.41	22,412.92	B+4.72A+25.37B
Between dams within sires	35	277,439.50	7,926.84	B+4.72A
Between full brothers	160	1,368,206.70	8,551.29	B

Table 31. Analysis of covariance of thyroid weight and testes weight - treated progeny at 10 weeks of age.

Source of covariation	D/F	Sum of products	Mean products	Components of covariance
Total	199	791,993.03		
Between dams' families	44	411,452.63		
Between sires' families	7	189,985.70	27,140.81	B+4.44A+25S
Between dams within sires	37	221,466.93	5,985.59	B+4.44A
Between full brothers	155	380,540.40	2,455.10	B

Table 32. Analysis of covariance of thyroid weight and testes weight - control progeny at 10 weeks of age.

Source of covariance	D/F	Sum of products	Mean products	Components of covariance
Total	202	507,766.25		
Between dams' families	42	168,195.35		
Between sires' families	7	57,935.07	8,276.44	B+4.72A+25.37S
Between dams within sires	35	110,260.28	3,150.29	B+4.72A
Between full brothers	160	339,570.90	2,122.32	B

Table 33. Components of covariance of body weight and thyroid weight - all classifications at 10 weeks of age.

Components of covariance	Female progeny		Male progeny	
	Treatment	Control	Treatment	Control
S	640.10	509.52	-877.00	388.15
A	3,670.45	511.57	7,145.59	80.92
SAA	4,310.55	1,021.09	6,268.59	469.07
B	10,139.52	1,587.10	10,462.26	3,274.77
B+SAA	14,450.07	2,608.19	16,730.85	3,743.84
B-S-A	5,828.97	566.01	4,193.67	2,805.70

Table 34. Components of covariance for body weight and testes weight, and between thyroid weight and testes weight -treated and control pregnancy at 10 weeks of age.

Components of covariance	Body weight and testes weight		Thyroid weight and testes weight	
	Treatment	Control	Treatment	Control
S	1,261.57	570.99	846.21	202.06
A	674.11	-132.30	795.16	217.79
B+A	1,895.68	438.69	1,641.37	419.85
B	4,753.96	8,551.29	2,455.10	2,122.32
B+S+A	6,649.64	8,989.98	4,096.47	2,542.17
B-S-A	2,858.28	8,112.60	813.73	1,702.47

Table 35. Estimates of genetic, environmental and phenotypic correlation between two of each characters: body weight at 10 weeks of age, thyroid weight and testes weight.

Characters	Sex	Treatment			Control		
		(1) $r_{Gxy}$	(2) $r_{Exy}$	(3) $r_{xy}$	$r_{Gxy}$	$r_{Exy}$	$r_{xy}$
Body weight & thyroid weight	Females	0.639	0.437	0.525	0.776	0.302	0.577
	Males	0.785	0.374	0.594	0.516	0.703	0.625
Body-weight & testes-weight	Females	0.534	0.417	0.474	0.130	0.398	0.330
	Males	0.387	0.172	0.306	0.400	0.457	0.431

(1) Genetic  $r$       (2) Environmental  $r$       (3) Phenotypic  $r$

Table 36. Partial correlations among body weight, thyroid weight, and testes weight at 10 weeks of age.

The variable characters	The constant character	Treatment	Control
"X Y"	"Z"	(1) $r_{XY.Z}$	(2) $r_{XY.Z}$
Body weight & thyroid weight	testes weight	0.741	0.536
Body weight & testes weight	thyroid weight	0.403	0.381
Thyroid weight & testes weight	body weight	-0.061	0.034
		0.562	0.567
		-0.097	0.087
		0.391	0.305

(1) Partial genic correlation.

(2) Partial phenotypic correlation.



on additive genetic variability being inconsistent and dependent upon the character in question. Thus, while the lack of thyroxine relatively decreases the additive genetic variance of body weight and thyroid weight, it increases that of body measurement and testes weight. These discrepancies appear to raise some controversy and the following is offered as an explanation:

Let  $\sigma_p^2$  = the phenotypic variance.

$\sigma_G^2$  = the additive variance due to the thyroid gland.

$\sigma_G^2$  = the additive variance due to all non-thyroidal causes, viz, all sources of additive genetic variance except that caused by thyroxine.

$\sigma_E^2$  = the environmental variance.

Accordingly, in the normal birds the heritability of a certain trait in the narrowest sense will be:

$$h_c^2 = \frac{\sigma_G^2 + \sigma_G^2}{\sigma_G^2 + \sigma_G^2 + \sigma_E^2} \tag{1}$$

Where the subscript c means the controls.

If the animal is deprived from thyroxine, then this heritability becomes:

$$h_t^2 = \frac{\sigma_G^2}{\sigma_G^2 + \sigma_E^2} \tag{2}$$

Where the subscript t means the treatment, and it is evident that whatever the size of  $\sigma_G^2$  may be, the  $h_c^2$  is always greater than  $h_t^2$ . In other words, this conclusion means that the control birds should always have a greater percentage of additive genetic variance than that of the treated birds. The above relationship has been observed with respect to the heritability of body weight and thyroid weight. However, it should be noticed that  $\sigma_G^2$  should always be the same in both the treated and the control groups. If  $\sigma_G^2$  in the treated birds is increased over that of the control, then  $h_t^2$  is greater than  $h_c^2$ . This relationship has been observed with respect to the heritability of body measurements and testes weight and it implies that the expression of the additive genotype of the non-thyroidal causes of development is apt to be changed under the thiouracil treatment (cf. Juhn, 1945).

Finally, it has been pointed out in the introduction that growth is the outcome of mitotic activities. It is desirable before the discussion comes to an end to dwell for a while upon the relationship between mitosis and the hormone thyroxine. It is well established that the fundamental function of this hormone is in metabolism. When no thyroxine is secreted the metabolic rate is reduced to a minimum as is the case when there is starvation. Some studies on the cytological consequences of starvation in animals have shown that mitosis is hampered under such conditions and this primarily occurs because of a chemical change in the chromosomes themselves. Darlington and Mather (1949) summarized this situation as follows: The chromosomes fail to reproduce, or to

separate in mitosis and the coiling of the chromosome thread does not occur. These authors attribute such failure of mitosis to the lack of nucleic acid which "regulates the reproduction of the chromosomes".

In accordance with these findings, Mittler and Herman (1950) have recently demonstrated that a solution of thyroid powder will induce abnormal mitotic figures in the root of the onion (*Allium cepa*). However, other mammalian hormonal substances such as adrenaline, insulin and parathyroid powder failed to induce these abnormalities. Their findings, though not confirming the effect of thyroxine on mitosis, definitely showed that such an effect is due to "an unknown water soluble factor in desiccated thyroid tissue".

## SUMMARY AND CONCLUSIONS

Eight New Hampshire sires were mated to an average of five females each, and the offspring of each hen were divided into two groups. One group served as controls while the other group was fed a ration containing 0.2 per cent thiouracil. These two groups of full-sibs were compared with respect to estimates of heritability, genetic and phenotypic correlations. The characters studied were thyroid weight, testes weight, body weight, and the three body measurements; keel length, body depth and shank length. The assumption is made that differences between the two groups, with respect to the above mentioned statistics, show to what extent the polygenes of the characters concerned need thyroxine for their action. The results and conclusions were as follows:

1. The control birds consistently grew more rapidly than the treated ones. The differences in rate of growth was most noticeable between the second and sixth weeks of age, after which time the two groups had almost comparable rates of growth. Between the sixth and the tenth weeks of age the treated birds weighed about 60 per cent as much as that of the controls.

2. The females were more resistant to the treatment than the males with respect to retardation of growth. This sex difference became more conspicuous as the birds became older.

3. Since the treated birds did not show complete arrest of growth, it was concluded that some other growth factors must be operating.

4. At ten weeks of age the keel length and shank length of the treated birds were 25 per cent less than those of the controls. Body depth was only about 20 per cent less in the former than in the latter.

5. The body measurements of the treated females were less affected by the treatment than those of the males.

6. On the average, the thyroid weight of the treated birds at ten weeks of age was about seventeen times that of the control birds. Practically no sex dimorphism was noticed with respect to the thyroid weight.

7. The average testes weight of the treated males was about 35 percent less than that of the normal birds. The average testicular weight per gram of body weight was, however, constant in both groups (0.129 and 0.120 centigrams for the treated and the control groups, respectively).

8. The phenotypic variance of the treated birds is much greater than that of the control birds in all the studied characters.

9. The phenotypic variance of body weight in the treated birds was relatively higher than that of body measurements.

10. The estimate of the percentages of the total genetic variance was partially obscured by a common environmental factor which might be due to the extension of the hatching season over a comparatively long period of time, or to a high frequency of accidental raising of maternal half-sibs together. Besides this common factor of environment, the increase of the dam's contribution over that of the sire's is due either to dominance or to maternal effects

or to both.

11. With respect to body weight, the differences between the contributions of the two parents showed a certain rhythm which was different in direction between the two sexes, it was concluded that sex hormones play a role in growth. Therefore, it was inferred that the production of sex hormones may be tied up with dominance or maternal effects or with both.

12. The percentages of the additive genetic variance of body weight were in general in the control greater than in the treated birds. The opposite, however, was true with respect to body measurements and testes weight. This discrepancy with respect to the action of the lack of thyroxine on additive variance was explained on the supposition that polygenic action differed under the treatment.

13. The goitergenic capacity of the thyroid gland was entirely caused by non-additive genes, the number of which is to be determined. Polygenes do not contribute to this capacity.

14. Genetic thyroid I activity in the normal birds at ten weeks of age, as indicated by the heritability of the thyroid weight, was mostly under polygenic control rather than non-additive gene action. Its heritability (percentage of additive variance) was at least 0.139 and 0.081 in the females and males, respectively. These estimates revealed that a good share of the variability in thyroidal activity is due to environmental factors.

15. In the normal males, the testes weight at ten weeks of age had a heritability (percentage of additive variance) of

0.158 which similarly indicates that most of its phenotypic variability is due to environmental causes.

16. The inference was made that the z-chromosome in the female has no major effects yet it is polygenically active.

17. Body weight was highly correlated with thyroidal activity in both the control and the treated groups. This fact was true on both phenotypic and genotypic levels.

18. In the control birds body weight was not correlated with testes weight. This situation was reversed under the treatment, which fact showed that there might be a compensatory contribution from the testes to body weight.

19. Thyroid weight and testes weight had positive correlations in the normal birds (0.391 and 0.305 on the genetic and phenotypic levels, respectively). Such a correlation was almost lost in the treated birds (-0.061 and 0.034, respectively). Hence, the genes concerned with the goitrogenic capacity apparently have no effect on the testes size.

20. The variability in body weight accounted for a good share of the correlated variability between thyroid weight and testes weight.

21. It is generally concluded that gene action is considerably altered if the bird is deprived of thyroxine and that this alteration takes different forms dependent on the kind of the character and the age of the individual.

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